3rd Annual UAB Huntsville Regional Medical Campus Research Day

Abstract and Poster Compendium

3rd Annual UAB Huntsville Regional Campus Research Day

Tuesday, March 24, 2020

Poster Session – Call for Abstracts

Submission Deadline
Monday, February 10, 2020

11:00am – 2:00 pm – Auditorium
3rd Floor, UAB Huntsville Regional Medical Campus

We are seeking submissions for the 3rd Annual UAB Huntsville Regional Campus Research Day and hope that you will consider submitting an abstract in one of the following categories:

1) ***Research Abstracts:*** Submissions may fit one or more of the following categories:
   - Education Innovation
   - Quality & Safety

2) ***Clinical Vignettes:*** Clinical vignette should describe a clinical condition that:
   - Illustrates unique or important teaching points;
   - Provides insight into clinical practice, education, or research;
   - Illustrates an important clinical problem such as a diagnostic, therapeutic or management dilemma.

Eligibility

- UAB Huntsville Regional Medical Campus medical students, core or clinical faculty, and residents, UAB Nurse Practitioner students/faculty, Auburn-Harrison SOP students and faculty, ACOM/VCOM students and clinical faculty based in North Alabama, and Huntsville Hospital clinical staff.
- Abstracts submitted to other meetings / journals in 2020 but not yet published are eligible.

Structure

Documents should be in a Word document, no more than 500 words (images, figures and references can be added later once accepted). Please include name of author(s), departmental affiliation, appointment/position, email address, and submission category. An example (attached) has been provided for you.

Research Abstract submissions should include: Description, Methods, Results, Discussion.

Clinical Vignette submissions should include Learning Objectives, Case Presentation, and Discussion.

Blinded peer review judging will determine acceptance and oral presenters. The top oral presenter and the top 3 poster award winners in each section will be announced on the day of the event and receive cash prizes. Those accepted will be notified by March 5 with further instructions.

Your Faculty Mentor should approve before submission.

Deadline is 12:00pm, CST, February 10, 2020. Send your submissions to: alanbacker@uabmc.edu

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Awards

Best Student Oral Presentation

Trichodysplasia Spinulosa: A Rare "Hair-Like" Dysplasia
Skyler Jones, Marla Davis

Best Resident Oral Presentation

Is It Getting Hot In Here? A Case of Multi-organ Failure and DIC in Heat Stroke
Katie Glosemeyer, William Humphrey

Clinical Vignette Poster Presentations

1st Place – Rare bacteria associated with spontaneous bacterial peritonitis (SBP)
Mrudula Thiriveedi, Kelsey Ivey, Farrah Ibrahim

Tie, 2nd Place – The Yin and Yang of Hepatitis C infection (HCV) and B-Cell Non-Hodgkin Lymphoma
Paul St. Clair, Katherine E. Glosemeyer, Farrah Ibrahim

Tie, 2nd Place – A Case of the Human Metapneumovirus
Joseph Shaw, Ainy Aziz, Ali Hassoun

Best Research Abstract Poster Presentation

Direct Primary Care in Rural Communities
Dusty Trotman; David Bramm, M.D.

Section I: Clinical Vignettes

49,XXXXY Klinefelter Syndrome

Jennifer Lamar MS3, Janaki Nimmagadda M.D., Clinton Martin M.D.
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Learning Objectives:

1. Understand how 49 XXXXY Klinefelter Syndrome occurs
2. Recognize the challenges faced with 49 XXXXY Klinefelter Syndrome
3. Management and treatment options available
4. Importance of early intervention

Case Presentation: 26-year-old short, thin, Caucasian male with 49, XXXXY Klinefelter Syndrome presented with functionally impairing hypersexuality and impulsivity. The patient underwent genetic testing at birth after presenting with low set ears and microphallus. He had marked physical and mental delays during childhood with an IQ of 45. He had limited verbal skills with an inability to read, write, and perform basic mathematics. His medical history included brittle bones treated with biweekly Testosterone injections, olecranon abnormalities, dysmorphic facial features, recurrent upper respiratory and urinary tract infections, anxiety, and repetitive behaviors, obsessions, and compulsions.

At 16 years, he sought treatment for hypersexuality and impulsivity, which was unresponsive to Paxil, but well controlled with Zoloft. His current dose of Zoloft was 50 mg but his parents stated an increase in inappropriate sexual behaviors and gestures which was impairing his sleep, public decency, and volunteer employment. During the interview he was quiet and timid, shielding his face with his hands.

Discussion: 49, XXXXY Klinefelter Syndrome occurs from random double maternal nondisjunction. It is the rarest and most severe form of Klinefelter Syndrome. Although there are common features among all Klinefelter variants, each additional X chromosome alters the phenotype. In additional to dysmorphic facial features, short stature, musculoskeletal abnormalities, these individuals have a significant decline in cognitive function, marked impairment in communication and social skills, and fluctuating behavioral issues. Care for these individuals requires a multi-disciplinary approach. Albeit they have cognitive impairments, these individuals have normal non-verbal skills and visual perception leaving these individuals with a discordance between receptive and expressive communication skills. Issues such as impulsivity, irritability, hyperactivity, anxiety, temper outbursts, and obsessive-compulsive behaviors are commonly reported. With delayed speech and motor development, early interventions are imperative to promote healthy alternative ways of learning and interacting with society. Using sign language as a means of communication and implementing mostly visual and constructive tasks as a learning style may lessen the behavioral patterns experienced by these individuals. Pharmacological therapies are effective

for moderate to severe behavioral problems, but early intervention with consistent behavior management based on a reward system is the most effective therapy.

49 XXXXY Klinefelter Syndrome

Jennifer Lamar, Janaki Nimmagadda M.D., Clinton Martin M.D., Katherine Moody M.D.
UABSOM Huntsville Campus, Department of Psychiatry
University of Alabama at Birmingham School of Medicine

Abstract

- 49, XXXXY occurs from double maternal nondisjunction[1]
- Rarest, most severe variant of Klinefelter syndrome (KS) with an incidence of 1:85,000 to 1:100,000 and no inheritance pattern or correlation with maternal age[1]
- More prominent findings compared to KS or other KS variants[2]
- Physical and mental development impairments correlate with the number of X chromosomes[3]
- Common features include ocular hypertelorism, epicanthal folds with up-slanting palpebral fissures, flat nasal bridge, hyperextensible joints, radioulnar synostosis, short stature, hypotonia, hypergonadotropic hypogonadism, and underdeveloped genitals[1,2,6]
- Average IQ between 20-60 because each additional X chromosome lowers the IQ 15-16 points[3]
- Decreased independent daily living, communication, and social skills with increasing X chromosomes, with communication skills most significantly affected[3,4]
- Normal non-verbal skills and visual perception suggesting better performance with visual and construction tasks than those requiring intact verbal fluency[4,5]
- Low tolerance and behavioral issues likely result from frustration with discordance receptive vs expressive impairment[3,5,6]

Case Report

26 year old short, thin, Caucasian male with 49, XXXXY Klinefelter Syndrome presents with functionally impairing hypersexuality and impulsivity. The patient underwent genetic testing after presenting with low set ears and microphallus at birth. He had marked physical and mental delays during childhood with an IQ of 45. He has limited verbal skills with an inability to read, write, and perform basic mathematics. His medical history includes brittle bones treated with biweekly Testosterone injections, olecranon abnormalities, dysmorphic facial features, recurrent upper respiratory and urinary tract infections, anxiety, and repetitive behaviors, obsessions, and compulsions.

At 16 years, he sought treatment for hypersexuality and impulsivity, which was unresponsive to Paxil, but well controlled with Zoloft. His current dose of Zoloft is 50 mg but his parents state an increase in inappropriate sexual behaviors and gestures impairing his sleep, public decency, and volunteer employment. During the interview he is quiet, timid, and shields his face with his hand.

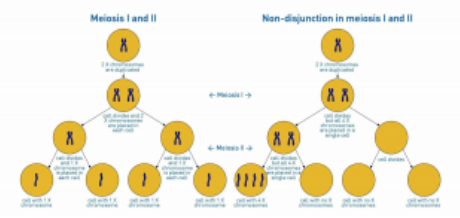
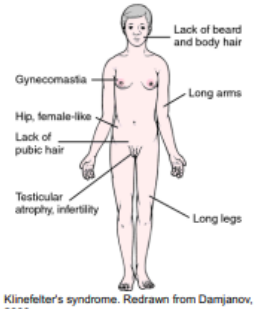


Figure 1: Normal (left) and abnormal (right) meiosis I and II[5]



Klinefelter's syndrome. Redrawn from Damjanov, 2000.

Miller-Keane Encyclopedia and Dictionary of Medicine, Nursing, and Allied Health, Seventh Edition. © 2003 by Saunders, an imprint of Elsevier, Inc

Discussion

- Care of an individual with 49, XXXXY should entail a multi-disciplinary approach[6]
- Behavioral issues such as irritability, obsessive-compulsive behaviors, anxiety, impulsivity, and temper outbursts are commonly seen[2]
- ADHD symptoms such as hyperactivity and behavioral dysregulation is commonly reported but lacks adequate studies[2]
- Speech and motor delay is imminent in 49, XXXXY; early interventions are critical to reduce future impairment[2]
- With a significant deficit in verbal communication, alternate forms of communication such as signing has proved to be effective for better cognitive function and improved behavior[6]
- Pharmacologic therapies are recommended to help control moderate to severe behavioral dysfunction[5]
- Some of the most effective treatment is early intervention with consistent behavior management based on a reward system[5]

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THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

Title: A case of anti-NMDA-receptor Encephalitis causing Psychosis

Authors: Kenneth Holt MS4; Clinton Martin M.D.

Abstract: Anti-NMDA-receptor encephalitis has been growing in recognition as a major cause of encephalitis. One study in 2012 found Anti-NMDA-receptor encephalitis to be more prevalent of a cause of encephalitis in young persons than any individual viral cause (Gabel, 2012). Recently, increased awareness of anti-NMDA-receptor encephalitis has caused the disease to rise to front of differentials for patients previously thought to have encephalitis of viral or unknown etiology. In this case report we discuss a patient who presented with seizures and episodic confusion who was diagnosed with Anti-NMDA-receptor encephalitis.

Discussion: Anti-N-methyl-D-aspartate Receptor (NMDAR) encephalitis is an autoimmune condition that can occur in the presence or absence of neoplasms. Clinical syndrome manifests with memory and behavioral disturbances, catatonia, agitation, psychosis, seizures and dyskinesias.

Probable anti-NMDA receptor encephalitis:

Diagnosis can be made if 3 of the following criteria have been met

1. Rapid onset (less than 3 months) of at least 4 of the 6 following major groups of symptoms:

    - Abnormal (psychiatric) behavior or cognitive dysfunction
    - Speech dysfunction (pressured speech, verbal reduction, mutism)
    - Seizures
    - Movement disorder, dyskinesias, or rigidity\abnormal postures
    - Decreased level of consciousness
    - Autonomic dysfunction or central hypoventilation

2. At least 1 of the following laboratory study results:

    - Abnormal EEG (focal or diffuse low or disorganized activity, epileptic activity, or extreme delta brush)
    - CSF with pleocytosis or oligoclonal bands

3. Reasonable exclusion of other disorders

Diagnoses can also be made in the presence of 3 of the above group symptoms accompanied by her systemic teratoma.

Definite anti-NMDA receptor encephalitis:

Diagnosis can be made in the presence of 1 or more of the 6 major groups of symptoms and IgG anti-GluN1 antibodies, after reasonable exclusion of other disorders.

References:

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Poster was self-printed for presentation and a PDF image was unavailable for this compendium.

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Submission category: Clinical Vignette

A case of Invasive Aspergillosis with Endocarditis in an Immunocompetent Patient

Learning Objectives:
To understand the risk factors, diagnosis and course of Invasive Aspergillosis.

Case Presentation:
A 66 y/o Caucasian male with COPD, chronic smoking, former alcohol use, and poor dentition, presented to an outside hospital with worsening cough, confusion, fever, and decreased oral intake. Patient was found to have right-sided pneumonia, thought to be secondary to aspiration. Due to increasing oxygen requirement and declining renal function, he was transferred to our hospital and was found to be in acute hypoxemic respiratory failure with septic shock, requiring intubation and pressor support. Patient was initially treated with meropenem and levofloxacin, but sputum cultures later grew Aspergillus, prompting addition of Voriconazole. Aspergillus antigen was positive at 1.369. CT scan of the chest showed bilateral pneumonia without cavitation. After stabilization of his respiratory status and transfer to the floor, patient began coughing and desaturating with all oral intake. Subsequent barium swallows showed oropharyngeal dysphagia with silent aspiration. Brain MRI was ordered to investigate the etiology of dysphagia which showed four lesions suspicious for infection vs. malignancy. Craniotomy with biopsy was done which revealed acute necrotizing granulomatous inflammation with septated fungal hyphae (acute angle branching consistent with Aspergillus) as well as gram positive cocci. Blood cultures remained negative throughout his hospital course. TEE was then done to investigate source of infective emboli to the brain which showed fimbriated MV endocarditis, consistent with fungal disease process and confirming cause of CNS dissemination. Patient is currently being evaluated for possible surgery for fungal endocarditis. After 6 weeks of antifungal therapy, his Aspergillus Ag is negative (<0.5), though 1,3-beta-D-glucan is still positive at 158.

Discussion:
Invasive aspergillosis (IA) is a rapidly-progressive and often fatal disease, typically affecting immunocompromised hosts. While *Aspergillus fumigatus* is the most common culprit, other species have also been linked to invasive disease. Colonization most commonly occurs via inhalation into the lungs (60%). Fever, cough, hemoptysis, and dyspnea are frequent, but nonspecific findings. Invasion across tissue planes into vasculature leads to hematogenous spread and can affect multiple organs, including the skin, brain, eyes, liver, kidneys, and heart.

Risk factors for IA include neutropenia, transplant recipients, glucocorticoid therapy (short and long term), COPD, malignancy, and chronically impaired cellular responses. Definitive diagnosis of invasive aspergillosis remains difficult as imaging is inconsistent and sputum and blood cultures lack sensitivity. Antigenic testing of components of fungal cell wall, PCR testing,

galactomannan or beta-D-glucan, are reasonably specific for invasive aspergillosis, with galactomannan also having use in tracking response to medication. Prompt treatment with voriconazole, the antifungal of choice, is essential for survival. Duration of treatment varies with type of organ involvement. Lifelong therapy is necessary once colonization of the heart is established, and early surgical intervention is crucial. This is due to the profound mortality of Aspergillus endocarditis which is almost 100%.

THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

INVASIVE ASPERGILLOSIS WITH ENDOCARDITIS IN AN IMMUNOCOMPETENT PATIENT

SARA-ELIZABETH CARDIN, BRYAN GRISSET, DO, KELSEY IVEY, MD, PAREKHA YEDLA, MD

Learning Objective

- Understand the risk factors, diagnosis, and course of Invasive Aspergillosis (IA)

Case Presentation

- A 66 y/o Caucasian male with COPD and chronic smoking history presented to an outside hospital with worsening cough, confusion, fever, and decreased oral intake.
- Found to have right-sided pneumonia, thought to be secondary to aspiration. Due to increasing oxygen requirement and declining renal function, he was transferred to our hospital

Hospital Course

- Upon arrival to our hospital, found to be in acute hypoxemic respiratory failure with septic shock, requiring intubation and pressor support.
- Patient was initially treated with meropenem and levofloxacin, but sputum cultures later grew Aspergillus, prompting addition of Voriconazole.
- CT scan of the chest showed bilateral pneumonia without cavitation.
- Later began desaturating with oral intake. Subsequent barium swallows showed oropharyngeal dysphagia with silent aspiration.
- Brain MRI ordered to investigate the etiology of dysphagia, which showed four lesions suspicious for infection vs. malignancy (Image 1)

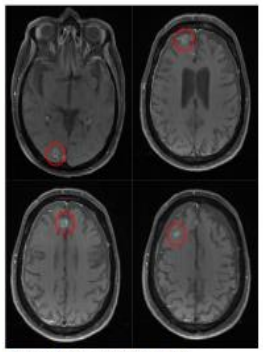

Image 1: MRI of 4 brain lesions (red circles)

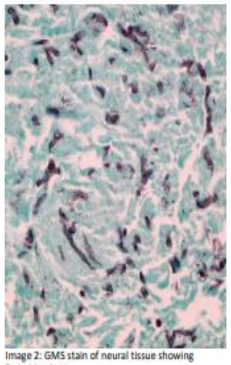

Image 2: GMS stain of neural tissue showing fungal hyphae

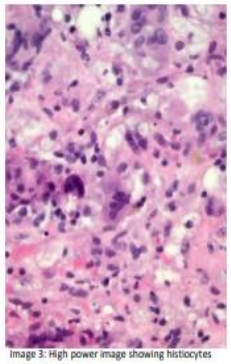

Image 3: High power image showing histiocytes and giant cells

- Craniotomy with biopsy revealed findings consistent with fungal hyphae (Image 2) as well as gram positive cocci.
- Blood cultures remained negative.
- TEE to investigate infective emboli source to the brain showed fimbriated MV endocarditis, consistent with fungal disease process and confirming the cause of CNS dissemination.

Discussion

- Invasive aspergillosis (IA) is a rapidly-progressive and often fatal disease, typically affecting immunocompromised hosts.
- While *Aspergillus fumigatus* is the most common culprit, other species have also been linked to invasive disease.
- Colonization most commonly occurs via inhalation into the lungs (60%).
- Fever, cough, hemoptysis, and dyspnea are frequent, but nonspecific findings.
- Invasion across tissue planes into vasculature leads to hematogenous spread and can affect multiple organs, including the skin, brain, eyes, liver, kidneys, and heart.
- Risk factors for IA include neutropenia, transplant recipients, glucocorticoid therapy (short and long term), COPD, malignancy, and chronically impaired cellular responses.
- Definitive diagnosis of invasive aspergillosis remains difficult as imaging is inconsistent, and sputum and blood cultures lack sensitivity.
- Antigenic testing of components of fungal cell wall, PCR testing, galactomannan or beta-D-glucan, are reasonably specific for invasive aspergillosis, with galactomannan also having use in tracking response to medication.
- Prompt treatment with voriconazole, the antifungal of choice, is essential for survival.
- Duration of treatment varies with type of organ involvement. Lifelong therapy is necessary once colonization of the heart is established, and early surgical intervention is crucial. This is due to the profound mortality of Aspergillus endocarditis which is almost 100%.

Acknowledgements

Special thanks to Dr. Frank Honkanen and the Huntsville Hospital Pathology Department for the histologic images

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Title: "A Case of the Human Metapneumovirus"

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Submission Category: Clinical Vignette

Learning Objectives

Human metapneumovirus (hMPV) is an enveloped, negative-sense RNA virus first described in 2001 by Dutch investigators studying children with unspecified viral respiratory infections. Serology from that study suggested that the virus has likely been circulating for at least 60 years and that virtually all humans are exposed in childhood. Most cases of severe infection are in children <5 years old, with the immunocompromised, elderly, and those using antibiotics as the majority of adult infection. In this case report, we describe a clinical course of viral pneumonia in an elderly woman who tested positive for hMPV.

Case Presentation

A 73 year old Caucasian female with a history of bronchiectasis and recurrent UTI who was brought to the ED for fever and altered mental status. She had one week history of shortness of breath, productive cough of white sputum, nasal congestion, intermittent headaches, back pain, and fatigue. Recently she was treated for UTI with oral nitrofurantoin, on day 7 of a 20-day course. Her temperature was 39.8˚ C, and her O2 saturation was 81%. All other vitals and routine labs were within normal limits. Bilateral coarse breath sounds with scant wheezing were found on otherwise unremarkable physical exam. She was placed on supplemental O2 via nasal cannula and received a PA/lateral chest X-ray. Her saturation improved to 91%, and the X-ray revealed a small, patchy airspace opacity at the base of the right lung which represented bronchiectasis or a possible consolidation. Blood, urine, and sputum cultures were drawn, and rapid PCR was negative for influenza A and B. She received initial doses of ceftriaxone and steroid therapy and was admitted to the inpatient service for further management of community-acquired pneumonia (CAP).

She was found to be positive for hMPV via nasal swab PCR, prompting initiation of contact and droplet isolation protocols. Blood culture results were negative, sputum cultures grew normal flora, and her urine cultures were positive for *E. coli* and *P. aeruginosa* which thought to be colonization. She remained on antibiotics. Her respiratory condition improved; she was soon afebrile with O2 saturations >90% on room air, and on the 5th day after admission she was discharged home with instruction for clinic follow-up in 2 weeks.

Discussion

Common viral causes of CAP include influenza, adenovirus, parainfluenza, RSV, and hMPV. The symptoms of hMPV in most adult infection are non-specific and include cough, nasal congestion, and dyspnea, but data on its role in lower airway diseases requiring hospitalization, even in low-risk populations, are emerging. Diagnosis in clinical settings is done with reverse-transcriptase PCR, and treatment is supportive. In vitro studies of ribavirin have shown possible activity against hMPV, but no clinical data exists currently.

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A Case of the Human Metapneumovirus

Joseph Shaw, Qurrat-ul-ain Aziz, D.O., M.P.H. and Ali Hassoun, M.D.

Introduction:

- Human metapneumovirus (hMPV) is an enveloped, negative-sense RNA virus first described in 2001 that has likely been circulating for at least 60 years in virtually all humans.
- In this case report, we describe a clinical course of viral community-acquired pneumonia in an elderly woman who tested positive for hMPV.

Background:

- hMPV is part of Pneumoviridae family
- Made separate family in 2016; has 2 genera *Metapneumovirus* (includes hMPV) and *Orthopneumovirus* (includes RSV)
- Often self-limiting URI, but in high-risk patients (infants, elderly, immunocompromised) can require hospitalization
- Transmitted by close-contact via large particle aerosols, droplets, and fomites
- Incubation period ~5-9 days

Symptom	Frequency (%)
Cough	100
Nasal Congestion	85
Rhinorrea	75
Dyspnea	69
Hoarseness	67
Wheezing	62
Fever	**4***

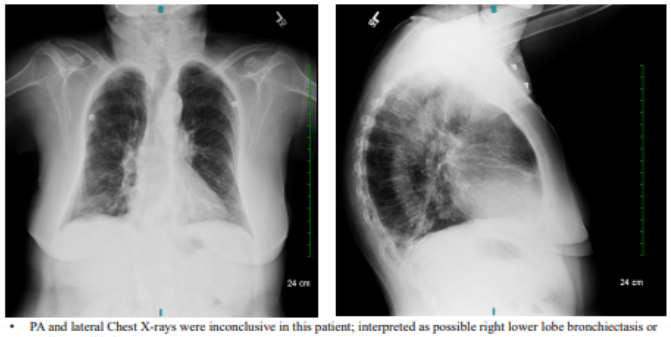
Clinical Course:

- A 73 year old Caucasian female with a history of bronchiectasis and recurrent UTI was brought to the ED for fever and altered mental status.
- One week history of shortness of breath, productive cough of white sputum, nasal congestion, intermittent headaches, back pain, and fatigue.
- Recently treated for UTI with oral nitrofurantoin, on day 7 of a 20-day course.
- Vitals: temperature 39.8˚ C, and her O2 saturation was 81% on room air.
- Physical Exam: Bilateral coarse breath sounds with scant wheezing
- Labs: routine labs were within normal limits. Blood, urine, and sputum cultures were negative, and rapid PCR was negative for influenza A and B. Positive for hMPV via nasal swab PCR
- Imaging: X-ray revealed a small, patchy airspace opacity at the base of the right lung which represented bronchiectasis or a possible consolidation.

Course of Treatment:

- Supplemental O2 via nasal cannula and saturation improved to 91%,
- Ceftriaxone and steroid therapy
- Placed on contact precautions for history of C. diff and droplet isolation protocols for hMPV
- Symptoms improved and she was soon afebrile with O2 saturations >90% on room air, and on the 5th day after admission she was discharged home with instruction for clinic follow-up in 2 weeks.

Imaging:



- PA and lateral Chest X-rays were inconclusive in this patient; interpreted as possible right lower lobe bronchiectasis or small consolidation.

Discussion:

- Common viral causes of CAP include influenza, adenovirus, parainfluenza, RSV, and hMPV
- The symptoms of hMPV in most adult infections are non-specific and include cough, nasal congestion, and dyspnea, but data on its role in lower airway diseases requiring hospitalization, even in low-risk populations, are emerging.
- Fever is relatively rare.
- Imaging is not required for diagnosis and is often unremarkable.
- Diagnosis in clinical settings is done with reverse-transcriptase PCR, and treatment is supportive.
- In vitro studies of ribavirin have shown possible activity against hMPV, but no clinical data exists currently.
- At minimum, contact isolation is recommended.

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A Case of Tracheobronchomalacia in an Elderly Male

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Learning Objective

1. To raise awareness about tracheobronchomalacia, an underdiagnosed cause of dyspnea in adults.
2. Understanding the pathophysiology and treatment options.

Case Presentation

Patient is an 81 year old Caucasian male with past medical history significant for atrial fibrillation and severe advanced Parkinson's who presented to emergency department for a fall and worsening shortness of breath. He had a productive cough which was being treated with antibiotics from his primary care physician. Patient has no prior history of known lung disease, asthma, COPD, or cigarette smoking. Vital signs notable for heart rate of 114 and respiratory rate of 23. Physical exam revealed inspiratory crackles, coarse breath sounds bilaterally, and loud expiratory tracheal breath sounds. CT of the chest showed pulmonary edema as well as severe tracheobronchomalacia (TBM) that extended to bilateral main bronchi. This was a new finding in comparison to a previous study. EKG showed atrial flutter with rapid ventricular response. Echocardiogram shows EF of 30% and severe global hypokinesis. Patient was given furosemide 40mg IV daily and started on BiPAP to help with TBM symptoms, but could not tolerate it on multiple occasions. With regards to his atrial flutter, patient underwent cardioversion after failed pharmacologic treatment and converted to sinus rhythm. Per pulmonary consultant, patient was not an ideal candidate for definitive surgical treatment. Furthermore, patient's code status was DNR and he did not wish for aggressive treatment. Patient's dyspnea improved with diuretics and nebulized breathing treatments. He was discharged home with conservative management.

Discussion

Tracheomalacia (TM) refers to segmental or diffuse tracheal weakness. It is referred to tracheobronchomalacia when it extends to the bronchus. Incidence of TBM is reported to range in between 4% to 23% in patients with respiratory symptoms undergoing bronchoscopy. The two types are primary, where it is associated with early childhood congenital defects and secondary, where it is acquired later in life. Acquired TBM is commonly associated with tracheal trauma such as during intubation or with chronic inflammation as in chronic obstructive pulmonary disease, cigarette smoking, gastroesophageal reflux disease, or relapsing polychondritis. In healthy patients, the posterior membranous portion of the trachea bows in slightly toward the lumen during exhalation and to a greater degree on forced exhalation or cough. In patients with TBM, they will have excessive bowing of this membranous portion even during normal tidal breaths. Some patients may present without any signs and symptoms, but the condition is usually regarded as progressive and the patient may eventually present with dyspnea, stridor, and difficulty expectorating secretions. Patients with TBM tend to be associated with more frequent respiratory infections and extended recovery times. Gold standard diagnosis of TBM is through functional bronchoscopy, but may also be evaluated through inspiratory/expiratory dynamic chest CT (reported accuracy rates as high as 97%), and pulmonary

function tests. Mainstay of treatment is to receive tracheal bronchoplasty, tracheal stenting, CPAP, or tracheostomy that is downstream from site of closure.

A Case of Tracheobronchomalacia in an Elderly Male

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Learning Objectives

•To raise awareness about tracheobronchomalacia, an underdiagnosed cause of dyspnea in adults.
•Understanding the pathophysiology and treatment options.

The Patient

- 81 year old Caucasian male with history of atrial fibrillation and severe advanced Parkinson's.
- CC: Shortness of breath.
- No prior history of known lung disease, asthma, COPD, or cigarette smoking.
- Vital Signs: Only notable for HR 114 and RR 23.
- Physical Exam: Significant for tachycardia, inspiratory crackles, coarse breath sounds bilaterally, and loud expiratory tracheal breath sounds on auscultation, otherwise unremarkable.
- CT Imaging: pulmonary edema and severe tracheobronchomalacia extending to bilateral main bronchi.
- Echocardiogram: Ejection fraction of 30% and severe global hypokinesis.

Hospital Course and Follow Up

- Pulmonary Edema: IV furosemide for diuresis.
- Tracheobronchomalacia: Started on BiPAP, but patient could not tolerate on multiple occasions.
- Atrial flutter: Underwent cardioversion after failed pharmacologic treatment and converted to sinus rhythm.
- Per pulmonary consultant, patient was not an ideal candidate for definitive surgical treatment. Patient did not wish for aggressive treatment.
- Dyspnea improved with diuretics and nebuilized breathing treatments.
- Discharged home with conservative management.
- Seen in clinic several weeks afterwards with persistent tracheal breath sounds but improved dyspnea.

Tracheobronchomalacia

- Tracheomalacia (TM) refers to segmental or diffuse tracheal weakness.
- It is called tracheobronchomalacia (TBM) when it extends to the bronchus.
- The incidence of TBM is reported to range in between 4% to 23% in patients with respiratory symptoms undergoing bronchoscopy.
- Primary TM is associated with early childhood congenital defects.
- Secondary TM is acquired later in life.

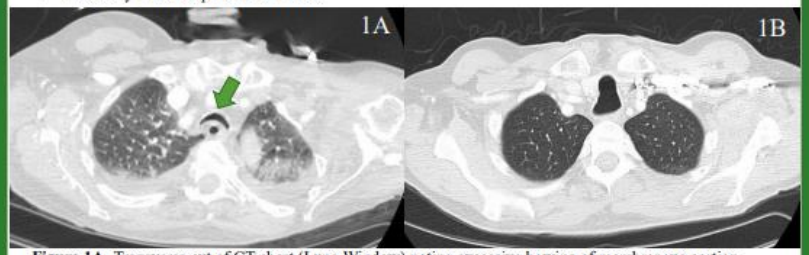

Figure 1A- Transverse cut of CT chest (Lung Window) noting excessive bowing of membranous portion of trachea. **Figure 1B**- Same approximate transverse cut of a CT of same patient four years prior.

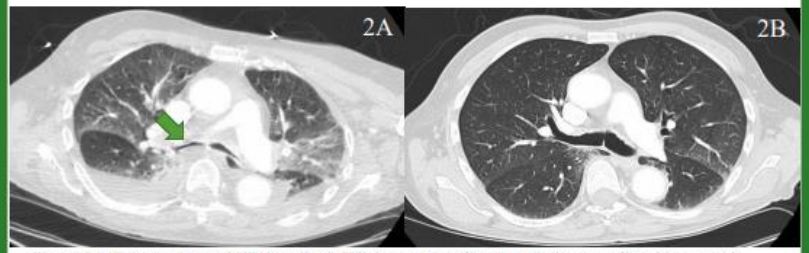

Figure 2A- Transverse cut of CT chest (Lung Window) noting airway narrowing extending down to main bronchi. **Figure 2B**- Same approximate cut of same patient four years prior.

Pathophysiology

- Acquired TM is commonly associated with trauma or chronic inflammation:
  - Tracheal trauma during intubation
  - Chronic obstructive pulmonary disease
  - Cigarette smoking
  - Gastroesophageal reflux disease
  - Relapsing polychondritis
- In healthy patients, the posterior membranous portion of trachea bows in slightly toward the lumen during exhalation and to a greater degree on forced exhalation or cough.
- In patients with TBM, the membranous portion of trachea will have excessive bowing even during normal tidal breaths.
- Some patients may present without any signs and symptoms, but the condition is usually regarded as progressive and patients may eventually develop dyspnea, stridor, and difficulty expectorating secretions.

Diagnosis

- Gold standard diagnosis of TMB is through functional bronchoscopy.
- May also be evaluated through inspiratory/expiratory dynamic chest CT:
  - Taking CT scan during inspiration and during expiration.
  - Measure changes in tracheal cross sectional area between the scans.
  - Reported accuracy rates as high as 97%.
- Pulmonary function testing will show variable obstruction depending on air flow speed.

Treatment Options

- Tracheobronchoplasty
- Tracheal stenting
- Tracheostomy downstream from site of closure
- CPAP

Clinical Takeaways

- TBM may be an underdiagnosed cause of dyspnea in adults.
- Dynamic chest CT may be a good alternative diagnostic test on patients who do not wish for aggressive testing as in bronchoscopy.

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A Fatal Disease with Encouraging Outcome

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Learning Objectives

1. Identify and diagnose Hemophagocytic Lymphohistiocytosis(HLH), a fatal hyperinflammatory syndrome early
2. Understand the spectrum of diagnoses associated with extreme elevation of ferritin

Case presentation

A 59-year-old male with diabetic foot infection and osteomyelitis, receiving intravenous Vancomycin and Piperacillin-Tazobactam for 3 weeks, presented with high fever and rigors. Work up to rule out sepsis including line related infections, endocarditis, pneumonia and urinary tract infection was negative. PICC line removed and sent for culture. Antibiotics changed to Meropenem and Daptomycin, considering drug fever. Laboratory results was significant for pancytopenia involving three cell lines. Anemia work up reveled extremely high ferritin of 33,519 ng/mL. This led to further investigation of causes of extremely high ferritin, including HLH, Malignancies, HIV and liver injury. Echocardiogram, CTA chest and cultures of blood and urine ruled out ongoing infection. Patient met 5 out of 8 HLH-2004 criteria (two not tested) and HScore predicted 80-88% probability. Patient had fever, splenomegaly, hypertriglyceridemia, high ferritin, and elevated sCD25. Bone marrow aspiration and NK cell activity not done. In addition, he had elevated AST, ALT, LDH and D-dimer. Hematologist was consulted, and as the patient started improving clinically, a conservative approach was adopted. Fever resolved, and ferritin levels began trending down. Patient followed with hematologist after discharge, had a ferritin level that normalized, and the patient recovered completely.

Discussion

HLH is a hyperinflammatory, hyperferritinemic syndrome that results from immune system's inability to restrict stimulatory effects of various triggers. Primary HLH (genetic) is more common in children, while secondary HLH, triggered by Infections (mainly viruses like EBV, also bacteria, parasites and fungi), malignancies, autoimmune disorders and others is the predominant form in adults. It commonly presents as a febrile illness with multiple organ involvement. Lack of specific markers, clinical picture mimicking sepsis or malignancy, and low index of suspicion causes challenges in diagnosis leading to high fatality. Heterogenicity of adult HLH excludes a "one size fits all" protocol for management. Stable patients who respond to prompt treatment of HLH trigger can be managed conservatively. In rapidly deteriorating patients treatment options include dexamethasone and etoposide, intrathecal methotrexate and hydrocortisone in CNS involvement, IV IG or Rituximab in viral infections, and

allogenic hematopoietic stem cell transplant in refractory patients. Patients are at risk of developing Posterior Reversible Encephalopathy Syndrome (PRES), which should be recognized and treated promptly. Supportive care including prompt management of organ dysfunction, appropriate transfusions, prevention and treatment of bleeding and/or infections. Though serum ferritin is not a specific marker for diagnosis, it is useful to monitor response to therapy.

Adult HLH is associated with high mortality (24- 75%), especially with associated malignancy. Patients with infectious and autoimmune triggers tend to have better outcomes. Treatment entails suppression of overactive immune system and prompt treatment of underlying cause. Treatment should not be delayed while awaiting molecular studies or ancillary tests. A multidisciplinary approach is necessary for proper management.

Poster was self-printed for presentation and a PDF image was unavailable for this compendium.

An unusual cause of headache and fever in a young adult

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Submission category: Clinical vignette

Learning objectives:

An approach to non-infectious etiology of headache and fever in a young adult.

Case Presentation:

A 22-year-old Caucasian German exchange student with no significant past medical history came to the emergency department with pulsatile headache in the occipital region for 4 weeks which worsened 3 days prior to presentation. She reported an episode of vomiting and near syncope on the morning of admission. She had a fever of 102.7 degrees Fahrenheit upon arrival and her physical exam was normal without any skin or joint findings. Labs were significant for white cell count of 0.97 $x10^3$/mcL with an absolute neutrophil count of 600, hemoglobin of 7.2 g/dL and normal platelet count. Cerebrospinal fluid (CSF) was clear with negative meningoencephalitis panel. Extensive work up was done to rule out an infectious cause due to her history of travel to Northeastern United States few months prior. Human immunodeficiency virus, Epstein bar virus, Cytomegalo virus, Parvo virus, Rickettsia serology, Lyme and Tick panel were negative. Work up for anemia and leukopenia showed normal iron and B12 levels. Patient was started empirically on cefepime in the interim. Due to lack of improvement in her clinical course, bone marrow biopsy was done on day three of hospitalization which showed B-cell acute lymphoblastic leukemia with 91% blasts on flow cytometry. CSF flow cytometry could not be done as the fluid was not sufficient, so possible leptomeningeal involvement as the cause of headache could not be established. After a long discussion with the patient and family (in Germany), she was flown to Germany on a Med flight for further treatment.

Discussion:

Acute lymphoblastic leukemia (ALL) is a hematologic malignancy of undifferentiated lymphoid precursor cells, which leads to excessive production of abnormal lymphoblasts in the bone marrow and subsequent hematopoietic failure. B cell acute lymphoblastic leukemia/lymphoma (B-ALL/LBL) is most common in children, with a second peak in adults more than 60 years old, rarely seen in young adults. The cause of B-ALL/LBL is unknown, but it may be associated with ionizing radiation and/or as-yet unidentified infectious agents.

Symptoms include malaise, bleeding, infections, bone pain or a combination of these. Less than 10% may have symptomatic central nervous system (CNS) involvement at diagnosis. Headache is a common symptom among childhood survivors of ALL. Diagnosis of B-ALL/LBL requires demonstration of B lymphoblasts with the characteristic immunophenotype in peripheral blood, bone marrow, or other involved tissue.

The most important cytogenetic abnormality in adult ALL is the Philadelphia chromosome, found in 20 to 30% of patients and is associated with poor prognosis. Although ALL in children is curable, survival in adult patients (older than 19 years) remains inferior despite the adoption of pediatric ALL regimens. Treatment includes chemotherapy and stem cell transplantation. CNS prophylaxis is an essential part of ALL therapy.

Conclusion:

In young adults who present with fever, headache, cytopenias and negative infectious work up, it is prudent to consider hematological malignancies in the initial differential diagnosis.

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An unusual cause of headache and fever in a young adult

Mrudula Thiriveedi, MD[1]; Parekha Yedla, MD[2]
UAB Huntsville, AL[1]; Associate Professor, UAB Medicine Huntsville, AL[2]

LEARNING OBJECTIVES:

- An approach to non-infectious etiology of headache and fever in a young adult.

CASE:

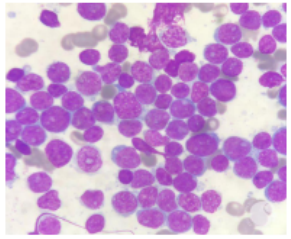
- A 22-year-old Caucasian German exchange student with no past medical history presented with pulsatile headache in the occipital region for 4 weeks.
- Reported an episode of vomiting and near syncope on the morning of admission.
- She had a fever of 102.7 degrees Fahrenheit upon arrival.
- Physical exam was normal without any skin or joint findings.
- Cerebrospinal fluid (CSF) was clear with negative meningoencephalitis panel.
- Labs revealed leukopenia with significant neutropenia and anemia.

Labs	
WBC count	0.97 x 10^3/mcL
Neutrophils	6.3%
Lymphocytes	88.3%
Hemoglobin	7.2 g/dL
Platelet count	181 x 10^3/mcL

- Extensive work was negative including Human immunodeficiency virus, Epstein bar virus, Cytomegalo virus, Parvo virus, Rickettsia serology, Lyme and Tick panel.
- Work up for anemia and leukopenia showed normal iron and B12 levels.
- Patient was started empirically on Cefepime in the interim.
- Due to lack of improvement in her clinical course, bone marrow biopsy was done on day three of hospitalization.
- Pathology showed B-cell acute lymphoblastic leukemia with 91% blasts on flow cytometry.
- CSF flow cytometry could not be done as the fluid was not sufficient, so possible leptomeningeal involvement could not be established.
- After a long discussion with the patient and family (in Germany), she was flown to Germany on a Med flight for further treatment.

DISCUSSION:

- Acute lymphoblastic leukemia (ALL) is a hematologic malignancy of undifferentiated lymphoid precursor cells, which leads to excessive production of abnormal lymphoblasts in the bone marrow.
- B-ALL is most common in children, with a second peak in adults more than 60 years old, rarely seen in young adults[1].
- The cause is unknown, but it may be associated with ionizing radiation and/or as-yet unidentified infectious agents.
- Symptoms include malaise, bleeding, infections or bone pain.
- Less than 10% may have symptomatic CNS involvement at diagnosis.



Bone marrow filled with blasts in ALL

ALL prognostic factors	Good	Poor
Age	2-10 y/o	<1 y/o; adult
WBC		>50,000 blasts
Phenotype	Precursor B-cell	Mature B-cell, null cell
Cytogenetics	High hyperploidy	Pseudo-diploid t(9;22), t(8;14)

Discussion continued..

- Headache is a common symptom among childhood survivors of ALL[2].
- Diagnosis of B-ALL/LBL requires demonstration of B lymphoblasts with the characteristic immunophenotype in peripheral blood or bone marrow.
- The most important cytogenetic abnormality in adult ALL is the Philadelphia chromosome, found in 20 to 30% of patients and is associated with poor prognosis.
- Although ALL in children is curable, survival in adult patients (older than 19 years) remains inferior despite the adoption of pediatric ALL regimens.
- Treatment includes chemotherapy and stem cell transplantation. CNS prophylaxis is an essential part of ALL therapy.

CONCLUSION:

- In young adults who present with fever, headache, cytopenias and negative infectious work up, it is prudent to consider hematological malignancies in the initial differential diagnosis.

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“Antidepressant choice in hepatic dysfunction”

Morgan Read, Clinton Martin, M.D.

Learning Objective: To investigate the literature for dosing adjustment and best therapy for treatment of depression in patients with chronic liver disease.

Case Presentation: This review was inspired by a 62-year-old patient with stage IV liver cirrhosis, uncontrolled diabetes mellitus and additional comorbidities. The patient was experiencing an increase in depressive symptoms: low energy, trouble sleeping, irritable mood, anhedonia, and helplessness. Currently taking Duloxetine 30 mg three times per day.

