A report of heat stroke in two Nigerian siblings

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Abstract

Infants and children are at higher risk of heat stroke for several reasons. We report these cases to highlight the danger of leaving children unsupervised in vehicles, aid prompt diagnosis, and management of heat stroke. Two Nigerian siblings aged ranges 5 and 3 years old, were trapped inside an unlocked vehicle and subsequently developed heat stroke. Both children presented with hyperthermia, severe dehydration, convulsions, and loss of consciousness. One of them also had hematuria. They were treated by spraying water onto their bodies to bring down the temperature, intravenous fluid resuscitation, oxygen therapy, and anticonvulsants. Both eventually recovered and were discharged with no obvious neurologic sequelae, but are being followed-up.

Key words: Childhood, heatstroke, hyperthermia, vehicular entrapment

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Introduction

The earliest record of heat stroke in the medical literature can be traced back to 24 BC during the Roman empire when a military campaign conducted by Aelius Gallus into Arabia Felix was cut short by the untimely death of the soldiers due to the sun and desert.[1]

Heat stroke is a severe form of heat related illness and is defined as core temperature ≥ 40°C accompanied by central nervous system dysfunction.[2] It is a pediatric emergency. It can be classic (nonexertional) when it is due to a hot environment or nonclassic (exertional) heat stroke when it is due to physical activity in hot weather (commonly seen in athletes and military personals). Nonexertional heat stroke is more common in children.[1] Infants and children are at higher risk for heat stroke due to inefficient sweating, higher basal metabolic rate and their inability to care for themselves and control their environment. McLaren et al.[1] stated that the temperature within a locked car can rise to dangerously high levels of 93°C, rapidly despite a lower ambient temperature. They also stated that partly open windows are not effective in decreasing either the rate of heat rise or the maximum temperature attained. In USA, in 2013, at least 23 deaths of children left unattended in vehicles have been reported.[4] In Nigeria, there are no available statistics, but there are media reports of children found dead inside cars and never reached medical care.[5] There are very few report of heat stroke in Nigeria in the medical literature.[6] The objectives of this report are to sharpen the clinicians’ skills at making a prompt diagnosis of such a case and increase the clinicians’ understanding of the disease process and the management of the disease.

Case Report

Two male siblings, aged 3 (Case 1) and 5-year-old (Case 2) were rushed into the emergency unit by their parents. They were said to be missing for 3 h and later found unconscious inside a car. The boys, who were apparently healthy, were left under the care of an aunt while the mother had gone to the market. The father had left his car unlocked in the compound because he had a bag of rice in the trunk and planned to retrieve it later during the day. While playing in the compound, the boys entered the vehicle and got trapped. On presentation, both had hyperthermia, generalized tonic clonic convulsions and...
severe dehydration. Case 1 had hematuria, while the Case 2 had concentrated urine. Their anthropometric parameters were within normal limit. The essential clinical findings are as highlighted in Table 1.

A clinical diagnosis of heat stroke was made in both cases. They were resuscitated by exposing, cooling the body using tap water and fans. Intravenous fluid - 100 ml/kg of Ringers lactate was used to correct the severe dehydration. Oxygen was administered via intranasal catheter at a flow rate of 1.5 L/min. Convulsions were aborted with intravenous diazepam and maintained on phenobarbitone. The temperature dropped to normal level within 11 and 24 h on admission in the Cases 2 and 1, respectively.

The urine analysis revealed blood + + and protein + and numerous red blood cells on microscopy in the Case 1, but normal parameters in the Case 2. Laboratory investigation results of the serum urea, electrolytes and creatinine on admission and 48 h later are as highlighted in Table 2. They had normal random blood sugar, white blood cell counts, and liver function tests. They were monitored closely and after 4 and 16 h on admission, the younger sibling (Case 2) and elder sibling (Case 1), respectively became fully conscious.

They were discharged on the third day of admission with normal systemic examination findings. The parents were counseled on the prevention of heat stroke and after several follow-up visits, neurological examination, and biochemical parameters had remained normal.

