Bilateral hippocampal hyperintensity and elevated cardiac enzyme levels due to exertional heat stroke

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Abstract

Heat stroke is a life-threatening illness with high mortality rates ranging from 21% to 63%, even though aggressive cooling and adequate treatment. It can lead to multiple organ damage. The neurologic, renal, gastrointestinal, hepatic, and hematologic involvements are common. The cardiac involvement is less found and most often, it causes the heart failure without elevated cardiac enzymes or electrocardiography (ECG) change. Even heat stroke universally involve the central nervous system, the reports of brain images after heat stroke are rare. We report a 12 year-old child who suffered from heat stroke with elevated cardiac enzymes, but with normal cardiac function; furthermore, novel magnetic resonance imaging (MRI) findings are also reported.

Keywords: bilateral hippocampal hyperintensities; cardiac enzymes; heat stroke; MR imaging

1. Introduction

Heat stroke is a life-threatening illness with a high mortality rate that ranges from 21% to 63%, even when there is aggressive cooling and adequate treatment. This is because heat stroke can lead to multiple organ damage. Furthermore, neurological, renal, gastrointestinal, hepatic, and hematologic involvement are common. Cardiac involvement is less often and heat stroke generally causes heart failure without an elevation of cardiac enzymes or any electrocardiography (ECG) change. Even when heat stroke universally involves the central nervous system, reports that use brain images after heat stroke are rare. We report a 12 year-old child who suffered from heat stroke with elevated cardiac enzymes, but with normal cardiac function; furthermore, novel magnetic resonance imaging (MRI) findings are also reported.

2. Case report

A 12-year-old boy who weighed 70 kg was found to be undergoing changes in consciousness after climbing a hill (height: 237 meters) over a 2-hour period with his classmates on a hot and humid summer day. According to the Central Weather Bureau, Taiwan, the temperature and the relative humidity on that day were 34.1°C and 66% respectively. He initially received first aid at a rural hospital where delirium with vomiting and diarrhea were recorded. Two hours later he...
was transferred to our hospital. His temperature was initially measured as 38°C, but this was recorded later as reaching almost 40°C after he had been transferred to the intensive care unit. The patient's Glasgow Coma Scale (GCS) was estimated to be E3V4M5. The patient's heart rate was 146 beats per minute and his respiratory rate was 36 per minute. The results of a physical examination were unremarkable except sweating.

An ECG disclosed sinus tachycardia. The blood studies results were as follows: white blood cells 31,200/mm³; platelets 85,000/mm³; sugar 124 mg/dL; sodium 144 mEq/L; potassium 3.1 mEq/L; calcium 9.2 mg/dL; uric acid 9.9 mg/dL; myoglobin 380 ng/ml; aspartate aminotransferase (AST) 306 U/L; alanine aminotransferase (ALT) 209 U/L; blood urea nitrogen 22 mg/dL; creatinine 1.43 mg/dL, and procalcitonin 24.36 ng/mL. The creatine kinase/creatinine kinase-MB (CK/CK-MB) ratio was 580 U/L/12.1 ng/ml and troponin-I was measured at 2.248 ng/mL. Furthermore, the level of brain natriuretic peptide was found to be 13.8 pg/mL and echocardiography of the heart showed normal chamber sizes and wall motion with a left ventricular ejection fraction of 65%.

Cooling was used on the patient, namely evaporation (air conditioning temperature set at 25°C) together with hydration (normal saline 1400 cc challenge follow by NaCl 0.225% with dextrose 5% provided at 90cc per hr). These were effective and his GCS had improved to E3V5M6 at 3 hours after our management had begun. Over two days in hospital, the body temperature of the patient was found to range between 36–37°C. On the 3rd day, he regained consciousness (GCS E4V5M6). His renal function, cardiac enzymes, and procalcitonin gradually returned to normal levels during his hospital stay. The patient's AST and ALT values peaked at 72 hours after arrival and then slowly declined to almost normal levels on discharge (Table 1).

We arranged for electroencephalography (EEG) and a brain MRI on the 5th day after admission. The EEG did not reveal any evidence of cortical dysfunction or epileptiform discharges. However, the brain MRI did show bilateral hippocampal and parahippocampal gyral abnormal hyperintensity (Fig. 1). Finally, he was discharged on the 8th day after arrival with only mildly elevated AST and ALT levels.