Discussion: Liver function has dynamic effects on pharmacokinetics: biotransformation, plasma protein binding, liver blood flow, and biliary excretion. CYP450 is the major player in antidepressant metabolism and is variably affected in CLD patients. Mirtazapine is a preferred antidepressant for liver failure patients. It demonstrates ~33% reduction in clearance and increase in half life. Start with 50% normal dose and mindful titration. SSRI with appropriate dose adjustments are also preferred. Liver transplant patients require different considerations, focusing more on drug interactions and analyzing drug-specific effects on the P450. Escitalopram is widely recognized for its safety in respect to drug interactions and can be useful in OLT patients with extensive medication lists. Suggest dosing in normal loading dose with a 50% decrease in maintenance dose vs hepatic healthy patients.

Antidepressant choice in hepatic dysfunction?

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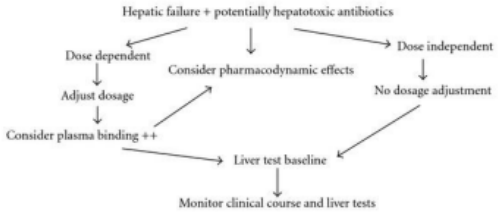
Introduction

- Approximately 1/3 cirrhotic patients experience depressive features, particularly hepatitis C patients
- Both those with chronic liver disease (CLD) and post orthotopic liver transplant (OLT) recipients with depressive symptoms have higher mortality rates than their non-depressed counterparts
- Patients with hepatic dysfunction have increased susceptibility to adverse effects of medications as well as exaggerated responses of desired effects
- Official guidelines for prescribing and dosing antidepressants in patients with hepatic dysfunction are lacking and a full knowledge of the pharmacokinetic and adverse effect profile of medication is essential to guide clinical decision-making.

Case Report

This review was inspired by a 62-year-old patient with stage IV liver cirrhosis, uncontrolled diabetes mellitus and additional comorbidities. The patient was experiencing an increase in depressive symptoms: low energy, trouble sleeping, irritable mood, anhedonia, and helplessness. Currently taking Duloxetine 30 mg three times per day..



Discussion

- Whether to use or avoid the medication with known hepatotoxicity in a patient with liver disease represents a major clinical controversy
- CYP450 is the major player in antidepressant metabolism and is variably affected in CLD patients
- **Mirtazapine is a preferred antidepressant for liver failure patients**. It demonstrates ~33% reduction in clearance and increase in half life. Start with 50% normal dose and mindful titration
- OLT patients require different considerations, focusing more on drug interactions and analyzing drug-specific effects on the P450
- **Escitalopram is widely recognized for its safety in respect to drug interactions and can be useful** in OLT patients with extensive medication lists. Suggest dosing in normal loading dose with a 50% decrease in maintenance dose vs hepatic healthy patients

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THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

Atypical Presentation of Neurosyphilis

John Gooch, MD PGY2, Department of Family Medicine UAB Huntsville Regional Campus;

Marshall Pritchett III, MD PGY1, Department of Family Medicine UAB Huntsville Regional Campus;

Nessy Abraham-Phillip, MD Department of Internal Medicine UAB Huntsville Regional Campus

Learning Objectives:

- Recognize the atypical presentations of advanced syphilis
- Understand the atypical diagnosis associated with new onset seizures

Case Presentation:

34-year-old African American morbidly obese man admitted to the hospital for syncope proceeded by lightheadedness. He was outside on a warm day with poor oral intake throughout the day. Loss of consciousness lasted less than 1 minute. No history of head trauma and no witnessed seizure. He had quick recovery to baseline mental status and was transported by ambulance to a local emergency department. In the emergency department, he had an episode of tonic-clonic seizure lasted less than 1 minute in duration which with decreased level of consciousness and he was emergently intubated for airway protection. CT scan of the brain without contrast was normal. While in the intensive care unit patient had no further seizure activity. Patient was able to be titrated off of sedation and extubated successfully. Neurological examination did not reveal focal deficit. MRI of the brain with and without contrast was unremarkable. Initial EEG did not reveal epileptiform seizure activity. HIV test was positive. Absolute CD4 count was greater than 300. Upon this result, patient had other routine sexually transmitted infection studies obtained which demonstrated a positive rapid plasma regain (RPR). Although patient had no repeat gross focal seizure activity or episodes of syncope and patient's neuro exam was nonfocal he remained confused and slow to respond to questions. Lumbar puncture was obtained. RPR in the cerebrospinal fluid which was positive. VDRL confirmed presence of treponemal antibody in the cerebrospinal fluid. CSF encephalitis panel was unremarkable for any other pathogens. A repeat electroencephalogram was obtained which did demonstrate focal epileptiform activity in the right hemisphere. Patient was immediately started on intravenous ampicillin and had a rapid neurologic recovery.

Discussion:

Although there is an overall decrease in incidence of syphilis, the incidence of neurosyphilis has increased over the past decade in immunocompromised populations. With the implementation of antibiotics, the incidence of neurosyphilis has decreased dramatically, but the percentage of symptomatic neurosyphilis still remains as high as 13%. Meningeal neurosyphilis are generally characterized by thickening of meninges. This can potentially lead to blockage around the foramina of the fourth ventricle resulting in hydrocephalus. Gummas are the result of leptomeningeal inflammatory reaction. Vascular neurosyphilis can cause arteritis and thrombosis leading to cerebral infarction. The clinical manifestations of Meningovascular neurosyphilis are secondary to the underlying pathology because body treponemal infection. Parenchymatous neurosyphilis however, is caused by direct

infection of treponema pallidum in brain tissue. Parenchymatous neurosyphilis is clinically appreciated by general paresis and tabes dorsalis. However, parenchymal neurosyphilis can also present with atypical features such as acute disseminated encephalomyelitis, stroke-like features, seizure and seizure-like activity, status epilepticus, cognitive decline, and mood disorders.

In patients with new onset seizures, neurosyphilis should be considered. MRI and abnormal EEG findings are nonspecific to treponemal infection and serum studies such as RPR and VDRL should be performed in patients suspicious for neurosyphilis. If serology results positive, CSF studies should be examined to diagnose neurosyphilis.

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Something on Your Mind

An Atypical Presentation of Neurosyphillis

John Gooch, MD PGY2, Department of Family Medicine UAB Huntsville Regional Campus;
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Nessy Abraham-Phillip, MD Department of Internal Medicine UAB Huntsville Regional Campus

Learning Objectives

· Recognize the atypical presentations of advanced syphilis
· Understand the atypical diagnosis associated with new onset seizures

Discussion:

Although there is an overall decrease in incidence of syphilis, the incidence of neurosyphilis has increased over the past decade in immunocompromised populations. With the implementation of antibiotics, the incidence of neurosyphilis has decreased dramatically, but the percentage of symptomatic neurosyphilis still remains as high as 13%. Meningeal neurosyphilis are generally characterized by thickening of meninges. This can potentially lead to blockage around the foramina of the fourth ventricle resulting in hydrocephalus. Gummas are the result of leptomeningeal inflammatory reaction. Vascular neurosyphilis can cause arteritis and thrombosis leading to cerebral infarction.

Case Presentation:

34-year-old African American morbidly obese man admitted to the hospital for syncope proceeded by lightheadedness. He was outside on a warm day with poor oral intake throughout the day. Loss of consciousness lasted less than 1 minute. No history of head trauma and no witnessed seizure. He had quick recovery to baseline mental status and was transported by ambulance to a local emergency department. In the emergency department, he had an episode of tonic-clonic seizure lasted less than 1 minute in duration which with decreased level of consciousness and he was emergently intubated for airway protection. CT scan of the brain without contrast was normal.

While in the intensive care unit patient had no further seizure activity. Patient was able to be titrated off of sedation and extubated successfully. Neurological examination did not reveal focal deficit. MRI of the brain with and without contrast was unremarkable. Initial EEG did not reveal epileptiform seizure activity. HIV test was positive. Absolute CD4 count was greater than 300. Upon this result, patient had other routine sexually transmitted infection studies obtained which demonstrated a positive rapid plasma regain (RPR). Although patient had no repeat gross focal seizure activity or episodes of syncope and patient's neuro exam was nonfocal he remained confused and slow to respond to questions. Lumbar puncture was obtained. RPR in the cerebrospinal fluid which was positive. VDRL confirmed presence of treponemal antibody in the cerebrospinal fluid. CSF encephalitis panel was unremarkable for any other pathogens. A repeat electroencephalogram was obtained which did demonstrate focal epileptiform activity in the right hemisphere. Patient was immediately started on intravenous ampicillin and had a rapid neurologic recovery.

Discussion Cont.

The clinical manifestations of Meningovascular neurosyphilis are secondary to the underlying pathology because body treponemal infection. Parenchymatous neurosyphilis however, is caused by direct infection of treponema pallidum in brain tissue.

Parenchymatous neurosyphilis is clinically appreciated by general paresis and tabes dorsalis. However, parenchymal neurosyphilis can also present with atypical features such as acute disseminated encephalomyelitis, stroke-like features, seizure and seizure-like activity, status epilepticus, cognitive decline, and mood disorders. In patients with new onset seizures, neurosyphilis should be considered. MRI and abnormal EEG findings are nonspecific to treponemal infection and serum studies such as RPR and VDRL should be performed in patients suspicious for neurosyphilis. If serology results positive, CSF studies should be examined to diagnose neurosyphilis.

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Auto Immune Hemolytic Anemia (AIHA) due to Bactrim

Jose Cavo, MD, Sujatha Baddam, MD, Farrah Ibrahim, MD

University of Alabama at Birmingham – Huntsville Regional Medical Campus Internal Medicine Residency Program

Learning objectives:

1. To recognize Bactrim as a cause of Autoimmune Hemolytic Anemia.
2. To learn about Auto Immune Hemolytic Anemia

Case Presentation:

A 69-year-old Caucasian male admitted for shortness of breath, generalize weakness and fatigue. Past medical history significant for iron deficiency anemia for six years. Patient is a truck driver. Six weeks ago, he had a positive urine test during DOT physical examination and Bactrim started. He did not have urinary symptoms. Three weeks after starting Bactrim, he developed chest pain while driving. He went to the nearest emergency department and was found to have a Hemoglobin of 4 gm/dL. He was diagnosed with warm antibodies AIHA and transfused four units of PRBCs. Repeat urinalysis was positive again, and he was prescribed Bactrim for urinary tract infection and discharged home. Several days prior to this admission, he had worsening shortness of breath, weakness and developed jaundice. Laboratory results were significant for hemoglobin of 3.8 gm/dL with elevated MCV, MCHC and RDW, an indirect hyperbilirubinemia of 1.9 mg/dL, elevated LDH at 630 EnzU/L, low haptoglobin level <10 mg/dL and positive Direct Coombs, Broad Spectrum Coombs, Anti-IgG Coombs and Complement specific Dat. Prednisone 60 mg orally daily started with gradual resolution of symptoms and improvement of hemoglobin levels.

Impact/Discussion:

Auto Immune Hemolytic Anemia is caused by warm agglutinins. The term 'warm agglutinin' is a misnomer as in fact it rarely causes agglutination of the red blood cells. Warm agglutinins are IgG antibodies that react with surface protein antigens on RBCs at a body temperature. Most cases are idiopathic but common causes include preceding viral infection, autoimmune diseases, connective tissue disorders, and immune deficiencies, malignancies of immune system, previous transfusions/transplants and drugs. The list of antibiotics is extensive and Bactrim has one of the weakest associations. Stronger antibiotic associations seen with beta lactams, including penicillin and cephalosporin. There are no specific symptoms with AIHA, patients instead present with symptoms of anemia in general including fatigue, exertional dyspnea, dyspnea at rest, palpitations, paleness, and jaundice. Physical exam may reveal pallor and splenomegaly. The presence of lethargy, confusion, and dyspnea with tachycardia constitutes a medical emergency. Laboratory workup usually consistent with a hemolytic anemia with decreased levels of hemoglobin and hematocrit, with spherocytes on peripheral blood smear, low haptoglobin level, elevated LDH, and indirect hyperbilirubinemia. Diagnosis is made once hemolytic anemia is suspected, with a positive direct antiglobulin test (DAT, also known as Direct Coombs test),

and less commonly with a positive indirect Coombs. Treatment involves volume resuscitation with blood transfusions, glucocorticoids, discontinuation of possible offending agents and evaluation for secondary causes.

Conclusion:

In this case, an otherwise healthy male developed AIHA after exposure to Bactrim. However, most cases of AIHA are idiopathic, the timing of AIHA after exposure to Bactrim strongly points to Bactrim as the culprit. AIHA is a medical condition of ongoing red blood cells autoimmune destruction that may present as a medical emergency. Early diagnosis and removal of offending agent and initiation of therapy can be lifesaving.

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Autoimmune Hemolytic Anemia (AIHA) due to Bactrim

Jose Cavo, M.D., Sujatha Baddam, M.D., Farrah Ibrahim, M.D.

Learning Objective:

To recognize Bactrim as a cause of and to learn about Autoimmune Hemolytic Anemia

Etiologies

- Most often it is idiopathic
- Post viral illness
- Autoimmune diseases
- Immune deficiencies
- Malignancies
- Connective tissue diseases
- Previous transplants/transfusions
- Drugs

Presentation

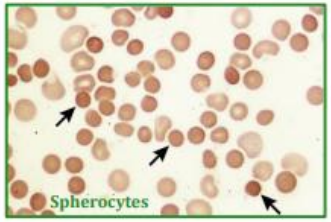
- Lethargy
- Exertional dyspnea
- Dyspnea at rest
- Palpitations
- Pallor
- Jaundice
- Splenomegaly
- Confusion
- Tachycardia

Laboratory Evaluation

- CBC with manual differential
- Reticulocyte count
- Lactate dehydrogenase
- Haptoglobin
- Fractionated Bilirubin
- Direct Coombs Test

Case Presentation:

- **Chief Complaint**: 62 year old male with past medical history of iron deficiency anemia presented with severe and worsening shortness of breath, generalized weakness and fatigue for the past several days.
- **ROS**: jaundice, chest pain
- **6 weeks earlier**- started on Bactrim for abnormal UA during a work-mandated physical exam
- **3 weeks earlier-** goes to ED with shortness of breath and is found to have hemoglobin of 4 mg/dL. Is diagnosed with AIHA and transused with PRBCs. UA again abnormal and given another course of Bactrim.
- **Physical exam:** jaundice, pallor
- **Labs:** hemoglobin 3.8 mg/dL, indirect bilirubin 1.9mg/dL, LDH 630 EnzU/L, haptoglobin level <10 mg/dL and positive Direct Coombs.
- **Treatment:** Was started on Prednisone 60 mg orally daily, with gradual resolution of symptoms and improvement of hemoglobin levels.



Management
Volume resuscitation with blood transfusion
Glucocorticoids
Discontinue offending agent

Diagnosis
Hemolytic Anemia: **↓Hemoglobin\Hematocrit** **↓Haptoglobin** **↑Lactate Dehydrogenase** **↑Direct bilirubin** **↑Reticulocytes**
Peripheral blood smear: **Spherocytes**
Direct Antiglobulin Test **+ DAT (Coomb's)**

Discussion:

- AIHA is caused by warm agglutinins, which is a misnomer as it rarely causes RBC agglutination
- Warm agglutinins are IgG antibodies that react with surface protein antigens on RBCs at body temperature.
- AIHA is a condition of ongoing RBC autoimmune destruction that may present as a medical emergency
- Many drugs, including many antibiotics are associated with AIHA. Beta-lactams have the strongest associations.
- Bactrim has a weak association but in this case, the patient was only exposed to Bactrim, strongly pointing to Bactrim as the culprit.

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Bizarre Behavior on Ambien

Nicole Lassiter, Clinton Martin, MD

Submission Type: Clinical Vignette

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Learning Objectives:

- Identify risk factors for parasomnias with hypnotic-sedative use
- Discuss the importance of depression and safety screening for patients on zolpidem

Case Presentation:

The patient is a 48-year-old female with a past medical history of bipolar 1 disorder with moderate mania, insomnia, schizophrenia, and ulcerative colitis who presented to the psychiatrist in ~2012 with complaints of insomnia. She had experienced years of difficulty with sleep onset prior to presentation and was started on Ambien 10 mg.

One week after initiating Ambien at bedtime, the patient experienced parasomnias including somnambulism and sleep-driving. She reportedly had multiple episodes of going to bed and awakening in odd scenarios. She once drove to a graveyard in the middle of the night. Her daughter once found her nude in the driveway of their home howling at the moon. She has no recollection of getting out of bed or driving. She also reports disturbing nightmares while on Ambien. She still felt like she was in a dream upon wakening the next day likening the sensation to hallucinations. She says she was awake but could discern that she was still dreaming. She also experienced new onset headaches after beginning Ambien and worsening depression with suicidal ideation.

She denies any personal or family history of somnambulism or other parasomnias. She denies concomitant alcohol or drug ingestion while taking the medication. The only other medication she was taking at the time was Geodon.

After two months of taking Ambien, patient decided to wean herself off due to negative side effects. She also had no improvement of insomnia. She had headaches until one week after discontinuation of Ambien and sleep-related disorders until one month after discontinuation. She also tried Lunesta, another sedative-hypnotic, in the past for insomnia but underwent no complex sleep-related disorders while taking it and saw no improvement of symptoms.

Discussion:

Insomnia continues to plague 60 million Americans at any given time, and it remains the culprit behind five million doctor's visit each year. Some patients turn to pharmacotherapy for help. Sedative-hypnotics including Ambien are tools used to combat this sleep disturbance. Ambien acts as an agonist at the benzodiazepine site $GABA_A$ receptors with α1 subunits. While it has a less addictive profile than benzodiazepines and is mostly harmless, some of its side effects can have negative implications.

Although rare with only 66 cases reported to the FDA over a 26-year period, complex sleep-related disorders can be seen with Ambien use. As described in the case presentation, these behaviors consist of sleepwalking, sleep eating, sleep driving, and anterograde amnesia. In some cases, such behaviors have led to serious injury or death from drowning, motor vehicles crashes, and more. Hence, it is crucial that providers proceed with caution and properly screen patients before prescribing Ambien.

Potential risk factors for emergence of complex sleep-related disorders while on sedative-hypnotics include primary sleep disorders such as obstructive sleep apnea, history of parasomnia, simultaneous use of other sedating substances including alcohol, sedative ingestion outside of normal bedtime, and living alone. Chances are also increased with increasing dosage. The appearance of complex sleep-related disorders can occur ranges and can occur after the first dose of Ambien or after several. Women are particularly susceptible, and one study showed that younger patients may be more vulnerable.

All patients beginning Ambien should be assessed for potential complication with complex sleep-related disorders. Additionally, patients who take Ambien consistently should be monitored for emergence of these parasomnias, nightmares, and worsening depression. Access to methods of self-harm should also be explored as there are cases of suicide attempts during sleep.

***This vignette was chosen for oral presentation on Research Day.**

Conversion Disorder

William Nolan, MS3, University of Alabama at Birmingham School of Medicine at Huntsville, Huntsville, Alabama. Faculty Advisor: Dr. Tarak Vasavada, Department of Psychiatry, Huntsville Hospital

Conversion disorder is a psychiatric condition characterized by neurologic symptoms, such as seizures, weakness, paralysis, or sensory deficits, which are unexplained by any known physiologic causes or observable imaging findings. Patients are typically convinced that they have a serious neurological condition, which makes psychiatric intervention difficult to initiate. Conversion disorder is a diagnosis of exclusion, which requires extensive neurological, metabolic, and psychiatric work-up prior to proper identification.

A 35 year old woman with a past medical history significant for depression, anxiety, and post traumatic stress disorder presented to the emergency department with generalized weakness, paresthesias, difficulty speaking, and intermittent diplopia that have been worsening over the past 13 months. Prior to symptom onset, she reports being functionally normal and stable on all of her psychiatric medications. One year ago, the patient was admitted for lower extremity paresthesias and received extensive neurological and psychiatric work-up. Ultimately, she was diagnosed with a folate deficiency and discharged with the appropriate medications. However, she claims that these treatments were ineffective and she became progressively disabled to the point where she was completely bed-bound. She has not seen a physician since her admission one year ago, and she has stopped taking her medications due to difficulties refilling prescriptions. Although she lives with family, they find it difficult to care for her, as she remains unable to move and reports feeling extreme physical pain whenever she is touched. She wears diapers and cannot remember the last time she bathed. Her nutrition has been poor, as her husband usually feeds her fast food. She also suffers from difficulty swallowing, which limits her diet. For the past 2 weeks, she has developed difficulty speaking and generalized pain, especially in her abdomen, which sometimes radiates into her throat and makes her feel like she is choking. Upon admission, neurology, neurosurgery, and psychiatry were consulted. Despite having positive neurological and musculoskeletal findings on physical exam, CTs and MRIs revealed no structural abnormalities, and repeat electromyography showed no evidence of nerve dysfunction. Her labs were unremarkable and unchanged since her last admission. Per psychiatry, her constellation of symptoms appeared to be consistent with conversion disorder, and treatment was initiated with vyvanse, gabapentin, alprazolam, duloxetine, and mirtazapine. Additionally, a note was sent to her PCP advising against changes to any medications.

This case illustrates the complicated process of diagnosing conversion disorder in the modern medical setting. Patients present with serious but chronic symptoms that cannot be explained by any objective measures, and it's difficult for physicians to diagnose and treat this disorder. Furthermore, because patients are convinced they have a serious neurological disease, they become disillusioned with physicians and avoid stable, consistent medical care. Even those patients who do wish to see a physician

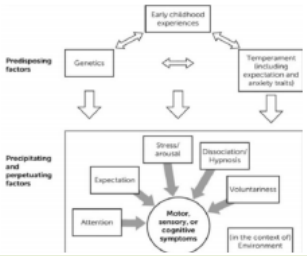
are unable to follow-up due to their disability and require additional assistance throughout the treatment process.

Prolonged Conversion Disorder: exacerbated by misdiagnosis?

William Nolan MS3, Tarak Vasavada, MD
UABSOM Huntsville Campus; Department of Psychiatry
University of Alabama at Birmingham School of Medicine

Introduction

- Conversion disorder is a psychiatric condition characterized by neurologic symptoms, such as motor or sensory deficits, which are unexplained by any known physiologic causes or observable imaging findings.
  - Symptoms are not intentionally produced, which differentiates it from factitious disorder or malingering.
  - Patients most commonly present with paralysis, blindness, or mutism, but a variety of other neurological symptoms have been documented, such as pseudo-seizures, anesthesia, ataxia, deafness and nerve pain.[1]
- Conversion disorder is surprisingly common with an estimated 20-25% of admitted patients to a general medical service having conversion symptoms
  - Conversion disorder is more common in females (2:1-10:1)
  - Onset can occur at any age, but it is more common in between the age of 10-35.[2]
- There has been an observed comorbidity between conversion disorder and other psychiatric conditions: Anxiety and Depression, other somatization disorders, histrionic personality disorder, dependent personality disorder, and antisocial personality disorder
  - A reported 25% of psychiatric outpatients were found to have at least one conversion symptom.[3]
- Patients are typically convinced that they have a serious neurological condition, which makes psychiatric intervention difficult to initiate. Because the condition is short-lived and self-resolving, it can be difficult to identify the true etiology of the neurologic symptoms unless the patient receives extensive work-up during the event.
  - One study found that true neurological disorders were found to have developed prior to or following up to 65% cases of conversion disorder, however another report found that up to 50% of conversion disorder cases were mistakenly labeled nonpsychiatric medical disorders.[4,5]
- In 90% of cases, symptoms of conversion disorder usually last for days to weeks and often resolve spontaneously without treatment.
  - Most of these resolved cases (75%) never have another episode of conversion disorder.[3]



Case Description

- A 35-year-old woman with a past medical history significant for depression, anxiety, ADHD, and post-traumatic stress disorder presented to the emergency department with generalized weakness, paresthesias, difficulty speaking, and intermittent diplopia that have been worsening over the past 13 months. Prior to symptom onset, she reports being functionally normal and stable on her psychiatric medications.
- One year ago, the patient was admitted for lower extremity paresthesias and received extensive neurological and psychiatric work-up. Ultimately, she was diagnosed with a folate deficiency and discharged with the appropriate medications. However, she claims that these treatments were ineffective and she became progressively disabled to the point where she was completely bed-bound. She has not seen a physician since her admission one year ago, and she has stopped taking her medications due to difficulties refilling prescriptions.
- As she remains unable to move and reports feeling extreme physical pain whenever she is touched. She wears diapers and cannot remember the last time she bathed. Her nutrition has been poor, as her husband usually feeds her fast food. She also suffers from difficulty swallowing, which limits her diet. For the past 2 weeks, she has developed difficulty speaking and generalized pain, especially in her abdomen, which sometimes radiates into her throat and makes her feel like she is choking. Upon admission, neurology, neurosurgery, and psychiatry were consulted.

Neurological Exam:
AOx3
CN II-XII intact
Speech: Fluent, comprehension intact
Decreased muscle tone throughout with bilateral upper and lower extremity wasting, bilateral UE strength 4/5, bilateral lower extremity strength 3/5
No pronator drift
Allodynia throughout
No dysmetria or tremor
Reflexes in upper and lower extremities absent

- Despite having positive neurological and musculoskeletal findings on physical exam, CTs and MRIs revealed no structural abnormalities, and repeat electromyography showed no evidence of nerve dysfunction.
- Her labs were unremarkable and unchanged since her last admission. During hospital stay, patient refused to work with physical therapy. Rehab was offered but patient was unwilling.
- Her significant psychiatric history was considered a major factor in her hospital course. Adequate treatment and therapy for her psychiatric conditions were deemed imperative to the overall improvement of her symptoms.

Discussion

- This case illustrates the complicated process of diagnosing prolonged conversion disorder in the modern medical setting. Patients present with serious but chronic symptoms that cannot be explained by any objective measures, and it's difficult for physicians to diagnose and treat this disorder when the symptoms have been present for such a long period of time.
- •Furthermore, because patients are convinced, they have a serious neurological disease, they become disillusioned with physicians and avoid stable, consistent medical care. Even those patients who do wish to see a physician are unable to follow-up due to their disability and require additional assistance throughout the treatment process.
- •Several studies have focused that high numbers of conversion disorder progress from an acute event to a recurrent or chronic condition and highlighted the importance of early diagnosis and explanation of these findings as psychiatric in nature to dissuade patients from pursuing expensive testing.[4,5,6,7]
- •Furthermore, it appears that just as in this patient, chronic conversion disorder can present alongside other comorbidities, such as nutritional deficiencies, psychiatric disorders, and musculoskeletal complaints, which all create red herrings and clinical outliers for the diagnosing physician.
- •In this case, it becomes clear that consulting physicians in the inpatient setting must stay open-minded to the possibility of prolonged conversion disorder as it can often become difficult to provide outpatient care to these patients when the are discharged without adequate therapy or assistance.

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THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

Title: Cytomegalovirus Induced Thrombocytopenia with No Platelets in an Immunocompetent Young Male

Authors: Sujatha Baddam, MD[1], Jose Cavo, MD[1], Kushdeep Chahal, MD[1]

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Type: Clinical vignette

Learning Objective

1. To recognize Cytomegalovirus as a cause of severe thrombocytopenia in immune competent people
2. To learn about immune thrombocytopenic purpura and treatment options

Case Presentation

A 36-year-old Caucasian male without any past medical history presented to emergency room (ER) with flu like symptoms for five days associated with subjective fevers, anorexia, nausea, cough and weight loss of 15 lb. in two weeks. He also reported possible tick bite while working in the yard two days prior to admission. No dizziness, vomiting, diarrhea or any bleeding were reported. Denied any smoking, alcohol use or any illicit drug use. No significant family history was reported. On evaluation he was afebrile, normotensive with normal heart rate and respiratory rate. Physical examination was unremarkable. Initial laboratory data revealed hemoglobin of 11.2, platelet count 4 x109, white cell count of 13,100 with 4.5% atypical lymphocytes, aspartate aminotransferase of 41, alanine aminotransferase of 49, and creatinine of 1.4. He tested positive for Influenza A, CMV Immune globulin (Ig)M and IgG antibody. Serological tests for tick panel including anaplasma, babesia, Lyme disease and ehrlichia were negative. Epstein-Barr virus (EBV) antibody, parvo virus antibody, hepatitis screen, HIV screen, auto antibodies including anti-nuclear antibody and anti-double stranded DNA were negative. Coombs test was negative.

Further work up includes ADAMTS13 activity was normal. No laboratory evidence of ADAMTS13 deficiency. After excluding all other causes, diagnosis of ITP was made. He was started on Tamiflu for Influenza A and high dose intra venous (IV) methyl prednisone for ITP. After platelet transfusion and two days of IV steroids platelet count improved 43 x 10^9 and he was discharged home with prolonged prednisone taper. Five days later, he presented to ER with severe epistaxis. Laboratory data revealed platelet count of 0 x10^9. Serum CMV-DNA was determined by PCR showed viral load of 8,790 copies/ml. Ultrasound abdomen showed mild splenomegaly. He received three doses of IVIG (1g/kg). Platelet count failed to improve after administration of IVIG. Bone marrow biopsy revealed hyper cellular marrow with trilineage hematopoiesis with no increase in CD 34 blasts. Per infectious disease and hematology recommendations, he was started on valganciclovir (900 mg PO

BID). One month later, platelet count improved to 150 x 109 and CMV viral load dropped to 413 with subsequent resolution of patient's symptoms.

Discussion

Secondary ITP is an acquired thrombocytopenia caused by autoantibodies against platelets. Many patients with ITP are asymptomatic. For those who do have symptoms, initial presentations of ITP are petechiae, purpura and epistaxis, with a more severe progression to intracranial hemorrhage or gastrointestinal bleeding leading to a fatal outcome, if treatment is not started on a promptly manner. CMV induced thrombocytopenia in immunocompetent adults seems to be rare. we are presenting a case of CMV induced ITP which failed to improve after standard treatment with high dose steroids and IVIG but responded to anti-viral therapy with valganciclovir. In conclusion, it may be worthwhile to test for CMV infection in patients presenting with ITP. Further research is needed in order to establish treatment guidelines for CMV induced ITP in immunocompetent adults.

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Cytomegalovirus Induced Thrombocytopenia with No Platelets in an Immunocompetent Young Male

Sujatha Baddam, MD[1], Jose Cavo, MD[1], Kushdeep Chahal, MD[1] [1]University of Alabama, Birmingham, Huntsville regional campus

Learning Objectives

1. To recognize Cytomegalovirus as a cause of severe thrombocytopenia in immune competent people
2. To learn about immune thrombocytopenic purpura and treatment options

Background

Immune thrombocytopenic purpura (ITP) is a common cause of acquired thrombocytopenia in an otherwise asymptomatic adult. It is generally believed to be caused by auto-antibodies against platelet antigens that destroy platelets peripherally and autoreactive cytotoxic T cells, as well as humoral and cellular autoimmunity directed at megakaryocytes, causing impaired platelet production. Cytomegalovirus (CMV) is a known cause of cause of morbidity and mortality in patients with immunosuppressed states. We are presenting a case of CMV induced thrombocytopenia in an otherwise healthy immunocompetent male who failed to improve after standard treatments with high dose steroids and intra venous immune globulin (IVIG).

Case Presentation

A 36-year-old Caucasian male without any past medical history presented to emergency room (ER) with flu like symptoms for five days associated with subjective fevers, anorexia, nausea, cough and weight loss of 15 lb. in two weeks. He also reported possible tick bite while working in the yard two days prior to admission. Denied any smoking, alcohol use or any illicit drug use.

On evaluation vitals were normal. Physical examination was unremarkable. Initial laboratory data revealed hemoglobin of 11.2, platelet count 4 x109, white cell count of 13,100 with 4.5% atypical lymphocytes, and creatinine of 1.4. He tested positive for Influenza A, CMV Immune globulin IgM and IgG antibody. Serological tests for tick panel including anaplasma, babesia, Lyme disease and ehrlichia were negative. Epstein-Barr virus (EBV) antibody, parvo virus antibody, hepatitis screen, HIV screen, auto antibodies including anti-nuclear antibody and anti-double stranded DNA were negative. Coombs test was negative.

Further work up includes ADAMTS13 activity was normal. After excluding all other causes, diagnosis of ITP was made. He was started on Tamiflu for Influenza A and high dose intra venous (IV) methyl prednisone for ITP. After platelet transfusion and two days of IV steroids platelet count improved 43 x 10^9 and he was discharged home with prolonged prednisone taper. Five days later, he presented to ER with severe epistaxis. Laboratory data revealed platelet count of 0 x10^9. Serum CMV-DNA was determined by PCR showed viral load of 8,790 copies/ml. Ultrasound abdomen showed mild splenomegaly. He received three doses of IVIG (1g/kg). Platelet count failed to improve after IVIG. Bone marrow biopsy revealed hyper cellular marrow with trilineage hematopoiesis with no increase in CD 34 blasts. Per infectious disease recommendations, he was started on valganciclovir (900 mg PO BID). One month later, platelet count improved to 150 x 109 and CMV viral load dropped to 413 with subsequent resolution of patient's symptoms.

Discussion

Secondary ITP is an acquired thrombocytopenia caused by autoantibodies against platelets. Many patients with ITP are asymptomatic. For those who do have symptoms, initial presentations of ITP are petechiae, purpura and epistaxis, with a more severe progression to intracranial hemorrhage or gastrointestinal bleeding leading to a fatal outcome, if treatment is not started on a promptly manner. CMV induced thrombocytopenia in immunocompetent adults seems to be rare. we are presenting a case of CMV induced ITP which failed to improve after standard treatment with high dose steroids and IVIG but responded to anti-viral therapy with valganciclovir. In conclusion, it may be worthwhile to test for CMV infection in patients presenting with ITP. Further research is needed in order to establish treatment guidelines for CMV induced ITP in immunocompetent adults.

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Clinical Vignette Abstract:

Dalbavancin is an Effective and Safe Outpatient Treatment Option for Pyogenic Discitis Caused by Methicillin-Resistant Staphylococcus Aureus (MRSA).

Authors: Syed Shabee Hassan[1], Hafiz Muhammad Fazeel[2], *Ali Hassoun[2]

Learning Points:

Dalbavancin is:

- a bactericidal intravenous agent that inhibits call wall cross linking and reduces cell wall formation.
- a safe agent with reliable activity against gram positive bacteria including MRSA and VISA.
- an excellent option for outpatient administration owing to its long half-life, single agent dosing and freedom from regular plasma drug monitoring.
- a useful agent for patients with intravenous drug abuse due to lack of need for central intravenous device.

Case Summary:

Case-1

81-year old female presented to outpatient clinic for non-healing lumbar wound secondary to wound dehiscence following a recent spine surgery. She had worsening back pain and lumbar wound 10x6 cm which had necrotic tissue, surrounding redness and a serosanguinous drainage. Initial wound culture showed MRSA, labs were unremarkable, and MRI confirmed lumbar osteomyelitis and discitis. Patient declined daily iv antibiotic therapy and PICC line. So, dalbavancin once weekly infusions were given for 8 weeks in outpatient setting. At the end of therapy, patient symptoms resolved and wound closed completely. She had no further recurrence at 6 months follow up

Case-2

31-year old Caucasian female with history of active IV drug abuse and multiple remote MRSA infections presented to the emergency with 4-day history of stabbing right lower back pain and fever.
On presentation, she was febrile with Tmax of 101.6 F, tachycardic and tachypneic. Physical exam was significant for right sacroiliac tenderness and inability to bear weight on right leg due to pain. Labs were remarkable for leukocytosis and blood culture positive for MRSA. MRI lumbar spine and pelvis revealed an iliopsoas abscess and associated sacral osteomyelitis and discitis in L5-S1 vertebrae. She underwent CT guided drainage of the abscess. Patient was initially treated with daily telavancin till blood culture sterilization. She was discharged home on once weekly dalbavancin infusion for 5 weeks and close follow-up with ID. On her last follow up, her symptoms and signs had resolved with no recurrence.

Discussion

Dalbavancin is a bactericidal agent with a long (>300 hours) half-life, which can make it an excellent option for invasive MRSA infections that require prolong iv therapy. It does not need drug level monitoring and does not require regular home care for infusion. In addition, lack the requirement of

permanent line for infusion minimizes the risk of catheter associated infections and non-infectious complications.

Even though use of Dalbavancin for osteomyelitis is reported in few publications, studies reporting the efficacy of Dalbavancin for discitis are scarce. Further studies are needed to confirm its benefit in this serious infection.

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Dalbavancin is an Effective and Safe Outpatient Treatment Option for Pyogenic Discitis Caused by Methicillin-Resistant Staphylococcus Aureus (MRSA)

Syed Shabee Hassan[1], Hafiz Muhammad Fazeel[2], *Ali Hassoun[2]
[1] PGY I UAB Huntsville Regional Campus, Department of Internal Medicine, Huntsville, AL
[2] Observer, Alabama Infectious Disease Center, Huntsville, AL

Case-1
An 81-year old female presented to outpatient clinic due to worsening back pain and a non-healing lumbar wound secondary to wound dehiscence following a recent spine surgery.

Physical Exam: A lumbar wound 10x6 cm which had necrotic tissue, surrounding redness and a serosanguinous drainage.

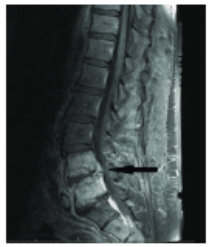
Labs/Imaging: wound culture showed MRSA, labs were unremarkable, and MRI confirmed lumbar osteomyelitis and discitis.

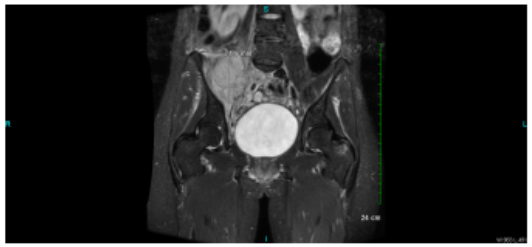
Course: Patient declined daily iv antibiotic therapy and PICC line. So, dalbavancin once weekly infusions were given for 8 weeks in outpatient setting. At the end of therapy, patient's symptoms resolved and wound closed completely. She had no further recurrence at 6 months follow up

Learning Points

Dalbavancin is:

- a bactericidal intravenous agent that inhibits call wall cross linking and reduces cell wall formation.
- a safe agent with reliable activity against gram positive bacteria including MRSA and VISA.
- an excellent option for outpatient administration owing to its long half-life, single agent dosing and freedom from regular plasma drug monitoring.
- a useful agent for patients with intravenous drug abuse due to lack of need for central intravenous device.





Discussion

- Dalbavancin is a bactericidal agent with a long (>300 hours) half-life, which can make it an excellent option for invasive MRSA infections that require prolong iv therapy.
- It does not need drug level monitoring and does not require regular home care for infusion.
- In addition, lack the requirement of permanent line for infusion minimizes the risk of catheter associated infections and non-infectious complications.
- Even though use of Dalbavancin for osteomyelitis is reported in few publications, studies reporting the efficacy of Dalbavancin for discitis are scarce. Further studies are needed to confirm its benefit in this serious infection.

Case-2
A 31-year old Caucasian female with history of active IV drug abuse and multiple remote MRSA infections presented to the emergency with 4-day history of stabbing right lower back pain and fever.

Vitals/Physical Exam: Temperature of 101.6 F, HR 104 and RR 21.
Right sacroiliac tenderness and inability to bear weight on right leg due to pain.

Labs/Imaging: WBC count 23,400. Blood cultures positive for MRSA. MRI lumbar spine and pelvis revealed an iliopsoas abscess and associated sacral osteomyelitis and discitis in L5-S1 vertebrae.

Course: She underwent CT guided drainage of the abscess. Patient was initially treated with daily telavancin till blood culture sterilization. She was discharged home on once weekly dalbavancin infusion for 5 weeks and close follow-up with ID. On her last follow up, her symptoms and signs had resolved with no recurrence.

Clinical Vignette Abstract

Title: Delay in Early Diagnosis and Treatment of HSV-1 Encephalitis Predisposes to Increased Morbidity and Mortality

Authors: Syed Shabee Hassan[1], Hafiz Muhammad Fazeel[3], Ali Hassoun[1]

Learning Points:

1. HSV-1 encephalitis is most common infectious cause of sporadic encephalitis.
2. A high index of suspicion is required as delay in early treatment is associated with significant morbidity and mortality.
3. Most common cause of delay in therapy is failure to consider HSV as one of the differential diagnoses.
4. MRI is best imaging tool and HSV-1 PCR on CSF is best serologic tool for diagnosis.

Case Summary:

48-year old female presented to local Emergency with 6-days history of high-grade fever (104 F), body aches, confusion and lethargy. Testing for flu was negative. She was discharged home after symptoms improved with empiric antibiotics and fluid hydration. She returned to the Emergency the following day with persistent high-grade fever (Tmax: 103 F), headache, new onset altered mentation with inability to speak. Physical exam was significant for expressive aphasia without any other focal neurologic deficits. Labs were remarkable for mild leukocytosis (WBCs: 12.27). MRI brain revealed an increased T2 signal in bilateral temporal lobe parenchyma and non-stenotic narrowing of cerebral vessels. Lumbar puncture for CSF studies was done and empiric Acyclovir, Vancomycin and Piperacillin-Tazobactam started. EEG came non-diagnostic. She was transferred to a tertiary care center for further evaluation. CSF analysis showed clear fluid with WBC 71, lymphocytic predominance, glucose 72, protein 68 and RBC 89. MRI and CSF findings were consistent with a viral encephalitis. HSV-1 PCR returned positive confirming a diagnosis of HSV Encephalitis (HSVE). Acyclovir was continued for 3 weeks and family was counselled on long-term morbidity particularly amnesia, behavioral abnormalities and cognitive impairment.

Discussion:

HSVE is most common viral encephalitis with a bimodal age distribution that presents with fever, confusion, headache, seizures and focal neurologic deficits although atypical presentations, as in our patient, have also been reported. Contrary to HSV-2, primary CNS infection by HSV-1 is possible and patients with old age and immunocompromised status are at increased risk. The proposed pathophysiology is transmission of virus from nasal mucosa through the olfactory and trigeminal nerves. This correlates with the parenchymal changes in temporal lobes that are most commonly associated with HSVE. CT scan brain is the usual initial test performed but it has poor sensitivity. MRI with and without contrast is best radiologic option. HSV-1 PCR done on CSF is confirmatory test although PCR is negative in first 2 days and PCR-negative infection has also been reported. EEG serves the supportive role, but our patient lacked the typical findings including periodic discharges, generalized or focal slowing and electrographic seizures. HSVE is a necrotizing brain infection and delay in treatment is associated with significant morbidity and mortality (>70%). Most common reported causes of delayed therapy are low diagnostic suspicion and waiting for preliminary CSF results. So, having a high suspicion and early initiation of therapy offer the best chance for better outcome.

