Central Diabetes Insipidus caused by Therapeutic Hypothermia after Cardiopulmonary Resuscitation with Carbon Monoxide Poisoning：A Case Report

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Research

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Abstract

Central diabetes insipidus(DI) usually has hypernatremia and increased urine output as the main clinical manifestations. It is also a rare complication of therapeutic hypothermia after cardiopulmonary resuscitation and carbon monoxide poisoning, but it may be fatal if it is not recognized in time. This case describes a patient who experienced cardiac arrest due to carbon monoxide poisoning, and then successfully restored his spontaneous heart rate after cardiopulmonary resuscitation. However, the patient experienced unexpected hypernatremia and increased urine output during therapeutic hypothermia, and was diagnosed with central DI as a complication of cerebral edema. After treatment, he eventually developed spontaneous breathing and corrected electrolyte imbalances.Central DI should be taken seriously as a possible complication of increased urine output during therapeutic hypothermia after carbon monoxide poisoning cardiopulmonary resuscitation, and pituitary vasopressin should be used to treat central DI.

Introduction

Severe carbon monoxide poisoning is usually caused by deliberate poisoning, accidental fire toxic gas exposure and inhalation injury[1]. The cognitive dysfunction, which is a characteristic of carbon monoxide poisoning, is also aggravated with cerebral edema and persistent brain damage[2]. Therefore, special intensive care and support are necessary, which is also identify by the American Heart Association as the fifth chain of survival in advanced cardiopulmonary life support[3]. After successful recovery of spontaneous circulation in cardiac arrest patients, hypothermia therapy (TH) has become the standard therapy for brain protection, and TH has also been recommended as an important protect method for neuroprotection in carbon monoxide poisoning[4, 5].

Central diabetes insipidus (DI) is caused by a lack of antidiuretic hormone, and the mainly clinical manifestations are hypernatremia and polyuria with hypertonic dehydration[6]. The most common causes of central DI are tumors and trauma in the neuropituitary area, but in a few cases it can also occur by severe hypoxic/ischemic brain injury[7]. Therefore, the central DI during therapeutic hypothermia is greatly increased after successful cardiopulmonary resuscitation, especially with carbon monoxide poisoning aggravates brain damage. We describe here a patient who experienced central DI due to complications from cardiac arrest caused by carbon monoxide poisoning after resuscitation and treated with therapeutic hypothermia.

Case Description

Chief complaints

A 40-year-old male patient was found fainted at the scene of the fire and had a cardiac arrest.

History of present illness

The patient was receive CPR and sent to the emergency department of the Strategic Support Force Specialized Medical Center by firefighters on June 27, 2021 .Rescue measures such as strengthening the heart, supplementing blood volume, and assisting ventilation under tracheal intubation. After 30 minutes, the patient's spontaneous circulation (ROSC) was restored. In order to confirm the diagnosis, related examinations such as arterial blood gas analysis, TCD, and CT examination of the chest and brain were performed. According to the examination results, he was diagnosed with severe carbon monoxide poisoning, cerebral edema, severe brain injury, and 10% superficial second-degree burn of the skin. After being given high concentration oxygen (60%) combined with Peep (6cmH20), brain dehydration and skin burns, he was transferred to the ICU for advanced life support.

When he arrived in the ward, Sedated to reduce irritation and started using external cooling mattresses and headgear for therapeutic hypothermia. To protect the burned airway, a tracheotomy was performed under local anesthesia. Within 8 hours after ROSC, she reached the target body temperature (35°C) and head temperature (33°C). Therapeutic hypothermia is maintained for 24 hours. Due to the discovery of gastrointestinal bleeding, an emergency gastroduodenoscopy was performed, and the results showed hemorrhagic gastritis. The bleeding stopped after treatment with local thrombin. After 19 hours of therapeutic hypothermia, the patient developed polyuria (urine volume per hour>1000mL), total urine volume was 6.5L (input volume 4.3L).