### 3. Discussion

Elevated troponin-I can happen during cardiovascular disease, such as coronary artery disease, congestive heart failure, arrhythmias, hypertension, myocarditis and pericarditis, as well as during various noncardiovascular disease, such as sepsis, pulmonary embolism, renal failure and trauma. However, the echocardiography and blood pressure of our patient were normal, which excluded most cardiac diseases. There was no evidence of infection in our case except for an abnormally high procalcitonin level, which in recent years has been used as a broadly used parameter for systemic infection and sepsis. However, procalcitonin is known to be elevated during both sepsis and heat stroke. Thus the clinical evidence was able to reliably differentiate heat stroke from sepsis. In addition, the patient recovered fully without treatment with antibiotics. Only a few reports have looked for elevated cardiac enzymes after heat stroke. We postulate that elevated cardiac enzymes associated with either preserved cardiac function or with transient cardiac dysfunction may be a specific result of heat stroke.

The use of MRI after heat stroke have been described only on rare occasions. The findings have included cortical laminar necrosis affecting the vascular boundary zones, delayed cerebellar atrophy, and hyperintense lesions affecting the external capsules, putamen, thalamus, and cerebellum. The pathogenic mechanism associated with these findings may be cerebral ischemia, which is caused by a rise in intracranial pressure after generalized peripheral vasodilatation.

### Table 1

<table>
<thead>
<tr>
<th></th>
<th>First day</th>
<th>Third day</th>
<th>Fifth day</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC (1000/µL)</td>
<td>31.2</td>
<td>9.6</td>
<td>9.8</td>
</tr>
<tr>
<td>Platelets (1000/µL)</td>
<td>349</td>
<td>85</td>
<td>171</td>
</tr>
<tr>
<td>BUN (mg/dL)</td>
<td>22</td>
<td>18</td>
<td>6</td>
</tr>
<tr>
<td>Cr (mg/dL)</td>
<td>1.43</td>
<td>0.37</td>
<td>0.34</td>
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<tr>
<td>AST (U/L)</td>
<td>306</td>
<td>471</td>
<td>85</td>
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<tr>
<td>ALT (U/L)</td>
<td>209</td>
<td>881</td>
<td>431</td>
</tr>
<tr>
<td>Procalcitonin (ng/mL)</td>
<td>24.36</td>
<td>5.35</td>
<td>0.58</td>
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<tr>
<td>CK (U/L)</td>
<td>580</td>
<td>690</td>
<td>154</td>
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<tr>
<td>CK-MB (ng/mL)</td>
<td>12.1</td>
<td>26.0</td>
<td>2.64</td>
</tr>
<tr>
<td>Troponin-I (ng/mL)</td>
<td>2.248</td>
<td>2.32</td>
<td>0.17</td>
</tr>
</tbody>
</table>

WBC = white blood cells; BUN = blood urea nitrogen; Cr = creatine; AST = aspartate aminotransferase; ALT = alanine aminotransferase; CK = creatine kinase.
Sudhakar PJ et al.\textsuperscript{13} presented a new finding indicating symmetrical bilateral T2-weighted hyperintensity involving the hippocampi together with the previous mentioned cerebellar and cerebral cortical involvement. He postulated the cause to be a combination of direct thermal injury and hypoxic–ischemic insult. Hippocampal hyperintensity has been reported to be associated with mesial temporal sclerosis, with limbic encephalitis and with prolonged seizure.\textsuperscript{14} Our patient had not suffered from any previous problems. He fully recovered consciousness and his EEG findings were normal. No seizures or other neurological defects were documented during his hospital stay or later during follow-up. We therefore conclude that heat stroke can be one of the differential diagnosis when there is bilateral hippocampal hyperintensity after brain MRI.

In conclusion, both an elevation of cardiac enzymes linked to preserved cardiac function and bilateral hippocampal hyperintensities after MRI may be specific findings associated with heat stroke.

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\textbf{Ethics approval}

Not needed.

\textbf{Conflicts of interest}

None declared.

\textbf{Acknowledgments}

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\textbf{References}