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Delay in Early Diagnosis and Treatment of HSV-1 Encephalitis Predisposes to Increased Morbidity and Mortality

Syed Shabee Hassan[1], Hafiz Muhammad Fazeel[2], *Ali Hassoun[2]
[1] PGY I UAB Huntsville Regional Campus, Department of Internal Medicine, Huntsville, AL
[2] Observer, Alabama Infectious Disease Center, Huntsville, AL

Learning Points

- HSV-1 encephalitis is the most common infectious cause of sporadic encephalitis.
- A high index of suspicion is required as delay in early treatment is associated with significant morbidity and mortality.
- Most common cause of delay in therapy is failure to consider HSV as one of the differential diagnoses.
- MRI is the best imaging tool and HSV-1 PCR on CSF is best serologic tool for diagnosis.

Case Summary

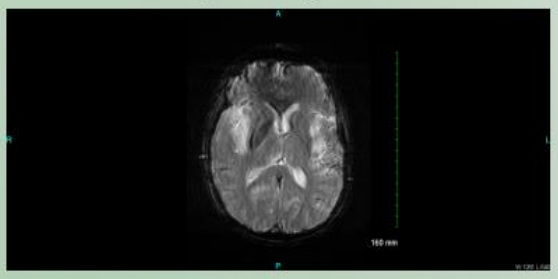
A 48-year old female presented to local emergency with 6-day history of high-grade fever (104 F), body aches, confusion and lethargy. Testing for flu was negative. She was discharged home after symptoms improved with empiric antibiotics and fluid hydration. She returned to the Emergency the following day with persistent high-grade fever (Tmax: 103 F), headache, new onset altered mentation with inability to speak.

Physical exam: Expressive aphasia without any other focal neurologic deficits.

Labs/Imaging: Mild leukocytosis (WBCs: 12.27). MRI brain revealed an increased T2 signal in bilateral temporal lobe parenchyma and non-stenotic narrowing of cerebral vessels. Lumbar puncture for CSF studies was done. EEG came non-diagnostic.

Course: Empiric Acyclovir, Vancomycin and Piperacillin-Tazobactam was started. She was transferred to a tertiary care center for further evaluation. CSF analysis showed clear fluid with WBC 71, lymphocytic predominance, glucose 72, protein 68 and RBC 89. MRI and CSF findings were consistent with a viral encephalitis.

HSV-1 PCR returned positive confirming a diagnosis of HSV Encephalitis (HSVE). Acyclovir was continued for 3 weeks and family was counselled on long-term morbidity particularly amnesia, behavioral abnormalities and cognitive impairment.



Discussion

- HSVE is the most common viral encephalitis with a bimodal age distribution that presents with fever, confusion, headache, seizures and focal neurologic deficits although an atypical presentation, like in our patient, has also been reported.
- Contrary to HSV-2, primary CNS infection by HSV-1 is possible and older patients and immunocompromised are at increased risk.
- The proposed pathophysiology is transmission of virus from nasal mucosa through the olfactory and trigeminal nerves. This correlates with the parenchymal changes in temporal lobes that are most commonly associated with HSVE.
- CT scan brain is the usual initial test performed but it has poor sensitivity. MRI with and without contrast is best radiologic option. HSV-1 PCR is the confirmatory test although PCR is negative in first 2 days and PCR-negative infections have also been reported.
- HSVE is a necrotizing brain infection and delay in treatment is associated with significant morbidity and mortality (>70%). Most common reported causes of delayed therapy are low diagnostic suspicion and waiting for preliminary CSF results. So, having a high suspicion and early initiation of therapy offer the best chance for better outcome.

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Diagnostic and Treatment challenges in Hemolytic Uremic Syndrome

Dhivya Velu MD[1]; Swetha Bheemanathni MD[2]; Ali Hassoun MD, FACP, FIDSA[3]

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LEARNING OBJECTIVES:

1. Recognize the symptoms and signs in Hemolytic Uremic Syndrome with all necessary diagnostic work ups
2. Address and rule out the other possible differential diagnosis with similar presentation as of typical HUS and their treatment options

CASE PRESENTATION:

55-year-old Caucasian female presented with complaints of non-bloody diarrhea and abdominal cramping. Patient had significant travel history to Israel from where the symptoms started. Several other co-travelers were reported to have similar symptoms but are self-resolved. Initial abdominal imaging revealed diffuse inflammatory changes in colon consistent with colitis, hepatosplenomegaly and mild ascites. Preliminary labs showed normal hematocrit and normal white blood cell counts with low platelets of 76000/mm3 concerning for severe ongoing disease process. Further labs revealed indirect hyperbilirubinemia with elevated serum creatinine. By then, Diarrhea has resolved. Eventually, Patient developed severe hemolytic anemia in form of elevated LDH and significantly low haptoglobin and declining platelet counts. Stool PCR studies were positive for Enterohemorrhagic *E. coli* and Shiga like Toxin Gene Stx2, pointing towards the diagnosis of HUS. Antibiotics were deferred since beginning and hydration with intravenous fluids was the only modality of treatment used. Insignificant ADAMTS13 levels helped in ruling out Thrombotic Thrombocytopenic Purpura (TTP). Despite resolution of symptoms with conservative treatment, the patient deteriorated clinically with worsening creatinine to 7.7 mg/dl and low platelets. There were no indications to initiate hemodialysis or other interventions with either eculizumab or plasmapheresis as per ASFA (American Society for Apheresis) criteria. Patient was monitored continuously throughout, until renal function started to improve with creatinine levels trending down, indicating slow resolution of disease. Patient improved overall and was discharged home.

DISCUSSION:

Hemolytic Uremic Syndrome (HUS) is a disease of non-immune (Coombs negative) hemolytic anemia, low platelet counts and renal impairment. Typical HUS, caused by Stx producing E. coli are often self-limited with better prognosis, though occasionally may end up with permanent damage. Rarely, Stx HUS results from *Shigella dysenteriae* infections which are associated with significant complications including septic shock, disseminated intravascular coagulation resulting in high mortality.

In this case, the diagnosis of HUS is obvious with the typical features of presentation. However, the differentials that we have mentioned above should be excluded, as timely intervention is needed for

better prognosis. Failure to initiate treatment on time like with plasmapheresis or complement inhibitor, in case of other differentials like Atypical HUS and TTP may lead to irreversible damage. Secondary HUS, though not well known, are reported to result from Streptococcus pneumoniae or viral infections. Thrombotic thrombocytopenic purpura mainly acquired form, which initially presents with similar features of HUS including severe microangiopathic hemolytic anemia (MAHA) and thrombocytopenia (most of the time lacking classic pentad features) can be easily ruled with Serum ADAMTS-13 levels or activity where it is expected to be severely low <10%. Even though, the case mentioned above is well known and well-studied, detailed work up to rule out the other possible differentials is mandatory despite the features of presentation.

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Disseminated Nocardiosis in an immunocompetent patient while on Daptomycin therapy

Dhivya Velu MD[1]; Swetha Bheemanathni MD[2]; Ali Hassoun MD, FACP, FIDSA[3]

Department of Internal Medicine, UAB Huntsville Regional Medical Campus AL[1]; North Alabama Hospitalists, Hunstville hospital, AL[2]; Alabama Infectious Disease Center, Huntsville, AL[3]

LEARNING OBJECTIVES:

1. Diagnose Nocardial infections in an immunocompetent and know the steps in management.
2. Recognize the role of Daptomycin in atypical presentation of Nocardiosis

CASE DISCUSSION:

76-year-old Caucasian female presents with painful swelling in right thigh for 2 months following a trivial blunt trauma to the affected site. Relevant medical history significant for Chronic Methicillin Sensitive Staphylococcus Aureus (MSSA) Abdominal Aortic graft infection on lifelong suppressive daptomycin therapy. Diffuse intramuscular abscess was evident from examination, given additional symptoms of low-grade fever and night sweats. Systemic examination otherwise unremarkable. Labs showed elevated inflammatory markers with normal blood cell counts. Initial imaging revealed multiple small ovoid fluid collections the largest one being 7.3 x 2.4 cm with no gas ruling out myonecrosis. Empirical antibiotics were started on Day 1 of encounter after obtaining blood cultures. Abscess was drained, and aspirated fluid was sent for cultures. Within 48 hours, Blood cultures started growing Gram positive rods. On the other side, AFB staining of abscess fluid with Ziehl Neelson method revealed Acid fast bacilli suggesting either Tuberculosis or Nocardia being the definite organism involved. Final confirmation done using Modified Kinyoun acid fast staining which stained the pathogen red confirming not Tuberculosis but Nocardia. Specific Antimicrobial therapy was initiated with Meropenem and continued the chronic daptomycin therapy. Repeat blood cultures at 72 hours were negative, indicating response to chosen therapy. At the end of 3 weeks, thigh aspirate cultures showed Nocardia growth revealing site of microbial seeding. Further tests identified the species to be Nocardia nova. Complete imaging was done and ruled out possible dissemination to any solid organs. On treatment, symptoms including abscess improved and patient discharged home. Follow up visits confirmed resolution of disease.

DISCUSSION:

Nocardiosis was primarily an opportunistic infection occurring in immunocompromised individuals, the main source being inhalation resulting in pneumonia and secondly, by direct cutaneous inoculation from soil as in abscess. Also 10% of cases have reported no valid predispositions. There are very few cases of bacteremia reported in literature, secondary to endovascular foreign objects, with evident poor outcome in terms of increased mortality despite specific treatment. Cultures require longer incubation period up to 2- 3 weeks. Nocardiosis involve 2 major pathogenic species- Nocardia brasiliensis and Nocardia asteroides complex which includes subspecies, Nocardia nova. Guidelines recommend Trimethoprim- Sulfamethoxazole as the first line drug of choice and other agents include amikacin, imipenem, meropenem and linezolid for a minimum of 3-6 months in immunocompetent and 6-12

months in immunocompromised individuals. In this case, the only possible explanation for the source could be infection through an unattended trivial penetration injury to abscess site.

Inspite of active Nocardia in blood stream, patient remained clinically stable throughout, with no organ involvement which depicts the rarity of this presentation. This could be explained by Daptomycin ongoing therapy in this patient as few studies have showed that the drug might have invitro activity against Nocardia but with very high Minimum Inhibitory Concentration (MIC).

Disseminated Nocardiosis in an immunocompetent patient while on Daptomycin therapy

THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

Dhivya Velu MD[1]; Swetha Bheemanathni MD[2]; Ali Hassoun MD, FACP, FIDSA[3]

Department of Internal Medicine, UAB Huntsville Regional Medical Campus AL[1]; North Alabama Hospitalists group, AL[2]; Alabama Infectious Disease Center, Huntsville, AL[3]

Learning objectives:

1. Diagnose Nocardial infections in an immunocompetent and know the steps in management.
2. Recognize the role of Daptomycin in atypical presentation of Nocardiosis

Case description:

ID : 76 year old Caucasian female

HPI :

Painful swelling in Right thigh x 2 months with occasional fever and night sweats

PMH :

h/o chronic methicillin sensitive Staphylococcus aureus (MSSA) Abdominal Aortic graft infection on lifelong suppressive Daptomycin therapy.

Allergic h/o: Sulfa drugs, Vancomycin

Vitals: T 37 C

BP 130/80 mm Hg

HR 92/min NSR

RR 18/min

Local examination:

Diffuse soft to firm non pulsatile palpable swelling on the right lateral aspect of thigh +.

Systemic examination: unremarkable.

Labs:

ESR > 80, CRP 8.2

9.92	10.9 / 34.2	352	140 / 4.1	101 / 25	19 / 1.2	105

HbA1C 7.0

Imaging:

MRI showed multiple small ovoid fluid collections the largest one being 7.3 x 2.4 cm.

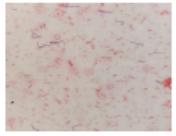
Initial diagnosis: *ABSCESS*

Further work up and management:

Day 1 Abscess was drained,
Aspirated fluid and blood samples sent for gram stain/ culture and
Empirical broad spectrum antibiotic therapy was initiated.

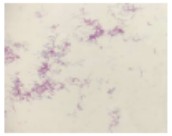
Day 2 Blood cultures 2/2 started growing *GRAM POSITIVE RODS.*

On the other hand, *AFB staining using Ziehl Neelson method revealed Acid fast bacilli.*



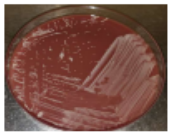
Given the results,
Mycobacteria spp. vs Nocardia spp. was suspected.
Rx was escalated to *Meropenem.*

Modified Kinyoun Acid fast staining was performed which stained the organism red confirming it to be NOCARDIA.



Multiple imagings including MRI Brain and CT chest/abd/pelvis were done to rule out other systemic involvement which were negative.

Meanwhile, *Culture on chocolate agar medium grew filamentous white colonies, characteristic of Nocardia.*



Day 4 At the end of 72 hrs, repeat blood cultures were obtained which remained negative indicating response to chosen therapy.

Patient was discharged safe to home on Meropenem to be continued for minimum of 3 months.

3 weeks Thigh aspirate cultures showed evidence of gram positive/Acid fast rods, confirming the site of microbial seeding.

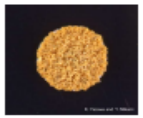
Further species testing revealed the organism to be ***Nocardia nova*** *sensitive to* ***Meropenem***.

Follow up

Follow up visits confirmed resolution of disease.

Discussion:

- Nocardiosis was primarily an opportunistic infection occurring in immunocompromised individuals, the main source being inhalation resulting in pneumonia and secondly, by direct cutaneous inoculation from soil as in abscess.



Nocardia nova
(online image)

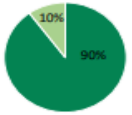
- Also 10% of cases have reported no valid predispositions.
- There are very few cases of bacteremia reported in literature, secondary to endovascular foreign objects, with evident poor outcome in terms of increased mortality despite specific treatment.
- Cultures require longer incubation period up to 2- 3 weeks.
- Nocardiosis involve 2 major pathogenic species

Nocardia asteroids group (90%)

1. *N. asteroides* complex (which contains multiple subspecies)
2. *N. farcinica* and
3. *Nocardia nova*

Others (10%)

1. *N. brasiliensis,*
2. *N. otitidiscaviarum,*
3. *N. transvalensis*



- **TREATMENT:**

Guidelines recommend *Trimethoprim- Sulfamethoxazole as the first line drug of choice* and other agents include amikacin, imipenem, meropenem and linezolid for a minimum of

i. 3-6 months in immunocompetent and
ii. 6-12 months in immunocompromised individuals.

In this case,

- The only possible explanation for the source could be infection through an unattended trivial penetration injury to abscess site.
- In spite of active Nocardia in blood stream, patient remained clinically stable throughout, with no organ involvement which depicts the rarity of this presentation.
- This could be explained by Daptomycin ongoing therapy in this patient as few studies have showed that the drug might have invitro activity against Nocardia but with very high Minimum Inhibitory Concentration (MIC).

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Submission Category: Clinical Vignette

Title: Don't Cough Too Hard You Might Herniate a Lung

Learning Objectives:

1. Diagnose lung herniation on examination findings
2. Understand the disrupted physiology of lung herniation.
3. Recognize lung hernia as a differential diagnosis for atypical chest pain.
4. Identify the causation for abdominal ecchymosis in the setting of lung herniation

Case:

A 77-year-old gentleman with COPD, nonobstructive coronary artery disease, and 50 pack-years smoking presents with right-sided chest pain. Patient had productive cough for three weeks. Primary care physician prescribed glucocorticoids, which improved the presumed COPD exacerbation. Patient noticed a constant burning pain one week prior to admission with an associated tearing sensation after productive coughing. A progressively worsening ecchymosis appeared on his abdomen. Patient presented to hospital when the pain became more severe. The patient had a paroxysmal breathing pattern noted with flail chest findings on this right lateral posterior chest wall. A mass was noted to herniate with expiration. Chest x-ray noted a lucency extending lateral to right lower ribs. CT Chest noted a posterior lung hernia with associated tenth rib fracture. Cardiothoracic surgery recommended conservative measures until the patient had increased dyspnea and hypoxia. Urgent thoracotomy with mesh placement for chest wall repair and diaphragm repair was performed for acquired spontaneous lung hernia.

Discussion:

Spontaneous lung herniation, consisting of lung parenchyma traversing beyond the confines of the chest wall, is a relatively uncommon diagnosis. Case reports document chest wall hernias occurring after surgical intervention or trauma, and less commonly from violent coughing episodes. Violent coughing, as well as sneezing or heavy lifting, induces a sudden elevation in transthoracic pressure causing rib or cartilage fracture, creating a defect for intercostal herniation. Associated risk factors predisposing patients to lung hernias include hyperinflation from COPD and presumed impaired healing and tissue integrity from oral steroid use, obesity, and diabetes mellitus. Both history and physical examination can reveal the diagnosis that is confirmed with chest imaging. The patient will often present with pain and a paradoxical breathing pattern with localized chest wall retraction during inspiration and herniation of lung parenchyma with expiration.

The patient's normal physiology was disturbed. During inspiration, the negative pleural pressure caused the chest wall defect and hernia sac to move inwards resulting in hernia reduction. With expiration, the hernia moved outwards and reappeared due to a more positive pleural pressure.

Indications for operative repair of lung hernias are similar to other anatomic hernias: strangulation, incarceration, or increasing symptoms. This patient's hernia orifice was large at 5.3 cm, which is why the patient was first observed and treated with conservative measures. Patient developed increased pain and dyspnea later and was taken for operative management. With spontaneous intercostal lung herniation, the intercostal musculature integrity can be disrupted with the rib fracture leading to severe muscle strain manifesting as localized pain and abdominal ecchymosis as seen in this patient.

Consideration of lung herniation as the presentation of atypical chest pain should be given especially in patients with comorbidities or risk factors such as this patient: significant tobacco use disorder, COPD, and prolonged coughing presenting with localized chest pain and ecchymosis.

Don't Cough Too Hard, You Might Herniate a Lung!

THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

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Learning Objectives

- Diagnose lung herniation on physical examination.
- Understand the disrupted physiology with lung herniation.
- Recognize lung hernia as a differential diagnosis for atypical chest pain.
- Identify the causation for abdominal ecchymosis in lung herniation.

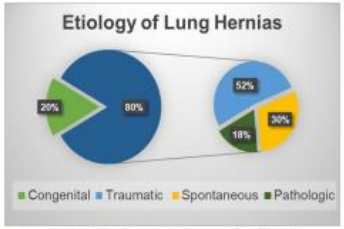


Figure 1. Etiology of Lung Hernias by percentage of representation. Spontaneous Lung herniation comprises 24% of all lung hernias.

Spontaneous Lung Herniation

- Risk Factors:
  - COPD hyperinflation
  - impaired healing & tissue integrity
    - 1.) oral steroid use
    - 2.) obesity
    - 3.) diabetes mellitus

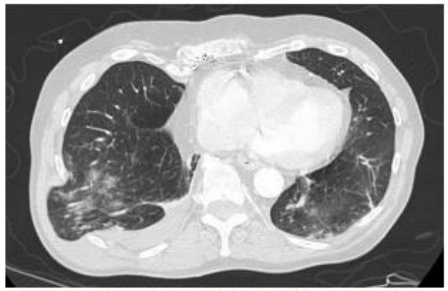
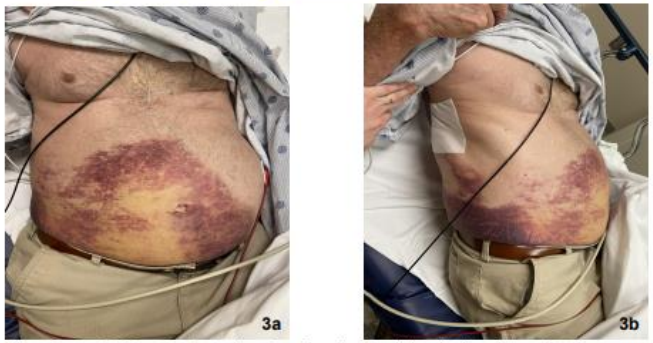


Figure 2. Transverse cut of CT Chest (Lung Window) noting a 5.3 cm posterior lung hernia.

CC: Right Chest Pain

- 77 year-old male with history of COPD, CAD, and 50 pack-year smoking history
- OP COPD Exacerbation treatment – Medrol dose pack
- Constant burning pain + tearing sensation after productive cough
- ***Ecchymosis*** and ***Paradoxical breathing*** pattern

Violent cough → **Sudden ↑ transthoracic pressure** → **Rib fracture** → **Intercostal muscle tear** → ***Ecchymosis***



Figures 3a. & 3b. – **3a.** Anterior view of patient's abdominal ecchymosis. **3b.** Anterolateral view of abdominal ecchymosis and chest wall defect on inspiration.

Physiology

- Chest wall defect → ***paradoxical breathing***
  - Inspiration = ▬ pleural pressure causes chest wall defect to sink into thoracic cavity.
  - Expiration = More ✚ pleural pressure causing hernia to bulge externally.

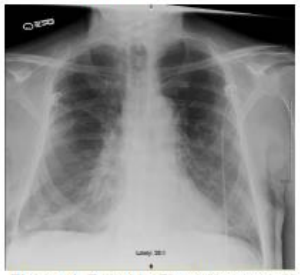


Figure 4. Portable Chest Xray notable for lucency lateral to the right rib cage.

Hospital Course and Follow Up

- Conservative management
- Acute dyspnea + hypoxia → urgent thoracotomy
- Mechanical ventilation
- Discharged home safely

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Title: "*Enterococcal* Bacteremia Is More Common Than You Think"

Authors: Joseph Shaw (MS4), Ainy Aziz, D.O., M.P.H. (Internal Medicine PGY-1), and Ali Hassoun, MD (Infectious Disease)

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Submission Category: Clinical Vignette

Learning Objectives

The genus *Enterococcus* consists of at least 38 gram-positive facultative anaerobes that characteristically thrive under hostile conditions. They conjugate with other bacteria and incorporate new genetic information with notable ease, contributing heavily to their resistance as well as their diverse pathogenicity. In humans, *Enterococcus* colonizes many tissues including the gut, skin, oropharynx, and vaginal mucosa. Infection can present as UTI, bacteremia, sepsis, or meningitis, the vast majority (~90%) are caused by *E. faecalis*. In this vignette, we describe three patients with vancomycin-susceptible *E. faecalis* bacteremia, as well as further discuss key points on *Enterococcus* infection.

Case Presentation

The first patient is a 68 year old male with a history of COPD, bipolar disorder, and CKD who presented with complaints of dyspnea, chest pain, and dysphagia for 1 day. He was afebrile, tachycardic, and tachypneic without leukocytosis. He required up to 12 L supplemental oxygen. His blood cultures were positive for *E. faecalis,* and TEE was negative for evidence of endocarditis. He received daptomycin for bacteremia, but his respiratory status continued to markedly decline and treatment was discontinued and patient was transferred to hospice.

Our next patient is a 76 year old male with a history of end-stage CHF (LVEF ~20%), A-fib, and COPD who presented increased shortness of breath and sudden 5-pound weight gain. He had mild leukocytosis and normal vitals, requiring supplemental O2. *E. faecalis* was found on blood culture that was treated with vancomycin. Cardiology was consulted, and TEE was negative for endocarditis. His bacteremia resolved, but worsening end-stage CHF led to the recommendation of inpatient hospice upon discharge.

The final patient is an 87 year old female who had 1 day of generalized weakness, rigors, body aches, and fever of 104˚C in the setting of chronic cough and recent treatment of bronchitis with azithromycin. She has history of CAD with stenting three months prior. Blood cultures positive for *E. faecalis* and treated with vancomycin. TEE was negative for endocarditis. Her condition improved, and was discharged with 6-week course of vancomycin.

Discussion

Enterococcus enters the bloodstream via the urinary tract, GI tract, wounds, and IV catheters. Confirmed enterococcemia has an associated mortality of 15-35%. Approximately 30% of community-acquired *E. faecalis* bacteremia is associated with endocarditis, while in nosocomial *E. faecalis* infection (~10% of all nosocomial bacteremia) it is rare. Antimicrobial treatment is prompted by ≥2 positive blood cultures, one positive blood culture in the presence of clinical sepsis, or blood culture in addition to positive culture from another site. Choice of antibiotic is based largely on sensitivities, but in serious illness or suspected endocarditis, combination therapy with ampicillin and ceftriaxone is recommended. In less worrisome cases, monotherapy with ampicillin, vancomycin, or daptomycin (if vancomycin-resistant) is acceptable.

Enterococcal Bacteremia Is More Common Than You Think

THE UNIVERSITY OF ALABAMA AT BIRMINGHAM
Joseph Shaw, Qurrat-ul-ain Aziz, D.O., M.P.H., and Ali Hassoun, M.D.

Learning Objectives:

- The genus *Enterococcus* consists of at least 38 gram-positive facultative anaerobes that thrive under hostile conditions.
- In this case series, we describe three patients with vancomycin-susceptible *E. faecalis* bacteremia, as well as further discuss key points on *Enterococcus* bacteremia.

Background:

- In humans, *Enterococcus* colonizes the gut, skin, oropharynx, and vaginal mucosa.
- Enterococcus can enter the bloodstream via the urinary tract, GI tract, wounds, and IV catheters.
- Infection can present as UTI, bacteremia/sepsis, endocarditis, or meningitis.
- The vast majority (~90%) of infections are caused by *E. faecalis*.

Case Presentation:

Patient #1

- 68 year old male with a history of COPD, bipolar disorder, and CKD who presented with complaints of dyspnea, chest pain, and dysphagia for 1 day.
- Vitals: Temp 36.9˚C, HR 103, RR 21 requiring up to 12 L supplemental oxygen.
- Physical Exam: Appeared in NAD, with diffusely decreased breath sounds on otherwise unremarkable exam.
- Labs: His blood cultures were positive for *E. faecalis,* and TEE was negative for evidence of endocarditis.
- Imaging: Multiple chest X-rays done during hospitalization revealed a scant, stable right-sided pleural effusion and worsening interstitial opacities bilaterally.
- **Hospital Course:** He received daptomycin for bacteremia, but his respiratory status continued to markedly decline. Treatment was discontinued, and the patient was transferred to hospice.

Patient #2:

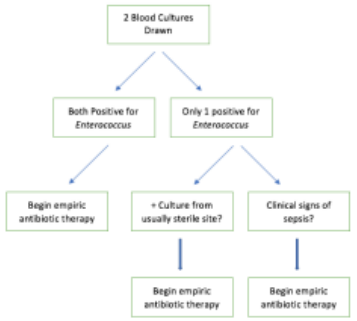
- 76 year old male with a history of end-stage CHF (LVEF ~20%), A-fib, and COPD who presented increased shortness of breath and sudden 5-pound weight gain.
- Vitals: WNL, requiring 2-3L supplemental O2.
- Physical Exam: Slightly increased work of breathing, but otherwise unremarkable.
- Labs: He had mild leukocytosis (WBC 16 with PMN predominance). *E. faecalis* was found on blood culture.
- **Hospital Course:** He was treated with vancomycin. Cardiology was consulted, and TEE was negative for endocarditis. His bacteremia resolved, but worsening end-stage CHF led to the recommendation of inpatient hospice upon discharge.

Patient #3:

- 87 year old female who had 1 day of generalized weakness, rigors, body aches, and fever in the setting of a chronic cough and recent treatment of bronchitis with azithromycin. She had a history of CAD with stenting three months prior.
- Vitals: Temp of 40˚C, with baseline hypertension. Otherwise WNL.
- Physical Exam: In ED patient was altered, but was A/Ox4 soon after empiric antibiotics were given.
- Labs: Blood cultures were positive for *E. faecalis,* TEE was negative for endocarditis. **Hospital Course:** Treatment was started with vancomycin. Her bacteremia resolved and she was afebrile, and she was discharged home with a 6-week course of vancomycin.

Discussion:

- Confirmed enterococcemia has an associated mortality of 15-35%.
- Approximately 30% of community-acquired *E. faecalis* bacteremia is associated with endocarditis, while in nosocomial *E. faecalis* infection (~10% of all nosocomial bacteremia) it is rare.
- Choice of antibiotic is based largely on sensitivities.
- Combination therapy (with ampicillin and gentamycin) is used in the presence of endocarditis or critical illness.
- Otherwise, monotherapy is recommended.



- **Algorithm for deciding to initiate empiric antibiotic therapy**

Monotherapy Agents	Dose	Notes
Ampicillin	1-2 g IV q4-6h	-Best choice if susceptible
Penicillin G	18-30 million U IV q24h	-Alternative to ampicillin
Vancomycin	15 mg/kg IV q12h	-Good choice as empiric or if ampicillin-resistant
Daptomycin	8-10 mg/kg IV q24h	-Alternative to vancomycin; less nephrotoxicity -Weekly CPK checks (risk for myopathy)

- **Monotherapy antibiotic treatment for *E. faecalis* bacteremia**

Fusobacterium necrophorum causing empyema

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Submission category: Clinical Vignette

Learning objective: Importance of adding empiric anaerobic coverage in complicated parapneumonic effusion and empyema.

Case presentation: A 48-year old Caucasian male presented to the ER with a chief complaint of shortness of breath and chest pain for 4 days. Past medical history includes intracranial hemorrhage and hypertension. Patient complained of cough with yellowish sputum production associated with chest pain. Patient denied smoking, recent travel and recreational drug use. Patient was septic on admission. On physical examination, patient was hypotensive with blood pressure of 94/67 mmHg, tachycardic, tachypneic with low grade fever of 100.3 F, decreased breath sounds on the left side. Chest x-ray showed extensive opacification of the left lung. CT chest w/o contrast showed large empyema in the left thorax. Lab results showed Leukocytosis (17,000) elevated creatinine (3.2). Pleural fluid analysis was suggestive of exudate. Patient was started on Zyvox and Rocephin empirically. Infectious diseases and pulmonology were consulted. Chest tube was placed and Intrapleural tPA was started. Patient received three rounds of tPA. HIV test was negative. IgG and IgA levels are normal. IgM level was low (34). Blood cultures showed no growth. Urine legionella and pneumococcal antigen was negative. Mycoplasma IgM antibody is negative. Pleural fluid culture was positive for 2+ Fusobacterium necrophorum. Antibiotic regimen was switched to Zosyn on day 4. Repeat CT chest w/o contrast showed improvement in fluid collection. Chest tube was removed on day 8. Patient was discharged to rehab facility on Rocephin to finish the course of 6 weeks of antibiotics.

Discussion: Empyema and parapneumonic effusions are common complications of pneumonia. Most common causes are Streptococcus and Staphylococcus aureus, followed by Anaerobes found in the oropharynx which includes Fusobacterium species, Prevotella species and Bacteroides species. In our patient, Fusobacterium necrophorum was the causative agent. Fusobacterium necrophorum is a gram-negative non- spore forming anaerobic bacillus. It causes invasive systemic infections like Lemierre's syndrome, endocarditis in adults and Cancrum oris in immunocompromised children. Conditions that would increase the risk includes poor dental hygiene, diabetes mellitus, malignancy, oropharyngeal infections and immunosuppression. Parapneumonic effusion and empyema are very uncommon in the absence of Leimierre's syndrome. A review of literature showed only 3 cases of empyema caused by Fusobacterium necrophorum. In two of these cases, patients were diagnosed with Leimierre's

syndrome, and in the third case there was no evidence of Leimmere's syndrome. Here, we are presenting a rare case of empyema caused by Fusobacterium necrophorum, with low IgM level and no evidence of Leimmere's syndrome. Treatment for empyema should be initiated immediately with antibiotics and drainage to reduce the complications. Empiric antibiotics in empyema should target against anaerobes, streptococcus if community acquired and MRSA in hospital acquired infections. Fusobacterium produces Beta- lactamase enzyme. Hence, beta- lactamase resistant antibiotic should be used, like ampicillin-sulbactam, piperacillin- tazobactam or monotherapy with carbapenems. Antibiotics are recommended for 4 to 6 weeks. In case of treatment failure in empyema, need for additional drainage options should be considered, which include intrapleural tPA or video assisted thoracic surgery.

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Fusobacterium necrophorum causing empyema

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Learning objective

- Importance of adding empiric anaerobic coverage in complicated parapneumonic effusion and empyema.

Case presentation:

- A 48-year old Caucasian male presented to the ER with a chief complaint of shortness of breath and chest pain for 4 days.
- Past medical history includes intracranial hemorrhage and hypertension. Patient complained of cough with yellowish sputum production associated with chest pain. Patient denied smoking, recent travel and recreational drug use.
- On physical examination, patient was hypotensive with blood pressure of 94/67 mmHg, tachycardic, tachypneic with low grade fever of 100.3 F, decreased breath sounds on the left side.

Lab results

- Leukocytosis (17,000) elevated creatinine (3.2).
- Pleural fluid analysis was suggestive of exudate.
- HIV test was negative.
- IgG and IgA levels are normal.
- IgM level was low (34).

Imaging

- Chest x-ray showed extensive opacification of the left lung.
- CT chest w/o contrast showed large empyema in the left thorax.

Culture results

- Blood cultures showed no growth.
- Pleural fluid culture was positive for 2+ Fusobacterium necrophorum.

Treatment

- Patient was started on Zyvox and Rocephin empirically before Pleural fluid culture results.
- Infectious diseases and pulmonology were consulted.
- Chest tube was placed and Intrapleural tPA was started. Patient received three rounds of tPA.
- Antibiotic regimen was switched to Zosyn on day 4.
- Repeat CT chest w/o contrast showed improvement in fluid collection.
- Chest tube was removed on day 8.
- Patient was discharged to rehab facility on Rocephin to finish the course of 6 weeks of antibiotics

Imaging

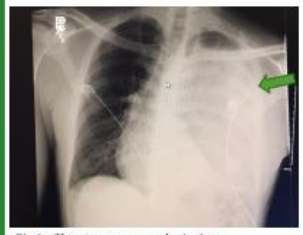

Fig1: Chest x-ray on admission

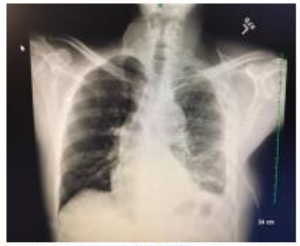

Fig2: Chest x ray on day 8 after chest tube placement and tPA

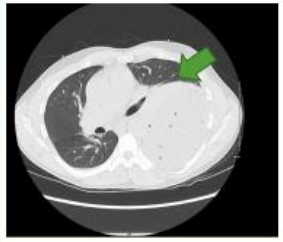

Fig3: CT chest on admission

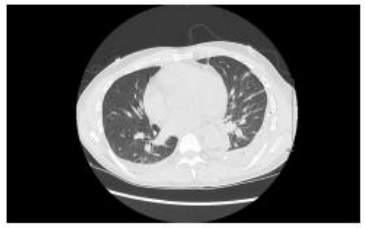

Fig4: CT chest on day 8 after chest tube placement and tPA

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Discussion:

- Empyema and parapneumonic effusions are common complications of pneumonia.
- Most common causes are Streptococcus and Staphylococcus aureus, followed by Anaerobes found in the oropharynx which includes Fusobacterium species, Prevotella species and Bacteroides species.
- In our patient, Fusobacterium necrophorum was the causative agent.
- Fusobacterium necrophorum is a gram-negative non- spore forming anaerobic bacillus.
- It causes invasive systemic infections like Lemierre's syndrome, endocarditis in adults and Cancrum oris in immunocompromised children.
- Conditions that would increase the risk includes poor dental hygiene, diabetes mellitus, malignancy, oropharyngeal infections and immunosuppression. Parapneumonic effusion and empyema are very uncommon in the absence of Leimierre's syndrome. A review of literature showed only 3 cases of empyema caused by Fusobacterium necrophorum.
- Here, we are presenting a rare case of empyema caused by Fusobacterium necrophorum, with low IgM level and no evidence of Leimmere's syndrome. Empiric antibiotics in empyema should target against anaerobes, streptococcus if community acquired and MRSA in hospital acquired infections.
- Fusobacterium produces Beta- lactamase enzyme. Hence, beta- lactamase resistant antibiotic should be used, like ampicillin-sulbactam, piperacillin- tazobactam or monotherapy with carbapenems.

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Have you ever been told you have a murmur?

Mariam Riad MD, Erin D. Britt DO, Roger Smalligan MD, MPH, John Fanning MD, FACP.

University of Alabama in Birmingham Huntsville Regional Campus, Department of Internal Medicine.

Learning Objective:

1. Recognizing the signs and symptoms of acute aortic regurgitation.
2. Highlight the identifiable risk factors that can alert physicians for developing high suspicion for Aortic Dissection (AD).

Case presentation

An 81-year-old man presented to the clinic with a chief complaint of 5 days of persistent cough with clear sputum and dyspnea. His past medical history includes type 2 diabetes, placement of ICD/pacemaker for ischemic cardiomyopathy four years ago with recent ejection fraction measured at 35-40%, history of coronary artery disease, status post coronary artery bypass graft surgery three months before presentation. On presentation, his dyspnea was exacerbated by lying supine and with exertion. His vital signs were BP 138/72 mm Hg, HR 80 beats/min, temperature 36 C, room air O saturation 96%. His physical exam revealed the patient had mild resting dyspnea. Neck had no JVD or bruits. Carotid pulses were 3+ and symmetric. Cardiac exam showed normal S1 and S2 with grade 2/6 diastolic murmur, no rubs, or gallops. Lungs had mildly diminished sounds on the right. Chest x-ray was read as mild right basilar opacity, possible atelectasis, or infiltrate. With his history of effusions and cardiomyopathy, he was sent home and asked to take an increased dose of furosemide. He returned to the clinic the following day, with worsening dyspnea on exertion and severe orthopnea. He also complained of chills but no subjective or objective fever. He was admitted to our inpatient service and his ECG showed a paced rhythm. Because the etiology of his dyspnea was unclear, he underwent CT imaging of his chest that revealed Type A dissection of the ascending thoracic aorta with an intimal flap. The dissection did extend down to the aortic root. An echocardiogram showed moderate aortic insufficiency. We consulted cardiovascular surgery, who felt the patient needed to be transferred to a tertiary care center for further evaluation and eventually discharged with no surgical intervention because of the high risk of the surgery. A one month follow up evaluation was scheduled at the tertiary care facility.

Discussion

AD is a life-threatening disease. Its annual incidence is 5 to 30 in 1 million and has a high mortality rate, up to 50%. Type A carries a worse prognosis and usually treated surgically and can be associated with aortic regurgitation. This patient had an echocardiogram done two years prior that showed no aortic valve disease. The cardiologist at that time found no murmur. Reviewing the previous records or asking the patient “Have you ever been told you have a murmur?” could have identified this as murmur as new. This finding along with significant sudden onset of severe orthopnea could have alerted the clinic physicians that this was a possible dissection.

This case was an atypical, significantly benign presentation for a type A ascending thoracic aortic dissection. There were no symptoms of chest pain, typical for AD. Our patient has more than one identifiable risk factor according to the International Registry of Acute Aortic Dissection (IRAD), including

age, gender, ethnicity, and recent previous cardiac surgery. IRAD also recognizes four distinct time periods for AD: hyperacute (symptom onset to 24 hours), acute (2–7 days), subacute (8–30 days), and chronic (>30 days). The overall survival rate was progressively lower through the four time periods.

Have You Ever Been Told You Have a Murmur?

THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

Mariam Riad MD, PG1, Erin Britt DO, PG2, Roger D. Smalligan MD, MPH, John Fanning MD, FACP
UAB-Huntsville Regional Medical Campus, Department of Internal Medicine.

Objectives

1. Recognize the signs and symptoms of acute aortic regurgitation (AR).
2. Highlight the identifiable risk factors for developing high suspicion for aortic dissection (AD).

Case

- 81 y/o M presenting for 5 days of persistent cough with sputum and dyspnea.
- Dyspnea worse with lying supine and exertion.

PMH	PSH
▪ NIDDM ▪ Ischemic CM EF 35-40% ▪ CAD	▪ ICD 4 yrs ago ▪ CABG 3 mo ago

- Post op. pleural & pericardial effusions s/p operative drainage.
- Vital signs: Bp 138/72 mm Hg, HR 80 bpm, Temp 36 C, O_2 96% on room air.
- Physical exam:
  - •Mild resting dyspnea
  - •No JVD or bruit
  - •Normal S1, S2
  - •Grade 2/6 diastolic murmur
  - •Diminished breath sounds on right lower lung field

Diagnostic tests

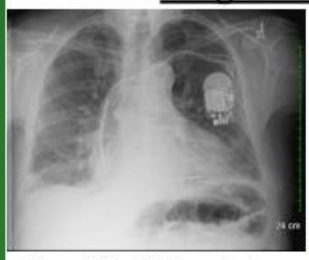

Figure 1. Chest X-Ray prior to admission showing pleural effusion and cardiomegaly

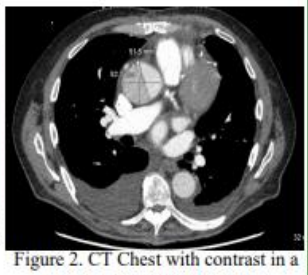

Figure 2. CT Chest with contrast in a patient with aortic dissection

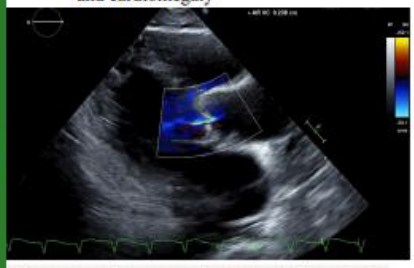

Figure 3. 2D Transthoracic echocardiogram with doppler showing aortic regurgitation

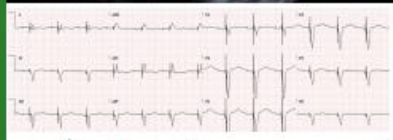

Figure 4. EKG with AV paced rhythm

7.82 x10^3/mcL	9.8	603 x10^3/mcL	141	103	27
	34.1%		5.0	29	1.0

MVC 73.5 fL, RDW 18.6%, Fe 6%, BNP 12,656 pg/ml (Baseline 5,854 pg/ml)

Values are in mg/dl unless mentioned otherwise

Outcome

- Patient was discharged from clinic after one dose of Lasix to return next day with persistent symptoms.
- He was then admitted to our inpatient service and Cardiothoracic surgery consulted and patient was transferred to tertiary care center.
- No surgical intervention pursued due to extremely high perioperative risk. Close follow up on blood pressure and recheck CT in 6 months.

Discussion

1. There were no symptoms of chest pain.
2. This case was an atypical benign presentation of Type A chronic aortic dissection and new onset aortic regurgitation.
3. Reviewing the previous records or asking the patient "Have you ever been told you have a murmur?" could have identified this murmur as new.
4. There were four identifiable risk factors according to the international registry of aortic dissection (IRAD) including age, gender, ethnicity, and recent previous cardiac surgery.
5. These findings along with sudden onset of orthopnea could have alerted the physician for possible aortic dissection.
6. Identifying possible risk factors and maintaining a high suspicion of dissection is of utmost importance.
7. It can potentially provide earlier diagnosis and more timely management of this frequently fatal disease.