History of past illness

The patient had hypertension.

Personal and family history

The patient had no history of smoking and familial cancer, but the patient drank about 800ml of beer before the accident.

Physical examination

The patient was still in a coma. The pupil does not respond to light, and the corneal reflection is not elicited. Vital signs: blood pressure 148/81 mmHg, heart rate 97 beats/min, body temperature 36.0°C, mechanical ventilation intubation (A/C mode, frequency 20 beats/min, oxygen concentration 60%, Peep6cmH20), SpO297%.

Laboratory examinations

When he arrived in ICU. Preliminary laboratory results showed that hemoglobin was 20.6 g/dL, carboxyhemoglobin was 40%, pH 7.29, Pco251.2mmhg, Po285.7mmhg, sodium 141.4mM/L, potassium 3.4mM/L, chloride 110.7mM/L.After 19 hours of therapeutic hypothermia,blood sodium concentration was 155.0mM/L the next day, and it rose to 165.8mM/L within three days, See Figure 1 for details. Urine specific gravity continued to be less than 1.005.

Imaging examinations

Head CT disclosed ventricular contraction and parenchyma edema(Figure 2),signaling brain edema.

Final Diagnosis

Central diabetes insipidus secondary to therapeutic hypothermia after cardiopulmonary resuscitation with carbon monoxide poisoning.

Treatment

The diagnosis of central diabetes insipidus was diagnosed. The patient received pituitary 24U (8ml/h) treatment. In the following 3 days, urine volume gradually decreased, blood osmotic pressure, urine osmotic pressure, blood sodium concentration and carbon Oxyhemoglobin returned to normal.

Outcome And Follow-up

In the afternoon of the 4th day after ROSC, the patient developed weak spontaneous breathing and was transferred to a secondary hospital by his family to continue treatment.

Discussion

In recent years, therapeutic hypothermia therapy has been widely regarded as one of the most reliable neuroprotective therapies for the treatment of various brain diseases and injuries, including hypoxic-ischemic encephalopathy caused by various reasons such as stroke, trauma, and cardiac arrest [8]. Therapeutic hypothermia therapy affects the increase of intracellular calcium ions and the release of excitatory neurotransmitters, leading to mitochondrial dysfunction and increased production of reactive oxygen species, causing central complications[9], but the specific mechanism is believed to be unable to be explained separately Its protective effect in the neurological field[10]. However, it is certain that therapeutic hypothermia can improve the clinical outcome of coma survivors of hypoxic-ischemic encephalopathy [11].

Central diabetes insipidus is a heterogeneous disease characterized by polyuria and hypernatremia caused by a lack of antidiuretic hormone. In many ischemic and hypoxic brain injuries, central diabetes insipidus can be caused by the destruction of neurons in the supraoptic nucleus and paraventricular nucleus of the hypothalamus or neuronal edema. Studies have shown that the probability of central diabetes insipidus in patients with irreversible brain injury is more than 11% [12], and only one death case reported central diabetes insipidus during therapeutic hypothermia [13]. Therefore, we emphasize that patients who are given therapeutic hypothermia after cardiac resuscitation should prevent the occurrence of central diabetes insipidus, especially when there are incentives such as carbon monoxide poisoning that can increase the degree of brain damage.

Conclusion

In short, therapeutic hypothermia is an important treatment for advanced life support after cardiac resuscitation. This patient suffered cardiac arrest due to carbon monoxide poisoning, and developed central diabetes insipidus during therapeutic hypothermia after cardiopulmonary resuscitation. After symptomatic treatment, he eventually developed spontaneous breathing. Cardiac arrest caused by carbon monoxide poisoning will increase the risk of central diabetes insipidus. Doctors should pay attention to this risk when giving therapeutic hypothermia and treat it as a possible complication during treatment.

Declarations

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Author contributions

Shi L wrote the manuscript and collected the patient's clinic information; Song HJ carried out critical revision and correction of the manuscript; all authors read and approved the final manuscript for submission and publication.

