CASE REPORT

Malignant Hyperpyrexia in Cervical Spine Injury

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Abstract:

Pyrexia is an elevation of body temperature above the normal range due to an increase in the hypothalamic thermoregulatory set point. Hyperpyrexia is an extreme elevation of body temperature equal to or greater than 41.5 °C (106.7 °F). Malignant hyperpyrexia is a rare and idiopathic extreme elevation of core body temperature above 42 °C characterized by the acute onset of hyperthermia, coagulopathy and shock. It is potentially life-threatening and can rapidly progress to severe liver and renal impairment, acidosis and encephalopathy. Reported here is a case of malignant hyperpyrexia in a patient with injury of the cervical spine.

Keywords: Malignant hyperpyrexia, hyperthermia, cervical spine injury, thermoregulation.

Case History:

A 43-year-old Chinese man was brought to the Accident and Emergency Department with a history of a heavy object having fallen on his head. He was fully conscious with a Glasgow Coma Scale (GCS) of 15 and complained of headache, neck pain, bilateral upper limb weakness and inability to move his lower limbs. Physical examination confirmed high paraplegia with bilateral symmetrical upper limb weakness.

A computed tomography (CT) scan showed subluxation of the cervical vertebrae C6 over C7, with a comminuted fracture of the left transverse process, lamina and articular facet of C6, plus widening of the C5-C6 and C6-C7 joints. MRI confirmed contusion of the spinal cord with a small hematoma at the level of C6. A CT scan of the chest showed multiple rib fractures on the right side with right haemothorax. No other significant injuries were identified.

He was admitted to the intensive care unit and placed on methyl prednisolone for 24 hours, intravenous fluids, deep vein thrombosis (DVT) prophylaxis, analgesics and incentive spirometry.

The next day the right lower lobe of the lung collapsed with increased right haemothorax and respiratory compromise. He was intubated and a right chest tube was inserted. Central venous and intra-arterial pressure monitoring was done and IV antibiotics injection piperacillin/Tazobactum were started. Over the next 48 hours chest tube drainage decreased and his respiratory parameters improved. On the fourth day after admission he was extubated. He became febrile on the seventh day and chest X-ray showed persistent collapse of the lower lobe of the right lung refractory to standard treatment and unresponsive to bronchoscopy. He was re-intubated on the tenth day in hospital.

On the eleventh day his temperature rose to 40 °C. Staphylococcus aureus was isolated from the sputum and found to be sensitive to the current antibiotic regimen. Cultures of blood and urine were negative.

Six days later his temperature rose to 42.5 °C. Diclofenac sodium and paracetamol were added, along with cooling measure such as tepid sponging and cooling mattress, but the patient became hypotensive requiring inotropic support. Trans-pulmonary thermodilution with continuous cardiac output monitoring was instituted and repeat cultures of blood, sputum and urine were obtained. Despite these measures his condition deteriorated and he became unresponsive with bilateral dilated fixed pupils. Cranial CT demonstrated generalised cerebral oedema with loss of grey and white matter differentiation.

Treatment with broad spectrum antibiotics injection vancomycin, Meropenam, and antifungal agent injection flucanozole was started. Laboratory investigations showed extreme elevation of D-dimer and myoglobin levels and elevated liver, renal, and cardiac enzymes. His temperature continued to be above 40 °C for the next six days. Rhabdomyolysis, ischemic hepatitis and acute renal failure requiring dialysis were evident. Two days later MRI of the brain showed minimal improvement. Cultures of blood, sputum and urine remained negative. Percutaneous tracheostomy was required and renal parameters improved slowly. Hemodialysis was stopped on hospital day 33.

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After 70 days in the ICU he remained ventilator dependent, quadriplegic and had a GCS of six. Electroencephalography showed extreme slowing of waves suggestive of ischemic hypoxic injury. He was transferred to a long-term care unit with a diagnosis of malignant hyperpyrexia following traumatic cervical spine injury.

**Discussion:**

The human body has a remarkable capacity to maintain its core temperature consistently between 36.5 and 37.5°C. Body temperature is regulated by neural feedback mechanisms that operate primarily through the hypothalamus that also contains heat and cold temperature sensors. Under the control of these mechanisms sweating begins at a temperature of 37°C and continues to increase as the body temperature increases. If the temperature falls below 37°C, a variety of mechanisms such as cessation of sweating, vasoconstriction and shivering occur to conserve body temperature.

However, spinal cord and local reflex pathways also contribute to thermoregulation. Most patients with high spinal cord injuries have an impaired ability to regulate body temperature because of autonomic dysfunction and damage to central and peripheral pathways. Extremely high temperatures have been seen also in cervical and upper thoracic spinal cord injuries with no apparent causes for the fever. Previously this has been called 'Quad Fever'; the temperature rises to 42°C and above and shock-like features are seen.

Malignant hyperpyrexia has no known aetiology or predisposing cause and should not be confused with malignant hyperthermia, which is triggered by volatile anesthetics and depolarizing muscle relaxants in predisposed individuals. Malignant hyperthermia is a pharmaco-genetic disorder characterised by a defect in ryanodine receptors and skeletal muscle calcium release channels which causes hyperthermia, muscle rigidity, hypercapnia and arrhythmias.

Malignant hyperpyrexia has characteristics that mimic the "hemorrhagic shock and encephalopathy syndrome" seen primarily in infants. Both these conditions are characterised by hyperthermia, vasomotor instability, coagulation disorders and encephalopathy. Severe hyperthermia can also occur in drug-induced diseases such as neuroleptic malignant syndrome or the serotonin syndrome.

Fever in the ICU setting is common but sustained fever above 42°C is unusual and suggests a malignant form of hyperpyrexia. Our patient developed malignant hyperpyrexia to 42.5°C on the 17th day. Both blood and urine cultures were negative. He was found to have severe cerebral oedema with loss of the grey-white interface. Concurrently he developed rhabdomyolysis and acute renal failure. Doppler study of the lower limbs was negative even though the patient had elevated D-dimer levels. Repeated cultures did not isolate any organism. Our patient remained febrile above 40°C for the next six days but became afebrile after a few days. Such a fever, when not fatal, has been seen to abort after a few days for no apparent reason. This patient did not receive anticholinergic, neuroleptics, phenothiazines, tricyclic antidepressants, selective serotonin-uptake inhibitors or monoaminoxidase inhibitors typically known to cause hyperthermia.

**Summary:**

Thermoregulation requires an intact spinal cord and posterior hypothalamus. In this patient it appears that the interruption of spinal cord pathways disturbed the critical balance controlling thermo-regulation leading to malignant hyperpyrexia and the consequences of uncontrolled sustained high fever.

**References:**