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Herpes Esophagitis Can Resemble Candidiasis

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Submission category: Clinical Vignette

Learning objective:

Presentation and evaluation of Patients with Herpes Esophagitis

Case Presentation: A 21-year old Caucasian male presented with a chief complaint of fever and severe odynophagia for 2 days. Past medical history was unremarkable. Patient could not swallow liquids or solids. Patient had intermittent fever. No relief with Tylenol. Patient denies any nausea, skin rash, weight loss and night sweats. Patient denies tobacco, alcohol or recreational drug use. Physical examination including oral exam was unremarkable. CTA chest was normal. CBC, CMP are within normal limits. HIV test was non-reactive. Throat culture was negative. Patient upper GI endoscopy showed esophageal plaques suspicious for Candidiasis. Patient was empirically started on fluconazole for suspected esophageal candidiasis. Absolute CD4 count was low (186) and CD3 count was low (376). IgG and IgM levels normal. IgA level was low (68). Esophageal pathology showed acute ulcerative esophagitis consistent with herpes esophagitis, HSV immunohistochemical stain is positive, and Gomori methenamine silver stain is negative for fungal elements. Patient was started on IV Acyclovir and Micafungin as the suspicion for esophageal candidiasis was still high. Patient symptoms improved, switched to PO Valtrex and Diflucan to finish the course for 2 weeks.

Discussion:

Herpes esophagitis more likely occurs in immunocompromised patients and is less common in immunocompetent patients. It is most likely caused by HSV1. Here, we are presenting a case of Herpes esophagitis with a low absolute CD4 count and negative HIV test. Idiopathic CD4 lymphocytopenia (ICL) is a rare condition. Patients with ICL are more prone to have opportunistic infections, with the most common being cryptococcus followed by mycobacterial and candida infections. The prevalence of herpes simplex, Pneumocystis and cytomegalovirus is very low in patients with ICL.

Clinical features of herpes esophagitis include dysphagia, odynophagia, retrosternal discomfort, heart burn, nausea, vomiting and weight loss. Diagnosis is confirmed by endoscopy, biopsy should be taken from the edge of the ulcer. Biopsy usually shows Cowdry type A inclusion bodies which are very typical for HSV infection. Cell culture and virus isolation are the gold standard for diagnosis. The duration of treatment in immunocompromised patients is 2 to 3 weeks, and a shorter duration of 2 weeks is recommended in immunocompetent patients. Oral Acyclovir is used as the first line as it has more effective anti HSV activity and it is less expensive. IV acyclovir can be used in patients who has severe odynophagia. Other antiviral agents which can be used are famciclovir and valacyclovir. Patients who do not improve after 5 to 7 days of treatment, drug resistant virus should be suspected. Foscarnet is the drug of choice in that scenario.

Herpes Esophagitis Can Resemble Candidiasis

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1. Department of Internal Medicine, UAB-Huntsville, Huntsville, AL, United States 2. Alabama Infectious Diseases Center, Huntsville, AL, United states.

Learning Objective

- Presentation and evaluation of Patients with Herpes Esophagitis

Case Presentation:

- 21-year old Caucasian male presented with a chief complaint of fever and severe odynophagia for 2 days.
- Past medical history was unremarkable.
- Patient could not swallow liquids or solids.
- Patient had intermittent fever. No relief with Tylenol.
- Patient denies any nausea, skin rash, weight loss and night sweats. Patient denies tobacco, alcohol or recreational drug use.
- Physical examination including oral exam was unremarkable.

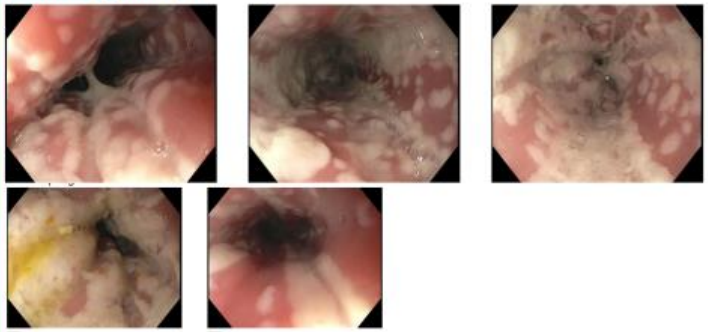
Labs:

- CBC, CMP are within normal limits.
- HIV test was non-reactive.
- Absolute CD4 count was low (186) and CD3 count was low (376).
- IgG and IgM levels normal. IgA level was low (68).

Diagnostic tests:

- CTA chest was normal.
- Upper GI endoscopy showed esophageal plaques suspicious for Candidiasis.

Endoscopy Images



Pathology

- Esophageal pathology showed acute ulcerative esophagitis consistent with herpes esophagitis.
- HSV immunohistochemical stain is positive, and Gomori methenamine silver stain is negative for fungal elements.

Treatment

- Patient was empirically started on fluconazole for suspected esophageal candidiasis before the pathology report.
- Patient was started on IV Acyclovir and Micafungin, as the suspicion for esophageal candidiasis was still high.
- Patient's symptoms improved, switched to PO Valtrex and Diflucan to finish the course for 2 weeks.

Discussion:

- Herpes esophagitis more likely occurs in immunocompromised patients and is less common in immunocompetent patients. It is most likely caused by HSV1.
- Here, we are presenting a case of Herpes esophagitis with a low absolute CD4 count and negative HIV test. Idiopathic CD4 lymphocytopenia (ICL) is a rare condition.
- Patients with ICL are more prone to have opportunistic infections, with the most common being cryptococcus followed by mycobacterial and candida infections.
- The prevalence of herpes simplex, Pneumocystis and cytomegalovirus is very low in patients with ICL.

- Clinical features of herpes esophagitis include dysphagia, odynophagia, retrosternal discomfort, heart burn, nausea, vomiting and weight loss.
- Diagnosis is confirmed by endoscopy, biopsy should be taken from the edge of the ulcer.
- Biopsy usually shows Cowdry type A inclusion bodies which are very typical for HSV infection. Cell culture and virus isolation are the gold standard for diagnosis.
- The duration of treatment in immunocompromised patients is 2 to 3 weeks, and a shorter duration of 2 weeks is recommended in immunocompetent patients.
- Oral Acyclovir is used as the first line as it has more effective anti HSV activity and it is less expensive. IV acyclovir can be used in patients who has severe odynophagia.
- Other antiviral agents which can be used are famciclovir and valacyclovir.
- Patients who do not improve after 5 to 7 days of treatment, drug resistant virus should be suspected. Foscarnet is the drug of choice in that scenario.

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1.Zaidi, S. A., & Cervia, J. S. (2002). Diagnosis and Management of Infectious Esophagitis Associated with Human Immunodeficiency Virus Infection. Journal of the International Association of Physicians in AIDS Care, 1(2), 53–62.

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Hidden in plain sight - Tuberculous dissemination involving Aorta

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LEARNING OBJECTIVES:

1. Recognize the possibility of tuberculosis dissemination to major vessels like Aorta.
2. Know the steps in management of Disseminated tuberculosis

CASE PRESENTATION:

45-year-old Asian female with history of sputum positive cavitary Tuberculosis (TB) on active Antitubercular therapy (ATT) presented with headaches and blurry vision. On examination, the patient appeared cachectic with a BMI of 14.5. Neurological examination revealed subtle deficits in form of right abducens palsy and mild left upper extremity mono-paresis. Systemic examination unremarkable. Imaging confirmed dissemination to brain as multiple intracranial tuberculomas. Labs were unremarkable. Quantitative drug assay was done to check therapeutic ATT levels which revealed subnormal C_{max} of Isoniazid and Rifampicin. Incidentally, CXR showed military mottling with increased opacity in the medial left upper region, concerning for mass vs consolidation. Further work up revealed a new Saccular pseudoaneurysm (33x31mm) arising from the Aortic arch, just distal to the origin of Left Subclavian artery more likely to be Mycotic, with enlarged Aortopulmonary and bilateral hilar lymph nodes. Specimen sampling is unreliable as patient already on active treatment even if it is subtherapeutic, hence false negative results are expected. Further Whole Body 18F FDG PET-CT imaging revealed increased FDG uptake in brain tuberculomas and saccular pseudoaneurysm in Arch of Aorta confirming ongoing disease activity. Treatment with Weight based ATT regimen- Isoniazid, Rifampicin, Pyrazinamide and Ethambutol (HRZE) initiated with dose adjusted to therapeutic serum levels. Two months later being on ATT, follow up imaging showed interval reduction in the size of previously noted pulmonary lesions but a significant increase in the size of saccular pseudoaneurysm was noted. Surgical option was postponed as patient remained asymptomatic of aneurysm with neurological symptoms having resolved. Meanwhile, the patient developed sudden onset massive hemoptysis indicating impending aneurysmal rupture which necessitated Thoracic Endovascular Aortic Repair (TEVAR). 6 months from Surgery, follow up Chest imaging confirmed resolution of disease.

DISCUSSION:

Tuberculosis is primarily airborne in origin but disseminates within, via blood or contiguous connections. Among blood vessels, the most commonly involved sites are noted to be abdominal aorta and cerebral vessels. Aortic involvement either as aortitis or aneurysm is very rarely reported. “Specimen negative Tuberculosis” is a different clinical entity just as we described here, more common in patients with relapse or re-infection who are non-compliant with primary ATT regimen and those treated inadequately. In such cases, diagnostic and treatment options are based on clinical expertise along with consideration of risk factors/regional prevalence. Imaging with characteristic features like multiple ring enhancing lesions, military pattern together plays a vital role in diagnosing dissemination in such cases

as above. In this patient, history of Sputum positive tuberculosis and development of multisystem involvement within treatment phase despite negative microbiological evidence has made the clinical decision of dissemination obvious. Successful treatment in this patient in terms of good prognosis and preserved survival is mainly because of the combined multidisciplinary approach in management involving both medical and surgical aspects.

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Authors: Sabrina Matosz, MD; Noaman Ahmad, MD; Ali Hachem, MD; Farrah Ibrahim, MD

Department: UAB- Huntsville Internal Medicine, The Cancer Center of Huntsville

Clinical Vignettes

Title:

Histiocytic Sarcoma: A case report

Learning objectives:

Histiocytic Sarcoma (HS) is a rare and aggressive hematopoetic neoplasm. There have only been a few case reports of primary splenic HS. Herein, we describe a case of primary HS of the spleen. Although HS is very uncommon, clinicians and pathologists must consider it as a differential diagnosis.

Case presentation:

52-year-old woman presented to the Emergency Department complaining of one-week duration of exertional dyspnea with nausea. CT chest with contrast showed a segmental branch pulmonary embolus and a suspicious infiltrating mass in the pancreatic tail that warranted a CT abdomen and pelvis. This revealed splenomegaly of 17 x 11 x 19 cm, a splenic lesion of .17 x .18 x .14 cm, and an irregularly shaped mass in the tail of the pancreas that encased the splenic artery and vein and multiple enlarged retroperitoneal lymph nodes. These findings necessitated an endoscopic ultrasound and the cytology of the pancreatic tail mass showed malignant cells with abundant necrosis; however, extensive immunohistochemistry could not delineate the cell origin. Therefore, the patient went for an exploratory laparotomy with splenectomy, distal pancreatectomy, partial gastrectomy and omental mobilization. The impression thus far was pancreatic adenocarcinoma with metastatic disease. However, the specimen from the spleen was immunoreactive for CD68 and CD45. The overall feature with the positive staining for histiocytic marker CD68 was most compatible with HS. It was negative for markers to rule out T cell, B cell, and myeloid lineage. Patient had a difficult course with PET scan one month after surgery revealing extensive metastatic disease and multiple admissions to the hospital due to malnutrition and left parietal cortical stroke. Patient was eventually started on CHOP chemotherapy with repeat imaging showing no improvement and neoplastic fever which indicated a poor response. The patient has now completed four cycles of ICE chemotherapy with her last CT abdomen showing overall improvement.

Discussion:

HS is a rare hematologic malignancy of unknown etiology. WHO currently defines HS as a neoplastic proliferation with morphological and immunophenotypic features of mature tissue histiocytes. It is difficult to differentiate HS by radiological evaluation alone and must be confirmed by immunohistochemistry. The diagnosis of HS is difficult to achieve since histiocytic lesions have been

shown to share molecular genetic or cytogenic features with original leukemia or lymphoma. Interestingly, many reports show an association between HS and other hematologic malignancies including lymphocytic leukemia/lymphomas such as follicular lymphoma. Therefore, pan-cytokeratin immunohistochemistry is needed to exclude tumors of epithelial origin when diagnosing tumors showing cells with pleomorphic morphology. HS cells should be positive for one or more histiocytic markers including CD68, CD163, lysozyme but negative for CD1a, CD21, CD35, CD30, T cell, B cell, and myeloid lineage markers.

In conclusion, only a limited number of cases have been reported despite HS being an aggressive hematopoietic neoplasm. The recognition of this neoplasm and its clinicopathological features are still not clearly understood. However, it is important to recognize and distinguish this aggressive neoplasm and to increase the diagnostic accessibility for pathologists and to make an appropriate therapeutic choice for clinicians.

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Histiocytic Sarcoma: A case report

Sabrina Matosz, MD[1]; Noaman Ahmad, MD[1]; Ali Hachem, MD[2]; Farrah Ibrahim, MD[1]

Dept. of Internal Medicine, UAB Huntsville[1]; The Cancer Center of Huntsville, Alabama[2]

Learning Objective:

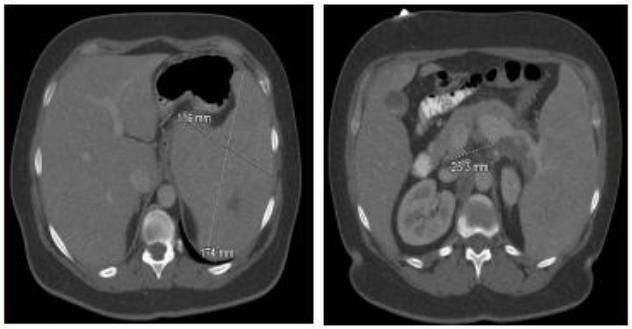
- Histiocytic Sarcoma (HS) is a rare and aggressive hematopoietic neoplasm. Median age of 52 with no apparent gender difference
- There have only been a few case reports of primary splenic HS

Herein, we describe a case of primary HS of the spleen. Although HS is very uncommon, clinicians and pathologists must consider it as a differential diagnosis

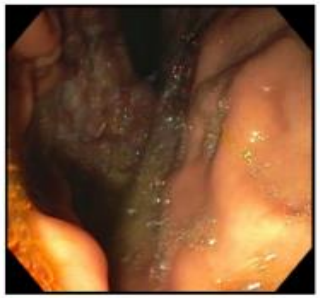
- The World Health Organization currently defines HS as a neoplastic proliferation with morphological and immunophenotypic features of mature tissue histiocytes
- It is difficult to differentiate HS by radiological evaluation alone and must be confirmed by immunohistochemistry

Case presentation:

52 year old woman presents to the ED due to one week duration of exertional dyspnea with nausea. CT chest w/ contrast was performed that showed segmental branch pulmonary embolism and suspicious infiltrating mass in pancreatic tail



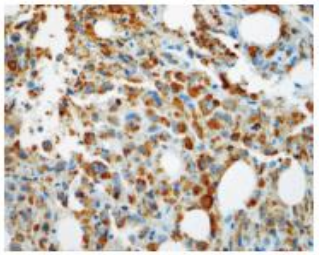
CT abdomen and pelvis: splenomegaly 17 x 11 x19 cm, splenic lesion of .17 x .18 x .14 cm and an irregularly shaped mass in the tail of the pancreas that encased the splenic artery and vein and multiple enlarged retroperitoneal lymph nodes



Endoscopic ultrasound: mass noted with irregular margins. Sonographic evidence suggesting invasion into celiac trunk and splenic artery. FNA for cytology was performed with cytology reporting malignant cells with abundant necrosis; however, extensive immunohistochemistry could not delineate the cell origin

The patient went for an exploratory laparotomy with splenectomy, distal pancreatectomy, partial gastrectomy and omental mobilization. The impression thus far was pancreatic adenocarcinoma with metastatic disease

The diagnosis of HS is difficult to achieve since histiocytic lesions have been shown to share molecular genetic or cytogenic features with original leukemia or lymphoma. Therefore, pan-cytokeratin immunohistochemistry is needed to exclude tumors of epithelial origin when diagnosing tumors showing cells with pleomorphic morphology



(Tumor cells expressing CD68)

HS cells are immunohistochemically positive for one or more histiocytic markers such as CD68, CD163, and lysozyme, but negative for CD1a, CD21, CD35, CD30, T cell, B cell, and myeloid lineage markers

Results:

- The overall feature with the positive staining for histiocytic marker CD68 was most compatible with HS. It was negative for markers to rule out T cell, B cell, and myeloid lineage
- The patient has now completed four cycles of ICE chemotherapy for her diagnosis of HS with her last CT abdomen showing overall improvement

Conclusion:

Only a limited number of cases have been reported despite HS being an aggressive hematopoietic neoplasm. The recognition of this neoplasm and its clinicopathological features are still not clearly understood. However, it is important to recognize and distinguish this aggressive neoplasm and to increase the diagnostic accessibility for pathologists and to make an appropriate therapeutic choice for clinicians.

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TITLE: Idiopathic Proliferative Fibrosing Mediastinitis Mimicking Malignancy

AUTHORS: C. Bennett Parker, Roger D. Smalligan

INSTITUTIONS: Internal Medicine, University of Alabama School of Medicine, Huntsville, AL, United States.

ABSTRACT BODY:

Learning Objective 1: Recognize the clinical features of fibrosing mediastinitis as a rare cause of a proliferative lung mass
Learning Objective 2: Recognize the importance of patient education in clinically ambiguous disease processes
Case: A 49 YO AAM with an ambiguous past medical history involving a solitary nodule in the lower lobe of the left lung with unknown etiology, progressive hilar adenopathy causing SVC syndrome, and multiple episodes of syncope presented to the ED with an episode of total loss of consciousness preceded by palpitations and lightheadedness after walking 20-30 feet. Notably, the patient has never used tobacco products or illicit drugs. Family medical history is significant for breast cancer in the patient's mother, lung cancer in his father, and multiple cancers in his extended family. On presentation, vital signs were stable, and orthostatic testing was negative. Diagnostic imaging, including head CT and chest X-ray, showed no acute abnormalities with identification of a patent superior vena cava with stent placement and hilar adenopathy; EKG showed sinus rhythm with occasional PVCs. The patient reported history of lymphoma and treatment with unknown chemotherapy. Chart review revealed extensive workup from multiple medical centers for a lung mass and hilar adenopathy causing SVC syndrome, leading to considerations of lymphoma with nondiagnostic biopsies; however, the lung pathology is now believed to be due to idiopathic proliferative fibrosing mediastinitis (IPFM). The patient was treated with 4 weeks of rituximab and showed improvement of the left lower lobe nodule from 2.6 x 1.6 cm to 2.2 x 1.5 cm. The patient's hospital course involved supportive care and telemetry, and the patient was given a Holter Monitor for the two days following discharge from the hospital to rule out cardiogenic source of syncope. He was educated on the diagnosis of IPFM and its distinction from malignant etiologies, and he was scheduled to receive serial CT scans every 6 months to monitor progression of the lung nodule and hilar adenopathy.

Discussion: Dedication to patient education in the setting of a clinically ambiguous disease process can prevent inaccuracies in a patient's medical history and lead to better care. Communication with the patient demonstrated a belief that his disease process was malignant and life-threatening; however, chart review revealed a consensus understanding among the patient's various physicians that the nodule and hilar adenopathy are due to IPFM. The pathophysiology of fibrosing mediastinitis is poorly understood but may involve fungal antigens leaking into the mediastinal space, resulting in inflammation and subsequent fibrosis. Our patient's nondiagnostic biopsies, FDG-avid lesion on PET scan, and negative immunohistochemical staining for lymphoma present a puzzling clinical picture. A trial of rituximab resulted in stability with slight improvement of the mediastinal lesion. The patient was pleased with the revelation of a likely benign disease process and will be monitored with serial CT scans.

Poster was self-printed for presentation and a PDF image was unavailable for this compendium.

Title: Infective Endocarditis as a complication of Hypertrophic Obstructive Cardiomyopathy.

Authors and Affiliation: E Roumaya, N Tangutur; UAB School of Medicine Family Medicine, North Alabama Hospitalist, Huntsville Hospital. Family Medicine Resident and Hospitalist.

Submission Category: Clinical vignette

Learning Objective: Describe a rare correlation between infective endocarditis and hypertrophic cardiomyopathy, and the need for antibiotic prophylaxis.

Introduction: Infective endocarditis has been seen as a complication of hypertrophic cardiomyopathy. With an incidence in approximately 3.8 per 1000 person-years, infective endocarditis in obstructive hypertrophic cardiomyopathy is rare, but has a high morbidly and mortality.

Case Presentation: A 44-year old Hispanic Male with history of alcohol abuse presented with increase in fevers, chills, and shortness of breath, which worsened over 3 months. During initial evaluation, echocardiogram revealed an ejection fraction of 60% with early aortic stenosis and mild insufficiency. He also had positive blood cultures for Strep Viridans. Subsequent transthoracic echocardiogram revealed a large mobile mitral valve vegetation measuring 19 x 10 mm with severe mitral regurgitation, small vegetation on the aortic valve, and obstructive hypertrophic cardiomyopathy. He was placed on gentamicin and ceftriaxone for antibiotic therapy. Underwent a mitral valve repair and aortic vale repair. From time of surgery, he went into complete heart block and received a permanent pacemaker on post-op day 5. He was started on Coumadin with was deemed stable for discharge home on post-op day 9 from valve repair and post-op day 4 from pacemaker placement. He completed 2 weeks of IV gentamicin during hospitalization and will be discharged with ceftriaxone to complete a total course of 4 weeks.

Discussion:

Hypertrophic cardiomyopathy is a genetic condition, which leads to asymmetric septal wall thickening. Which can lead to outflow obstruction and mitral valve abnormalities. Infective endocarditis is more frequent in obstructive hypertrophic cardiomyopathy, especially those who also have atrial dilation. There has been controversy in studies regarding the need for antibiotic prophylaxis in people with hypertrophic cardiomyopathy to prevent infective endocarditis. Multiple studies revealed those with an obstructive pattern and atrial dilation are more likely to develop infective endocarditis, and it is recommended that these people receive antibiotic prophylaxis. The most common valve to be affected is the septal aspect of the mitral valve. This is likely due to the turbulent blood flow and contact during systole between the mitral anterior leaflet and septum causing endocardial damage. Due to the mitral

valve being most commonly affected, surgical intervention with mitral valve replacement is commonly done in which resolution of outflow obstruction usually occurs.

Conclusion: As in our case, this patient presented with Streptococcal Viridans bacteremia with hypertrophic obstructive cardiomyopathy. Infective endocarditis is more common in those with outflow obstruction and aortic dilation. Therefore it would have been recommended that this patient received antibiotic prophylaxis to prevent infective endocarditis.

Infective Endocarditis as a Complication of Hypertrophic Obstructive Cardiomyopathy

E Roumaya MD, N Tangutur MD; UAB-Huntsville Department of Family Medicine, North Alabama Hospitalist, Huntsville Hospital

Introduction

- Infective endocarditis is a known but rare complication of hypertrophic obstructive cardiomyopathy.
- An incidence of approximately 3.8 per 1000 person-years.
- Associated with a high morbidity and mortality.
- Although controversial, current guidelines do not recommend infective endocarditis antibiotic prophylaxis in hypertrophic obstructive cardiomyopathy.

Clinical Vignette

- A 44-year old Hispanic Male with history of alcohol abuse presented with a 3 month history of worsening fevers, chills, and shortness of breath.
- Outside facility transthoracic echocardiogram revealed an ejection fraction of 60% with early aortic stenosis and mild insufficiency.
- Blood cultures grew Strep viridans.
- Repeat transthoracic echocardiogram revealed a large mobile mitral valve vegetation measuring 19 x 10 mm with severe mitral regurgitation, small vegetation on the aortic valve, and hypertrophic obstructive cardiomyopathy.
- Transesophageal echocardiogram confirmed findings.
- Treated with IV gentamicin and IV ceftriaxone.
- CV surgery performed a successful mitral valve repair and aortic vale repair.
- Surgery was complicated by complete heart block for which he received a permanent pacemaker on POD 5.
- Bridged to Coumadin for anticoagulation while inpatient.
- Discharged home on POD 9 on day 14/14 of IV gentamicin and day 14/28 of IV ceftriaxone.
- Completed a full 4 week IV antibiotic course with ID outpatient.

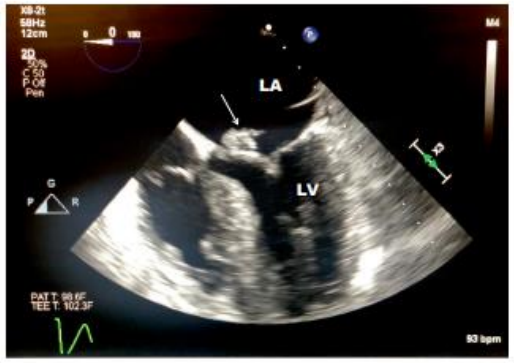

Figure 1. Transesophageal echocardiogram showing a vegetation (arrow) on the mitral valve. The thickened septum as result of hypertrophic obstruction cardiomyopathy can be identified in the inferior-middle of the image.

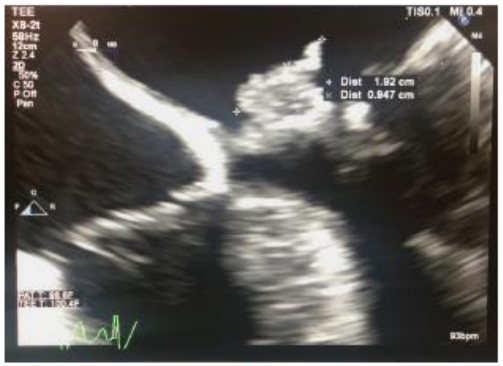

Figure 2. Transesophageal echocardiogram illustrating mitral valve vegetation measuring 1.92 cm x 0.947 cm.

Discussion

- Hypertrophic cardiomyopathy is a genetic condition, which leads to asymmetric septal wall thickening.
- This thickening leads to outflow obstruction and mitral valve abnormalities.
- Commonly the septal aspect of the mitral valve is affected through endocardial damage secondary to turbulent blood flow, and septal contact of the anterior leaflet during systole.
- When identified, surgical intervention is often necessary to relief the outflow obstruction. Commonly this is a mitral valve replacement or repair.
- These abnormalities and obstruction lead to left atrial dilatation.
- Left atrial dilatation has been suggested as a risk factor for infective endocarditis.
- There has been controversy in studies regarding the need for antibiotic prophylaxis in people with hypertrophic cardiomyopathy to prevent infective endocarditis.
- Multiple studies suggest antibiotic prophylaxis in those with hypertrophic cardiomyopathy exhibiting an obstructive pattern and atrial dilation given the increased risk of infective endocarditis.

Conclusion

- This patient presented with late stage disease identified through imaging and blood work revealing infective endocarditis.
- The degree of mitral valve dysfunction, septal enlargement, and atrial dilatation suggest this patient was high risk and may have been a candidate for prophylactic antibiotics.
- However, his disease was not identified until his presentation with symptomatic infective endocarditis.
- Improvements in community based healthcare delivery systems may have allowed this patient to receive periodic health evaluations which would have relieved the system of the high cost of care required by complications of late stage disease.

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Invasive Group A Streptococcal Postpartum Endometritis and Confirmed Toxic Shock Syndrome Associated with Multi-Organ Infarcts and Severe Reactive Arthritis

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Learning Objective

To present an atypical and rare case presentation of streptococcal toxic shock syndrome (S-TSS) complicated by multiple organ infarcts.

Case presentation

A 41 old woman presented 5 days after an uncomplicated vaginal delivery with vague abdominal pain. Physical examination was notable for hypotension which responded quickly to intravenous fluid and mild lower abdominal tenderness on palpation (Blood pressure 71/39 mmHg, heart rate 93 beats/min, respiratory rate of 22 breath/min, oxygen saturation 95% on room air and temperature of 36.4 Celsius). Laboratory results were significant for a high anion gap metabolic acidosis of 18, elevated alkaline phosphatase (ALP), bilirubin, and mild lactic acidosis (Total bilirubin 3.6 mg/dl [0.1-1.2], direct bilirubin 3.4 mg/dl [0.1-0.3], and ALP 283 IU/L [20140], Lactic acid 4.0 mmol/L [0.5-1]). Complete blood count was significant for normocytic anemia (hemoglobin 10.9 g/dl [12-15]) and new onset thrombocytopenia (platelet 84 x10^3/mcL [150-450x10^3]). Vaginal examination and ultrasound showed an enlarged uterus consistent with postpartum findings with no evidence of retained products of pregnancy. The patient was fluid resuscitated in the emergency room and blood, urine and cervical cultures were obtained. She was started on broad spectrum antibiotic therapy with intravenous penicillin and clindamycin for a preemptive clinical diagnosis of postpartum endometritis with probable toxic shock syndrome. At less than 24 hours, two sets of blood and cervical cultures were positive for group A β-hemolytic Streptococcus. The patient subsequently developed multi-organ infarcts, acute respiratory distress syndrome requiring noninvasive respiratory support, and severe reactive arthritis. Our patient improved gradually throughout her hospital stay and was discharged to a rehab facility on antibiotic therapy for four total weeks due to the possible presence of embolic phenomenon.

Discussion

Our patient was diagnosed with invasive GAS and STSS associated with confirmed splenic infarct, multiple suspected renal infarcts and severe reactive arthritis. To our knowledge, this has not been previously reported in the medical literature. Torda et al reported a case in Australia in 2005 of probable S-TSS and multiple associated splenic and pulmonary infarcts. Progression of the disease could potentially be prevented by maintaining a high index of suspicion for puerperal sepsis in the post-partum period, especially when patients present with vague complaints of fatigue and persistent abdominal pain coupled with hypotension. Furthermore, physicians should be aware that negative imaging does not rule out the presence of endometritis, as evident in our patient's case. This further directs focus on clinical findings in order to attempt early diagnosis and implementation of effective and aggressive treatment management. Careful consideration to the patient's postpartum clinical presentation with implementation of an interdisciplinary approach should be utilized.

***This vignette was chosen for oral presentation on Research Day.**

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Submission Category: Clinical Vignette

Title: Is It Getting Hot in Here? A Case of Multiorgan Failure and DIC in Heat Stroke.

Learning Objectives:

1. Diagnose heat stroke based on core temperature and change in mental status.
2. Recognize and treat hyperthermia early.
3. Identify risk factors for heat-related deaths, including mental illness.
4. Manage multi-organ failure in heat stroke by understanding the pathophysiology.

Case:

A 35-year-old male with bipolar disorder and drug abuse history was found unresponsive. The patient was last seen having audiovisual hallucinations. At an outside hospital, the patient's temperature was 42.4^{o} C. Patient was intubated and given vasopressors before being transferred. On presentation, the patient had a core temperature of 39.9^{o} C, warm, erythematous skin, minimal diaphoresis, bloody stool output, and coma. Witnessed seizure occurred and lorazepam was given. The patient was admitted to the ICU. Laboratory studies were notable for metabolic acidosis secondary to lactic acidosis, rhabdomyolysis, elevated troponins, coagulopathy, AKI, and hemoconcentration. CT head and MRI brain were negative. CT chest, abdomen, and pelvis noted liquid within the colon. Urine drug screen negative. The patient developed progressive oliguric kidney failure requiring CVVHD, reliance on vasopressors, crescendo-decrescendo transaminitis, worsening hyperbilirubinemia, and coagulopathy consistent with DIC. Patient was diagnosed with non-exertional heat stroke with multiorgan failure.

Discussion:

Heat stroke is a life-threatening condition defined as a core body temperature greater than or equal to 40^{o} C with a mental status change. From 2006-2010, 3332 U.S. deaths were attributed to heat stroke. Heat stroke is classified into non-exertional and exertional. Non-exertional heat stroke is often fatal and seen in the elderly and individuals with co-morbidities. Exertional heat stroke occurs in individuals who are physically active in high temperatures such as military personnel and marathon runners. Observational studies have identified additional risk factors for heat-related death including psychiatric illness and inability to care for oneself.

Core temperatures above 40^{o} C are associated with physiologic adaptations and irreversible brain injury. The organs most sensitive to hyperthermia include brain and liver. With hyperthermia, sympathetic nervous system shunts blood flow to cutaneous arterioles to assist with evaporative heat loss, resulting in a decrease in splanchnic blood flow. The liver experiences direct heat injury and hypoperfusion from ischemia thus leading to hepatocyte apoptosis.

The colon also experiences decreased perfusion, which leads to gut edema and permeability to allow release of endotoxins. Endotoxins are not metabolized due to hepatic injury and thus activate systemic

inflammatory response (SIRS), exacerbating volume contraction and cardiac hypoperfusion. Interestingly, heat also leads to a reduction in cerebral blood flow and increased permeability of the blood-brain barrier leading to encephalopathy. Excessive heat causes endothelium heat injury and activates platelet aggregation leading to micro-thromboses, consumptive coagulopathy, and DIC.

The treatment of heat stroke is supportive and centers on reducing the hyperthermia to limit irreversible brain injury and subsequent multiorgan failure. A case series on heat-related liver injury and failure reported survival with liver transplantation. Transferring a patient to a liver transplant center should be done if irreversible brain injury is ruled out and acute liver failure is present. Prevention of heat-related deaths focuses on limiting risk factors and optimizing protective risk factors.

***This vignette was chosen for oral presentation on Research Day.**

Isolated painful Horners syndrome as the façade of an underlying silent Dissection

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LEARNING OBJECTIVES:

1. Identify carotid artery dissection when presenting with ocular manifestations.

2. Therapeutic decision making in carotid artery dissections

CASE PRESENTATION:

38-year-old male with past medical history of hypertension and migraines presented with unequal pupils preceded with symptoms of right eye pain and conjunctival injection. Review of systems positive for right sided neck and scapular pain. Examination revealed right pupillary constriction, right eyelid drooping and loss of cilio-spinal reflex suggesting partial Horner syndrome. Rest of the neurological examination was noted to be intact. CT Angiography involving head and neck was done which revealed abnormality in form of narrowing and irregularity noted in the extracranial portion of distal right internal carotid artery with dense soft tissue thickening leading to near total occlusion of the vessel, suggesting Right internal carotid artery dissection, with possible secondary thrombosis. Heparinization was immediately initiated. Four vessel cerebral angiography was performed which revealed promising flow across the anterior communicating artery from left to right, being the reason behind preserved cranial functions. Asymptomatic presentation of carotid artery dissection with or without evident thrombosis warrants medical treatment with either antiplatelets or anticoagulants. Preferred treatment used here was anticoagulation using Warfarin with low molecular weight heparin (Lovenox) bridging, given the significant thrombosis associated with dissection and Surveillance in 3 months advised. Repeat cerebral angiography in 3 months revealed near complete resolution of Right internal carotid artery dissection. However, treatment with coumadin continued for a total of 6 months duration to avoid carotid thrombo-embolic complications.

DISCUSSION:

More than 60% of Internal Carotid artery dissections (CADs) are known to present with ocular manifestations mainly in form of Horner syndrome with or without visual disturbances. Carotid artery dissections often under-recognized as cause of Horner syndrome and could be missed easily especially when patient lacks any other neurological signs. Acute onset Horner syndrome with or without unilateral neck pain or headache and visual disturbances warrants investigation of possible CAD. Conventional angiography is the preferred diagnostic test but however CT/MR Angiography often serves as the first step due to their feasibility. In case of presentation with neurological deficits, ischemia/infarct involving middle cerebral artery are more common in complicated dissections associated with embolism. Hence strong suspicion of carotid artery dissection warrants immediate heparinization as the first step. Treatment options aim at preventing thrombo-embolic events

associated with dissections. Antiplatelets are preferred in case of strongly doubted dissections or clinically confirmed dissection with no evident active thrombosis. Anticoagulants on the other hand are the preferred agents in case of evident active thrombosis. Choice of drugs either warfarin with therapeutic INR goal of 2.0-3.0 or newer drugs like apixaban or rivaroxaban often depends on clinical expertise and affordability, with a duration of 3- 6 months. In case of contraindication to medical treatment or hemodynamic instability, endovascular intervention with stenting is preferred. Prognosis is highly variable in case of dissections anywhere. Isolated local deficits are known to have better prognosis with complete resolution; however, recurrence is possible.

Isolated painful Horner syndrome as the façade of an underlying silent Dissection

THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

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Learning objectives

1. Identify carotid artery dissection when presenting with ocular manifestations.
2. Therapeutic decision making in carotid artery dissections

Case presentation

HPI
- 38 yo Caucasian male presenting with c/o unequal pupils. Symptoms started 2 days prior with right eye pain and conjunctival injection.

Relevant history
- Former smoker, actively consumes 1-2 beer daily
- h/o Hypertension and Chronic migraines on Nadolol 20mg qHS and Rizatriptan PRN

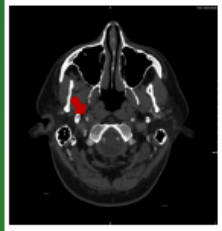
Exam
- Eyes asymmetry : Right eyelid drooping +
- Pupil size : R < L
- Loss of ciliospinal reflex on Right side; Intact on left

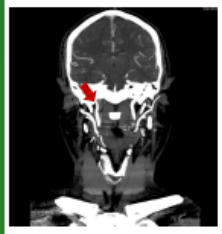
Vitals

T : 36.7 C
PR : 64/min
RR : 17/min
BP : 167/117 mmHg RUE, 164/110mmHg LUE

Imaging
- CT Orbits w/ contrast
- CTA Head and Neck
- Cerebral Angiogram

1. CT orbit w/ contrast
- No significant abnormality noted.





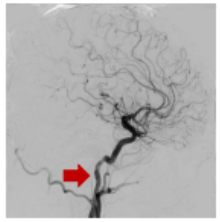
2. CTA Head &Neck revealing abnormality in form of narrowing and irregularity in the extracranial portion of distal right internal carotid artery with dense soft tissue thickening leading to near total occlusion of the vessel, suggesting dissection

3. Four vessel Cerebral Angiogram revealing promising flow across the anterior communicating artery from left to right, being the reason behind preserved cranial functions.

Treatment
- Heparinization initiated as soon as dissection was suspected.
- Later, treatment was narrowed down to Warfarin with Lovenox bridging given significant thrombosis associated with dissection.

Follow up

In 3 months,



Four vessel Cerebral angiography revealed near complete resolution of Right internal carotid artery dissection.

Continued treatment –
Warfarin continued for a total of 6 months duration to avoid carotid thrombo-embolic complications.

Discussion

- More than 60% of Internal Carotid artery dissections (CADs) are known to present with ocular manifestations mainly in form of Horner syndrome w/ or w/o visual disturbances.
- Carotid artery dissections often under-recognized as cause of Horner syndrome and could be missed easily especially when patient lacks any other neurological signs.
- Acute onset Horner syndrome with or without unilateral neck pain or headache and visual disturbances warrants investigation of possible CAD.
- Conventional angiography is the preferred diagnostic test but however CT/MR Angiography often serves as the first step due to their feasibility.
- In case of presentation with neurological deficits, ischemia/infarct involving middle cerebral artery are more common in complicated dissections associated with embolism.
- Hence strong suspicion of CAD warrants immediate heparinization as the first step.
- Treatment options aim at preventing thrombo-embolic events associated with dissections.
- Antiplatelets are preferred in case of strongly doubted dissections or clinically confirmed dissection with no evident active thrombosis.
- Anticoagulants on the other hand are the preferred agents in case of evident active thrombosis.
- Choice of drugs either warfarin with therapeutic INR goal of 2.0-3.0 or newer drugs like apixaban or rivaroxaban often depends on clinical expertise and affordability, with a duration of 3- 6 months.
- In case of contraindication to medical treatment or hemodynamic instability, endovascular intervention with stenting is preferred.
- Prognosis is highly variable in case of dissections anywhere. Isolated local deficits are known to have better prognosis with complete resolution; however, recurrence is possible.

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Title: "*I've got MAC on my finger!*"

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Submission Category: Clinical Vignette

Learning Objective:

Mycobacterium Avium Complex (MAC) is an acid-fast Gram-positive bacillus that typically affects immunocompromised patients and can present in various ways. Risk factors for MAC infections includes AIDS with CD4 count of less than 100 and other immunocompromise diseases such diabetes and cancer. The most common presentation includes pulmonary disease and disseminated infections. Pulmonary disease presents with cough, fatigue, malaise, dyspnea, hemoptysis, weight loss, and fever and normally affects individuals with underlying lung disease. Disseminated disease is another common presentation with bacteremia with seeding to other organs. Other presentations include pericarditis, soft tissue abscesses, skin lesions, lymph node involvement, central nervous system lesions, and bone infection. This case study will discuss MAC infection in bone and its treatment.

Case Presentation:

A 63-year-old female with history of ADHD and hypertension presented with a left index finger injury from a rose bush in 2/2018. She presented with swelling, pain, and redness at the site, but she did not have any fevers or chills. She had an I&D of the finger on 4/2018. 6 weeks later, her finger was still swollen and painful and MRI of the finger was negative. She was seen by an infectious disease physician on 11/2018 and was referred for biopsy of the hand with bacterial, fungal, AFB stain and culture as well as a repeat MRI. The left index finger on physical exam was indeed erythematous and tender. Biopsy was positive for MAC and patient was treated with clarithromycin 500 mg BID, ethambutol 400 mg, and rifampin 300 mg BID. Her immunodeficiency work up including HIV were negative. Patient was treated for one year, her symptoms and signs resolved with no recurrence.

Discussion:

MAC infection involving the skin and soft tissues and bones are rare. Modes of transmission includes trauma or surgical procedures. Diagnosis for bone infection is made through biopsy. It is important to detect MAC infection as soon as possible since skin, soft tissue, and bone infection can lead to abscess formation and fistulas. Treatment options includes debridement and antibiotics. Unfortunately, there are no clear guidelines for treatment of such infections. Antibiotics of choice to treat MAC infections includes a macrolide, ethambutol15mg/kg daily, and rifamycin. Options for macrolide includes azithromycin 250-300 mg/day or clarithromycin 500-1000 mg/day. If the bacteria are resistant to macrolides, aminoglycosides with fluoroquinolones can be added for coverage. For choices of rifamycin,

rifabutin 150-300 mg/day or rifampin 600 mg/day can be used. Recommended duration of treatment is at least 6 months.

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"I've got MAC on my finger!"

