

Case Report

Postoperative hyperthermia after resection of a seminoma from the thalamus and third ventricle

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Abstract: Hyperthermia is relatively common in inpatients, but hyperthermia occurring in the immediate postoperative period after undergoing neurosurgery has some unique characteristics. This case report concerns a patient who developed immediate postoperative hyperthermia up to 39.3°C (the axillary temperature) in the post-anesthesia care unit (PACU) after resection of a seminoma from the thalamus and third ventricle. Having been re-intubated and mechanically ventilated, the elevated temperature was treated on the PACU by cooling the skin with ice and antipyretic drugs. Within 2 hours after the surgery, the patient's body temperature had fallen to 37.8°C and vital signs were stable. The patient was then transferred to the neurology intensive care unit for further management. The patient was discharged 70 days after surgery with normal body temperature. During excision of a space-occupying lesion in the thalamus or hypothalamus, clinicians must be mindful of the possibility of hyperthermia and administer appropriate treatments immediately.

Keywords: Neurosurgery, fever, hyperthermia, hypothalamus

Introduction

Hyperthermia, usually caused by infection, inflammation, trauma or drugs, is common in clinical practice. Despite the wide variety of causes, anesthesiologists must pay particular attention to new-onset hyperthermia during the immediate postoperative period, referred to henceforth as postoperative hyperthermia. Postoperative hyperthermia may be provoked by surgical stimulation or trauma, especially by neurosurgery, or other iatrogenic causes during anesthesia or surgery, including blood transfusion and over-administration of intraoperative warming [1]. Hyperthermia results in elevation of the metabolic rate, increased oxygen demand, a hyperdynamic cardiovascular system and elevated intracranial pressure (ICP), all of which can be detrimental for postoperative recovery [2, 3]. Neurosurgery, particularly when undertaken in or around the thermoregulatory center in the hypothalamus, may cause postoperative hyperthermia; in these circumstances the high temperature would be particularly detrimental to neurological recovery [2]. While the extent of brain injury that can be caused by abnormalities of body temperature is well rec-

ognized by neurosurgeons [4], few studies have examined the diagnosis and appropriate management of hyperthermia occurring in the post-anesthesia care unit (PACU) immediately after neurosurgery.

Case presentation

A 27-year-old man was admitted to West China Hospital for investigation of a 4-month history of weakness. His family and social histories were unremarkable. He had a normal body temperature (BT) of 36.5°C, a heart rate (HR) of 75 beats/min, and a blood pressure (BP) of 120/80 mmHg. Magnetic resonance imaging (MRI) of the brain revealed multiple bilateral cystic masses in the right thalamus and hypothalamus. Preoperative blood analysis and serum electrolyte concentrations were normal. The nature of the space occupying lesions could not be determined by imaging alone, so he was scheduled for resection of the lesions in the bilateral thalamus and the third ventricle.

The patient was anesthetized with propofol, remifentanyl and sevoflurane. During surgery, electrocardiography, invasive blood pressure

and pulse oximetry were continuously monitored and recorded, and maintained within normal ranges. Surgery lasted 3 hours. The total volume of intravenous fluid administered was 2,300 ml. Urine output was 850 ml, and blood loss was approximately 300 ml. The histopathological examination revealed that the tumor was a seminoma. Ten minutes after surgery had ended, the patient was transported to the PACU with an endotracheal tube in place. On arrival, the patient's BP was 119/59 mmHg with a pulse of 84 beats/min. When spontaneous breathing returned, the endotracheal tube was removed. At that time, the patient's HR was 84 beats/min, BP 119/59 mmHg and SpO₂ 98%. No abnormal nervous system signs were observed. However, half an hour later, the patient began to shiver. Treatment with IV tramadol 100 mg was not effective. The patient then began to sweat and became tachypnea. His body temperature was recorded as 39°C, and flaked ice was applied to the patient's body avoiding the ears, fingers and toes. Intramuscular antipyretic drugs were also administered. The patient's HR increased to 132 beats/min, and his BP was recorded as 118/78 mmHg. Arterial blood gas analysis revealed the following: pH 7.366; PaCO₂ 30.7 mmHg, PaO₂ 81.2 mmHg, serum sodium concentration 150.6 mmol/l; serum potassium concentration 3.52 mmol/l; ionized calcium concentration 0.882 mmol/l; plasma glucose 7.4 mmol/l, and serum lactate concentration 10.4 mmol/l. We immediately administered methylprednisolone 80 mg, hydrocortisone 100 mg, 500 ml of 5% NaHCO₃, and calcium gluconate 2.0 g intravenously and continued physical cooling with ice.