### Table 1: Clinical parameters of the two siblings on presentation

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Case 1</th>
<th>Case 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>Axillary temperature</td>
<td>40°C</td>
<td>39.5°C</td>
</tr>
<tr>
<td>Hydration status</td>
<td>Severe dehydration</td>
<td>Severe dehydration</td>
</tr>
<tr>
<td>Glasgow Coma Scale</td>
<td>6/15</td>
<td>7/15</td>
</tr>
<tr>
<td>Pulse rate (beats/min)</td>
<td>190</td>
<td>167</td>
</tr>
<tr>
<td>Blood pressure (mmHg)</td>
<td>70/40</td>
<td>70/40</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>60</td>
<td>40</td>
</tr>
</tbody>
</table>

### Table 2: Urea, electrolytes, creatinine and PCV values on admission and after 48 h on admission

<table>
<thead>
<tr>
<th>Laboratory parameters</th>
<th>Case 1</th>
<th>Case 2</th>
<th>Normal values (mmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urea</td>
<td>Admission 48 h 6 3.6 3.2 2.1-6.9</td>
<td>Admission 48 h 6 3.6 3.2 2.1-6.9</td>
<td>Sodium 140 145 133 138 130-146</td>
</tr>
<tr>
<td>Sodium</td>
<td>140 145 133 138 130-146</td>
<td>190 167 130 138 130-146</td>
<td>Chloride 110 112 95 100 94-108</td>
</tr>
<tr>
<td>Chloride</td>
<td>110 112 95 100 94-108</td>
<td>190 167 130 138 130-146</td>
<td>Potassium 4.6 4.0 5.0 3.2 3.0-5.6</td>
</tr>
<tr>
<td>Potassium</td>
<td>4.6 4.0 5.0 3.2 3.0-5.6</td>
<td>190 167 130 138 130-146</td>
<td>Bicarbonate 14 22 24 24 20-28</td>
</tr>
<tr>
<td>Bicarbonate</td>
<td>14 22 24 24 20-28</td>
<td>190 167 130 138 130-146</td>
<td>Creatinine 11 95 60 45 30-116</td>
</tr>
<tr>
<td>Creatinine</td>
<td>11 95 60 45 30-116</td>
<td>190 167 130 138 130-146</td>
<td>PCV (%) 39 32 36 30 30-40</td>
</tr>
</tbody>
</table>

PCV = Packed cell volume

### Discussion

Heat stroke refers to a condition in which an individual exposed to high environmental temperature experience hyperthermia and neurologic dysfunction which may manifest as disorientation, lethargy, delirium, convulsions, and coma. It is also associated with multi organ dysfunction such as renal dysfunction, coagulation abnormalities, fluid and electrolyte imbalances. Mortality rate is between 10% and 70% depending on severity of the condition and age of the patient. Hyperthermia decreases cerebral perfusion leading to hypoxia and ischemia. The effect of hypoxia, ischemia and oxidative damage to the hypothalamus has been implicated in the multiple organ dysfunction seen in heat stroke due to the pathological changes through the hypothalamic-pituitary-adrenal axis mechanisms.

In children, heat stroke can be fatal and may result in neurologic disability. Our patients had hyperthermia-temperature 40.5°C (approximately rectal = 41°C) in Case 1 and 39.5°C (approximately rectal = 40°C) in Case 2. They had features of neurologic dysfunction-convulsions and unconsciousness. Endotoxins are implicated in the tissue injury associated with heat stroke, these stimulate the production and or release of tumor necrosis factor-alpha, interleukin-1, and -6 and interferon-γ. These elevated cytokines play key roles in the pathogenesis of heat stroke with the resultant neurological and multi organ dysfunction, apart from direct thermal injury. They also had features of fluid and electrolyte imbalance-severe dehydration, hypotension, acidosis (Case 1). Arterial blood gas analysis was not done because of cost. The severe dehydration in heat stroke is secondary to excessive sweating and insensible water loss from tachypnea.