Qurrat-ul-ain Aziz, D.O., M.P.H. , Joseph Shaw, and Ali Hassoun, M.D.

Learning Objective:

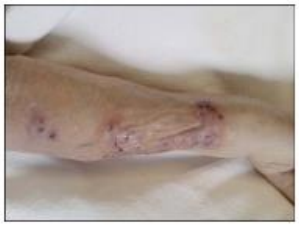
Mycobacterium Avium Complex (MAC) is an acid fast Gram positive bacilli that typically affects immunocompromised patients and can present in various ways.

Risk factors

- AIDS with CD4 count of less than 100
- Diabetes
- Cancer

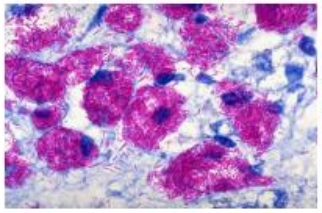
Presentation

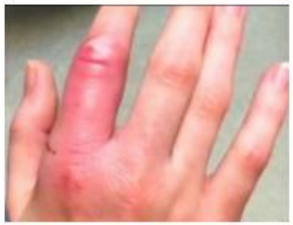
- Pulmonary disease
- Disseminated disease
- Bacteremia
- Pericarditis
- Soft tissue abscesses
- Skin lesions
- Lymph node involvement
- Central nervous system lesions
- Bone infection



Case Presentation:

- **Chief Complaint**: 63 year old female with history of ADHD and hypertension presented with a left index finger injury from a rose bush in 2/2018.
- **ROS**: swelling, pain, and redness at the site. No fever or chills
- **4/2018**- I&D of the finger
- **6/2018-** still swollen and painful and MRI of the finger was negative
- **11/2018-** seen by ID and referred for biopsy of the hand with stain and culture as well as a repeat MRI
- **Physical exam:** erythematous and tender left finger.
- **Labs:** HIV and other immunodeficiencies and results were negative
- **Biopsy:** positive for MAC
- **Treatment:** Clarithromycin 500 mg BID, Ethambutol 400 mg, and Rifampin 300 mg BID with end date on 1/16/2020. Her swelling had decreased and her finger was no longer tender.





Antibiotics	
Macrolide	Azithromycin- 250 to 300 mg/day Clarithromycin- 500 to 1000 mg/day
Ethambutol	15mg/kg daily
Rifamycin	Rifabutin- 150 to 300 mg/day Rifampin- 600 mg/day
Macrolide resistant	Aminoglycosides Fluoroquinolones

Discussion:

- MAC infection involving the skin and soft tissues and bones are rare
- Modes of transmission: trauma or surgical procedures
- Diagnosis- biopsy
- Can lead to abscess formation and fistulas
- Treatment- debridement and antibiotics
- Antibiotics of choice includes macrolide, ethambutol , and rifamycin
- Recommended duration of treatment is at least 6 months

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Management of Pediatric Tic Disorders and Co-Morbid Psychiatric Disorders

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Submission Category: Clinical Vignette

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Learning Objectives

1. Understand the comorbid disorders associated with tics.
2. Understand the optimal treatment options for tics with and/or without associated comorbid disorders.
3. Understand the benefit of behavioral therapy in the treatment of tics.

Case Presentation

A 6-year-old Caucasian male presents to the office with his mother and father for an evaluation of ADHD (Attention Deficit Hyperactivity Disorder). The parents noted that he began having behavioral issues at daycare at the age of 3. Last year, he had severe behavior issues in Kindergarten and his parents had to pull him out of school and home school him the rest of the year. His symptoms are characterized by a short attention span, impulsive and hyperactive behavior, easy distractibility, poor listening, poor grades, careless mistakes, losing items, avoiding tasks that require mental effort, difficulty playing quietly, fidgeting, excessive talking, difficulty waiting his turn, and interrupting others. The symptoms are present both at home and at school. Exacerbating factors include fatigue, distracting activities, classroom time, group play, and mental effort. Alleviating factors include, attention holding activities such as watching television. His symptoms are impairing his ability to function and learn. He was treated for ADHD in the past but is currently not on medication because the medication worsened his tics. His parents note that he has always had minor tics since he was very little, but they became much more apparent around 6 years of age. He exhibits both motor and vocal tics, characterized by neck jerking and humming, respectively. The patient notes that he is unable to control these tics, and they are worse when he is stressed.

He was prescribed methylphenidate for his ADHD symptoms. At the next visit, his parents noted that since starting the medication, his tics had worsened. He was then prescribed guanfacine as an adjunct to the methylphenidate, which the parents noted improved the severity of the tics. At his fourth clinical visit, the patient and his father unfortunately noted that his tics had returned to full severity and the neck jerks were causing the child pain.

Discussion

The optimal management of tic disorders should address a patient's hierarchy of impairments. A physician's focus should be on treating the most socially and occupationally debilitating condition, which is usually the comorbid condition, such as ADHD or OCD, and then continuing to monitor the child's tics. Medications approved for the management of tics include 1st and 2nd generation antipsychotics. Alpha-2 adrenergic agonists, such as clonidine and guanfacine are indicated for the treatment of tics and comorbid ADHD. Some patients have described worsening of their tics due to stimulant treatment for ADHD, but this association has not been proven in the literature. In addition to medication, it is very important to educate the family and teachers about the course and the prognosis of tic disorders. Treatment planning should include accommodations in the classroom such as an Individualized Education Plan/504 Plan. Children with moderate to severe tics and/or have comorbid conditions that respond to behavioral therapy should be considered for Comprehensive Behavioral Intervention for Tics (Habit Reversal Training), which entails awareness training, developing a competing response to the urge to tic, and social support. This service has been shown to significantly reduce tic severity and improve function and is provided at Children's Hospital of AL.

A Case of Tourette's Disorder

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Introduction

DSM- V Diagnostic Criteria for Tourette's disorder

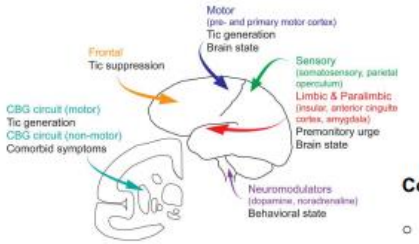
1. Both multiple motor and 1 or more vocal tics have been present at some time during the illness, although not necessarily concurrently.
2. The tics occur many times a day (usually in bouts) nearly every day or intermittently throughout a period of more than 1 year, and during this period there was never a tic-free period of more than 3 consecutive months.
3. The disturbance causes marked distress or significant impairment in social, occupational or other important areas of functioning.
4. The onset is before age 18.
5. The disturbance is not due to the direct physiological effects of substance (e.g., stimulants) or a general medical condition (e.g., Huntington's disease of post viral encephalitis).

- Tics- sudden, rapid, recurrent, non-rhythmic movement or vocalization
- Simple, complex, transient, or chronic
- Tourette's disorder- chronic tic disorder (CTD)
- Comorbid conditions with CTD: ADHD, OCD, learning disabilities, Autism spectrum disorder
- No evidence that stimulant medications increase tics, but may see exacerbation of tics in individual cases
- Behavioral interventions- Habit Reversal Training (HRT)
- Most verified behavioral approach to treatment of tics
- No empirical support for deep brain stimulation (DBS), repetitive magnetic stimulation, special diets and dietary supplements for treatment of CTD's

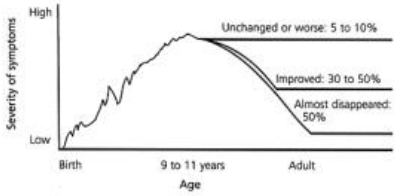
Case Report

The patient is a 6-year-old boy with a past medical history of ADHD being seen for the management of severe tics and behavior problems. His symptoms began at 3 years of age and at age 5, he had to be pulled out of public school due to worsening behavior problems. He currently suffers from both motor and vocal tics, characterized by neck jerking and humming. The tics are worsened by stress. He was first prescribed methylphenidate for his ADHD symptoms. At a follow up visit, his parents noted that since starting the medication, his tics had worsened. He was then prescribed guanfacine as an adjunct to the methylphenidate, which improved the severity of the tics. At his fourth clinical visit, the patient and his father unfortunately noted that his tics had returned to full severity and the neck jerks were causing the child pain, which prompted a referral for HRT.

Discussion



The diagram shows primary brain pathways involved in TDs. (Diagram from *Pathophysiology of Tic Disorders* by Yael D, et. al.)



Onset typically occurs before 7 years of age and the disorder is usually recognized 2 to 3 years after onset. In most children, the severity peaks at 9 to 11 years of age. About 5 to 10% of patients have been intensifying course with little or no improvement. In about 85% of patients, symptoms diminished during and after adolescence.

Problems associated with Tourette's syndrome

Coprolalia
- Obscene words and statements

Copropraxia
- Obscene gestures
- Touching others sexually
- Holding groin, Picking at buttock

Compulsive Behaviors
- Touching things
- Rechecking
- Washing hands repeatedly

Eco Phenomena
- Echolalia
- Palilalia
- Echopraxia

Most Common Tics

- ❑ Throat clearing
- ❑ Coughing
- ❑ Hiccupping
- ❑ Sniffing
- ❑ Eye blinking

Management

❑ **Medications:**
Alpha-2 adrenergic agonists; 1st and 2nd generation antipsychotics

❑ **Behavioral therapy**:
Habit Reversal Training

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THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

Mental Health and Sarcoidosis

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Learning Objectives:

1. Characterize Sarcoidosis as a disease
2. Understand the impact Sarcoidosis has on mental health
3. Consider the rational and pertinence of screening for Sarcoidosis in select patients

Case Presentation:

Patient is a 15-year-old Caucasian female with history significant for anemia presented to clinic for possible anxiety and depression. The patient states that she has been feeling "off" over the past year and that it has been gradually worsening. She attributes these mood changes to feeling like she has not adjusted to starting high school as well as her peers. She complains of feeling low on energy, losing interest in activities she was previously passionate about, changes in sleep and appetite and frequently feeling like a burden on her family. She also complains of new onset episodes of feeling excessively anxious around 3 times per week but denies symptoms related to panic attacks.

The patient has a maternal family history of sarcoidosis, anxiety, and depression. Two of her family members have officially been diagnosed with sarcoidosis, her maternal great grandmother in her early 60s as well as her maternal aunt in her 40s. Family reports that few other family members may also have sarcoidosis but have not been officially diagnosed. The family members who have been diagnosed with depression and anxiety are exclusively female, reportedly have symptom onset at a younger age, and have struggled with it throughout their lives. The family members who have been diagnosed with sarcoidosis also reportedly have more severe cases of anxiety and depression in comparison to other family members who have not displayed any sarcoid related symptoms.

Discussion:

Sarcoidosis is an inflammatory and immunological disease characterized by non-caseating granulomas with multiple organ system involvement. There is thought to be a genetic component to sarcoidosis as higher rates of the disease are seen in families especially with mother-child relationships. However, it is challenging to diagnose due to vague complaints such as fatigue. Furthermore, it is more mystifying to know when the disease actually manifests. Can one be asymptomatic for years without any overt somatic complaints? Some studies report up to 65% prevalence of anxiety/depression in patients with

asymptomatic or symptomatic sarcoidosis. While the cause is not known, those with the inflammatory condition had significant psychosocial stress prior their diagnosis. This is a reasonable observation considering the interplay between the immune system and stress is well documented. Is it possible for anxiety/depression to be present in a large population of Sarcoidosis patients prior to their diagnosis? With vague complaints, patients suffer on average of 5 years before being diagnosed. Is it valid to screen patients presenting with anxiety or depression who also have a strong family history for Sarcoidosis? We need more research in this area to guide clinical practice.

Mental Health and Sarcoidosis

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Introduction

Sarcoidosis is an immunological disease characterized by non-caseating granulomas involving multiple organ systems, commonly the lungs, skin, eyes, and heart. Neurologic manifestations are seen in 5% of patients and is attributed to granulomatous inflammation in the CNS.[5] Sarcoidosis is believed to have some genetic component as increased rates of disease are seen in family clusters (five times more likely in the sibling of patient), racial groups (three times more likely in African Americans), and specific HLA subtypes.[7]

Case Report

Patient is a 15-year-old Caucasian female with history significant for anemia presented to clinic for possible anxiety and depression. The patient states that she has been feeling "off" over the past year and that it has been gradually worsening. She attributes these mood changes to feeling like she has not adjusted to starting high school as well as her peers. She complains of feeling low on energy, losing interest in activities she was previously passionate about, changes in sleep and appetite and frequently feeling like a burden on her family. She also complains of new onset episodes of feeling excessively anxious around 3 times per week but denies symptoms related to panic attacks.

The patient has a maternal family history of sarcoidosis, anxiety, and depression. Two of her family members have officially been diagnosed with sarcoidosis, her maternal great grandmother in her early 60s as well as her maternal aunt in her 40s. Family reports that few other family members may also have sarcoidosis but have not been officially diagnosed. The family members who have been diagnosed with depression and anxiety are exclusively female, reportedly have symptom onset at a younger age, and have struggled with it throughout their lives. The family members who have been diagnosed with sarcoidosis also reportedly have more severe cases of anxiety and depression in comparison to other family members who have not displayed any sarcoid related symptoms.

Discussion and Future Direction

Sarcoidosis affects multiple organ systems lending to various somatic complaints, with fatigue ranking the most common.[8] Yet, the significant predictors of fatigue are cognitive dysfunction and depressive symptoms. Compared to the general population, those with sarcoidosis have significantly more anxiety and depression with the largest impact affecting the younger age groups. Dyspnea, SES, comorbidities, and quantity of organ systems affected contributes to the development of anxiety and depression.[4] Of the comorbidities, psychiatric complaints for anxiety and depression are roughly 65%.[2]

One Netherland study found a 4% prevalence of anxiety/depression in asymptomatic sarcoidosis patients and a 30% prevalence in symptomatic patients. Although this percentage is lower than other studies who report 60% - 66% prevalence, these values are substantially high considering the study eliminated those with significant comorbidities.[6]

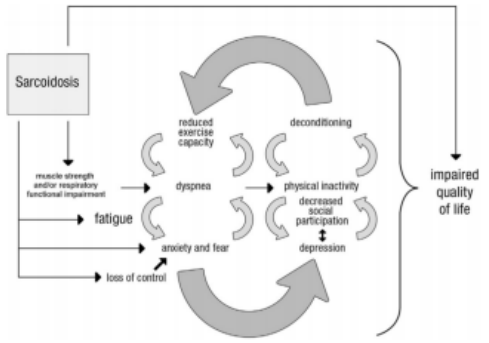

Fig. 1. Negative vicious circle of physical deconditioning: disabling symptoms in sarcoidosis can reduce daily physical activities, resulting in general deconditioning and a reduced quality of life. (Adapted from Swigris JJ, Brown KK, Make BJ, et al. Pulmonary rehabilitation in idiopathic pulmonary fibrosis: a call for continued investigation. Respir Med 2008;102(12):1678-85, and Marcellis RG, Lenssen AF, de Vries J, et al. Reduced muscle strength, exercise intolerance and disabling symptoms in sarcoidosis. Curr Opin Pulm Med 2013;19(6):528.)

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Literature cites approximately 450 cases of familial sarcoidosis with evidence of potential genetic factors. Familial sarcoidosis is more prevalent in mother-child relations.[1] While the cause of sarcoidosis is unknown, those with the inflammatory condition had significant psychosocial stress prior to their diagnosis.[6,9] This is a logical assumption considering stress alters the immune system and inflammatory response significantly impacting many diseases.

A study done by Yamada et al. found psychosocial stress and lack of sleep were prominent in patients one year prior to their diagnosis.[9] Although this study was limited to 55 newly diagnosed subjects, there is literature providing expansive details of patients' symptoms the years prior to diagnosis.

Is it possible for anxiety/depression, whether it be from psychosocial stressors or inflammatory effects, to be present in a large population of sarcoidosis patients prior to their diagnosis? Typically patients suffer on average 5 years before getting diagnosed prolonging their suffering and lowering their quality of life.[4] Is it valid to screen patients presenting with anxiety or depression and who have a family history for Sarcoidosis?

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THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

"The Mind-Body Connection: Managing Depression and Anxiety in Pediatric Patients with Crohn's Disease"

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Submission Type: Clinical Vignette

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Learning Objectives:

- Investigate the bidirectional relationship between Crohn's disease and depression
- Highlight the association between treating depression and anxiety in patients with Crohn's disease and higher rates of disease remission

Case Presentation:

A 17-year-old Caucasian female with a history of Crohn's disease (CD) and polyarticular juvenile idiopathic arthritis (PJIA) presented for an initial psychiatric evaluation with worsening depression over the past few months. No specific trigger for depression was identified. Depressed mood was associated with increased irritability causing interpersonal conflicts, increased weight gain, sedation, decreased concentration, and low self-esteem.

Patient was diagnosed with Crohn's disease at 13 years old. She was given prednisone to induce CD remission. While taking prednisone, she had a psychotic episode requiring hospitalization. Complicating her case, she had a family history of bipolar disorder. Due to concerns of distinguishing between steroid-induced psychosis and the first presentation of bipolar disorder, she was previously treated with antipsychotics. Additionally, previous trials of SSRIs to treat depression seemed to worsen her mood symptoms.

Over the course of 6 months since her initial evaluation, the patient has been treated with bupropion and lamotrigine and has experienced fewer depressive symptoms. She reports her mood has improved, along with her social anxiety, resulting in enhanced work performance. Her most recent PHQ-A assessment scaled her depression as a 6 out of a 27-point scoring system, indicating mild depression.

Discussion:

Patients with Crohn's disease have 2-3 times higher rates of depression and anxiety compared to the normal population. Depression and anxiety were originally thought to be caused by the psychological burden of living with a chronic illness. However, recent research implicates that CD and depression may have a genetic correlation and share similar pathophysiological mechanisms. Clinical studies reveal that antidepressant usage in patients with CD improved remission rates. These findings suggest patients with CD should be screened for depression and anxiety. Adequate management of both Crohn's disease and depression/anxiety may lead to a more benign disease course and relief of psychiatric symptoms.

The Mind-Body Connection: Managing Depression in Patients with Crohn's Disease

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Introduction

- The etiology of Crohn's disease is theorized as an abnormal inflammatory response to commensal bacteria in the gastrointestinal tract
- Depression has been associated with elevated levels of inflammatory markers[1]
- Patients with Crohn's disease tend to have higher rates of anxiety and depression compared to the general population[2]
- Pediatric patients are especially prone to developing depression and anxiety after diagnosis[3]
- Higher rates of anxiety and depression are associated with patients in active disease rather than patients in remission[4,5]
- Antidepressant use is associated with fewer relapses in disease activity[4,6]
- Screening for psychiatric illness is not a part of standard IBD treatment[3]
- Adequate management of both Crohn's disease and depression/anxiety may lead to a more benign disease course and relief of psychiatric symptoms

Case Report

A 17-year-old Caucasian female with a history of Crohn's disease (CD) presented for an initial psychiatric evaluation with worsening depression over the past few months. No specific trigger for depression was identified. Depressed mood was associated with increased irritability causing interpersonal conflicts, increased weight gain, sedation, decreased concentration, and low self-esteem.

Patient was diagnosed with Crohn's disease at 13 years old. She was given prednisone to induce CD remission. While taking prednisone, she had a psychotic episode requiring hospitalization. Complicating her case, she had a family history of bipolar disorder. Due to concerns of distinguishing between steroid-induced psychosis and the first presentation of bipolar disorder, she was previously treated with antipsychotics. Additionally, previous trials of SSRIs to treat depression seemed to worsen her mood symptoms.

Over the course of 6 months since her initial evaluation, the patient has been treated with bupropion and lamotrigine and experienced fewer depressive symptoms. She reports her mood has improved, along with her social anxiety, resulting in enhanced work performance. Her most recent PHQ-A assessment scaled her depression as a 6 out of a 27-point scoring system, indicating mild depression.

Bi-directionality of Brain–Gut Interactions in Patients With Inflammatory Bowel Disease, Gracie, David J. et al., Gastroenterology, Volume 154, Issue 6, 1635 - 1646.e3

Discussion

- Recent research implicates that CD and depression may develop secondary to activation of immune-inflammatory pathways[1]
- TNF-alpha is a pro-inflammatory cytokine that is elevated during active flare-ups in Crohn's disease[7]
- **Bupropion increases monoaminergic and dopaminergic tone by increasing intracellular cAMP and is hypothesized to lower TNF-alpha levels through this mechanism[7-8]**
- **Bupropion has been associated with inducing remission in CD and other autoimmune diseases[7-8]**
- Clinical studies reveal that antidepressant usage in patients with CD improved remission rates[6]
- These findings suggest patients should be screened for depression and anxiety at the time of initial diagnosis and during active flare-ups
- **Clinicians may consider bupropion as a possible first line treatment for managing depression in patients with CD[8]**

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Necrotizing granulomas in a young immunocompetent patient

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LEARNING OBJECTIVE:

1. Recognize and diagnose pulmonary histoplasmosis in a young healthy patient.
2. Manage histoplasmosis infection with the proper drugs and duration

CLINICAL PRESENTATION:

29-year-old African American female presented with atypical chest pain and dry cough lasting for a month. Patient stated that she was perfectly healthy until the symptoms began and persisted despite outpatient treatment. Review of systems significant for occasional low-grade fever and night sweats. No history revealing any recent travel, exposure to woods or soil components, contact with people of similar illness or significant loss of weight/appetite. Laboratory investigations revealed normal white blood cell counts. Chest X-ray revealed Left perihilar fullness. On further evaluation, Computed Tomography (CT) Chest with contrast confirmed enlarging left hilar adenopathy concerning for malignancy and stable Left Upper lobe nodules suggesting inflammatory etiology. Further PET CT imaging helped rule out malignancy. Decision was made to proceed with Video Assisted Thoracoscopic Surgery (VATS) Left Upper lobe wedge resection and VATS Mediastinal mass biopsy for further evaluation. Frozen sections intraoperatively did show necrotizing granulomas and further expert reports confirmed necrotizing granulomatous inflammation with associated yeast forms consistent with Histoplasma species. Meanwhile Serology tests revealed positive histoplasma antibodies of titers 1:64 adding to the diagnosis. Test results for other granulomatous diseases were negative. Hence, the combination of serology and tissue pathology helped in diagnosing the patient with Acute localized pulmonary histoplasmosis. Patient was started on Isavuconazole for a duration of 12 weeks. Further follow up visits confirmed clinical response to the treatment chosen, with no relapse of symptoms.

DISCUSSION:

Granulomatous lesions in lung mainly concerns for the possibility of infectious etiology which includes Tuberculosis, Fungal infections (Histoplasmosis, Aspergillosis, Blastomycosis, Coccidioidomycosis, Cryptococcosis) and others including include Sarcoidosis and Malignancy. Histoplasmosis, caused by fungus Histoplasma capsulatum, includes a spectrum of diseases ranging from self-limited respiratory illness to disseminated infection, as per CDC definition. This infection is known to be endemic to Northern America particularly Ohio and Mississippi river valleys. The mode of transmission is always through inhalation of spores from soil particles enhanced by bird/bat excrement, hence known as Cave disease. No cases have been reported with human-human transmission. Most commonly, the disease presents with pulmonary symptoms including dry or wet cough, atypical chest pain and febrile episodes more like a pneumonia. Sometimes vague presentation which are common in immunocompetent, most often delay diagnosis and the start of treatment which may complicate the clinical course. Early treatment is the key step in cure. Widely used treatment options include Amphotericin B, Amphotericin B lipid complex and Itraconazole. In this young patient, Isavuconazole was preferred for its excellent oral

bioavailability and better pharmacokinetics. Duration of treatment depends on the extent of involvement. It may vary between 6 to 12 weeks in an acute localised infection and months to 1 year in severe disseminated infection. Asymptomatic and mild pulmonary histoplasmosis may even resolve without treatment.

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Necrotizing granulomas in a young immunocompetent patient

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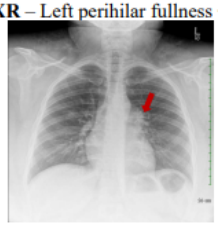
Learning objectives:

1. Recognize and diagnose pulmonary histoplasmosis in a young healthy patient.
2. Manage histoplasmosis infection with the proper drugs and duration.

Case description:

- **HPI** – 29 yo previously known perfectly healthy AA female presented with chest pain and dry cough for more than a month.
- No known exposure to contacts with sick people, woods to soil components.
- No significant prior medical illness.
- **Examination** – Lungs clear to auscultation with bilateral equal air entry and no added sounds. Other system examinations unremarkable.
- **CXR** – Left perihilar fullness +



- **CT Chest with contrast** confirmed enlarging left hilar adenopathy concerning for malignancy and stable left upper lobe nodules suggesting inflammatory etiology.
- **Video Assisted Thoracoscopic Surgery(VATS)** Left Upper lobe wedge resection and

Mediastinal mass biopsy was performed.

- **Serology**
  Histoplasma yeast antibody titers 1:64
- **Tissue pathology-** Necrotizing granulomatous inflammation with associated yeast forms consistent with Histoplasma species.

▲ ***Acute localized pulmonary histoplasmosis***
R_x Isavuconazole x 12 weeks

Discussion:

- Fungal infections causing granulomatous lung diseases

1. Necrotizing *(Cryptococcus, Histoplasma spp, Blastomyces spp.,Aspergillus spp.,Coccidiodes,Mucor)*
2. Non Necrotizing - *Candida spp*

- Other causes of granulomatous inflammation to be ruled out in a young patient

1. Non fungal Infections
   Bacterial – Tuberculous/Non tuberculous mycobacteria, Nocardia spp, Rickettsia, Q fever and Cat scratch disease.
2. Inflammation - Pneumonitis
3. Auto-immune- Sarcoidosis, SLE, Churg strauss, GPA
4. Malignancy- Lymphoma, Langerhan cell histiocytosis, secondaries.
5. Miscellaneous – Drugs (Methotrexate), Toxins (Beryllium, Zirconium), Pneumoconiosis

Pulmonary histoplasmosis

- CDC definition: Histoplasmosis, caused by fungus Histoplasma capsulatum, includes a spectrum of diseases ranging from self-limited respiratory illness to disseminated infection.
- Endemic areas: Northern America particularly Ohio and Mississippi river valleys.
- Mode of transmission: inhalation of spores from soil particles enhanced by bird/bat excrement, hence known as Cave disease. No cases have been reported with human-human transmission.
- Pulmonary symptoms: dry or wet cough, atypical chest pain and febrile episodes more like a pneumonia. Sometimes vague presentation which are common in immunocompetent.
- Diagnostic tests: Positive serology with specimen biopsy together yields high sensitivity/ specificity.

Types of Pulmonary histoplasmosis	Mild to Mod disease	Severe disease	Duration
Acute localized pulmonary disease	Only if symptoms persist > 4 weeks despite initial R_x. Itraconazole 200mg TID x 3 days then 200mg BID	1 to 2 weeks of Amphotericin, followed by Itraconazole 200mg TID x 3 days then 200mg BID	6- 12 weeks
Acute diffuse pulmonary disease	Same as above		
Chronic cavitary pulmonary histoplasmosis	Itraconazole 200mg TID x 3 days then 200mg BID		At least 1 year
Mediastinal syndromes/ Broncholithiasis/ Lung nodules	Medical R_x not proven useful unless symptomatic. Itraconazole 200mg TID x 3 days then 200mg BID +/- Surgical intervention.		6 – 12 weeks

In this young patient, Isavuconazole was preferred over conventional Itraconazole for its excellent oral bioavailability and better pharmacokinetics.

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Clinical Vignette
Title: "On heparin with thrombocytopenia, must be Heparin Induced Thrombocytopenia," – Think again.

Learning Objective: Clinicians should always act urgently if Thrombotic Thrombocytopenia Purpura (TTP) is suspected, but it is also important to keep all causes of hemolytic anemia and thrombocytopenia in mind.

Case Presentation: A previously healthy 49-year-old female presented to the emergency department for evaluation of headache of 3 days duration. Initial imaging showed acute right frontal lobe and intraventricular hematoma with a small volume subarachnoid hemorrhage due to an aneurysm from the mid right anterior cerebral artery. She underwent a successful intracranial aneurysmal coiling of the nine mm right A1/A2 aneurysm. Patient was doing well until six days post bleed when she developed progressive neurological decline with repeat CAT scan of the head showing slight increase in ventricular size and increasing edema. Due to the hydrocephalus, a left frontal ventriculostomy was performed and an external ventricular drain was placed. Angioplasty was also performed due to severe vasospasm involving the pericallosal anterior cerebral artery.

The patient was improving and DVT prophylaxis with heparin at a dose of 5,000 units subcutaneous twice a day was started on the fourth day of admission. Heparin was continued for ten days and then discontinued as the platelets decreased from 255,000 to 55,000 mcL. Heparin Induced Thrombocytopenia (HIT) was highly suspected. Further laboratory investigation showed: Haptoglobin <10 mg/dL, LDH 616 IU/L, D-Dimer 2.40 ug/ml, Absolute Reticulocyte Count 0.140 x10^6/mcl, Fibrinogen 206 mg/dL, Hemoglobin 8.2 g/dL, ADAMTS13 activity assay 50 IU/dL (Normal >70), and Heparin PF4 IgG Ab negative. Eventually, the patient was treated for TTP with seven rounds of plasmapharesis in addition to prednisone. At the time of discharge, the patient's platelet levels normalized to 158,000 mcL.

Discussion: TTP is a rare and life threatening thrombotic microangiopathy characterized by microangiopathic hemolytic anemia (MAHA), severe thrombocytopenia, and organ ischemia linked to disseminated microvascular platelet rich thrombi. It is most frequently acquired via ADAMTS13 autoantibodies but rarely; it is inherited via mutations of ADAMTS13 gene. It is a rare hematologic disease with an average annual prevalence of about 10 cases/million people.

The historical clinical pentad of fever, thrombocytopenia, MAHA, neurological symptoms, and renal insufficiency that used to define TTP appears obsolete as several cohort studies have noted that having all the five symptoms only occurs in less than 10% of patients with acute TTP. The only consistent

abnormalities in TTP include MAHA and thrombocytopenia, which can also occur in other conditions. TTP may mimic other pathology including sepsis, severe preeclampsia, immune thrombocytopenia, HIT, malignant hypertension, DIC, and disseminated cancer. Thus, recognition of TTP can be difficult because of variety of presentations and lack of specific diagnostic criteria. Prompt recognition of TTP is crucial because the disease responds well to plasma-exchange treatment but is associated with a high mortality rate when untreated.

In this patient, the initial diagnosis was HIT as the patient was on Heparin and subsequently developed thrombocytopenia. In conclusion, clinicians should be familiar with the clinical presentations and laboratory abnormalities in the disorders of primary hemostasis as these disorders can be fatal which require early diagnosis and proper treatment that differs depending on the condition.

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"On heparin with thrombocytopenia, must be Heparin Induced Thrombocytopenia," – Think again.

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Case Presentation:

A previously healthy 49-year-old female presented to the emergency department for evaluation of headache of 3 days duration

Day 1:
- MRI showed acute right frontal lobe and intraventricular hematoma with small volume subarachnoid hemorrhage due to an aneurysm from mid right anterior cerebral artery
- Underwent an intracranial aneurysmal coiling of the 9 mm right A1/A2 aneurysm

Day 4:
- Heparin 5000 units/mL subcutaneous injections q12 hours began for DVT prophylaxis

Day 6:
- Patient developed progressive neurological decline with repeat CT head showing slight increase in ventricular size and edema
- Due to the hydrocephalus, a left frontal ventriculostomy was performed and an external ventricular drain was placed
- Angioplasty was performed due to severe vasospasm involving the pericallosal anterior cerebral artery

Day 12:
- EVD removed
- Heparin for DVT prophylaxis was continued

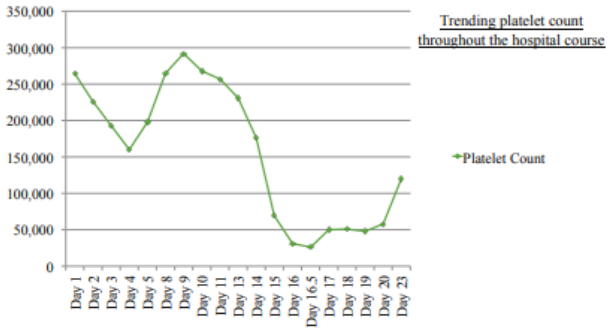
Day 15:
- Patient has been on Heparin for DVT prophylaxis now for 10 days but discontinued as platelets decreased from 255,000 to 69,000 mcL.
- Hematology-Oncology was consulted due to thrombocytopenia

Day 16:
- Platelets decreased to 55,000 mcL. Heparin-PF4 antibody has been ordered
- High concerns for Heparin Induced Thrombocytopenia (HIT)

Days 17-23:
- Platelets trended down to 31,000 mcL
- Completed a total of 7 plasma exchanges
- Prednisone was added as platelets only trended up to 50,000
- Eventually total bilirubin normalized and no signs of further hemolysis indicative of favorable response to plasmapharesis
- Discharge platelet count at 158,000 mcL

Learning objective:

Clinicians should always act urgently if Thrombotic Thrombocytopenia Purpura (TTP) is suspected, but it is also important to keep all causes of hemolytic anemia and thrombocytopenia in mind

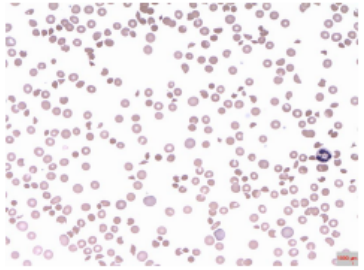


Labs	Results
Haptoglobin	<10 mg/dL
LDH	616 IU/L
D-Dimer	2.40 Ug/ml
Absolute Reticulocyte Count	0.140 x 10^6/mcl
Fibrinogen	206 mg/dL
Hemoglobin, Day 1	8.2 g/dL
PT; INR; PTT	14.4; 1.1; 26.8 seconds
RBC Morphology	Occasional polychromasia, anisocytosis, schistocytes, and ovalocytes
ADAMTS13 activity assay	50 IU/dL (Normal >70)
Heparin PF4 IgG Ab	Negative
Total, Direct, Indirect Bilirubin, Creatinine level	2.5; 0.3; 2.2; 0.8 mg/dL
ANA screen, hepatitis screen	Negative, Negative
4Ts for HIT score	<5%, low probability of HIT

TTP may mimic other pathology including sepsis, severe preeclampsia, immune thrombocytopenia, HIT, malignant hypertension, DIC, liver disease, and disseminated cancer
Thus, recognition of TTP can be difficult because of variety of presentations and lack of specific diagnostic criteria. Prompt recognition of TTP is crucial because the disease responds well to plasma-exchange treatment but is associated with a high mortality rate when untreated

TTP is a rare and life threatening thrombotic microangiopathy characterized by microangiopathic hemolytic anemia (MAHA), severe thrombocytopenia, and organ ischemia linked to disseminated microvascular platelet rich thrombi

- It is most frequently acquired via ADAMTS13 autoantibodies but rarely; it is inherited via mutations of ADAMTS13 gene
- It is a rare hematologic disease with an average annual prevalence of about 10 cases/ million people
- The historical clinical pentad of fever, thrombocytopenia, MAHA, neurological symptoms, and renal insufficiency that used to define TTP appears obsolete as several cohort studies have noted that having all the five symptoms only occurs in less than 10% of patients with acute TTP
- The only consistent abnormalities in TTP include MAHA and thrombocytopenia, which can also occur in other conditions



Conclusion:

In this patient, the initial diagnosis was HIT as the patient was on Heparin and subsequently developed thrombocytopenia. Clinicians should be familiar with the clinical presentations and laboratory abnormalities in the disorders of primary hemostasis as these disorders can be fatal which require early diagnosis and proper treatment that differs depending on the condition.

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Oritavancin in the treatment of Infective Endocarditis in IV Drug Abuser

Authors: Maha Al-Baghdadi MD, Ali Hassoun MD

Oritavancin is a lipoglycopeptide that is FDA-approved for the treatment of acute bacterial skin and skin structure infections. It inhibits cell wall biosynthesis leading to cell death. Prolonged intravenous (IV) antimicrobial therapy in IV drug abusers may be complicated by concern for IV catheter misuse, sometimes requiring prolonged hospitalization. Given the weekly dose pharmacokinetic, oritavancin may have been the preferred agent for long-term use in IV drug abusers.

Hospital course

A 26-year-old female with a longstanding history of IV drug abuse presented with cough and fever. Blood culture revealed MRSA bacteremia and CT scan demonstrated multiple areas of pulmonary congestion consistent with septic pulmonary emboli. She required mechanical ventilation and resuscitation with intravenous fluids and alpha agents. Transesophageal echocardiogram identified a tricuspid valve endocarditis. MRI of spine showed cervical spine discitis. Appropriate antibiotics were initiated with Daptomycin and Rifampicin. She underwent tricuspid valve replacement with epicardial pacing leads insertion to the left ventricle and right atrium, followed by pacemaker insertion because of continued ventricular standstill. She was extubated and made slow but steady progress complicated with acute kidney injury thought to be related to volume contraction, infection related and possibly antibiotic therapy. After antibiotics adjustment, her renal function improved, however it did not return to baseline. She was discharged on the 24th postoperative day. At the time of discharge, her repeat blood cultures remain negative, she was a in paced rhythm and vitally stable. She was treated with Orbactiv at a weekly dose of 1200 mg over 3-hr period for 5 weeks which was well tolerated with resolution of symptoms and no recurrence.

Discussion

IV drug abusers are at higher risk for infections with multidrug-resistant (MDR) pathogens. The concern regarding safe management of PICCs or ports in those patients result in patients requiring prolonged inpatient stays for IV antibiotics. Oritavancin is a semisynthetic lipoglycopeptide antibiotic that is FDA-approved for the treatment of acute bacterial skin and skin structure infections which is the most common indication of Oritavancin used in the previously reported cases. Oritavancin been reported in few publications for treatment of bioprosthetic and native valve endocarditis, methicillin-susceptible *Staphylococcus aureus* (MSSA) soft tissue infection and bacteremia, coagulase-negative staphylococcal bacteremia, entercoccal bacteremia, and recently multidrug-resistant VRE hardware-associated vertebral osteomyelitis. Long acting oritavancin has an emerging use to treat deep-seeded and serious infections in IV drug abusers. It may be equally effective as standard-of-care, offer built-in treatment adherence owed to their extremely long half-life, and secure earlier discharge and significant cost-savings. Oritavancin will contribute to improve the quality of care to all patients, especially IV drug abusers, in hospitals as well as in outpatient settings with continued treatment at the preferred environment of their choice. Prospective clinical trials are warranted.

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Oritavancin in the Treatment of Infective Endocarditis in IV Drug Abusers

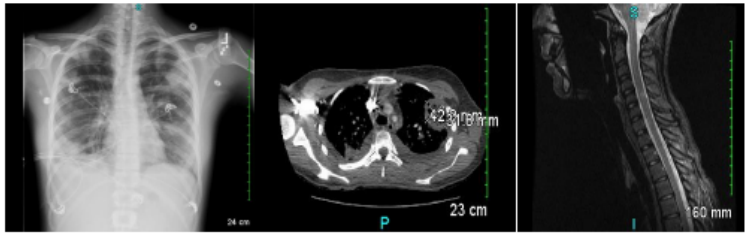
Maha Al-Baghdadi MD[1], Ali Hassoun MD[2] 1.UAB Huntsville Regional Campus 2.Alabama Infectious Disease Center

Introduction

- Oritavancin is a lipoglycopeptide that is FDA-approved for the treatment of acute bacterial skin and skin structure infections.
- It inhibits cell wall biosynthesis leading to cell death.
- Given the weekly dose pharmacokinetic, oritavancin may have been the preferred agent for long-term use in IV drug abusers.

Hospital course

- A 26-year-old female with a longstanding history of IV drug abuse presented with cough and fever.
- Blood culture revealed MRSA bacteremia
- CT scan demonstrated multiple areas of pulmonary congestion consistent with septic pulmonary emboli. She required mechanical ventilation and resuscitation with intravenous fluids and alpha agents.
- Transesophageal echocardiogram identified a tricuspid valve endocarditis.
- MRI of spine showed cervical spine discitis. Appropriate antibiotics were initiated with Daptomycin and Rifampicin.
- She underwent tricuspid valve replacement with pacing to the left ventricle and right atrium, followed by pacemaker insertion.
- She was extubated and made slow but steady progress complicated with acute kidney injury thought to be related to volume contraction, infection related and possibly antibiotic therapy.
- She was discharged on the 24th postoperative day. At the time of discharge, her renal function improved, her repeat blood cultures remain negative, she was a in paced rhythm and vitally stable.
- She was treated with Orbactiv at a weekly dose of 1200 mg over 3-hr period for 5 weeks which was well tolerated with resolution of symptoms and no recurrence.



Multiple septic pulmonary emboli Cervical spine discitis

Discussion

- IV drug abusers are at higher risk for infections with multidrug-resistant (MDR) pathogens. The concern regarding safe management of PICCs or ports in those patients result in patients requiring prolonged inpatient stays for IV antibiotics.
- Oritavancin is a semisynthetic lipoglycopeptide antibiotic that is FDA-approved for the treatment of acute bacterial skin and skin structure infections which is the most common indication of Oritavancin used in the previously reported cases.
- Oritavancin been reported in few cases for treatment of bioprosthetic/native valve endocarditis, methicillin-susceptible ***Staphylococcus aureus*** (MSSA) soft tissue infection and bacteremia, coagulase-negative staphylococcal bacteremia, entercoccal bacteremia, and recently multidrug-resistant VRE hardware-associated vertebral osteomyelitis.
- Long acting oritavancin has an emerging use to treat deep-seeded and serious infections in IV drug abusers.
- It is equally effective as standard-of-care, offer built-in treatment adherence owed to their extremely long half-life, and secure earlier discharge and significant cost-savings.

Conclusion

- Oritavancin will contribute to improve the quality of care to all patients, especially IV drug abusers, in hospitals as well as in outpatient settings with continued treatment at the preferred environment of their choice.
- Prospective clinical trials are warranted.

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Persistent Reflux Symptoms despite Aggressive Treatment - When Medication Fails

Nicholas Rivers B.S.[1], Mark Allen M.S.[1], Daniel Boyett M.D.[2], Roger Smalligan M.D.[3], John E. Fanning, M.D.[4]

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Category: Clinical Vignette
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Learning Objectives:

To discuss the presentation and types of diaphragmatic hernia.

Case:

A 43yo woman with a PMH of GERD and morbid obesity presented with two years of post-prandial chest and epigastric abdominal pain that was worse when supine. She had dysphagia and a sensation that food was trapped in her chest. Her symptoms did not improve with antacids, PPIs, and H2 antagonists. Upper GI endoscopy one-year prior revealed a medium-sized paraoesophageal hernia, confirmed on CT, and esophageal manometry was normal. Because of another year of persistent symptoms, a repeat CT and upper GI series in 2019 showed a large hiatal hernia with stomach in the chest. She was referred to a general surgeon for repair.