Informed consent statement

Written consent was obtained from the patient’s wife.

Conflict-of-interest statement

The authors declare that they have no competing interests.

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Figures

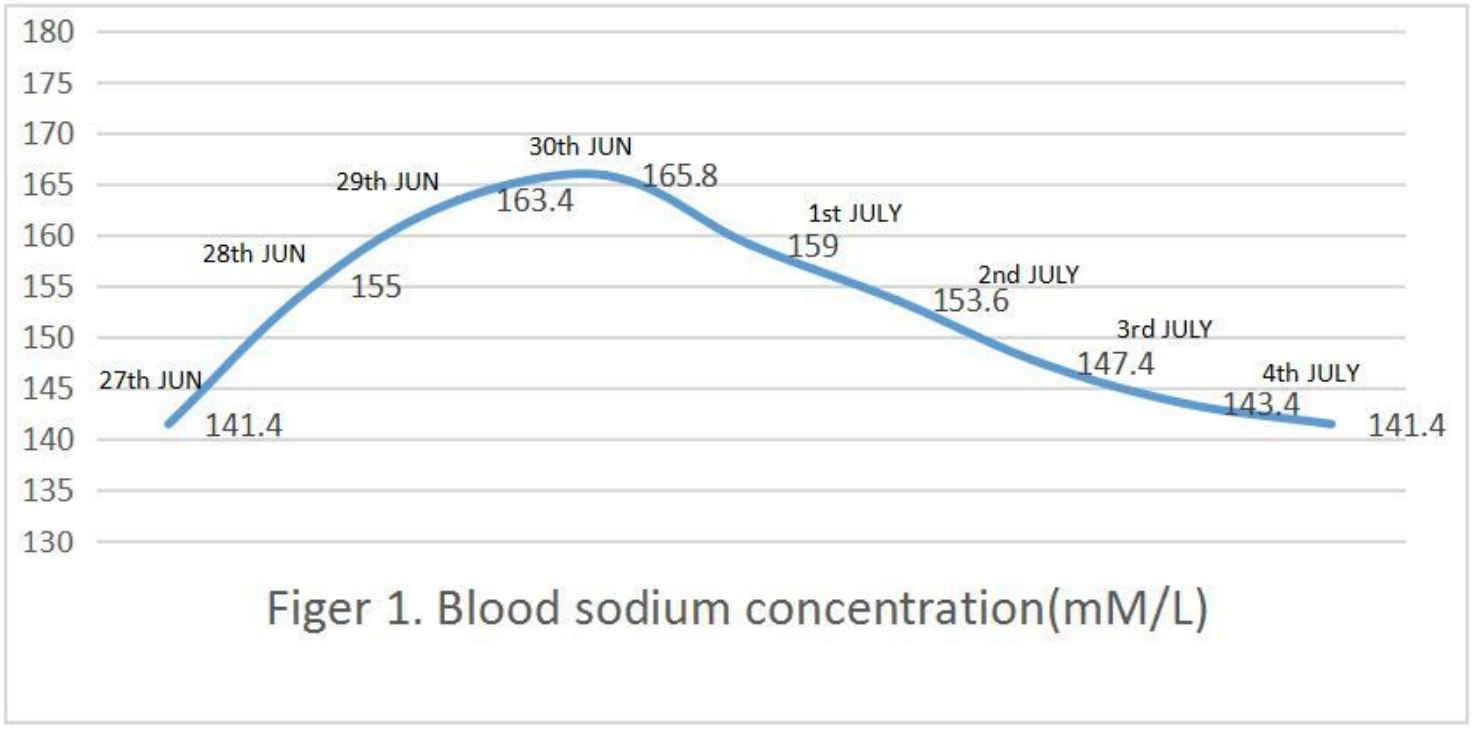

Figure 1

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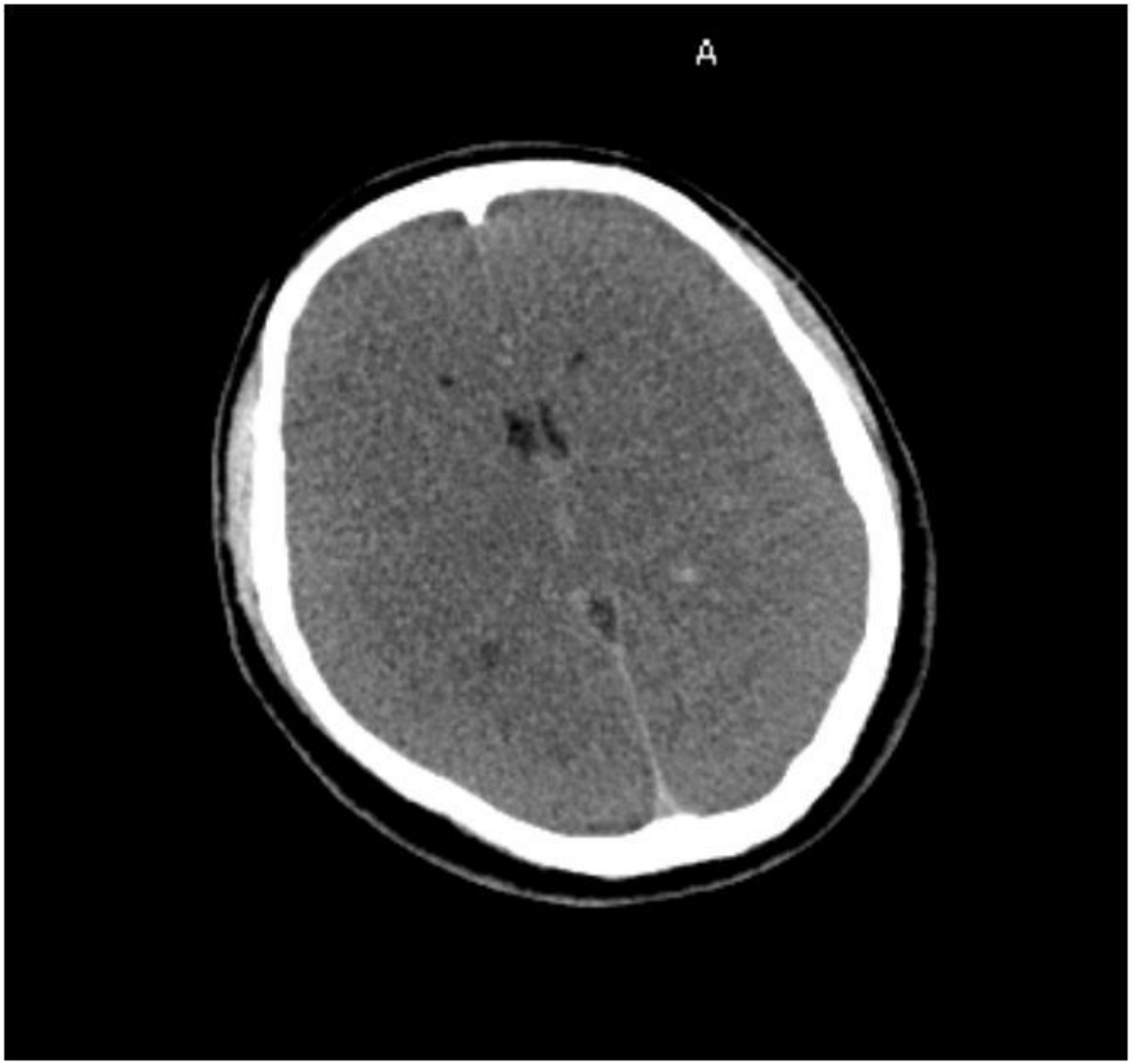

Figer 2: Head CT showed brain edema.

Figure 2

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