Case 1 also had hematuria at presentation, subsequently urine color cleared within 24 h of admission. A coagulation disorder is very unlikely because he had no bleeding in any other body orifice, liver function test and platelet counts were within normal limits in both patients. Urine microscopy showed numerous red blood cells, no myoglobin was detected. Heat stroke have been associated with hemorrhagic complications, these are said to be more commonly secondary to direct thermal injury than consumption coagulopathy.

The main objectives in the treatment of heat stroke are to lower the body temperature as quickly as possible and the provision of adequate support of the cardiovascular system in order to prevent irreversible organ damage and mortality. The main treatment strategies for heat stroke are immediate cooling of the body using various methods and support of organ system functions. Prompt measurement of core body temperature is necessary and rapid cooling methods employed. These can
be by external cooling methods like splashing of cold tap water onto the body. The external cooling technique, although not as effective as the internal cooling technique is the only available option in many resource limited countries. Advanced forms of external cooling technique include immersion cooling with the use of cooling blankets, ice packs placed on axilla, groin, head and neck. The limitation of the technique is peripheral vasoconstriction and shivering, which can be reduced by peripheral massage. Internal cooling techniques can be employed in severe cases such as cold intravenous fluid infusion, gastric, bladder and rectal cold water lavage, heat exchanged balloons, iced peritoneal lavage, cardiopulmonary bypass, and cold continuous hemofiltration.\(^{[9,12]}\) The use of antipyretics like paracetamol in classic heat stroke has not been clearly documented, but may be used in classic heatstroke where normal liver function has been confirmed. Argaud et al.\(^{[13]}\) used paracetamol in their patients with classic heat stroke but excluded 6 (5%) with hepatic and hematological dysfunction. These complications are said to be more frequent in exertional heat stroke where the use of antipyretics are not recommended.\(^{[14,15]}\) The use of antipyretics may be harmful in the presence of hepatic, hemorrhagic and renal complications because they may worsen the bleeding tendencies. Heled et al.\(^{[15]}\) have reported a death in a patient whose condition deteriorated with hemorrhagic complications after initial management with paracetamol. Drugs like dantrolene have proved ineffective in lowering core temperature.\(^{[16]}\) In our patients, we employed external cooling techniques by splashing the whole body with cold tap water and fanning with large fans. The most serious complication of heat stroke is multiple organ dysfunction. Our patients presented with encephalopathy and hypotension. They were successfully treated with appropriate supportive measures.

The major sequelae of heat stroke are mostly neurologic abnormalities and patients may present with convulsions, deep coma, flaccid paralysis, and hyperreflexia.\(^{[9,17]}\) Our patients presented with convulsion, but they responded to anticonvulsants, regained full consciousness and subsequent neurologic examination findings were normal. A computed tomography (CT) scan of the brain was not done because parents could not afford the cost. Some findings in brain CT scan of patients that have suffered from heat stroke include early cerebral edema, central pontine myelinolysis, vascular bounding zone infarcts, and later diffuse cerebral atrophy.\(^{[18]}\)

Prognosis is better if heat stroke is identified early and management with cooling methods, fluid resuscitation and electrolyte replacement is commenced early.\(^{[9,17]}\)

This type of heat stroke due to vehicular entrapment is preventable. In the case of our patients, the father did not realize that leaving his vehicle unlocked for some few hours was a danger to his children. Children should never be allowed to play in or around unlocked vehicles. Children should also not be left unattended in vehicles even if windows are open. In countries like USA, leaving a child unattended in vehicles is considered child neglect.\(^{[19,20]}\) Furthermore, not manually checking that car doors are locked properly even for remote controlled keys is considered child neglect.

This report highlight the importance of education of the public on the dangers of leaving children unattended to in vehicles and attempt to assist clinicians in the prompt diagnosis and effective management of heat stroke in children.

**References**


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