Intraoperatively, the patient had a large defect in the anteromedial portion of the left hemidiaphragm that appeared to be congenital in nature. Two-thirds of the distal stomach had herniated into the chest. There was also a small paraesophageal hiatal hernia. The patient was successfully repaired with resolution of her symptoms.

Discussion:

Gastroesophageal reflux disease is common in adults. Prevalence estimates range from 18-27% in North America. If no alarm symptoms are present, treatment routinely includes dietary changes, elevated sleep position, proton pump inhibitors, and/or H2 inhibitors. Failure to respond should include a stepwise work-up for causes of reflux symptoms.

Herniation of the stomach through the diaphragm can be chronic, traumatic, or congenital in nature. The most common type is the hiatal hernia; a chronic condition that affects adults. Risk factors include obesity and previous foregut surgery. Hiatal hernias can be sliding or paraoesophageal. They can be asymptomatic or present with GERD, epigastric pain, dysphagia and odynophagia. Fundoplication is indicated if a large symptomatic hernia is present. Traumatic rupture of the diaphragm due to blunt force injury should be urgently repaired.

Congenital diaphragmatic hernias (CDH) are due to embryologic defects in the diaphragm. Over 95% of CDH are due to defects in the left posterolateral diaphragm (Bochdalek hernia) and present at birth. These CDH have a high mortality rate and poor prognosis. Approximately 2% of CDH are the Morgagni type which are parasternal and caused by a defect in anteromedial diaphragm. These defects are extremely rare and can present at birth, but symptomatic adult cases of Morgagni hernia have been reported in the literature. Our patient denied any recent or lifetime trauma and her disease course was chronic. Her defect was anteromedial in location and distinct from the esophageal hiatus, suggesting the diagnosis of a Morgagni congenital diaphragmatic hernia. This case points out the importance of careful, stepwise and systematic work-up in patients with unresponsive reflux symptoms.

Persistent Reflux Symptoms Despite Aggressive Treatment - When Medication Fails

Nicholas Rivers, Mark Allen, Daniel Boyett MD, Roger Smalligan MD, John E Fanning MD
UAB School of Medicine, Huntsville Campus, Huntsville, AL

Learning Objectives

- Describe the delayed presentation of an adult with a congenital diaphragmatic hernia
- Discuss the differential and work-up for refractory reflux symptoms
- Define the types of diaphragmatic hernias

Case Presentation

A 43yo woman with a PMH of **GERD** and morbid obesity presented with two years of **post-prandial chest and epigastric abdominal pain** that was worse when supine. She had **dysphagia,** sensations that **food was trapped** in her chest and **occasional shortness of breath**. Her symptoms **did not improve with antacids, PPIs**, and H2 antagonists. Upper GI endoscopy one-year prior revealed a medium-sized paraoesophageal hernia, confirmed on CT, and esophageal manometry was normal. Because of **another year of persistent symptoms**, a repeat CT (Figure 1) and upper GI series in 2019 showed a large hiatal hernia with stomach in the chest. She was referred to a general surgeon for repair.

Intraoperatively, the patient had a large defect in the posterolateral portion of the left hemidiaphragm (Figure 2A) that appeared to be congenital in nature. Two-thirds of the distal stomach had herniated into the chest. There was also a small paraesophageal hiatal hernia. The hernia was successfully repaired with resolution of her symptoms.

Background

Gastroesophageal reflux disease (GERD) is common in adults, with estimates of prevalence ranging from 18-27% in North America[1]. In the absence of alarm symptoms, treatment routinely includes dietary changes, elevated sleep position, proton pump inhibitors, and/or H2 inhibitors. Failure to respond should include a stepwise work-up for causes of refractory reflux symptoms.

Differential Dx:
- Achalasia
- Hiatal Hernia
- Eosinophilic esophagitis
- Infectious esophagitis
- Pill esophagitis
- Gastroparesis
- Esophageal stricture or cancer

Workup for suspected hiatal hernia:
- Barium Swallow (most sensitive)
- Esophagogastroduodenoscopy
- High Resolution Esophageal Manometry

Discussion

Herniation of the stomach through the diaphragm can be chronic, traumatic, or congenital in nature. The most common type is the hiatal hernia; a chronic condition that affects adults. Risk factors include obesity and previous foregut surgery. Hiatal hernias can be sliding or paraoesophageal. They can be asymptomatic or present with GERD, epigastric pain, dysphagia and odynophagia. Fundoplication is indicated if a large symptomatic hernia is present. Traumatic rupture of the diaphragm due to blunt force injury should be urgently repaired.

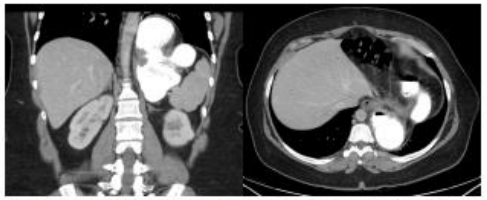


Figure 1 Pre-operative abdominal CT scan, showing a presumed type IV paraesophageal hernia.

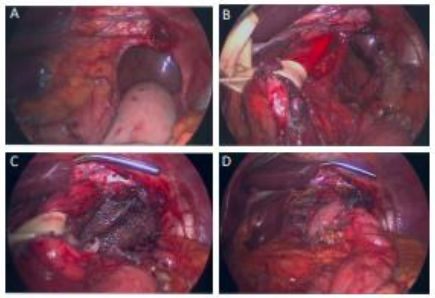
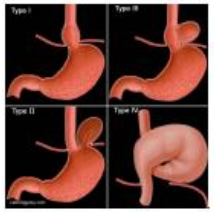
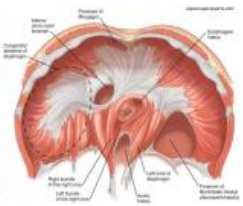


Figure 2 Intraoperative photos from laparoscopic camera. **A:** initial view of diaphragmatic defect after partially reducing the stomach. **B:** defect after esophagus and stomach were mobilized. **C:** mesh closure of defect. **D:** abdomen post repair and nisin fundoplication

Discussion





Congenital diaphragmatic hernias (CDH) are due to embryologic defects in the diaphragm. Over 95% of CDH are due to flaws in the posterolateral diaphragm (Bochdalek hernia), while 2% are parasternal or anteromedial in location (Morgagni hernia)[2]. These CDH typically present at birth with a high mortality rate and a poor prognosis. The remainder of CDH cases are characterized by central tendon defect or complete absence of the diaphragm. These defects are extremely rare and can result in lung hypoplasia and fetal demise. There have been approximately 100-150 cases of adult Bochdalek hernias reported in the literature worldwide[3]. While they are often asymptomatic incidental findings in adults, Bochdalek hernias can also cause GERD, bowel incarceration, pulmonary disease, and recurrent chest infections[4,5].

Our patient denied any recent or lifetime trauma and her disease course was chronic. Her defect was posterolateral in location and distinct from the esophageal hiatus, suggesting the diagnosis of a Bochdalek congenital diaphragmatic hernia. This case points out the importance of careful, stepwise and systematic work-up in patients with refractory reflux symptoms.

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Clinical Vignette

Protect Your Patient from Personal Bias, A Real Headache

Learning objectives:

1. How to recognize personal bias.
2. How to base diagnosis on establish guidelines to protect your patients from your bias.

Case Presentation:

A 41-year-old African American female with history of opioid-dependent chronic pain, heart failure, obesity, hypertension, COPD, OSA, fibromyalgia, lupus, chronic migraines, chronic menometrorrhagia due to uterine fibroids, and depression with anxiety requiring daily benzodiazepines presented to ED complaining of a pounding headache with photosensitivity and nausea. Vitals revealed blood pressure of 223/152. Head imaging was negative. Was started on Nicardipine drip. She had this problem on many occasions in the past; she runs out of pain medications, then develops intractable headache causing hypertension not responsive to antihypertensives. She insisted all she needed was her pain medications. PDMP report showed that in the past year she had filled 103 prescription for controlled substances by 23 different prescribers, using 9 different pharmacies in 3 different states. Review of hospital records revealed a history of multiple monthly hospital visits, usually for uncontrolled pain from various etiologies including dental caries, fibromyalgia, uterine fibroids, lupus, lower back pain, chest pain and headache. She denied vision changes/loss, tinnitus, back pain or retrobulbar pain. Neurological exam was intact. No papilledema. Hypertension was quickly controlled and was started on oral antihypertensives, but headache persisted. Neurology was consulted, and after thorough evaluation, nothing was added to the regimen. Plans for discharge were discussed during rounds with diagnosis of opioid seeking behavior vs analgesic overuse headache. On planned day of discharge, with very low suspicion, we decided to order lumbar puncture. Opening pressure was 42 confirming diagnosis of Increased Intracranial Pressure. Acetazolamide was started and the headache improved.

Impact/Discussion:

Caring for patients that are addicted to prescription or illicit drugs can be very difficult. Here,

a review of PDMP revealed the patient was a ‘drug seeker.’ In addition, despite several serious comorbidities, she was only concerned about her opioid analgesics to the point that she was dishonest about symptoms and medication adherence. Her headache description matched migraines and physical exam findings did not suggest IHH. A review of ‘headache workup’ in UpToDate led us to offer the

patient a lumbar tap to measure opening pressure. We had very little suspicion for IHH, but it was the only other thing we could offer. We even discussed during rounds whether it was worth putting her through a spinal tap and the possible complications given such a low suspicion. By relying on recommendations and placing our opinion aside, we were able to reach a diagnosis and initiate treatment.

Conclusion:

Overuse and abuse of controlled substances is a very common problem. It is important to be vigilant so as not to contribute to their use/abuse problem, but also important to provide the best care possible, including treatment of other conditions. The need of these patients for their 'drug' can make it very difficult to tease out important parts of the history. Though clinical suspicion is very important in many cases, relying on recommendations and guidelines may help deliver appropriate care when dealing with these difficult cases.

This abstract was chosen for oral presentation on Research Day.

Pulmonary Arterial Hypertension (PAH) Caused by Methamphetamine and Cocaine Abuse

Authors: Syed Shabee Hassan[1], MD, Alan Baggett[2], MD, FACP

Learning objectives:

- Assess and treat a patient with Pulmonary Hypertension
- Recognize illicit drugs as an important cause of Pulmonary Arterial Hypertension

Case: 41-year-old Caucasian female presented to the ED with progressively worsening dyspnea on exertion which began 2 months ago and 1-day history of chest tightness. On arrival, she was tachycardic, and hypotensive. She had no pertinent PMH. She was a 20 pack-year active smoker, active methamphetamine and cocaine smoker. Physical exam was significant for parasternal heave and JVP of 10 cm. Labs were remarkable for negative troponin, urine screen positive for methamphetamines and cocaine. EKG showed sinus tachycardia, right strain pattern. CT Chest ruled out pulmonary embolism, showed enlarged main pulmonary artery as well as right ventricle and atrium, findings consistent with PAH. Transesophageal echo showed enlarged right ventricle and RV hypokinesis. She then underwent a right heart catheterization to confirm right-sided pressures. The PCWP was 5 mmHg (6 - 16), mean PA pressure 47 mmHg (normal < 20 mmHg), pulmonary vascular resistance 16 Wood units (1.9 – 3.1), and the cardiac index was 1.8 (2.8 – 4.2). These findings were consistent with severe pulmonary hypertension and reduced cardiac index, as well as normal right- and left- filling pressures. She was diagnosed with PH Group 1, admitted to the CCU for vasodilator therapies, and discharged home on Sildenafil and Ambrisentan dual-therapy with plans for close follow-up.

Discussion: PH is classified under 5 groups of causes: PAH, left heart disease, lung disease/hypoxia, pulmonary artery obstruction and idiopathic PH respectively. It is important to distinguish the cause of PH, as treatments and prognosis varies greatly. Assessment involves CT chest which in our patient ruled out ILD (Group 3), pulmonary embolism (Group 4) and TTE which ruled out Group 2. RHC confirmed a low PCWP, as well as severely elevated pulmonary artery pressure, which was consistent with Group 1.

PAH is a rare disease that affects 5 – 52 people in a million, with a female to male ratio of 1.7 – 4.8:1. After idiopathic and heritable causes, drugs and toxins are the most important etiology. Cocaine and methamphetamine use have both been associated with a high risk of PAH, and deserve respect as an important cause in the right clinical setting. This case of a young, otherwise healthy, female highlights the steps needed to diagnose PAH, as well as emphasizes the need for a thorough social history to recognize rare causes of this already under-recognized condition.

Conclusion: PH should always be considered in young patients with new or sub-acute dyspnea on exertion. While connective tissue disease and heritable causes are significant, illicit drugs continue to be

an important cause in PAH. RHC is an essential tool to help diagnose and guide therapy, which includes PDE-5 inhibitors and ERAs.

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Pulmonary Arterial Hypertension (PAH) Caused by Methamphetamine and Cocaine Use

Syed Shabee Hassan, MD, Alan Baggett, MD, FACP

UAB Huntsville Regional Campus, Department of Internal Medicine, Huntsville, AL, USA.

Learning objectives

- Assess and treat a patient with pulmonary hypertension (PH)
- Recognize illicit drug use as an important cause of pulmonary arterial hypertension

Case

A 41-year-old Caucasian woman presented to the Emergency with progressively worsening dyspnea on exertion which began 2 months ago and 1-day history of chest tightness.

Vitals: BP 108/66 mmHg, Heart rate 125/min, RR 16/min.

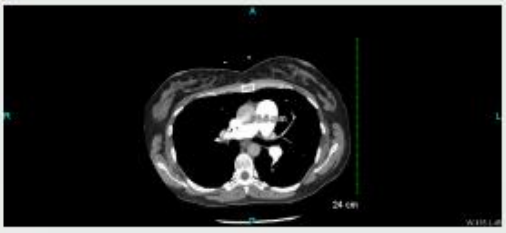
Past Medical History: None.

Social History: 20 pack-year active smoker, active methamphetamine and cocaine smoker.

Physical exam: Parasternal heave and JVP of 10 cm.

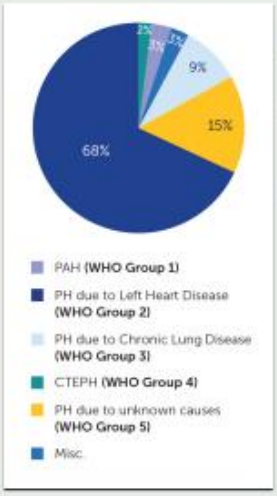
Labs/Diagnostics: Troponin negative, urine screen positive for methamphetamines and cocaine. EKG showed sinus tachycardia, right strain pattern. CT Chest ruled out pulmonary embolism, showed enlarged main pulmonary artery as well as right ventricle and atrium, findings consistent with PAH. Transesophageal echo showed enlarged right ventricle and RV hypokinesis.

She then underwent a right heart catheterization to confirm right-sided pressures. The PCWP was 5 mmHg (6 - 16), mean PA pressure 47 mmHg (normal < 20 mmHg), pulmonary vascular resistance 16 Wood units (1.9 – 3.1), and the cardiac index was 1.8 (2.8 – 4.2). These findings were consistent with severe pulmonary hypertension and reduced cardiac index, as well as normal right- and left- filling pressures. She was diagnosed with PH Group 1, admitted to the CCU for vasodilator therapies, and discharged home on Sildenafil and Ambrisentan dual-therapy with plans for close follow-up.



Discussion

- PH is classified under 5 groups of causes: PAH, left heart disease, lung disease/hypoxia, pulmonary artery obstruction and idiopathic PH respectively.
- It is important to distinguish the cause of PH, as treatments and prognosis varies greatly.
- Assessment involves CT chest which in our patient ruled out interstitial lung disease (Group 3), pulmonary embolism (Group 4) and transthoracic echo which ruled out Group 2. Right heart cath confirmed a low PCWP, as well as severely elevated pulmonary artery pressure, which was consistent with Group 1.
- PAH is a rare disease that affects 5 – 52 people in a million, with a female to male ratio of 1.7 – 4.8:1.
- After idiopathic and heritable causes, drugs and toxins are the most important etiology. Cocaine and methamphetamine use have both been associated with a high risk of PAH, and deserve respect as an important cause in the right clinical setting.
- This case of a young, otherwise healthy, female highlights the steps needed to diagnose PAH, as well as emphasizes the need for a thorough social history to recognize rare causes of this already under-recognized condition.



Conclusion

- PH should always be considered in young patients with new or sub-acute dyspnea on exertion.
- While connective tissue disease and heritable causes are significant, illicit drugs continue to be an important cause in PAH. RHC is an essential tool to help diagnose and guide therapy, which includes PDE-5 inhibitors and ERAs.

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Rare bacteria associated with spontaneous bacterial peritonitis

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Submission category: Clinical vignette

Learning objectives:

Rare bacteria associated with spontaneous bacterial peritonitis (SBP).

Differentiating spontaneous from secondary causes of peritonitis.

Case:
A 47-year-old African American female with recent diagnosis of decompensated alcoholic liver cirrhosis was admitted with altered mental status. Patient was lethargic upon arrival with distended abdomen. Vital signs were significant for heart rate of 104 per minute and respiratory rate of 30 per minute. Labs were significant for blood glucose of 40 mg/dL, lactate of 17 mmol/L, white cell count of 18.53 $x10^3$/mcL, creatinine of 1.8 mg/dL, total bilirubin of 13.1 mg/dL, albumin of 2.8 g/dL, international normalized ratio of 3.9 and ammonia of 119 mcmol/L. Ascitic fluid analysis revealed total nucleated count of 49,300 cells/mm^3 with 90% neutrophils, total protein of 1.8 g/dL, glucose of less than 2 mg/dL and lactate dehydrogenase (LDH) of 474. Patient was started on cefepime and albumin for possible SBP.

Computed tomography scan of the abdomen did not show evidence of perforation or abscess. She was treated with sodium bicarbonate and dextrose for refractory lactic acidosis and hypoglycemia, likely due to liver failure. Patient also received lactulose enemas for hepatic encephalopathy. Model for End stage Liver-sodium score worsened to 37 from 23 two weeks ago. Ascitic fluid culture and two sets of blood cultures grew *Achromobacter xylosidans* which showed intermediate sensitivity to cefepime and sensitivity to Zosyn and Levaquin. She was not deemed to be a candidate for liver transplantation given her last drink was 3 months ago and the presence of bacteremia. Patient's clinical status worsened requiring pressor support at which point family decided to transition to comfort measures.

Discussion:

Spontaneous bacterial peritonitis (SBP) is defined as an ascitic fluid infection without an evident intra-abdominal surgically treatable source. SBP should be suspected in patients with ascites due to advanced cirrhosis who develop symptoms such as fever, abdominal pain/tenderness, and altered mental status. The diagnosis is established by a positive ascitic fluid bacterial culture and an elevated ascitic fluid absolute polymorphonuclear leukocyte count (≥250 cells/mm^3).

Secondary bacterial peritonitis is suspected if at least two of the three ascitic fluid findings are present which include total protein >1 g/dL, glucose <50 mg/dL and LDH more than the upper limit of normal for serum. Cultures showing a polymicrobial infection suggest gut perforation.

Most cases of SBP are due to gut bacteria such as E. coli and Klebsiella, though Streptococcal and Staphylococcal infections can also occur. *Achromobacter xylosidans* is a rare cause of peritonitis and bacteremia. It is an aerobic, gram negative rod widely distributed in the environment that mainly causes healthcare-associated infection. Most cases in the literature are described in patients with some form of immunosuppression, usually hematological malignancies, and significant association with prior antibiotic therapy within thirty days. Studies showed primary bacteremia as the common clinical presentation with catheter related infection as the main source of bacteremia. Other reported sites of infection include meningitis, abscesses, osteomyelitis, and pneumonia. Treatment is challenging due to the high prevalence of multidrug resistance.

Rare bacteria associated with spontaneous bacterial peritonitis (SBP)

THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

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LEARNING OBJECTIVES:

- Rare bacteria associated with SBP.
- Differentiating SBP from secondary bacterial peritonitis.

CASE:

- A 47-year-old female with recently diagnosed decompensated alcoholic cirrhosis was admitted with altered mental status.
- Fluid analysis consistent with portal hypertension during the previous admission 2 weeks prior, but no evidence of infection.
- During the present admission, patient was lethargic upon arrival with distended abdomen.
- Vital signs were significant for heart rate of 104 and respiratory rate of 31.
- Comparison of her labs and fluid studies is as follows:

Ascitic fluid	Current admission	Two weeks ago
TNC	49,300 cells/mm^3	60 cells/mm^3
Total protein	1.8 g/dL	1.5 g/dL
Glucose	< 2 mg/dL	119 mg/dL
LDH	474	47
Fluid albumin	0.7 g/dL	0.7 g/dL

Labs	Current admission	Two weeks ago
Lactate	17 mmol/L	2.1 mmol/L
WBC count	18.53 x 10^3/mcL	11.54 x 10^3/mcL
Sodium	134 mmol/L	137 mmol/L
Blood glucose	40 mg/dL	182 mg/dL
Creatinine	1.8 mg/dL	0.5 mg/dL
Total bilirubin	13.1 mg/dL	5.1 mg/dL
Albumin	2.8 g/dL	2.6 g/dL
INR	3.9	2.6

- Patient received Cefepime and albumin for possible SBP.
- CT of the abdomen showed no evidence of perforation or abscess.
- MELD-Na score worsened to 37 from 23 two weeks ago.
- Ascitic fluid culture and two sets of blood cultures grew *Achromobacter xylosidans* which was sensitive to Zosyn.
- Her clinical status continued to worsen at which point family decided to transition to comfort measures on day 3.

DISCUSSION:

- SBP is defined as an ascitic fluid infection without an evident intra-abdominal surgically treatable source.
- Suspect in cirrhotic patients with ascites who develop fever, abdominal pain/tenderness, and altered mental status.
- The diagnosis is established by elevated ascitic fluid PMN count (≥250 cells/mm^3) and positive bacterial culture.

Ascitic fluid findings	Spontaneous bacterial peritonitis	Secondary bacterial peritonitis
Total protein	<1 g/dL	>1 g/dL
Glucose	>50 mg/dL	<50 mg/dL
LDH	Lower than serum LDH	More than the upper limit of normal for serum.

Discussion continued..

- Our patient met all the criteria for secondary peritonitis, however, culture was monomicrobial.
- It is possible that infection could have been introduced by paracentesis two weeks prior.
- Most cases of SBP are due to gut bacteria such as E. coli.
- *Achromobacter xylosidans*[1] is a rare cause of peritonitis and bacteremia.
- It is an aerobic, gram negative rod widely distributed in the environment.
- Most cases in the literature are described in patients with immunosuppression and prior antibiotic therapy within 30 days.
- Primary bacteremia was the most common presentation[2].
- Other reported sites of infection include meningitis, osteomyelitis, and pneumonia.
- Treatment is challenging due to the prevalence of multidrug resistance.

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Rare Complication of a Common Problem: Bickerstaff Brainstem Encephalitis

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Tejanand Mulpur, Neurology, Huntsville Hospital

Abstract:

Bickerstaff brainstem encephalitis (BBE) is a rare post-infectious disease that effects the central nervous system. The underlying disease mechanism is unknown; however, the disease process is thought to be related to an autoimmune mechanism triggered by a previous infection. It is thought to exist on the same spectrum as the Miller-Fischer variant of Guillain-Barre syndrome. The symptoms can be progressive varied, making the diagnosis and appropriate treatment challenging.

A 62 year old female came to the ED with a headache and diplopia. The headache was bilateral and persistent for several days. On admission to the ED, she rated it as an 8/10, and no relief with ibuprofen. She denies nausea, vomiting, photophobia, or phonophobia. She endorsed numbness and tingling in her extremities that was progressively getting worse, moving up distally on both the upper and lower extremity, reaching her mid-forearm and knees in a symmetrical pattern. Her tongue felt “thick” and she was experiencing gait instability.

The patient’s relevant medical history includes a mild viral upper respiratory infection in the past week. She had a history of migraines but has not had an episode in several years. In the emergency department the patient was given a dose of steroids. However, her headache continued to get worse. She developed new onset of ptosis, dysarthria, and dysphagia. At this point, her symptoms no longer fit with her initial diagnosis of a complex migraine and was admitted. Neurological exam showed 4/5 strength in UE and LE, right CN III palsy and left CN VI. Reflexes were 2/2 throughout. An LP showed normal opening pressure, pleocytosis (total nucleated cells = 15), no neutrophil predominance, normal protein, normal glucose. On CBC, patient had leukocytosis (WBC 16.15) with increased abs neutrophils (13.9). IVIG was started prior to LP. Patient was transferred to the Neurology ICU due to risk of airway compromise.

On day 3 of receiving IVIG, with improvement to her ptosis and strength, patient developed afib with RVR. Given IV amiodarone, chemically converted, which was changed to Toprol XL after stable rhythm. Chemical stress test showed no evidence of ischemic changes. Echo showed normal findings except mild mitral valve regurgitation and aortic valve regurgitation. She was only continued with home ASA and prescribed a high dose statin (as CHADSVASc = 0). Patient’s dysphagia resolved on day 4 of treatment. On discharge to a rehab facility, she remained ambulating with a walker and continued to have lateral rectus palsy. Six weeks after admission, all of the patient’s symptoms completely resolved.

This case illustrates challenges in identifying post-infectious neurological diseases early. Bickerstaff encephalitis can range from more mild symptoms like ophthalmoplegia and gait disturbance to severe forms, with additional paralysis and altered consciousness. The case demonstrates awareness for the potential sequelae on the autonomic system in GBS variants. It highlights the importance of clinical history and physical in the diagnosis, as MRI changes and positive antibodies for anti-GQ1b IgG may not be present in all cases.

Rare Complication of a Common Problem: Bickerstaff Brainstem Encephalitis

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Learning Objectives

1. Identify signs of post-infectious neurological impairment
2. Determine when treatment should be initiated
3. Assess appropriate placement to an ICU setting

Hospital Course

A 62 year old female came to the ED with a headache and diplopia. The headache was bilateral and persistent for several days. On admission to the ED, she rated it as an 8/10, and no relief with ibuprofen. She denies nausea, vomiting, photophobia, or phonophobia. She endorsed numbness and tingling in her extremities that was progressively getting worse, moving up distally on both the upper and lower extremity, reaching her mid-forearm and knees in a symmetrical pattern. Her tongue felt "thick" and she was experiencing gait instability. Otherwise, the patient had no abnormal vital signs.

The patient had a mild viral upper respiratory infection in the past week. She had a history of migraines but has not had an episode in several years. In the emergency department the patient was given a dose of steroids. Initially, she was thought to have a complex migraine.

However, her headache continued to get worse. She developed new onset of ptosis, dysarthria, and dysphagia. Neurological exam showed 4/5 strength in UE and LE, palsy in right CN III and left CN VI. Reflexes were 2/2 throughout. Patient had leukocytosis (WBC 16.15) with increased abs neutrophils (13.9) but remained afebrile. Her diagnosis no longer fit a complex migraine and the patient was admitted. A lumbar puncture showed normal opening pressure, pleocytosis (total nucleated cells = 15), no neutrophil predominance, normal protein, normal glucose. IVIG was started prior to LP. Patient was transferred to the Neurology ICU due to risk of airway compromise.

On day 3 of receiving IVIG, with improvement to her ptosis and strength, patient developed atrial fibrillation with RVR. She was given IV amiodarone, chemically converted, which was changed to Toprol XL after stable rhythm. Chemical stress test showed no evidence of ischemic changes. Echo showed only mild mitral valve regurgitation and aortic valve regurgitation. Patient's dysphagia resolved on day 4 of treatment. On discharge to a rehab facility, she remained ambulating with a walker and continued to have lateral rectus palsy. Six weeks after admission, all of the patient's symptoms completely resolved.

Summary of Symptoms:	
Antecedent viral infection Leukocytosis, afebrile Increased TNC on LP, normal protein, Negative blood and CSF cultures	Basilar headache, ophthalmoplegia, ptosis, dysarthria, normal reflexes Improvement with IVIG

Discussion

- Bickerstaff brainstem encephalitis (BBE) is a rare post-infectious disease that effects the central nervous system. The incidence in the United States is not known, however, it is thought to be less than in Japan where the incidence is 0.78 per 100,000[3]
- The underlying disease mechanism is unknown; however, the disease process is thought to be related to an autoimmune mechanism triggered by a previous infection.
- It is thought to exist on the same spectrum as the Miller-Fischer variant of Guillain-Barre syndrome. Both diseases are associated with anti-GQ1b antibodies The symptoms can be progressive varied, making the diagnosis and appropriate treatment challenging.

Anti-GQ1b Syndrome			
BBE	MFS	Acute ophthalmoparesis	GBS with opthalmaplegia

- CSF albuminocytological disassociation supporting the clinical diagnosis; however, across the spectrum of GBS, MFS, and BBE, up to 25% can have normal CSF studies.[1]
- Serum Anti-Gq1b antibodies may further confirm the diagnosis, however, they are not required. A sample of 500 clinically defined cases of BBE only showed seropositivity in 62% of the cases.[4] There are additional antibodies that may be involved in myelin destruction causing related symptoms.
- EEG findings are not specific enough to be of true diagnostic value and may only show slow-range activity and may only be of utility early in the disease process with predominant altered sensorium if the it is unclear if the symptoms are originating from the peripheral nervous system or the central nervous system.
- MRI findings can be variable and do not correlate with the severity of illness. Associated findings include hypointense foci on T-1-weighted images and hyperintense foci on T2-weighted images in the brainstem, thalamic, or basal ganglia lesions.
- IVIG as early as there is clinical suspicion, often before confirmation with an LP, is the treatment of choice[2]. Plasmapheresis is also a treatment option.[5] In severe cases, Rituximab has been used[6]
- Several types of autonomic dysfunction, particularly of the cardiac system, have been described with GBS. However, there has been less involvement of the autonomic nervous system in Miller-Fischer Syndrome and Bickerstaff-Brainstem Encephalitis[8]. The patient in the case presented had an episode of atrial fibrillation on day 3 of receiving IVIG, approximately 5 days after initial symptoms presented. It is unlikely the arrythmia was a sequelae of BBE.

Conclusion

This case illustrates challenges in identifying post-infectious neurological diseases early. Bickerstaff encephalitis can range from more mild symptoms like ophthalmoplegia and gait disturbance to severe forms, with additional paralysis and altered consciousness. The case demonstrates awareness for the potential sequelae on the autonomic system in GBS variants. It highlights the importance of clinical history and physical in the diagnosis, as MRI changes, expected CSF findings, and positive antibodies for anti-GQ1b IgG may not be present in all cases. Treatment with IVIG for suspicion of GBS or variants should not be delayed in lieu of finding a diagnosis.

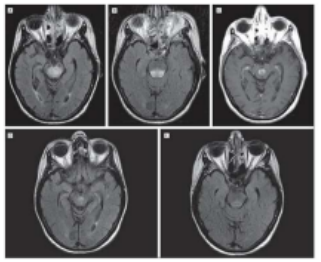

Figure 1. Representative image of BBE in an 81 year old patient of MRI with FLAIR (A-B) and T1 (C). Treatment with steroids resolved symptoms and lesions (D,E).
Source: *Roos RP, Soliven B, Goldenberg F, Badruddin A, Baron JM. An elderly patient with Bickerstaff brainstem encephalitis and transient episodes of brainstem dysfunction. Arch Neurol. 2008;65(6):821–824. doi:10.1001/archneur.65.6.821*

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Rare Genetic disorder Greig Cephalopolysyndactyly Syndrome (GCPS) : a case report .

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Submission Category: Clinical Vignette

Learning Objectives

- Clinical manifestations of Greig Cephalopolysyndactyly Syndrome (GCPS)
- Genetic cause of GCPS and inheritance pattern
- How to diagnose GCPS
- Treatment options for GCPS

Case Presentation

This is a 31-year-old Caucasian male who presents to the Family Medicine Center to establish care. He states that he was diagnosed with Greig Cephalopolysyndactyly Syndrome at an early age and has had seizures and migraines associated with the disorder. His seizures have been well controlled on antiepileptics, and his last seizure was approximately 10 years ago. He has had more than 60 orthopedic surgeries for syndactyly, but he is now able to ambulate with the assistance of ankle braces.

Discussion

Greig Cephalopolysyndactyly Syndrome (GCPS) is a rare genetic syndrome associated with polydactyly, syndactyly, and craniofacial abnormalities. Individuals with this syndrome typically have one of more extra fingers or toes or abnormally wide thumb or great toe. Many individuals with GCPS have permanently flexed fingers. It is also associated with wide set eyes, a broad nasal bridge, macrocephaly, and/or a high prominent forehead. Facial abnormalities are thought to be due to abnormally wide sutures. GCPS is rarely (less than 10%) associated with serious medical problems including intellectual disability, developmental delay, hydrocephalus or seizure disorder. It is an autosomal dominant disorder that affects males and females equally. There are over 200 cases reported in the literature. GCPS is caused by a mutation in the GL13 gene which is involved in gene expression and disrupts early development. A full deletion of the GL13 gene is thought to cause the severe form of the disease associated with intellectual disability and hydrocephalus. Diagnosis is based on clinical findings, typically at birth. CT and X-ray can be used to determine the extent of bone fusion. It is not uncommon to see advanced bone age on radiographic images. Genetic testing

is confirmatory, and individuals suspected to have GCPS based on clinical findings and imaging, should undergo genetic testing. Treatment is cosmetic or symptomatic only, such as surgery to correct polydactyly and syndactyly.

Rare Genetic Disorder Greig Cephalopolysyndactyly Syndrome (GCPS) : A Case Report

Amanda Stisher, MD, Hunter French, MD, Shivani Malhotra, MD
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Introduction

This is a 31-year-old Caucasian male who presents to the Family Medicine Center to establish care. He states that he was diagnosed with Greig Cephalopolysyndactyly Syndrome at an early age and has had seizures and migraines associated with the disorder. His seizures have been well controlled on antiepileptics, and his last seizure was approximately 10 years ago. He has had more than 60 orthopedic surgeries for syndactyly, but he is now able to ambulate with the assistance of ankle braces.

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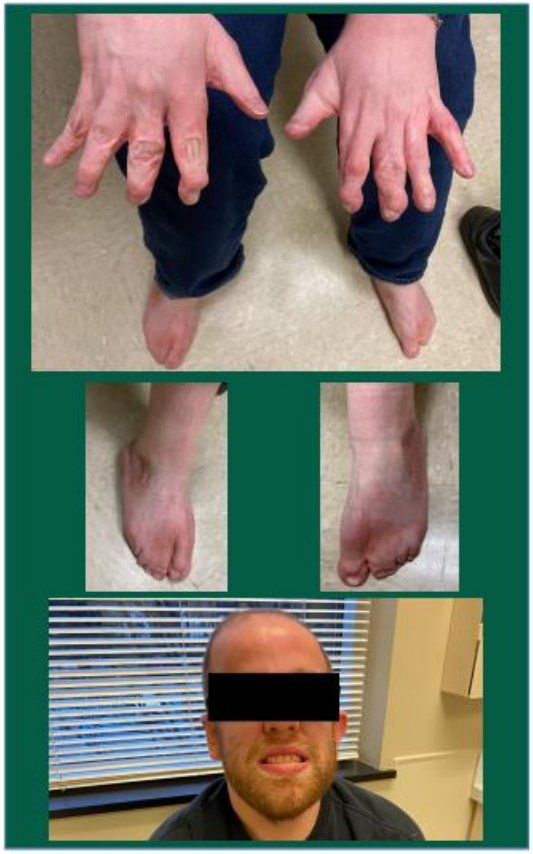
Photos



Discussion continued

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THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

2020 Huntsville Regional Campus Research Day

Kevin Narang, Dr. Katie Woods, Dr. Gayatri Venkatraman

"Severe Folate and Vitamin B12 deficiency in the setting of Anti-Epileptic Drug use"

58-year-old male with a history of traumatic brain injury (TBI) causing recurrent seizures that have been well controlled since 2013 on Tegretol, Keppra, and Phenytoin. Unfortunately, his health has worsened in recent years due to recurrent falls. Subsequent labs demonstrated Folate and Vitamin B12 deficiencies. Other medications include Metformin, Lipitor, and Lisinopril. Metformin is known to cause a slight decrease in Vitamin B12 levels by reducing intestinal absorption but is rarely enough to be symptomatic. The patient was started on oral supplementation with Folic Acid and B12. He followed up one year later with similar lab results and he was falling more frequently. His folate regimen was increased, and he began receiving daily B12 intramuscular injections for one week and then transitioned to weekly injections. The following summer labs were rechecked and both Folate and B12 were at goal. His symptoms improved with fewer falls and improved sleep. Folate and Vitamin B12 are key co-factors in neurologic and hematopoietic function. Both are found in a well-balanced diet but can be supplemented as well. There are several causes of deficiencies in either and an overlap between their clinical presentation. Folate deficiency is commonly seen in long-term anti-epileptic drug use. However, a vitamin deficiency was not initially considered in this patient and his worsening neurologic symptoms were presumed to be a manifestation of his recurrent seizures and TBI. This case highlights the importance of the medication management in patients with chronic conditions.

Folate and Vitamin B12 Deficiency in the Setting of Anti-Epileptic Drug Use

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Learning Objectives

- **Discuss the various etiologies, clinical findings, and complications of Folate and Vitamin B12 deficiency.**
- **Discuss the management of patients with a history of seizures and long-term anti-epileptic use with significant neurologic sequelae.**
- **Review the biochemical pathways involved with Folate and Vitamin B12 metabolism and how laboratory testing may differentiate between the two.**

Case Presentation

58 year-old Caucasian male with a history of seizures secondary to a TBI presents with recurrent falls.

- The patient sustained a traumatic brain injury during a motor vehicle collision 25+ years ago and developed recurrent seizures. His seizures have been well controlled for 5+ years on a regimen of Tegretol, Phenytoin, and Keppra, but the patient's health has deteriorated in the past few years. The patient presents with recurring falls. He denies any recent changes with his seizure medications. He states that the falls have been progressively worsening along with the quality of his sleep and peripheral neuropathy.
- No focal neurological deficits on physical exam.
- Additional PMH is notable for DM, HTN, HL, and peripheral neuropathy.
- Medications include Metformin, Lipitor, and Lisinopril.
- Denies alcohol, tobacco, or drug use.
- Family history of stroke, cardiovascular disease, and diabetes.
- Vitamin Supplementation with 5 mg of Folic Acid daily and Vitamin B12 1000 mcg intramuscular injections daily for one week and then weekly were required to overcome the deficiencies found on laboratory testing. The patient improved clinically and was de-escalated to more routine vitamin supplementation.

Table 1. Serum levels of Folate, Vitamin B12, Methylmalonic Acid, and Homocysteine.

	Oct '17	Oct '18	June '19	Nov '19
Folate (ng/mL)	<2	<2	>20	>20
Vit B12 (pg/mL)	238	254	540	819
MMA (nmol/L)	-	126	-	-
Homocysteine (umol/L)	-	47.3	-	-

Discussion

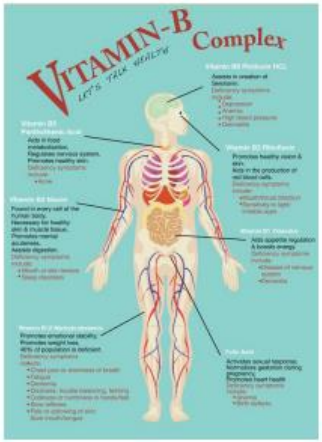

Figure 1. Folate and Vitamin B12 are key co-factors in neurologic and hematopoietic function. There are several causes of deficiencies in both with an overlap in their clinical presentation. Classic presentation includes megaloblastic anemia, neuropsychiatric manifestations, and GI symptoms, but these may vary. Both are found in a well-balanced diet, but may be supplemented as well.

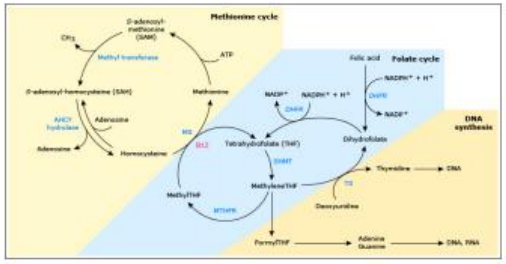

Figure 2. Methionine Cycle and Folate Cycle in the role of DNA Synthesis. Both Folate and Vitamin B12 play a role in the metabolism of homocysteine into methionine and hence homocysteine levels may be elevated in a deficiency of either. On the other hand, Vitamin B12 is a co-factor in the conversion of methylmalonic-CoA into succinyl-CoA in which a deficiency of Vitamin B12 will lead to accumulation of methylmalonic acid. If one or both of these vitamins are deficient, that will lead to a downstream effect of impaired DNA synthesis.

Future Recommendations

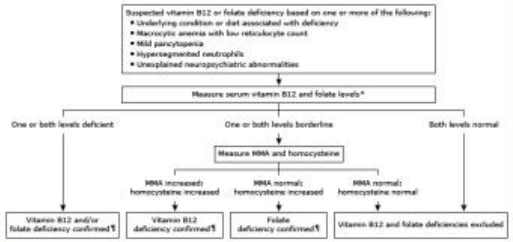

Figure 3. Algorithm for working up Vitamin B12 and Folate Deficiency - UpToDate.

Take Home Points

- Consider alternative etiologies when a patient's condition does not improve or worsens
- Patients with long-term medication use should be monitored either clinically or with laboratory testing to monitor for potential adverse effects
- Causes of Folate deficiency include decreased intake, increased demand, and decreased absorption. Unlike Vitamin B12, which is abundantly stored in the liver, Folate levels may deplete acutely causing signs or symptoms of deficiency

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Splenic Rupture in an Immunocompetent Patient with Acute CMV Infection

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2. Alabama Infectious Disease Center

Introduction

CMV infection presents with a wide range of clinical presentations with the mononucleosis-like illness (IM) being the most common one. Atraumatic splenic rupture due to CMV infection is a rare complication and has been reported few times in the literature.

Hospital Course

20-year-old Caucasian male with no pertinent PMH presented with intermittent RLQ abdominal pain of 1-week duration. The pain intensified 2-3 days before the admission and was associated with fever.

The patient had been tested for IM in the past with negative results. He reported multiple streptococcal throat infections in the past but denied any throat pain at presentation time. The patient denied any contact sport or trauma to the abdomen. The patient was hemodynamically stable, and he was treated conservatively. T: 37.4 °C, HR: 97, RR: 20, BP: 131/76, Hb 12.5, WBC 10.27 $x10^3$ (Lymphocytes: 7.68 $x10^3$), ALT 61, AST 59.

