Unusual presentation of more common disease/injury

Heat stroke presented with disseminated intravascular coagulation and bilateral intracerebral bleed

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Heat stroke is a life-threatening condition which is characterised by nausea, vomiting, confusion, disorientation and coma. Aggressive treatment in the form of intravenous fluids along with other symptomatic management can be life saving. Here we present an unusual case of heat stroke followed by disseminated intravascular coagulation, multiple organ dysfunction with bilateral intracerebral bleed who survived with judicious management and recovered without any neurological sequelae.

BACKGROUND

Heat stroke is a life-threatening condition characterised by high body temperature, altered mental status and hot dry flushed skin that can be fatal if proper and urgent assessment and treatment are not initiated rapidly.¹ A variable degree of organ involvement is present in the heat stroke. At the beginning, there is heat exhaustion, characterised by non-specific symptoms such as malaise, headache and nausea. If untreated, this illness results in heat stroke, a serious disease possibly involving central nervous system dysfunction, rhabdomyolysis, arrhythmias, disseminated intravascular coagulation (DIC) and hepatic failure, not uncommon followed by death.² Here we present an interesting case of heat stroke present with DIC and intracerebral bleed.

CASE PRESENTATION

A 38-year-old male patient presented in the emergency department with a history of sudden loss of consciousness during 10 km run in the examination for the subinspector post at around 12:00 h in the afternoon along with 3–4 episodes of vomiting. He had neither significant history of cardiovascular or respiratory disease, nor had similar episodes in the past. No history of drug ingestion or alcohol intoxication was found. His vitals were pulse rate 110/min, blood pressure 110/70 mm Hg, bilateral lung field was clear, GCS-E2M5V2, bilateral small size sluggish reacting pupils with non-illicitable planters and temperature was 105°F.

INVESTIGATIONS

His arterial blood gas analysis showed pH 7.29, pCO₂ 32 mm Hg, pO₂ 129 mm Hg, Na⁺ 138 mmol/l, K⁺ 3.7 mmol/l. Haemoglobin 15 g %, total leucocyte count was 11 000 cells/mm³, DLC N77L9M5, platelet count 48 000/μl blood, random blood sugar 195 mg/dl. Patient's fibrin degradation product was significantly raised and found to be 80 μg/dl (normal <5.0), prothrombin time 29.7 (normal 12.3 s), international normalised ratio 2.63, activated partial thromboplastin time 33.5 (control

26.38 s), urea 55 mg/dl, creatinine 1.2 mg/dl, total serum bilirubin 3.5 mg/dl, serum glutamic oxalacetic transaminase 797 IU/l, serum glutamic pyruvic transaminase 595 IU/l, serum alkaline phosphatase 72 IU/l. CT scan of the head was done which revealed intracranial intraparenchymal bleed in bilateral corona radiata region. Patient was diagnosed to be a case of heat stroke with DIC with bilateral intracerebral bleed (figure 1).

Figure 1 Intracranial intraparenchymal bleed in bilateral corona radiata region.

DIFFERENTIAL DIAGNOSIS

Patient presenting with high-grade fever with unconsciousness in emergency unit, differential diagnosis would be inflammatory brain disease in the form of cerebral malaria, viral encephalitis or meningitis, septic encephalopathy. Only the history of patient falling unconscious after exhaustion in hot humid condition, then a possibility of heat stroke is kept and the management of such patient can be life saving with supportive management.

TREATMENT

The patient was kept on conservative treatment in the form of intravenous mannitol and intravenous fluid (5–6 litres in 24 h) along with liver-supportive treatment.

OUTCOME AND FOLLOW-UP

His vitals got normalised with improvement in laboratory parameters which normalised completely in 5–6 days. Patient recovered without any neurological sequelae.

DISCUSSION

Heat stroke can cause many different reactions within the body that leads to neurological dysfunction, including decreased cerebral perfusion and aberrations in coagulation. Initially during hyperthermia, peripheral vasodilatation predominates to facilitate heat loss through the skin. To avoid a functional hypovolaemia, a compensatory vasoconstriction of the splanchnic and renal vasculature occurs, likely causing the symptoms of nausea, vomiting and diarrhoea. If heat stress continues, however, the compensatory vasoconstriction will eventually fail, further increasing the body temperature. Concurrently, cerebrovascular congestion and cerebral oedema occur with the hyperthermia, causing an increase in intracranial pressure. Combined with a failure of splanchnic vasoconstriction and decreased mean arterial pressure, cerebral blood flow falls.^{3 4} This results in cerebral ischaemia. An aberration in coagulation is caused by a decrease in protein C, protein S and antithrombin III, as well as alterations in vascular endothelium, creating a pattern resembling sepsis and DIC.³ This can cause haemorrhage within the brain, also resulting in neurological dysfunction.⁴ All of these physiological processes occur concurrently, causing the common outcome of neuronal dysfunction.

It is a condition which is characterised by nausea, vomiting, confusion, disorientation and coma. It can lead to rhabdomyolysis, hyperthermia, coma and multiorgan failure (MOF) along with DIC. It can lead to intracerebral haemorrhage in the region of cerebellum, thalamus, putamen, internal capsule and sometimes in the region of corona radiata. Aggressive treatment in the form of intravenous fluids along with other symptomatic management can be life saving.

There are cases of heat stroke with MOF with DIC and patients have survived with meticulous management^{5 6} but otherwise outcomes are fatal.⁷ However, such case of

intracerebral haemorrhage resulting from heat stroke and their outcome is rare. A review literature illustrated that neurological injury may not necessarily be transient.⁸ In a series, 24% of patients had no neurological impairment and 43% had minimal impairment but 33% had moderate-to-severe impairment of severe impairment of neurological function at discharge. Some authors speculate that this neurological injury may be related to hypernatraemic cerebral damage and in our case serum Na⁺ was normal. Boersma *et al*⁹ had reported from Netherlands where a 20-year-old mountain biker died due to heat stroke with cerebral haemorrhage secondary to DIC. An ischaemic and haemorrhage mechanism was proposed in a patient with heat stroke showing increased signal intensity of the cerebrum and cerebellum where patient noted to have persistent mental status changes and memory deficits and also developed difficulty with fine motor coordination, ataxia with shuffling gait.¹⁰

Learning points

- ▶ Heat stroke can be fatal and life threatening.
- ▶ With immediate supportive and judicious management it can be life saving.
- ▶ In our tropical country physical endurance test should be taken under strict medical supervision.
- ▶ Precious life of our police and military personal should not be put at stake.

Competing interests None.

Patient consent Obtained.

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