One hour and 15 minutes after the operation had ended, hyperthermia (BT 39.3°C), tachycardia (maximum HR of 148 beats/min) and diaphoresis persisted, and a gradual reduction in conscious level was observed. The patient then suffered a convulsion, and his urine output increased. The airway was secured with a cuffed endotracheal tube, facilitated by succinylcholine 80 mg and propofol 100 mg intravenously. At the same time, fluid resuscitation was administered according to our hospital's protocol. Later, the patient's HR increased to 158 beats/min, BP fell to 90/47 mmHg, end-tidal carbon dioxide concentration (ETCO₂) was recorded as 40 mmHg and his urine output was 700 ml within 2 hours. A central venous infu-

sion of norepinephrine 0.03 ug/kg/min commenced to elevate the blood pressure. With continued physical cooling and rehydration therapy, the patient's heart rate began to decrease gradually to 116 beats/min, his BP reached 122/65 mmHg and his body temperature reached 38°C. Repeat arterial blood gas analysis revealed the following: pH 7.393; PaCO₂ 32.6 mmHg; PaO₂ 133.7 mmHg; serum sodium concentration 151.4 mmol/l; serum potassium concentration 3.33 mmol/l; ionized calcium concentration 0.877 mmol/l; plasma glucose 6.6 mmol/l, and serum lactate concentration 10.6 mmol/l. We administered IV potassium and calcium supplementation and at the same time increased the rate of fluid infusion. Two hours later, the patient's body temperature was recorded as 37.8°C, and he was transferred to the neurological intensive care unit (NICU) for further treatment.

In the NICU, the patient was treated with controlled moderate hypothermia and was managed with strategies to achieve a negative fluid balance and ICP reduction, as well as other elements of supportive care. One month after the initial operation, the patient was fitted with a ventricular-peritoneal shunt and left lateral ventricular peritoneal shunt because of hydrocephalus. Anti-microbial treatment, attention to fluid and electrolyte balance and supportive care was continued, and the patient's condition stabilized. The patient was discharged home 70 days after the initial operation with normal body temperature.

Discussion

In this case, the normal preoperative body temperature, the conduct of neurosurgery involving the hypothalamus, the new and rapid onset of hyperthermia after surgery in the PACU and the relative lack of the response to antipyretic drugs is strongly suggestive of a diagnosis of hyperthermia induced by a hypothalamic injury. The diagnosis of hypothalamic insult-induced hyperthermia [5] was confirmed by the patient's relevant history, the area operated upon and clinical signs (HR, BP, ETCO₂, and arterial blood gas analysis). Hyperthermia is a relatively common complication during surgery; when it occurs the primary treatment is to remove and treat causative and aggravating factors. Accordingly, we set a lower ambient temperature, exposed the body surface to flaked ice

and administered antipyretics. The body temperature should be carefully monitored in patients undergoing neurosurgery in or near the thermoregulatory center, and rapid and effective cooling measures should be applied to restore temperature homeostasis should hyperthermia occur.

The hypothalamus plays a critical role in regulation of body temperature. More frequently, intraoperative thermal dysregulation results in hypothermia. Nevertheless, hypothalamic lesions or injuries may produce hyperthermia by elevating core body temperature, which must be addressed quickly-particularly in patients with neurological injury or disease. Any underlying pathological condition of the central nervous system may be adversely affected by hyperthermia, and clinical outcomes may be worsened by it. When the hypothalamic thermoregulatory center is injured, the body temperature can increase to 41-42°C. Cerebral blood flow and cerebral metabolic rate also increase with elevated body temperature (between 37°C and 42°C [6]. During neurosurgery, when the temperature lies between 37°C and 42°C, increasing ICP leads to cerebral ischemia and hypoxia. Further, cerebral hypoxia and cerebral edema have a severe adverse impact on outcomes.

When hyperthermia develops, controlled mild hypothermia (27°C-35°C) therapy should be initiated promptly. There is no consensus on whether drugs or physical cooling measures are superior for neurosurgery-induced hyperthermia, and more studies are needed to address this issue. Nevertheless, physical cooling measures allow cerebral function to be monitored, whereas drugs may themselves impair conscious level and mask other postoperative complications. The critical role of the hypothalamus in thermoregulation center means that injury to this area may also result in hypothermia.

Malignant hyperthermia (MH) is another important cause of hyperthermia in patients under anesthesia [7, 8]. Malignant hyperthermia is a pharmacogenetic disorder of skeletal muscle characterized by a hypermetabolic response to inhaled anesthetics and succinylcholine, resulting from increased myoplasmic calcium concentration [9]. To some extent, MH shows similar symptoms to neurosurgery-induced hyper-

thermia, including hyperthermia, convulsions and a hyperdynamic state [10]. In this case, although the patient developed symptoms similar to MH, such as hyperthermia, tachycardia and tachypnea, crucially the convulsion occurred after hyperthermia had developed and without a significant elevation in PaCO₂ or ETCO₂, making it highly likely to have been a febrile convulsion and essentially excluding the diagnosis of MH [11]. Further, most cases of MH occur shortly after the administration of succinylcholine or a volatile anesthetic [12, 13]. In this case, the patient developed a high temperature in the PACU more than 5 hours after the start of anesthesia.

This was the first case of postoperative hyperthermia provoked by an insult to the hypothalamus after neurosurgery in our institution. Hypothalamic syndrome should be considered among the differential diagnoses in patients with sustained fever after surgery in or near the thalamus. For surgery involving the hypothalamic region, core body temperature, arterial blood gas analysis, urine output and serum electrolyte concentrations should be measured continuously throughout the entire perioperative period. Clinicians must be aware of the possibility of hyperthermia as a perioperative complication of surgery involving the thalamus or hypothalamus, and administer appropriate treatments immediately.

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Disclosure of conflict of interest

None.

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