CT Abdomen revealed splenomegaly (craniocaudal dimension 17.3 cm) and 13 mm subcapsular hematoma with free fluids within both colic gutters and pelvis consistent with atraumatic splenic rupture with hemoperitoneum. Heterophile antibody testing of EBV was negative and serological studies revealed positive IgM antibodies to CMV.

Serial testing showed stable hemoglobin, the patient was hemodynamically stable, fever and leukocytosis resolved, and abdominal pain was minimal. No antiviral therapy was needed, and the patient was cleared for discharge with recommendations of avoiding strenuous activity and follow-up as an outpatient.

One month after discharge, the patient came back with left shoulder and mild abdominal pain of one-day duration. A repeat CT scan showed an increase in the size of splenic hematoma. The patient was conscious and oriented with stable vital signs and soft abdomen. Hb 14.3 WBC 6.75 $x10^3$, ALT 16, AST 18. The patient had laparoscopic splenectomy and discharged home in a stable condition.

Discussion

Primary CMV infection in immunocompetent hosts has a wide range of clinical manifestations, the most common is with a mononucleosis-type syndrome. Rare cases have been reported describing fulminant and multisystemic involvement like colitis, encephalitis and retinitis. Systemic symptoms and fever predominate in CMV mononucleosis. Tonsillar white coat, cervical lymphadenopathy, and hepatosplenomegaly are significantly associated with EBV-IM compared to CMV-IM.

The diagnosis of CMV is based upon serology by finding of IgM antibodies against CMV early in the course of the disease and the rise of IgG antibodies in the convalescent phase of the illness.

Systematic reviews of atraumatic splenic ruptures have shown that infection is the most common cause of these cases followed by neoplastic and hematological disorders. In a systematic review of 845 patients with atraumatic splenic ruptures, 137 (14.8%) cases were due to viral infections and the majority were caused by EBV or Malaria with only 13 cases (1.5%) attributed to CMV.

Different definitions have been postulated as to what represents splenomegaly and massive splenomegaly. One objective definition depends on the length of spleen on U/S with length> 13 cm considered splenomegaly and > 18 cm considered massive splenomegaly. Our patient presented with an enlarged spleen of a craniocaudal dimension of 17.3 cm (15.1 x 8.1 cm).

Conclusion

Although splenomegaly has been associated with CMV, atraumatic splenic rupture with hematoma due to CMV infection is a rare complication that requires a high index of clinical suspicion. It would be helpful for primary care physicians to differentiate between EBV-IM and CMV-IM based on symptoms, clinical signs, and laboratory data, such as complete blood count.

This abstract was chosen for oral presentation on Research Day.

Title: Syncope as an Unusual Clinical Manifestation of Pheochromocytoma

Authors: Sabrina Matosz, MD; Ankur Jindal, MD; Farrah Ibrahim, MD

Learning Objective: Most patients with pheochromocytoma have sustained or paroxysmal hypertension; however, some may present with hypotension, orthostatic hypotension, and even syncope.

Case: 60-year-old female transferred to our hospital for evaluation of syncope. She had a sudden episode of dizziness followed by loss of consciousness, lasted one minute, and associated with jerky movements. Patient regained consciousness fully and spontaneously without any intervention. She has been complaining of dizzy spells the last two months and ENT started her on meclizine for possible Meniere's disease. Vital signs on admission included a pulse of 84/minute and blood pressure of 113/68 mmHg. EKG showed nonspecific T wave changes. Troponin T, 5th generation was 378 ng/L. Echocardiogram demonstrated an ejection fraction of 55-60%.

Video electroencephalograph (VEEG) showed no epileptiform waves. MRI/CT head showed no acute intracranial abnormality. Triple-rule-out CT (TRO CT) angiography ruled out coronary artery disease, pulmonary thromboembolism, or aortic dissection. It did note a questionable left upper quadrant mass. CT abdomen and pelvis demonstrated a 5.0 x 6.5 x 6.7 cm heterogeneously enhancing mass arising from the left adrenal gland. Patient denied any abdominal pain, nausea, loss of vision, palpitations, headaches, diaphoresis, weight loss, or purple striae. No previous history of hypertension, malignancy, or multiple endocrine neoplasia syndromes. Terazosin was started and she underwent a left laparoscopic adrenalectomy.

Discussion: We report a female with no family history of hereditary pheochromocytoma syndromes and no history of hypertension, admitted for syncope. Seizure was on the differential diagnoses and Levetiracetam was started. However, the patient had no evidence of seizures on VEEG and no concerning pathology on head MRI. Cardiac workup was also unconvincing. Pertinent labs included 24-hour urine total metanephrines 8,479 mcg/24 hrs, plasma metanephrines 12 nmol/L, and 24-hour urine free catecholamines 74 mcg/24 hr. Surgical pathology confirmed pheochromocytoma.

Pheochromocytomas are catecholamine-secreting tumors that arise from chromaffin cells of the adrenal medulla. Pheochromocytomas are rare, with an annual incidence of 2 to 8 cases per 1 million people. The diagnosis of pheochromocytoma is based upon biochemical confirmation of catecholamine hypersecretion with 24h urine collection or plasma free metanephrines followed by imaging studies. The most common symptoms are headache, palpitations, diaphoresis, and hypertension. Although, most patients have hypertension, some patients present with hypotension and relatively rare, orthostatic hypotension. Syncope due to hypotension is unusual in this disease. Several reasons for hypotension and subsequently syncope could include hypovolemia, intermittent secretion of catecholamines, impairment of peripheral response to catecholamines, baroreflex failure, and adrenocortical insufficiency.

Conclusion: Syncope is unusual in patients with pheochromocytoma and this case serves to demonstrate an unpredicted presentation of this tumor. Although the classic triad of symptoms in patients with

pheochromocytoma consisting of episodic headache, sweating, and tachycardia, other signs and symptoms including syncope and orthostatic hypotension are rare but may occur.

This abstract was chosen for oral presentation on Research Day.

Clinical Vignette Abstract:

Treatment of Hairy Cell Leukemia by Cladribine Predisposes Patients to an Increased Risk of New-Onset Drug Allergies.

Authors: Syeda Sabeeka Batool[1], Mohamed Hasan[1], Hafiz Muhammad Fazeel[2], Ali B Hachem[3]

Affiliations:

1. PGY-II Resident, Internal Medicine, UAB-Huntsville, AL 35801
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3. Attending Physician, Hematology & Medical Oncology, The Cancer Center of Huntsville, AL 35801

Learning Points:

1. Cladribine is first line treatment for hairy cell leukemia (HCL) with high (>90%) remission rate.
2. Patients treated by Cladribine develop new-onset allergies which most of times present as cutaneous reactions.
3. Penicillins, Sulfonamides, and Allopurinol are the most common drugs reported to show such allergic reactions.
4. Our case uniquely reports new onset allergy towards Levofloxacin which is not highly reported in literature.

Case History:

A 24-years-old female was diagnosed with HCL after she presented with severe pancytopenia (< 25000/uL) and massive splenomegaly (29 cm). Her bone marrow biopsy showed 70% involvement with hairy cells which stained positive for V600E and BRAF mutation. She relapsed after initial remission with cladribine. So, she was restarted on cladribine with rituximab, and allopurinol was prescribed for hyperuricemia prophylaxis. She developed a diffuse rash and facial swelling within 48 hours of starting allopurinol which responded promptly to antihistamines and steroids. A few days later, she developed a similar allergic reaction towards levofloxacin which also responded promptly to stopping levofloxacin and antihistamines. She reported previous allergies towards penicillin and sulfonamides which developed after she was started on cladribine. Her BMI was 27.74 and systemic inquiry and labs were significant only for rash, leukopenia (1100 cells/uL) and thrombocytopenia (81000/uL).

Discussion:

Hairy cell leukemia is an uncommon (< 2%) adult leukemia that presents with splenomegaly and hairy cells accumulated in spleen and bone marrow. Diagnosis is made by typical histologic features of bone marrow and immunophenotypic profile (positive for CD20, CD22, CD103 and CD25). Cladribine, a purine analog, is now considered first line treatment for hairy cell leukemia and has shown high rates of complete remission (95%) and overall survival (97%). But cladribine use has been infrequently reported to cause cutaneous adverse reactions and rarer reports have also shown patients developing new onset allergic reactions to different drugs and antibiotics after being exposed to cladribine. The possible mechanism of this immunomodulated effect and reduced drug tolerance is an imbalance in T-cell immunity and memory cells secondary to CD4+ lymphopenia caused by purine analogues or the disease itself. The treatment of such reactions is similar to any other drug eruptions. Discontinuation of cladribine and the inciting drug is most important step. Resensitization with the drug is safe after recovery of CD4+ lymphocyte count.

Treatment of Hairy Cell Leukemia by Cladribine Predisposes to an Increased Risk of New-Onset Drug Allergies.

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Syeda Sabeeka Batool, MD, Mohamed Hasan, MD, Hafiz Muhammad Fazeel, MD, Ali B. Hachem, MD

- PGY-II, Department of Internal medicine, UAB- Huntsville Hospital, Huntsville, AL 35801
- Observer, Alabama Infectious Disease Center, Huntsville, AL 35801

CASE PRESENTATION

Presenting Complaint:

- 20 -old female with a new onset maculopapular skin rash

Physical Exam:

- Vitals stable and normal; BMI= 27.7 kg/m2.
- Generalized morbilliform rash diffusely spread over trunk and extremities.
- Palmo-plantar involvement positive with no mucosal involvement.
- Unremarkable otherwise.

Past Medical History:

- Hairy cell leukemia with severe pancytopenia (<25000/uL) and massive splenomegaly (29 cm)
- Multiple previous courses of Cladribine
- Recently developed an allergic skin rash towards penicillin and co-trimoxazole.
- Ex-Smoker, no recreational drug use

Routine Workup:

- Leukopenia (1100 cells/uL) and thrombocytopenia (81000/uL).
- Bone marrow biopsy showed 70% involvement with hairy cells which stained positive for V600E and BRAF mutation
- Otherwise unremarkable

Medications:

- Allopurinol for hyperuricemia prophylaxis
- Levofloxacin for bacterial prophylaxis.
- Patient had recently developed allergy towards penicillin and co-trimoxazole.

Treatment:

- Oral antihistamines and topical steroids for rash.
- Levofloxacin was stopped.

Follow-up:

- Complete resolution of rash.
- Patient continued to receive cladribine.

DISCUSSION

- Hairy cell leukemia is an uncommon (< 2%) adult leukemia that presents with splenomegaly and hairy cells accumulated in spleen and bone marrow (1).
- Diagnosis is made by typical histologic features of bone marrow and immunophenotypic profile (positive for CD20, CD22, CD103 and CD25).
- Cladribine, a purine analog, has shown high rates of complete remission (95%) and overall survival (97%) (2).
- Cladribine use has been rarely reported to cause cutaneous drug reactions.
- Rarer reports have shown patients developing new onset allergic reactions to different drugs and antibiotics after being exposed to cladribine.
- Penicillins, Sulfonamides, and Allopurinol are the most commonly reported drugs associated with these allergic reactions.
- The possible mechanism of this immunomodulated effect and reduced drug tolerance is an imbalance in T-cell immunity and memory cells secondary to CD4+ lymphopenia caused by purine analogues or the disease itself (3).
- The treatment of such reactions is similar to any other drug eruptions. Discontinuation of the inciting drug is most important step (2).
- Steroids are used in severe cases and resensitization with the drug is safe after recovery of CD4+ lymphocyte count (2).

Reported Risk of Allergic Skin Rash For Antibiotics in HCL Patients (1)

			Univariate model			Multivariate model		
Antibiotic	Morbilliform rash (n = 17)	No morbilliform rash (n = 80)	Odds ratio	*P* value	95% confidence interval	Odds ratio	*P* value	95% confidence interval
Penicillin, n (%)	5 (29.4)	5 (6.2)	6.25	.009*	(1.54, 25.8)	5.87	.022†	(1.26, 27.9)
Fluoroquinolone, n (%)	7 (41.2)	18 (20.0)	2.80	.069*	(0.90, 8.50)	1.46	.56	(0.38, 5.14)
Sulfonamide, n (%)	8 (47.1)	21 (26.2)	2.50	.095*	(0.84, 7.39)	2.46	.14	(0.73, 8.31)
Cephalosporin, n (%)	1 (5.9)	11 (13.8)	0.39	.386	(0.02, 2.25)			
Clindamycin, n (%)	1 (5.9)	2 (2.5)	2.44	.478	(0.21, 28.5)			
Vancomycin, n (%)	1 (5.9)	8 (10.0)	0.56	.600	(0.07, 4.82)			
Macrolide, n (%)	1 (5.9)	7 (8.8)	0.65	.698	(0.03, 4.04)			

*P value <.10.
†P value <.05.

LEARNING POINTS

- Patients receiving cladribine show an increased risk of new onset drug allergies.
- Our case reports new onset allergy towards Levofloxacin which has nit been previously reported in literature.
- Our case also reports development of allergy towards penicillin and sulfonamides which are rare but previously established in literature.
- Treatment is similar to other allergic reactions and outcome is almost always uneventful.

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*No conflict of interests disclosed for this case report.

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Trichodysplasia Spinulosa: **A rare "hair-like" dysplasia**

Learning Objectives:

- Appreciate unforseen consequences of long-term immunosuppression even after resolution of immunosuppression
- Recognize the clinical manifestations of HPV-8
- Consider potential treatment options

Case Presentation:

A 3-year-old had a history of right-sided facial and parameningeal embryonal rhabdomyosarcoma in October 2017. He completed a 13-week course of the RMS13 chemotherapy and radiation (proton) therapy clinical trial and remission was achieved, and he was subsequently monitored serially with CT imaging for recurrence. The patient became pancytopenic during therapy, and remained pancytopenic until approximately June 2019 at which time filgrastim was discontinued.

In September 2019 the patient developed sudden-onset roughness and red discoloration of the right cheek, which progressed over days to indurated, edematous skin of the nose, cheeks, glabella, and ears. The patient's mother described the lesion as being both tender and boggy. He was treated with Elidel and topical emollients and plastics was consulted for biopsy. Biopsy findings were notable for basal cell layer differentiation into nucleated eosinophilic cells with numerous trichohyalin granules and negative for viral inclusion bodies, which was consistent with *Trichodysplasia spinulosa*. The patient received a CT head at both this visit and at a January 2020 appointment, both of which were negative for intracranial abnormalities or recurrence of right infratemporal mass. Routine CBC and CMP monitoring was WNL throughout this time. Between these two visits, the patient's rash spread to diffusely involve the trunk, arms, and legs. The mother had started the patient on home topical clindamycin with no improvement. She was counseled on potential treatment options, and Valcyte 50 mg/mL oral was began.

Discussion:

Trichodysplasia spinulosa is characteristically described as a skin eruption with friable spinous processes and indurated erythematous papules that on biopsy show particles with size and appearance suggestive of papovavirus but inconsistency with papilloma or polyomavirus on PCR and immunohistiochemistry. Reported cases involve almost exclusively patients on immunosuppressive therapy, with normal leukocyte counts being virtually unseen thus far in initial presentation. Of the 14 reported cases in a literature review in 2010, two patients improved with topical 3% cidofovir, one improved with valganciclovir, one improved with changing of immunosuppressive regimen, and one improved with surgical shaving of spinules and treatment with tazarotene 0.5%; since that time additionally physical extraction of spicules has shown to be effective. Other treatments have included oral antibiotics and oral acitretin which were reported as unsuccessful; and topical imiquimod, topical steroids, antihistamines, and benozyl peroxide with mixed results. The condition overall is believed to be self-limiting, and several cases have improved following resolution of immunosuppression. Overall the most efficacious methods appear to be topical cidofovir and oral valganciclovir as well as manual exfoliation or extraction, or combinations of these agents; however, given that the rash is generally tender medication seems most effective in a pediatric population.

This abstract was chosen for oral presentation on Research Day.

Unilateral Graves Orbitopathy

Authors: Maha Al-Baghdadi MD, Ali Hassoun MD

Introduction

Graves orbitopathy is an autoimmune disease characterized by orbital inflammation of both adipose tissue and extra-ocular muscles (EOM). It is the most common cause of exophthalmos. Unilateral Graves orbitopathy is rare and described as a transient stage before the involvement of the other eye.

Hospital course

A 31 years-old Caucasian female with PMH of facial eczema and tobacco abuse. Patient presented to the emergency department complaining of right-eye swelling and pain. Symptoms developed gradually over one month, she attributed her symptoms to flare-up of her existing eczema. The patient had CT scan done in an outside facility that showed no significant findings and she was treated as cellulitis with a 10-day course of oral clindamycin with no significant improvement. She presented to our hospital ER with worsening right orbital swelling and pain. Her CT scan revealed right periorbital swelling suspicious for cellulitis. Patient was given vancomycin and ceftriaxone. On admission: T: 36.6°C, HR: 98, RR: 17, BP: 123/83, WBC Count 9.99 x10^3/mcL (Neutrophils 5.51, Lymphocytes 3.61). As her symptoms and signs were not suggestive of cellulitis, further evaluation revealed patient had heat intolerance, tremor and palpitations with evidence of exophthalmos on exam. Her thyroid function test showed TSH level <0.02 and free T4 2.68 (reference range: 0.8-1.7) consistent with hyperthyroidism. The patient was treated as Graves' orbitopathy with methimazole 10 mg TID, propranolol 20 mg TID and prednisone 30 mg once/day. The patient was discharged with instructions to follow-up as an outpatient. Initial Thyroid stimulating immunoglobulin (TSI) was negative. On follow-up visit, the patient stated that she was having more frequent palpitations and anxiety. Repeated TSI testing was positive and the free T4 was 3.14. The patient was instructed to continue steroid therapy for 6 weeks and be reassessed on regular basis.

Discussion

Clinical features of Graves orbitopathy include exophthalmos, lid retraction, periorbital swelling, ophthalmoplegia, and chemosis. Diagnosis is based on clinical features and supported by abnormal thyroid function tests and positive thyroid antibodies. Unilateral graves orbitopathy is defined as one or more of features of Graves orbitopathy involving only one eye without any signs in the contralateral eye. It is rare and reported in only 5-15% of all cases of Graves' orbitopathy. Few cases reported concomitant unilateral Graves orbitopathy and orbital cellulitis. The natural history of unilateral thyroid eye disease is unknown, and maybe misdiagnosed. It is important for physicians to keep in mind to seek alternate diagnosis if unilateral graves ophthalmopathy is encountered especially with normal or slightly abnormal thyroid function test. Important differential diagnoses include cellulitis, lymphoma, orbital pseudotumor and malignancy.

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Unilateral Graves Orbitopathy

Maha Al-Baghdadi MD[1], Ali Hassoun MD[2] 1.UAB Huntsville Regional Campus 2.Alabama Infectious Disease Center

Introduction

Graves orbitopathy is an autoimmune disease characterized by orbital inflammation of both adipose tissue and extra-ocular muscles (EOM).

It is the most common cause of exophthalmos.

Unilateral Graves orbitopathy is rare and described as a transient stage before the involvement of the other eye.

Hospital course

A 31 years-old Caucasian female with PMH of facial eczema and tobacco abuse.

Patient presented to the emergency department complaining of right-eye swelling and pain.

Symptoms developed gradually over one month; she attributed her symptoms to flare-up of her existing eczema.

The patient had CT scan in an outside facility that showed no significant findings and she was treated as cellulitis with a 10-day course of oral clindamycin with no improvement.

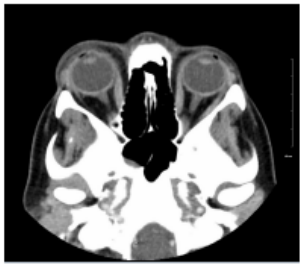
She presented to our hospital ER with worsening right orbital swelling and pain. Her CT scan revealed right periorbital swelling suspicious for cellulitis. Patient was given vancomycin and ceftriaxone.

On admission: T: 36.6°C, HR: 98, RR: 17, BP: 123/83, WBC Count 9.99 x10^3/mcL (Neutrophils 5.51, Lymphocytes 3.61).

Further evaluation revealed patient had heat intolerance, tremor and palpitations with evidence of exophthalmos on exam. Her thyroid function test showed TSH level <0.02 and free T4 2.68 (reference range: 0.8-1.7) consistent with hyperthyroidism.

The patient was treated as Graves' orbitopathy with methimazole 10 mg TID, propranolol 20 mg TID and prednisone 30 mg once/day. The patient was discharged with instructions to follow-up as an outpatient. Initial Thyroid stimulating immunoglobulin (TSI) was negative.

On follow-up visit, the patient stated that she was having more frequent palpitations and anxiety. Repeated TSI testing was positive and the free T4 was 3.14. The patient was instructed to continue steroid therapy for 6 weeks and be reassessed on regular basis.



Right periorbital swelling suspicious for cellulitis

Discussion

The diagnosis of Graves orbitopathy is based on clinical features and supported by abnormal thyroid function tests and positive thyroid antibodies.

Unilateral graves orbitopathy is defined as one or more of features of Graves orbitopathy involving only one eye without any signs in the contralateral eye.

It is rare and reported in only 5-15% of all cases of Graves' orbitopathy. Few cases reported concomitant unilateral Graves orbitopathy and orbital cellulitis.

The natural history of unilateral thyroid eye disease is unknown, and maybe misdiagnosed.

Conclusion

It is important for physicians to keep in mind to seek alternate diagnosis if unilateral graves ophthalmopathy is encountered especially with normal or slightly abnormal thyroid function test.

Important differential diagnoses include cellulitis, lymphoma, orbital pseudotumor and malignancy.

Visceral Artery Aneurysm: An Unusual Cause of Abdominal Pain

Authors: Maha Al-Baghdadi MD, Parekha Yedla MD

Learning objectives

Increase awareness of visceral artery aneurysm and pseudoaneurysms.

Understanding risk factors, complications, and management of these aneurysms.

Case

A 58-year-old Caucasian female with past medical history of coronary artery disease and asthma presented to the hospital due to shortness of breath for a few days' duration. Upon admissions her vital signs BP 114/64, HR 84, RR 30, temperature 37.3, O2 saturation 90% on 2 L nasal cannula. She had Bilateral wheezing and work-up revealed bilateral multifocal pneumonia as shown by CT chest. The patient was admitted, and antibiotics were initiated. A few hours after admission, the patient developed right upper quadrant, dull abdominal pain. Abdominal ultrasound showed a mass within the upper abdomen near the mesenteric root. CT abdomen with contrast showed right-sided peripancreatic aneurysm arising from the gastroduodenal artery, actively bleeding, with a large hematoma. Patient's hematocrit dropped from 8.5 g/dl to 6.6 g/dl and her B.P was 97/49. Interventional Radiology was consulted right away, an emergent mesenteric CT angiogram was done which revealed jejunal branch pseudoaneurysm and irregularity of the vessel with extravasation into a large hematoma. This was successfully embolized with a combination of coils and glue. The patient tolerated the procedure well, and there was no further drop in her hematocrit in the following days. The patient was discharged on day 6 after the procedure.

Discussion

Visceral artery aneurysms (VAA) and pseudoaneurysms (VAPA) are relatively rare with an incidence ranging from 0.1 to 2% of people. The splenic artery is the most common site and the inferior mesenteric artery is the least common site of aneurysmal disease. Gastroduodenal aneurysms account for only 1.5% of all VAA. These aneurysms are caused by multiple etiologies including inflammation from pancreatitis (the most common cause for VAPA), atherosclerosis (the most common cause for VAA), trauma, infection, vasculitis, and collagen vascular diseases.Patients may remain asymptomatic or present with symptoms like abdominal pain, nausea, vomiting, melena, or hematochezia. CT angiogram

is the diagnostic test of choice. These aneurysms have a high risk of rupture that can result in hemorrhage into the peritoneal cavity, retroperitoneal space or gastrointestinal tract (more commonly with VAPA than VAA). Prompt diagnosis and management of symptomatic patients with trans-catheter coil embolization or surgery is the key to decrease mortality from these aneurysms. Immediate treatment is usually recommended in patients with pseudoaneurysm regardless of their size in asymptomatic patients.Our patient did not have a history of trauma or pancreatitis which are the leading causes of pseudoaneurysms and will need further outpatient work up to rule out vasculitis as there was an irregularity of the jejunal branch.

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Visceral Artery Aneurysm: An Unusual Cause of Abdominal Pain

Maha Al-Baghdadi MD[1], Parekha Yedla MD[1] [1]UAB Huntsville Regional Medical Campus

Learning objectives

- Increase awareness of visceral artery aneurysm and pseudoaneurysms.
- Understanding risk factors, complications, and management of these aneurysms.

Case

- A 58-year-old Caucasian female with past medical history of coronary artery disease and asthma presented to the hospital due to shortness of breath for a few days' duration.
- Upon admissions her vital signs BP 114/64, HR 84, RR 30, temperature 37.3, O2 saturation 90% on 2 L nasal cannula.
- She had Bilateral wheezing and work-up revealed bilateral multifocal pneumonia as shown by CT chest.
- The patient was admitted, and antibiotics were initiated.
- A few hours after admission, the patient developed right upper quadrant, dull abdominal pain.
- Abdominal ultrasound showed a mass within the upper abdomen near the mesenteric root.
- CT abdomen with contrast showed right-sided peripancreatic aneurysm arising from the gastroduodenal artery, actively bleeding, with a large hematoma.
- Patient's hematocrit dropped from 8.5 g/dl to 6.6 g/dl and her B.P was 97/49
- Interventional Radiology was consulted right away, an emergent mesenteric CT angiogram was done which revealed jejunal branch pseudoaneurysm and irregularity of the vessel with extravasation into a large hematoma.
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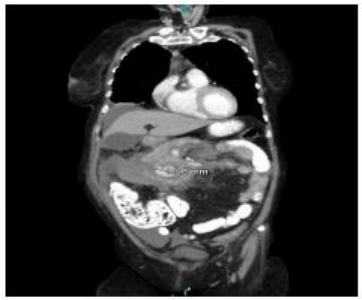
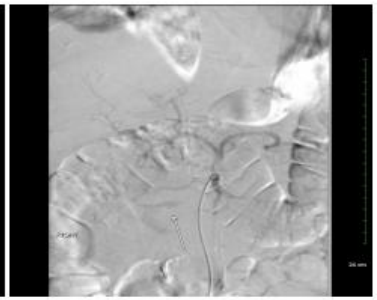


Gastroduodenal peripancreatic artery aneurysm with large hematoma



Angiogram of jejunal branch pseudoaneurysm showing irregularity

Discussion

- Visceral artery aneurysms (VAA) and pseudoaneurysms (VAPA) are relatively rare with an incidence ranging from 0.1 to 2% of people.
- The splenic artery is the most common site and the inferior mesenteric artery is the least common site of aneurysmal disease. Gastroduodenal aneurysms account for only 1.5% of all VAA.
- These aneurysms are caused by multiple etiologies: inflammation from pancreatitis (the most common cause for VAPA), atherosclerosis (the most common cause for VAA), trauma, infection, vasculitis, and collagen vascular diseases.
- Patients may be asymptomatic or present with symptoms like abdominal pain, nausea/vomiting, melena, or hematochezia.
- CT angiogram is the diagnostic test of choice.
- These aneurysms have a high risk of rupture that can result in hemorrhage into the peritoneal cavity, retroperitoneal space or gastrointestinal tract.
- Prompt diagnosis and management with trans-catheter coil embolization or surgery is the key to decrease mortality.
- Immediate treatment is usually recommended in patients with pseudoaneurysm regardless of their size in asymptomatic patients.
- Our patient did not have a history of trauma or pancreatitis which are the leading causes of pseudoaneurysms, will need further outpatient work up to rule out vasculitis as there was an irregularity of the jejunal artery.

TITLE: When It Doesn't All PAN Out – Remember SAM

AUTHORS (FIRST NAME, LAST NAME): Joe Mcilwain[1], Roger D. Smalligan[1]

INSTITUTIONS (ALL): 1. Internal Medicine, UAB-Huntsville, Huntsville, AL, United States.

ABSTRACT BODY:
Learning Objective #1: Recognize causes of microaneurysms in abdominal vasculature

Learning Objective #2: Distinguish between segmental arterial mediolysis (SAM) and polyarteritis nodosa (PAN)

Case: A previously healthy 56yo white woman presented with sudden abdominal pain radiating to her back with nausea but no fever, chills, vomiting, diarrhea, urinary symptoms, headache, jaw claudication or history of trauma. PMH: no DM, HTN, or arthritis. Meds: oxybutynin; Surgery: cholecystectomy; Family Hx: neg for autoimmune disease; P/S: no smoking, ETOH or drugs. PE: alert woman in pain, BP 110/65, P 89, R 17, lungs clear; heart RRR without murmurs; abdomen: soft but exquisitely tender to palpation; neuro was normal and skin without subq nodules or livido. Normal body habitus and no unusual joint laxity. Labs: WBC 12.9k, Hgb 13, MCV 87, platelets 200k, BUN 16, Cr .8. Images: Contrast abdominal CT: hemoperitoneum and luminal irregularities suspicious for multiple aneurysms involving the SMA but no signs of bowel ischemia. Diagnostic laparoscopy revealed 1500cc of blood and clots; however, her entire small bowel and colon were well-perfused. Extensive rheum/autoimmune workup was negative including rheumatoid factor, ANA, PR3-ANCA, MPO-ANCA, anti-CCP, dsDNA as well as hepatitis B and C. CT angiogram of the abdomen showed 5 splenic artery aneurysms (9mm), a right renal artery aneurysm and various small aneurysms throughout the splanchnic mesentery. CTA of the head was largely unremarkable. High dose steroids were used initially but were stopped once the diagnosis of SAM was made. A splenectomy was recommended for definitive diagnosis and due to the risk of bleeding but that is pending the healing of her intestinal lesions.

Impact/Discussion: Systemic arterial mediolysis is a rare, noninflammatory, vascular condition that affects medium to large abdominal arteries. SAM is characterized by lysis of smooth muscle in the medial arterial layer. The differential for SAM includes polyarteritis nodosa, fibromuscular dysplasia, and various connective tissue diseases. This case shows the importance of early differentiation between PAN and SAM. SAM is a self-limited disease process requiring supportive care whereas PAN has a poorer prognosis and requires strong immunosuppressant therapy which can be toxic. Both conditions affect middle-aged and older people. SAM affects both sexes equally while PAN has a slight propensity towards men. SAM tends to be relatively sudden in onset while PAN presents indolently with preceding systemic, inflammatory symptoms. SAM does not typically present with anemia, ESR elevation or an association with hepatitis B as is seen with PAN. A definitive diagnosis of SAM requires tissue evaluation but is often not pursued given sites involved.

Conclusion: Physicians need to include SAM in the differential diagnosis of patients with severe abdominal pain and vasculitic appearing lesions on angiography since misdiagnosis of PAN will lead to unnecessary immunosuppressant treatment with its associated serious side effects.

This abstract was chosen for oral presentation on Research Day.

Authors:

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Title: The Yin and Yang of Hepatitis C infection (HCV) and B-Cell Non-Hodgkin Lymphoma

Learning Objectives:

1. Diagnose the presence of elevated immunoglobulins in the setting of an elevated gamma gap
2. Recognize extra-hepatic manifestations of hepatitis C virus
3. Utilize antiviral therapy as a potential cure for indolent non-Hodgkin's lymphoma in patients co-infected with Hepatitis C

Case:

A 55-year-old gentleman with history of testicular cancer status post resection, radiation and splenectomy presented with abdominal pain, anorexia and constipation. Physical examination significant for bony prominence on left clavicle. Abdominal pain resolved with laxatives. Imaging revealed lytic lesion on medial left clavicle. Labs revealed elevated serum protein and albumin, positive hepatitis C antibodies and detectable viral load with elevated kappa free light chains and elevated kappa/lambda ratio. HIV antibodies were negative. Serum electrophoresis noted an increased level of polyclonal gamma globulins consistent with chronic inflammation. Biopsy of clavicular mass revealed Diffuse Large B-cell Lymphoma (DBCL).

Discussion:

HCV is a blood borne infection affecting approximately three million people in the United States. Known exposures for HCV include blood transfusions prior to 1992 and intravenous drug use. In addition to being a causative agent of cirrhosis and hepatocellular carcinoma, HCV infection can have numerous systemic manifestations and associations, including keratitis, cognitive impairment, peripheral neuropathy, increased atherosclerosis, type 2 diabetes, thyroid disease, Sjogren syndrome, renal disease, and, as in our patient, B-cell lymphomas.

Studies have shown both a higher prevalence of HCV in patients with Non-Hodgkin Lymphoma (NHL) than the general population and a significantly increased risk of NHL in HCV patients.

The mechanism of this effect is not fully understood, though pathogenesis is thought to be related to chronic stimulation of the immune system by the virus, oncogenic stimulation of B cells due to HCV intracellular replication, as well as oxidative damage triggered by HCV proteins.

The malignant B cells produce large amounts of immunoglobulins as a gap (referred to varyingly as a gamma gap, protein gap, or paraprotein gap) between the total serum protein and the serum albumin. The gold standard test to evaluate the protein gap is serum protein electrophoresis, which can reveal the monoclonal immunoglobulins produced by the malignant cells. In this case, polyclonal gamma globulins were present from concomitant hepatitis C infection and lymphoma.

Treatment of both NHL and HCV improve mortality. Goal of treatment with HCV is to achieve sustained viral response to limit further systemic manifestations of the virus. Patients who have successful treatment for HCV are at significantly reduced risk of NHL than their untreated or unsuccessfully treated counterparts. HCV patients with B-cell Non-Hodgkin's lymphoma, indolent lymphoma types can be potentially cured with antiviral therapy. In more aggressive lymphoma types, such as in DBCL, immunochemotherapy is recommended in addition to antiviral therapy. Current literature suggests that viral clearance is related to lymphoma response, prevention of potential liver damage associated with chemotherapy, and avoidance of DBCL relapse.

Given the close association of HCV with NHL, it is worth considering both conditions in a patient presenting with either and assisting patients to receive antiviral therapy to prevent further systemic manifestations of either disease process.

The Yin and Yang of Hepatitis C infection (HCV) & B-Cell Non-Hodgkin Lymphoma

THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

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Learning Objectives

- Diagnose the presence of elevated immunoglobulins in the setting of an elevated gamma gap
- Recognize extra-hepatic manifestations of hepatitis C virus (HCV)
- Utilize antiviral therapy as a potential cure for indolent non-Hodgkin's lymphoma in patients co-infected with Hepatitis C

Introduction

- HCV is a bloodborne infection affecting approximately 3 million people in the U.S.
- Systemic manifestations of HCV infection:
  - Peripheral neuropathy
  - T2DM
  - Renal disease
  - Atherosclerosis
  - Sjogrens Syndrome
  - Thyroid disorders
  - B-cell lymphomas
  - Hepatocellular Carcinoma

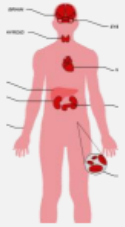

Figure 1. Diagram of systemic manifestations of HCV infection

- Higher prevalence of HCV in patients with Non-Hodgkin Lymphoma (NHL)
- Increased risk of NHL in patients with HCV
- Pathogenesis uncertain, possibly due to:
  - Chronic immune stimulation
  - Oxidative damage

The Patient

- 54 year old male with a history of testicular cancer in remission and splenectomy
- CC: Abdominal pain with associated anorexia and constipation
- Physical Exam: unremarkable except firm, non-mobile mass on the left clavicle

Investigations

139	103	29	110
4.9	25	1.3	9.6

	26
8.7	25
4.4	122
	0.7

11.4 | 15.6 / 46.9 | 313

Diff
N – 53
L – 34
M – 10

HCV PCR: 6,947
HCV Genotype: 2

Hep A: — HBV: —
HCV: + HIV: —

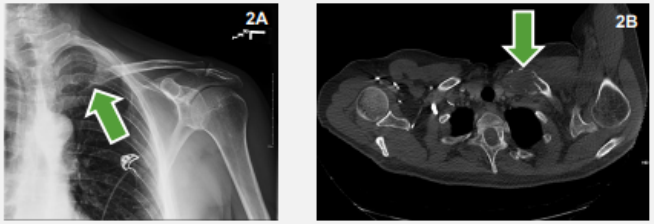

Figure 2A – Clavicular Xray noting bony lesion on head of left clavicle. Figure 2B – CT Chest noting expansile lytic lesion to medial left clavicle

Kappa/Lambda Flc Ratio: 4.73 (103.23/21.81)
UPEP: No monoclonal bands
Pathology: Diffuse Large B-cell Lymphoma

Outcome and Follow up

- PET CT (Jan 2020)– increased metabolic activity in expansile lesion left clavicle and level 2 LN in left side of neck.
- Bone Marrow Biopsy 1/10/20
- Rituximab + CHOP (cyclophosphamide, doxorubicin, vincristine) for 4-6 months
- Most recent PET CT (March 2020) shows no involvement of lymph nodes and improvement of clavicular lesion.
- Per oncologist, given PET improvement may stop chemotherapy and start radiation sooner given good response. Antiviral treatment has not been started.

Clinical Takeaways

- New USPFTF 2020 guidelines recommend HCV screening for all patients ages 18-79.
- Malignant B cells produce large amounts of immunoglobulins detectable as a gamma gap.
- Patients who have successful treatment for HCV are at significantly reduced risk of NHL than their untreated or unsuccessfully treated counterparts.
- Current literature suggests that HCV clearance is related to NHL response to chemotherapy, prevention of potential liver damage associated with chemotherapy, and avoidance of relapse.
- Given the close association of HCV with NHL, one should be alert for signs of NHL as well as hepatic and extra hepatic symptoms in a patient with HCV.

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Section II: Research Abstracts

TITLE: A Quality improvement project to improve documentation of site, type of deep venous thrombo-embolism and duration of anticoagulation in Internal medicine residency clinic

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INSTITUTIONS (ALL): 1. Internal Medicine, UAB, Huntsville regional medical campus, Huntsville, AL, United States.

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Type: Research

Background: Deep venous thrombosis (DVT) refers to formation of one or more blood clots in one of the body's large veins, most commonly in the lower limbs. Anticoagulation is the mainstay therapy for patients with DVT. The administration of anticoagulation is always associated with increased risk of bleeding. Aim of this study to improve documentation of site, type of DVT and duration of anticoagulation in our residency clinic, so that one can prevent complications associated with long term use of anticoagulants if there is no indication for it.

Methods: In order to improve documentation of type, site of DVT and duration of anticoagulation we conducted three-year retrospective review of clinic notes of 36 patients with ICD-10 defined DVT presented to Internal medicine residency clinic. Five patients are excluded from the study as they have line associated upper extremity DVT. Variables analyzed included age, gender, site of DVT, co- morbid conditions, documentation of provoked or unprovoked DVT, documentation of duration of anti-coagulation, name of anti-coagulation, cause of DVT and following with hematologist or not. Age at the time of diagnosis was not documented for most of the patients.

Results: We reviewed documentation of 31 patients with Diagnosis of ICD-10 defined lower extremity DVT in our Internal Medicine residency clinic. Among 31 patients, 14 (45%) have duration of anticoagulation documented and 17 (55%) did not have duration of anticoagulation documented. 13 (42%) have documented type of DVT whether it is provoked or unprovoked and 18 (58%) did not have documentation about type of DVT. Most patients were female (55%) and median age was 69yrs (range 32-88). Exact location of DVT was documented in only 10 (32%) patients and underlying cause was documented in 11 (35%) patients. Most commonly used anti-coagulant was Xarelto (38%) followed by coumadin (35%). HTN and obesity emerged as the prevalent co-morbid conditions accounting for 51% and 25%respectively. Other co morbid conditions were smoking (21%), Diabetes (16%) and any cancer (15%). 12 (39%) patients are following with hematology specialist as per chart review.

Conclusions: Less than 50% of the patients have documented type, site of DVT and duration of anticoagulation. Exact location was documented in only 32% patients. It Is very important to document location of DVT to determine need and length of anticoagulation. Some people with distal DVT may not even need any anticoagulation at all. In our clinic it is very less likely that patient will be seen by same

physician every time, so it is very vital to document every detail in the clinic notes to determine further course of action for the patient. Our next step would be to educate our residents to document details of DVT and anticoagulation to prevent overuse of anticoagulation. We would like to collect the data after few months of intervention to see an improvement in documentation.

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A Quality improvement project to improve documentation of site, type of deep venous thrombo-embolism and duration of anticoagulation in Internal medicine residency clinic

Sujatha Baddam[1] Farrah Ibrahim[1] [1.] Internal Medicine, UAB, Huntsville regional medical campus

Objectives

1. To improve documentation of site, type of DVT and duration of anticoagulation in our residency clinic
2. To prevent complications associated with long term use of anticoagulants if there is no indication for it.

Background

Deep venous thrombosis (DVT) refers to formation of one or more blood clots in one of the body's large veins, most commonly in the lower limbs. Anticoagulation is the mainstay therapy for patients with DVT. The administration of anticoagulation is always associated with increased risk of bleeding. Aim of this study to improve documentation of site, type of DVT and duration of anticoagulation in our residency clinic, so that one can prevent complications associated with long term use of anticoagulants if there is no indication for it.

Methods

In order to improve documentation of type, site of DVT and duration of anticoagulation we conducted three-year retrospective review of clinic notes of 36 patients with ICD-10 defined DVT presented to Internal medicine residency clinic. Five patients are excluded from the study as they have line associated upper extremity DVT. Variables analyzed included age, gender, site of DVT, co- morbid conditions, documentation of provoked or unprovoked DVT, documentation of duration of anti-coagulation, name of anti-coagulation, cause of DVT and following with hematologist or not. Age at the time of diagnosis was not documented for most of the patients.

Results

We reviewed documentation of 31 patients with Diagnosis of ICD-10 defined lower extremity DVT in our Internal Medicine residency clinic. Among 31 patients, 14 (45%) have duration of anticoagulation documented and 17 (55%) did not have duration of anticoagulation documented. 13 (42%) have documented type of DVT whether it is provoked or unprovoked and 18 (58%) did not have documentation about type of DVT. Most patients were female (55%) and median age was 69yrs (range 32-88). Exact location of DVT was documented in only 10 (32%) patients and underlying cause was documented in 11 (35%) patients. Most commonly used anti-coagulant was Xarelto (38%) followed by coumadin (35%). HTN and obesity emerged as the prevalent co-morbid conditions accounting for 51% and 25%respectively. Other co morbid conditions were smoking (21%), Diabetes (16%) and any cancer (15%). 12 (39%) patients are following with hematology specialist as per chart review.

Conclusions

Less than 50% of the patients have documented type, site of DVT and duration of anticoagulation. Exact location was documented in only 32% patients. It Is very important to document location of DVT to determine need and length of anticoagulation. Some people with distal DVT may not even need any anticoagulation at all. In our clinic it is very less likely that patient will be seen by same physician every time, so it is very vital to document every detail in the clinic notes to determine further course of action for the patient.

Disclosure

The authors declares that they have no relevant or material financial interests that relate to the research described in this abstract

A Real World Comparison of ExacTrac and Cone Beam CT for Image Guided Radiosurgery

Research Abstract – UAB Huntsville Research Day 2020

Roman Travis[1], Michael Taylor MS[2], John Gleason MD[2]
[1]UAB School of Medicine, Huntsville AL; [2]Alliance Cancer Care Huntsville AL

Abstract

Introduction: Stereotactic radiosurgery (SRS), a mainstay in brain tumor treatment, requires precise patient alignment to treat brain tumors while avoiding healthy tissue. Several imaging strategies are used to precisely align each patient to the machine. Two of these strategies ExacTrac x-ray and cone beam CT (CBCT) are often used redundantly. ExacTrac uses two orthogonal x-rays to prescribe a "shift" three translational axes and three rotational axes. CBCT prescribes the same 6-dimensional shift using a rapidly acquired CT-scan. Since a small amount of "noise" is always present, shifts are only applied when an axis is outside of a specified tolerance i.e. 0.8 mm. Since CBCT requires additional time and resources, this research project seeks to determine the concordance between the two modalities' prescribed shifts by calculating the root mean square (RMS) distance between each shift for a cohort of patients. We then compare this to typical tolerances used clinically.

Methods: 28 consecutive patients who received SRS at Alliance Cancer Care in 2019 were reviewed. At a typical treatment, the patient is first aligned using ExacTrac, then checked with CBCT, and finally rechecked with ExacTrac before the dose is delivered. The patient is shifted if any of the prescribed corrections exceed a predetermined tolerance. We retrospectively recorded each shift prescribed by the CBCT and the ExacTrac recheck. We compared the two for each patient by calculating the RMS distance between each shift for both the rotational and translational axes.

Results: The average RMS distance between CBCT and ExacTrac translations was 0.43 mm (range: 0.10-1.89 mm, SD: 0.34 mm). Only 1 (3.5%) difference in shift was beyond 1 mm. The average RMS difference between rotations was 0.70° (range: 0.28°-1.83°, SD: 0.38°). 5 (17.9%) rotational differences were beyond a tolerance of 1°.

Discussion: We found a high degree of concordance between prescribed translational shifts of each modality with an average difference well below typical tolerances. The patient with a translational RMS difference >1 mm was shifted only after the final ExacTrac recheck, not the CBCT. This suggests significant patient movement between scans rather than inaccurate measurement. The rotational measurements were less concordant with an average RMS difference approaching typical tolerances. Three of the five patients with differences over 1° were corrected before treatment suggesting increased rotational freedom of movement within the treatment mask. The other two were not corrected because the ExacTrac and CBCT shifts were both within tolerance. Only the difference between the two exceeded typical tolerances. This suggests the presence of slightly more noise along the rotational axes. Notably, every translational shift prescribed by CBCT large enough to necessitate a

shift was also captured by ExacTrac. This suggests that CBCT only confirmed the ExacTrac translations without triggering additional changes in treatment application. Our data shows high concordance between each modality's translational measurement and only marginal discordance between rotational measurements. This call into question the added value provided by CBCT when ExacTrac is already being used. Larger studies would be required to demonstrate the feasibility of ExacTrac-only positioning.

A Real World Comparison of ExacTrac and Cone Beam CT for Image Guided Radiosurgery

Roman Travis[1], Michael Taylor MS[2], John Gleason MD[2]

[1]UAB School of Medicine, Huntsville AL; [2]Alliance Cancer Care Huntsville AL

Background

Stereotactic radiosurgery (SRS) is a mainstay in brain tumor treatment. To deliver SRS, linear accelerators aim high energy photons at brain tumors while sparing nearby healthy tissue.

During delivery, treatment head rotates around the patient while constantly shaping its beam. After one "arc" is completed, the treatment "couch" is rotated allowing for the next arc to spare already treated healthy tissue while centering on the treated lesion.

SRS's highly conformal dose delivery reduces morbidity but requires incredibly precise alignment of the patient to the treatment machine. Previously, Gamma Knife aligned patients using a rigid frame which was screwed onto a patient's head for the entire treatment process. Modern frameless solutions eliminate the painful frame while maintaining accuracy.[3] There a several alignment systems available to ensure submillimeter accuracy.

Alliance Cancer Care employs two redundant bony alignment systems (image 1):

- **Cone Beam CT (CBCT)** – Head CT rapidly acquired by the machine.
  - + Precise imaging of bony anatomy
  - - Cannot be used when the couch is rotated for additional arcs to prevent collision with patient.
- **ExacTrac** – Two orthogonal head x rays used by computers to reconstruct 3D position and prescribe shifts.
  - + Can be acquired at any couch angle. Precise imaging of bone.
  - - Images are not easily interpreted by human operators.

Each measurement with ExacTrac and CBCT produces the 6 shifts required to perfectly align the patient: three in the X-Y-Z translational planes and three around the X-Y-Z rotational axes. If any of these exceeds a calculated tolerance (i.e. 0.8 mm or 0.8°), the shift is applied and rechecked.

Figure 1. The Initial Alignment Protocol at Alliance follows the above protocol. The shift at step 2 is always applied, never applied at step3, and rarely applied at step 4 – normally if the patient moves.

Research Question

Since CBCT requires additional time and resources, this project seeks to determine the concordance between the prescribed shifts of ExacTrac and CBCT by calculating the root mean square (RMS) distance between each shift for a cohort of patients. We then compare this to tolerances used clinically.

Methods

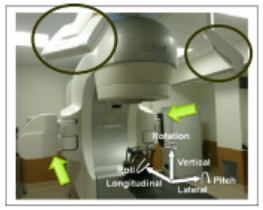

Image 1. Linac Treatment Diagram[1] demonstrating key components and six degree of freedom couch mobility.

Black Circles: Exactrac xray detectors

Green Arrows: Retractable CBCT

White Axis: Arrows demonstrate 6 degrees of freedom - 3 translational and 3 rotational

Study Design

•28 consecutive patients who received SRS at Alliance Cancer Care in 2019 were retrospectively reviewed.

- Only the initial alignment scans with the couch directly inline with the machine were reviewed since CBCT can only be applied there.
- Follow up treatments were excluded to avoid overweighting individual patients.

•The shifts prescribed by the CBCT in step 3 and the ExacTrac recheck in step 4 were recorded for each patient. See table 1.

Please note no shifts were applied between these measurements. The patient's position was unaltered by the system between steps 3 and 4.

•Both predicted shifts for each patient were compared by calculating the root mean square (RMS) difference between each prescribed shift for both the rotational and translational axes demonstrated below in equation 1 - essentially a three dimensional Pythagorean theorem.

$$RMS = \sqrt{(x_c - x_e)^2 + (y_c - y_e)^2 + (z_c - z_e)^2}$$

Equation 1. RMS calculation carried out once for translational and once for rotational shifts.

Results

The average RMS distance between CBCT and ExacTrac translations was 0.43 mm (range: 0.10-1.89 mm, SD: 0.34 mm). Only 1 (3.5%) difference in shift was beyond 1 mm. The average RMS difference between rotations was 0.70° (range: 0.28°-1.83°, SD: 0.38°). 5 (17.9%) rotational differences were beyond a tolerance of 1°.

	RMS Difference Mean	SD (Range)	% Exceeding 1 mm or 1°
Translation	0.43 mm	0.34 mm (0.10 – 1.89 mm)	3.5% (1 patient)
Rotation	0.70°	0.38° (0.28° - 1.83°)	17.9% (5 patients)

Table 1. RMS Difference Between ExactTrac and CBCT

Translational RMS distance averaged 0.43 mm with a range of 0.10 – 1.89mm. 1 patient exceed a strict tolerance of 1 mm. Rotational RMS difference averaged 0.70° with a range of 0.28° - 1.83°). 5 (17.95%) of patients exceeded RMS difference of 1°.

Conclusion

Translational Differences

- **There was a high degree of concordance between prescribed translational shifts of each modality with an average difference well below typical tolerances.**
  - The patient with a translational RMS difference >1 mm was shifted only after the final ExacTrac recheck, not the CBCT. This suggests significant patient movement between scans rather than inaccurate measurement.
  - Notably, every translational shift prescribed by CBCT large enough to necessitate a shift was also captured by ExacTrac. This suggests that CBCT only confirmed the ExacTrac translations without triggering additional changes in treatment application. This aligns with research showing sub-millimeter translational variation between the modalities.[1,2]

Rotational Differences

- **The rotational measurements were less concordant with an average RMS difference approaching typical tolerances.**
  - Three of the five patients with differences over 1° were corrected before treatment suggesting increased rotational freedom of movement within the treatment mask. The other two were not corrected because the ExacTrac and CBCT shifts were both within tolerance.
  - Only the *difference* between the two modalities exceeded typical tolerances. This suggests the presence of slightly more noise along the rotational axes. A prospective study showed smaller rotational variation of around 0.3°.[1] This smaller variation is possibly due to less patient movement in a research setting.

Our data shows high concordance between each modality's translational measurement and only marginal discordance between rotational measurements.

This work calls into question the added value provided by CBCT when ExacTrac is already being used. This is especially true for treatment plans having all tumors closer to the isocenter where rotational differences are minimized.

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Acknowledgements

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UAB Research Day – 2020

Title: Description of an Interprofessional, Community-Based Naloxone Nasal Spray Distribution and Education Events

Authors:

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Category: Education Innovation

Description: The opioid epidemic continues to sweep across the United States, with almost 400,000 people dying from an opioid overdose from 1999-2017.[1] The number of deaths have steadily increased throughout the years, with a spike in deaths due to synthetic opioids. Naloxone is a life-saving medication that can reverse the acute effects of opioid overdose and save lives, but must be available at the time of the overdose.[2] Recent efforts to increase naloxone availability include pharmacy distribution and opioid education and naloxone distribution programs. While naloxone prescriptions and sale have doubled in recent years, there is still a large need to reach the estimated 2.1 million people 12 and older who are affected by opioid use disorder. In Alabama, overdose-related deaths continue to increase while access to naloxone remains challenging.[3] In order to help address this issue, the UAB – Huntsville School of Medicine and Auburn University Harrison School of Pharmacy established community partners with Not One More Alabama (NOMA) and the Alabama Department of Mental Health in 2018. The goals of this partnership were to 1) educate the public on opioid overdose and naloxone use and 2) increase the access and distribution of naloxone in our community.

Methods: Since 2018, the UAB School of Medicine and Auburn University Harrison School of Pharmacy have participated in community-based events sponsored by NOMA to provide education and training to victims, friends, family members, and community members affected by opioid use disorder. Community-based events in Northern Alabama are held by NOMA and staffed by UAB and Auburn participants. Participants include medical and pharmacy students, medical and pharmacy residents, and faculty members. At the events, community members are educated by participants on recognizing signs and symptoms of opioid overdose, how to properly administer naloxone nasal spray (Narcan), and what to watch for after naloxone is administered. After recipients verify their understanding, they are given a naloxone nasal spray kit, which includes two doses.

Results: More than 30 students, residents and faculty have participated in these education and distribution events. Since 2018, a total of 534 kits (1,068 doses) of naloxone have been distributed at five events. At the largest event, the End Heroin Huntsville Walk, over 200 kits were distributed in 2018 and 2019. Additionally, UAB and Auburn have been able to increase their community presence and raise awareness and understanding in the community through these events.

Conclusion: Education on opioid overdose symptoms and distribution of naloxone nasal spray use at community-based events has increased the Huntsville area awareness of the opioid epidemic as well as access to this lifesaving medication.

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Description of Interprofessional, Community-Based Naloxone Nasal Spray Distribution and Education Events

THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

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Background

- 400,000 people have died from opioid overdose from 1999-2017.[1]
- Naloxone reverses effects of acute opioid overdose, must be available at time of overdose.[2]
- Efforts to increase naloxone:
  - Pharmacy distribution and opioid education
  - Naloxone distribution programs
- Naloxone prescriptions have doubled in recent years, but still 2.1 million people affected by opioid use disorder (OUD).[2]
- In Alabama, overdose-related deaths increase while access to naloxone remains challenging.[3]
- In 2018, UAB – Huntsville School of Medicine and Auburn University Harrison School of Pharmacy established community partners with Not One More Alabama (NOMA) and the Alabama Department of Mental Health to address this challenge.
- The goals of this partnership were to 1) educate the public on opioid overdose and naloxone use and 2) increase the access and distribution of naloxone in our community.

Methods

- Community-based naloxone distribution events
  - Not One More Alabama (Host)
  - UAB School of Medicine (Staff)
  - Auburn University Harrison School of Pharmacy (Staff)
- Participants include:
  - Medical/pharmacy students
  - Medical/pharmacy residents
  - Medical/pharmacy faculty
- Participants provide education and training to victims, friends, family members, and community members affected by OUD
  - Recognizing signs/symptoms of opioid overdose
  - How to properly naloxone nasal spray (Narcan) and handle overdose
  - What to watch for after naloxone administration
- After recipients verify their understanding, they are given a naloxone nasal spray kit, which includes two doses.

Results

- More than 30 students, residents, and faculty have participated
- Since 2018, a total of 534 kits (1,068 doses) of naloxone have been distributed at 5 events
- The End Heroin Huntsville walks are the largest events and have resulted in over 200 kits distributed in 2018 and 2019.
- UAB and Auburn have increased their community presence and raised awareness and understanding in the community through these events

Pictures from Events

Nalaxone Kits

Students and residents educating community members

UAB and Auburn personnel at the 2019 End Heroin HSV Walk

Discussion

- Events have been well received among community members and participants
- These events and partnership have increased access to naloxone for members of the community who are close to someone at risk of opioid overdose
- These events and partnership have helped raise awareness of the opioid epidemic in the northern Alabama community

Future Directions

- Continue this partnership and holding these events with the goal of increasing awareness of the opioid epidemic and access to naloxone
- Continue education on opioid overdose symptoms and the role of naloxone
- Leverage this partnership to explore other opportunities to combat this epidemic (i.e. grant funding, other initiatives, etc.)

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Knowledge that will change your world

Direct Primary Care in Rural Communities

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University of Alabama School of Medicine

- dtrotman@uab.edu
- Research abstract

Background
Many primary care physicians in rural communities are struggling with balancing efforts of accommodating a greater patient panel while not sacrificing the quality of care provided. Primary care physicians are seeing more patients and also allotting longer hours to nonclinical paperwork, thus increasing burnout rates in these rural areas. The recent increase in number of DPC clinics across the United States could eventually improve physician shortages in underserved communities.

Methods
A thorough literature search was used to define direct primary care, using PubMed and Google Scholar for "direct primary care", "concierge medicine", and "retainer medicine". The DPC Frontier website (https://mapper.dpcfrontier.com) was used to obtain contact information for the clinics for the 10 DPC clinics in the state of Alabama. These clinics were then contacted to collect data regarding the DPC clinic's services.

Results
Each DPC clinic on average had 2.0 ± 0.2 employees. Seven of the ten clinics were considered rural with populations less than 50,000. The average membership fee per individual was $60.5 ± 2.7. Average rates for couples were $52.5 ± 2.4 per individual. appointment lengths were on average, 48.3 ± 4.6 minutes. Eight of the ten DPC clinics dispensed medication at the clinic at wholesale prices. All ten DPC clinics offered discounted labs, discounted radiologic imaging services, and 24/7 direct communication with the DPC physician.

Conclusions
The services provided by the DPC clinics throughout Alabama included medications at discounted costs, basic lab tests, discounted radiologic imaging, as well as nutrition and exercise plans. Telemedicine resources such as RubiconMD allow the patient and physician to discuss more complex scenarios with specialists, obtain diagnosis and treatment knowledge immediately while still in the exam room. DPC physicians in rural communities could utilize services and consult multiple specialists at once, exponentially improving the quality of care with instant communication and clarity.
Direct Primary Care can offer more affordable healthcare to many individuals in rural communities with recent increases in insurance costs. An analyzed insurance premium average for Alabama in 2019 was $546, up from $256 in 2014, using the second-lowest cost silver (benchmark) insurance premium with a $3000 deductible for a 40-year-old, resulting in a total average premium cost for a year of $6,552. The average individual DPC membership in Alabama was $60.50 per month for an annual total of $726. DPC members can switch to plans with lower monthly premiums such as Libertyshare, which offers coverage with deductibles of $1,000 for an individual. DPC practices help to improved continuity, accessibility, and quality of care with the belief that these practices will help lower hospitalization rates, emergency department visits, and health care cost.

**This abstract was chosen for oral presentation on Research Day; poster provided for compendium.*

Direct Primary Care in Rural Communities

Dusty Trotman, MS3; David Bramm, M.D.

University of Alabama at Birmingham

Background

Many primary care physicians in rural communities are struggling with balancing efforts of accommodating a greater patient panel while not sacrificing the quality of care provided. Primary care physicians are seeing more patients and allotting longer hours to nonclinical paperwork, thus increasing burnout rates in these rural areas [1]. The recent increase in the number of DPC clinics across the United States could eventually improve physician shortages in underserved communities.

Methods

1. A thorough literature search was used to define direct primary care, using PubMed and Google Scholar for "direct primary care", "concierge medicine", and "retainer medicine".
2. The DPC Frontier website (https://mapper.dpcfrontier.com) tracks the DPC clinics throughout the US and includes contact information for the clinics, which was used to obtain locations and contact methods for the 10 DPC clinics in the state of Alabama as of October 2019.
3. The data from these clinics included the average number of staff in these clinics, subscription expenses, and additional services are provided to members (labs, medications, nutrition).
4. Then, our study focused on rural DPC clinics (defined as areas with populations fewer than 25,000 residents) and what primary care services are provided by the DPC models to the rural patient populations.

Figure 1. DPC Frontier website (https://mapper.dpcfrontier.com) to locate and contact the DPC clinics in Alabama (October 2019) [2].

Results

	Averages in Alabama
Employees per DPC clinic	2.0 (0.67)
Individual subscription price	$60.50 (8.64)
Couples subscription price	$52.50 (7.64)
Appointment time length	48.3 minutes (14.33)
Medication dispensary at clinic (%)	8 (80)
Discounted labs (%)	10 (100)
Discounted radiologic imaging (%)	10 (100)

Table 1. Direct Primary Care Clinic Statistics in Alabama.
Values are (SD) unless otherwise stated.

	Average DPC Costs - Alabama	Employer-Based – Alabama	Individual Silver plan - Alabama	Individual Silver plan - U.S.
Annual Costs or Premium	$726	$1,453	$6,552	$5,736
Catastrophic insurance	$1,000			
Average Deductibles	$3,288	$1,655	$3,000	$3,000
Annual Costs	$5,014	$3,108	$9,552	$8,736

Table 2. Alabama 2019 Insurance Premium Averages.
The average insurance plan was found using the second-lowest cost silver (benchmark) insurance premium for a 40-year-old for both Alabama and the United States. *Kaiser Family Foundation Marketplace Average Benchmark Premiums*[3]. Catastrophic insurance costs found using Liberty Health Share [4].

Labs	DPC prices	Retail	Savings (%)
CBC/diff	$1.50	$14.00	89.3
CMP	$3.43	$18.00	80.9
Hg A1c	$2.00	$17.00	88.2
Lipid Panel	$3.00	$23.00	86.9
TSH	$1.50	$29.00	94.8

Table 3. Example of Lab Prices Offered at an Alabama DPC Clinic.
Labs were ordered through LabCorp and dispensed at wholesale prices. Retail prices obtained using the Fair Price value through Healthcare Bluebook for Huntsville, Alabama[4].

Conclusion

- **Alabama has opened ten total DPC clinics since the state passed a DPC-defining law in 2017 [2].**
- **DPC physicians are reducing administrative workloads to allot more time for providing quality patient care. Rural healthcare could impact the most from DPC practices through the services offered.**
- **The services provided by the DPC clinics throughout Alabama include medications at discounted costs, wholesale labs, discounted radiologic imaging, as well as gym memberships, nutrition and exercise plans.**
- **By dispensing discounted medications in office, the patient obtains the medications before they leave the office, which provides improved compliance through routine tracking.**
- **Due to recent increases in insurance costs, Direct Primary Care can offer more affordable health care to many individuals in rural communities. An analyzed individual insurance premium for Alabama in 2019 was $546 on average, up from $256 in 2014, resulting in a total annual premium cost of $6,552 [3].**
- **DPC along with catastrophic insurance such as Liberty Health Share provides affordable coverage compared to individual insurance through a Silver plan. However, DPC with catastrophic plans is less viable financially compared with individuals with excellent employer-based insurance.**
- **DPC practices help to improved continuity, accessibility, and quality of care with the belief that these practices will help lower hospitalization rates, emergency department visits, and overall health care costs.**

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THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

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William Coleman, MD, Office for Family Health Education & Research, UAB School of Medicine Huntsville Regional Medical Campus, Director
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Submission Category: Research- Education Innovation

Title: Evaluating 'Tar Wars' and tobacco use habits among adolescents in two rural Alabama communities: a 7-year study.

Description: The long-term use of tobacco and nicotine containing products cause health problems encompassing the highest number of preventable deaths in the country. A decrease in tobacco-related health problems can only be accomplished if people no longer use tobacco and other nicotine containing products. Tobacco and nicotine prevention begins with youth education. One such tobacco prevention program is Tar Wars, developed by the American Academy of Family Physicians. This research involved a 7-year longitudinal analysis of the continued effectiveness of the Tar Wars program after its completion by a student.

Methods: An annual survey was conducted in two similar Alabama public school systems to evaluate retention of the Tar Wars information and document tobacco use among the students. An average of 1244 students per year in grades 5-12 were surveyed across 7 years (2012-2019). Students in the Scottsboro City School System were the "Treatment" group who received Tar Wars instruction in the 5th grade. Scottsboro used a paper format survey. Students in the Fort Payne City School System were the "Control" group who did not have the Tar Wars program. Fort Payne used an electronic format of the survey. The survey data from both school systems was analyzed for relevant statistics.

Results: The data indicated exposure to the Tar Wars program did not decrease overall tobacco use. However, overall tobacco use decreased in both the Treatment and Control groups from 10.80% in 2012 to 2.72% in 2019. While traditional cigarette use decreased throughout the survey years, 2017 saw a significant spike in vaping and e-cigarette use that also rose in 2018. Additionally, while not fully related to the Tar Wars program, the study found a possible correlation between a student's exposure to tobacco in the home and the likelihood of early tobacco use. Approximately 22% of students living with tobacco users have tried tobacco products themselves in comparison with 7% of students form non-tobacco homes having tried tobacco products.

Discussion: This 7-year longitudinal study found Tar Wars to be effective in increasing students' knowledge of tobacco in the short term, but there was no statistical evidence that the program decreases tobacco use among students as they progress into their teenage years. In addition, the study highlights an increasing use of e-cigarettes in the under-18 population. The results of this study led the two school systems to now participate in a newly developed tobacco and nicotine education program with lessons and activities for students throughout their middle and high school years.

Evaluating Tar Wars® and Tobacco use Habits Among Adolescents in Two Rural Alabama Communities: a 7-year study

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Office for Family Health Education & Research, UAB School of Medicine Huntsville Regional Medical Campus
University of Alabama at Birmingham

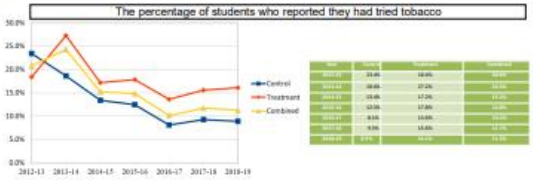
Introduction

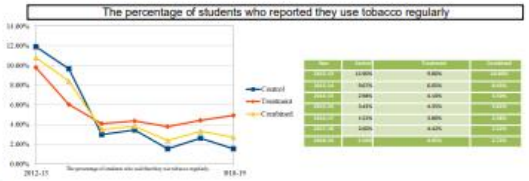
- The long-term use of tobacco and nicotine products cause health problems accounting for the highest number of preventable deaths in the country.
- Tobacco and nicotine prevention begins with youth education.
- Tar Wars® is a tobacco prevention and education program developed by the American Academy of Family Physicians in 1995
- This study is the culmination of a 7-year longitudinal analysis of the continued effectiveness of the Tar Wars® program after its completion, conducted by UAB medical students

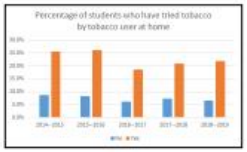
Methods

- The survey was administered in two similar Alabama public school systems to evaluate retention of the Tar Wars® information and to document tobacco use among the students.
- The Scottsboro and Fort Payne city school systems were chosen because of their similarities in geography and population size
- Students in the Scottsboro system were the "Treatment" group who received Tar Wars® instruction in the 5th grade.
- Students in Fort Payne were the "Control" group who did not participate in the Tar Wars® program
- 1244 students per year in grades 5-12 were surveyed across 7 years (2012-2019).
- The survey data was analyzed for relevant statistics.

Results







Discussion

- While exposure to Tar Wars® was not shown to have a longitudinal impact on tobacco use, the data indicates overall tobacco use decreased in both the Treatment and Control groups from 10.80% in 2012 to 2.72% in 2019.
- Although traditional cigarette use decreased throughout the survey years, 2017 saw a significant spike in vaping and e-cigarette use that also rose in 2018.
- As an incidental finding, the data indicates a possible correlation between a student's exposure to tobacco in the home and the likelihood of early tobacco use.

Conclusion

- This 7-year study found Tar Wars® to be effective in increasing students' knowledge of the effects of tobacco **use** in the short term, but there was no statistical evidence that the program decreases tobacco use among students as they progress into their teen years.
- The study highlights an increasing use of e-cigarettes in the under-18 population.
- The results of this study led the two school systems to help develop and participate in a new tobacco and nicotine education program with lessons for students throughout their middle and high school years.

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Acknowledgements & Contact Information

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THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

Multiplex Polymerase Chain Reaction (PCR) Panels in Pediatric Hospital Care: New Insights into Factors Driving Antimicrobial Use.

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(3) Women & Children's Hospital - Huntsville Hospital. (4) University of Alabama at Birmingham (UAB) School of Public Health.

Description:

Multiplex PCR panels are diagnostic tools that have become a part of regular pediatric hospital care. These panels allow identification of a wide spectrum of microbial targets in samples from the respiratory tract and cerebrospinal fluid (CSF). They have a rapid turnaround time and excellent sensitivity and specificity. However, it remains controversial whether their use leads to optimal use of antimicrobials in different hospital units. We aimed to determine if the use of several multiplex PCR panels was associated with appropriate antimicrobial therapy (AAT) in hospitalized children. We also sought to describe clinical and laboratory factors influencing antimicrobial decisions.

Methods:

We conducted a single-center, retrospective study of hospitalized pediatric patients from January 2015 to December 2018 who underwent testing with 1 or more multiplex PCR panels, including 4 different respiratory panels and 1 meningoencephalitis panel (MEP). We analyzed multiplex PCR panel results and their subsequent impact on antimicrobial treatment. Using logistic regression, we analyzed the clinical and laboratory factors associated with AAT (defined as targeted antimicrobial therapy based on clinical assessment and tests results).

Results:

There was a total of 1,002 PCR tests (817 respiratory tests and 185 MEP) in 951 patients during the study period. Mean length of hospital stay was 7 days. A total of 53.2% patients were admitted to the pediatric ICU. Test results were positive in 77.1% of respiratory panels and 16.7% of MEP. Co-detection was present in 44.2% of respiratory panels. Co-detection with bacteria was more commonly detected with Respiratory syncytial virus (RSV) as compared to Enterovirus or Rhinovirus. No co-detection was identified in MEP. Patients admitted to the floor were more likely to have AAT than ICU patients (82.5% vs 71.7%). ICU admission increased the odds of unnecessary antimicrobials [OR 1.6 95% CI 1.1-2.5]. A positive test result including bacteria + virus resulted in decreased odds of AAT [OR 0.4, 95% CI 0.3-0.8].

Statistical analysis concluded that age, CBC, CRP, season of sampling, comorbidity, and intubation were not significantly associated with AAT.

Conclusions:

We present new insights into factors driving antimicrobial use in pediatric hospital care who received multiplex PCR testing. ICU admission was significantly associated with unnecessary antimicrobial use after adjusting for potential confounders. Common tests ordered during hospital admission as CBC, CRP, blood culture and chest x-ray did not have an impact on antibiotic therapy decision-making. Frequently PCR results were either not acted upon or caused additional use of antimicrobials. Further investigation is warranted to better understand factors influencing antimicrobial use in pediatric hospitals.

This abstract was chosen for oral presentation on Research Day.

Reinforcement learning abnormalities in individuals at clinical high risk and in the first episode of psychosis

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Research Abstract

Background

Prior studies indicate that chronic schizophrenia (SZ) is associated with a specific profile of reinforcement learning abnormalities. These impairments are characterized by: 1) reductions in learning rate, and 2) impaired Go learning and intact NoGo learning. Furthermore, each of these deficits are associated with greater severity of negative symptoms, consistent with theoretical perspectives positing that avolition and anhedonia are associated with deficits in generating, updating, and maintaining mental representations of reward value hat are needed to guide decision-making. However, it is unclear whether these deficits extend to earlier phases of psychotic illness and when individuals are unmedicated.

Methods

Two studies were conducted to examine reinforcement learning deficits in earlier phases of psychosis. In study 1, participants included 35 participants with first episode psychosis (FEP) and 25 healthy controls (HC). Study 2 included 17 antipsychotic naïve individuals who met criteria for attenuated psychosis syndrome (APS) (i.e., those with a prodromal syndrome) and 18 matched healthy controls (HC). In both studies, participants completed the Temporal Utility Integration Task, a measure of probabilistic reinforcement learning that contained Go and NoGo learning blocks. Participants in the clinical groups also completed neuropsychological testing and standard clinical interviews designed to determine symptom severity and diagnosis.

Results

FEP displayed impaired Go learning and intact NoGo learning. In contrast, APS did not display impairments in Go or NoGo learning at the group level. Negative symptoms were not significantly associated with reinforcement learning in APS participants. However, greater impairments in Go learning were associated with increased cross-sectional risk for conversion on the NAPLS risk calculator score in the APS group.

Discussion

Findings provide new evidence for areas of spared and impaired reinforcement learning in early phases of psychosis. Similar to chronic SZ, FEP was associated with impaired Go learning, and intact NoGo learning. Reinforcement learning is more spared in those at clinical high-risk, except those at greatest risk for conversion, where Go learning deficits are more pronounced. These findings suggest that reinforcement learning deficits may emerge early among those who are at clinical high risk for developing psychosis and that they are already pronounced by illness onset in the first episode. Importantly, these reinforcement learning deficits do not appear to be a byproduct of illness chronicity or antipsychotic medication use, but rather a consequence of the illness itself.

Poster was self-printed and PDF file was not available for this compendium.

Kaaren Royster, BS, Alabama A&M University, MSW Intern

Jacob Calahan, Emily Blaine, Mary Clinton, Auburn University Harrison School of Pharmacy, PharmD Candidates 2020

Miranda Andrus and Haley Phillippe, PharmD, Auburn University Harrison School of Pharmacy

Meredith Lewis, PhD, LICSW, UAB Huntsville, Family Medicine Residency, Behavioralist

Submission Category: Research-Education Innovation

Title: Reinforcing Effective Diabetes Education in the Outpatient Setting

Diabetes continues to be a major health issue. In the US alone, there are 30 million patients affected with diabetes and about one in four of them are unaware. Up to 95% of all diabetic cases are Type 2 Diabetes Mellitus (T2DM). Risk factors for T2DM include visceral obesity, age $\geq$ 45 years old, family history, race (African American, Hispanic American and Asian American) and physical activity <3 times per week. People with diabetes are at a higher risk of complications such as kidney disease, heart disease, amputations, and eye complications. Through appropriate education and diabetes management, such as healthy eating habits, physical activity, and adherence to pharmacological therapy, patients can achieve improved outcomes and quality of life.[1,3]

Methods: In July 2015, Family Medicine Center began offering diabetes education groups to patients. The group included resident physicians and pharmacy staff. Social work staff works as the gatekeepers managing the group referrals ordered by Family Medicine Physicians using the electronic medical record. The monthly group schedule is emailed to physicians to reinforce referral options for patients. Each patient referred to a group is mailed an invitation letter. The letter includes details about the group to include meeting time, location, and the assigned staff for the group. Lastly, to recruit for a group, patients receive a reminder call about 48 hours before the group day.

Patients were educated in a group setting on multiple factors that influence their diabetes including healthy eating habits, physical activity, and T2DM pharmacotherapy. During the class, we used the plate method to interact with patients about how to improve their eating habits to ensure a well-balanced diet and proper carbohydrate consumption. We informed patients on physical activity goals to help reduce weight, maintain healthy glucose levels, and improve their overall health. This was achieved by reviewing the pathophysiology of diabetes, the complications of uncontrolled diabetes, symptoms of hyper- and hypoglycemia, and proper preventative measures (foot care, eye care, and remaining up to date on vaccines) with the class. All information was provided in patient-friendly terms to ensure complete understanding.

Results & Discussion: Since 2015, 68% of patients referred to a diabetic group attended with a total of 132 encounters. Engagement for group attendance starts with effective communication. Effective communication is the ability of healthcare providers to clearly articulate the task or recommendation to be completed by the patient (Mehl, 2019). Education in a group setting was pivotal for motivating patients and offering accountability. Barriers identified for patients included introverted patients being overshadowed by extroverted patients during the group session. Other barriers included obstacles in attending the group session itself, such as transportation issues or other obligations (i.e. work).

THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

REINFORCING EFFECTIVE DIABETES EDUCATION IN THE OUTPATIENT SETTING

KAAREN ROYSTER, BS, MSW CANDIDATE 2020; JACOB CALAHAN, EMILY BLAINE, MARY CLINTON, PHARMD CANDIDATES 2020; MIRANDA ANDRUS, HALEY PHILLIPPE, PHARMD; MEREDITH LEWIS, PHD, LICSW

Introduction

Diabetes continues to be a major health issue. In the US alone, there are 30 million patients affected with diabetes and about one in four of them are unaware.

Up to 95% of all diabetic cases are Type 2 Diabetes Mellitus. People with diabetes are at a higher risk of complications such as kidney disease, heart disease, amputations, and eye complications. Through appropriate education and diabetes management, such as healthy eating habits, physical activity, and adherence to pharmacological therapy, patients can achieve improved outcomes and quality of life.

Methodology

In July 2015, the UAB Family Medicine Center began offering diabetes education groups to patients. The group includes resident physicians and pharmacy staff. Social work staff manage the group referrals ordered by Family Medicine Physicians. Each patient referred to a group is mailed an invitation letter. The letter includes the meeting time, location, and the assigned staff for the group. Patients receive a reminder 48 hours before class.

The Class

Patients were educated in a group setting on multiple factors that influence their diabetes including healthy eating habits, physical activity, and T2DM pharmacotherapy. During the class, we used the plate method to interact with patients about how to improve their eating habits to ensure a well-balanced diet and proper carbohydrate consumption. We informed patients on physical activity goals to help reduce weight, maintain healthy glucose levels, and improve their overall health. This was achieved by reviewing the pathophysiology of diabetes, the complications of uncontrolled diabetes, symptoms of hyper- and hypoglycemia, and proper preventative measures (foot care, eye care, and remaining up to date on vaccines) with the class. All information was provided in patient-friendly terms to ensure complete understanding.

Results

Since 2015, 68% of patients referred to a diabetic group attended with a total of 132 encounters. Barriers identified for patients included introverted patients being overshadowed by extroverted patients during the group session. Other barriers included obstacles in attending the group session itself, such as transportation issues or other obligations (i.e. work).

Conclusion

Engagement for group attendance starts with effective communication. Effective communication is the ability of healthcare providers to clearly articulate the task or recommendation to be completed by the patient. Education in a group setting was pivotal for motivating patients and offering accountability.

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Meredith Lewis, PhD, LICSW, UAB Huntsville, Family Medicine Center, Clinical Social Worker

Hunter French, MD, UAB Huntsville, Family Medicine Center Residency, Resident Physician

Submission Category: Research-Education Innovation

Title: Take to the Stage: Intersections of Integrative Healthcare, Human Development, and Psychological Recovery

Description: At the core of patient-centered practice—it must be stated: Patients are not simply MRN numbers. Contextualizing patient's circumstances ultimately yields to greater psychotherapy intervention efficacy, using collaborative opportunities within an integrative healthcare setting. Utilizing Erikson's Stages of Development and Vogel-Scibilia et al.'s Stages of Psychological Recovery Model while assessing our patient data allows insight on how factors influence the respective crisis stage. From frequent "no-shows" to medical non-adherence, unhealthy patient practices are not always simple matters of will.

Methods: A retrospective chart review of patients from August 2019 to November 2019, were reviewed for data analysis. During this review, demographics were collected to include the patient's name, gender, race, diagnosis, and chief complaint. At the end of the chart review, data were analyzed to discover trends comparing Erikson's Stages of Development and Vogel-Scibilia et. al's Stages of Psychological Recovery Model respectively. Conclusions were drawn.

Results: For this chart review, 55 patients attended counseling sessions for 161 encounters. The patient's age range from 5 years old to 76 years old. Patients included 25% male and 75% female. 51% African American, 45% Caucasian American, and 4% Asian/White Latino. 52.7% of patients were in Erickson's *Generativity vs. Stagnation* and *Vogel-Scibilia et al.'s Purpose vs. Passivity*, respectively. 1.8% were in Erikson's *Initiative vs. Guilt* and Vogel-Scibilia et al.'s *Empowerment vs. Guilt*, respectively. 29% of patients were in the *Intimacy vs. Isolation* stage of both frameworks. 3.6% of patients were in the *Integrity vs. Despair* stage of both frameworks.

Discussion: Social work provides an invaluable asset to providing quality care to patients with mental health diseases. Consulting Erikson's Stages of Development and Vogel-Scibilia et al.'s Stages of Psychological Recovery Model provides an integral context for patient experience and orientation toward recovery within a healthcare setting. Mental health workers allow a primary care provider to treat a broader range of diseases within the behavioral health spectrum. Some diseases may not

respond to medications - and if they do will only see improvement in conjunction with cognitive-behavioral or another therapy provided by social and mental health workers.

Several conclusions are drawn from the analyzed data. First, over half of the patients attending psychotherapy were in the *Purpose vs. Passivity* stage (40-65 years of age). As psychotherapy provides a neutral atmosphere for change and goal development, it is assumed patients psychotherapy as an effective tool in navigating the crisis between attaining purpose and passively experiencing life. Furthermore, patients falling within *Integrity vs. Despair* chief complaints of dementia diagnosis, extensive loss, and physical decline. These experiences would be consistent with this crisis stage—marked by a sense of life reflection and reconciliation. Findings inform providers understanding of patients and the implementation of more compassionate, patient-centered practice.

THE UNIVERSITY OF ALABAMA AT BIRMINGHAM

Take to the Stage:

Intersections of Integrative Healthcare, Human Development, and Psychological Recovery

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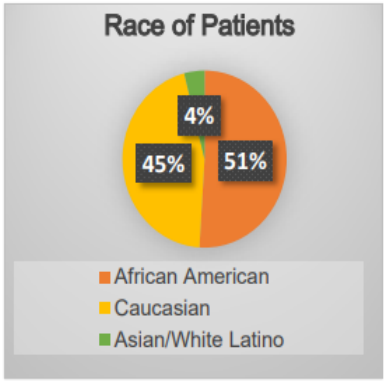
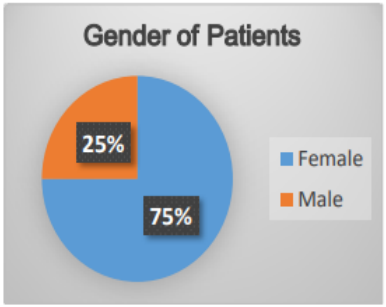
Introduction

At the core of patient-centered practice, it must be fully understood that patients are not simply MRN numbers. Contextualizing client circumstances ultimately yields to greater intervention efficacy. Utilizing Erikson's ***Stages of Development*** and Vogel-Scibilia et al.'s ***Stages of Recovery Model*** against our patient data allows us to gain possible insight on how an individual's journey might be influenced by his or her respective crisis stage. From frequent "no-shows" to medical non-adherence, unhealthy patient practices are not always simple matters of will power. The context provided by Erik Erikson's ***Stages of Development*** and Vogel-Scibilia et al.'s ***Stages of Recovery*** allows for even greater collaborative opportunities within an integrative healthcare setting.

Methodology

A retrospective chart review of patients from August 2019 to November 2019 was conducted for data analysis. During this review, demographics were collected to include the patient's name, gender, race, diagnosis, and chief complaint. At the end of the chart review, data were analyzed to discover trends comparing Erikson's ***Stages of Development*** and Vogel-Scibilia et. al's ***Stages of Psychological Recovery Model*** respectively. Conclusions were drawn.





Results

For this chart review, 55 patients attended counseling sessions for 161 encounters. The patient's age range from 5 years old to 76 years old. Patients included 25% male and 75% female. 51% African American, 45% Caucasian American, and 4% Asian/White Latino. 52.7% of patients were in Erickson's Generativity vs. Stagnation and Vogel-Scibilia et al.'s Purpose vs. Passivity, respectively. 1.8% were in Erikson's Initiative vs. Guilt and Vogel-Scibilia et al.'s Empowerment vs. Guilt, respectively. 29% of patients were in the Intimacy vs. Isolation stage of both frameworks. 3.6% of patients were in the Integrity vs. Despair stage of both frameworks.

Stage	Percentage
Initiative vs. Guilt Empowerment vs. Guilt	1.8%
Identity vs. Role Confusion New Self vs. Sick Self	12.7%
Intimacy vs. Isolation	29%
Generativity vs. Stagnation Purpose vs. Passivity	52.7%
Integrity vs. Despair	3.6%

Conclusion

Consulting Erikson's ***Stages of Development*** and Vogel-Scibilia et al.'s ***Stages of Recovery Model*** provides integral context for patient experience and orientation toward recovery within a healthcare setting. Several conclusions can be drawn from the gathered data. As it relates to the recovery model, over half of attending patients within the population were in the *Purpose vs. Passivity* stage. As psychotherapy provides a neutral atmosphere for change and goal development, it appears that use of counseling services is viewed as an effective tool in navigating the crisis between attaining purpose and passively experiencing life. Furthermore, patients falling within *Integrity vs. Despair* dealt with chief complaints of dementia diagnosis, extensive loss, and physical decline. These experiences would be consistent with this crisis stage—marked by a sense of life reflection and reconciliation. Findings inform understanding of clients and implementation of more compassionate, client-centered practice.

References

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