Case Report

# Acute Vertebrobasilar Territory Infarcts due to Heat Stroke

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Heat stroke is common in tropical country like India especially during the summer season. We report an unusual case of acute vertebrobasilar territory infarcts due to heat stroke. A middle-aged man developed hyperpyrexia (42.2°C) after strenuous fieldwork in a hot summer day. Next morning, he was found in altered sensorium. The brain imaging showed acute posteromedial midbrain and cerebellar infarcts. A diagnosis of acute ischemic stroke due to heat stroke was made, and the patient was put under the intensive care unit. Two weeks later, the patient became conscious, but had slurred speech and incoordination in all the 4 limbs. Six months after rehabilitation, the patient still have scanning speech and limb ataxia. Acute ischemic stroke worsened the prognosis in our patient. Acute infarct is a very rare neurologic manifestation of heat stroke. This case report emphasizes the importance of suspecting acute infarct in heat stroke patient. **Key Words:** Acute stroke—Heat stroke—Hyperthermia—Posterior circulation stroke.

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Heat stroke is a serious and potentially life-threatening medical emergency, which is characterized by core body temperature greater than 40°C, altered mental status, and multiple organ dysfunction because of failure of thermoregulation.<sup>1</sup> Acute severe heat stroke may be associated with rhabdomyolysis, disseminated intravascular coagulation, acute renal failure, liver damage, acute respiratory distress syndrome, electrolyte imbalance, and neurologic complications.<sup>1-5</sup> Various indicators of poor prognosis are degree of hyperthermia, duration of

exposure, and prolonged coma. Long-term neurologic sequelae occur in approximately 20% of patients. The prognosis of heat stroke is better with early diagnosis and prompt management, which includes fluid resuscitation, cooling measures, and electrolyte replacement.

### Case Presentation

A previously healthy 48-year-old man had strenuous fieldwork in hot summer day, after which he developed lethargy, vertigo, nausea, and increased body temperature. Next morning, he was found in altered state. There was no history of seizure, previous medication use, diabetes mellitus, hypertension, alcohol abuse, smoking, or cardiac disease. On physical examination, he had raised body temperature (42.2°C), dry skin, tachycardia, and high blood pressure (170/100 mm Hg). Glasgow Coma Scale score was 4 (E1V1M2). Pupils were small and reacting to light. Nystagmus was present bilaterally with fast component toward left side. Bilateral fundi were normal. The deep tendon reflexes were normal, and bilateral plantars were mute. There were no meningeal signs.

Preliminary investigations in emergency showed normal hemogram including total leukocyte counts.

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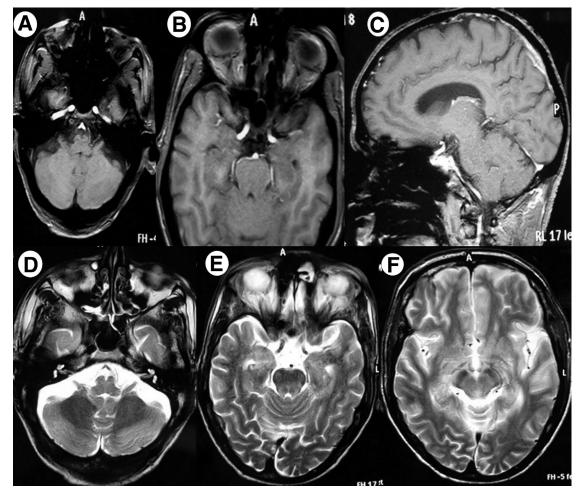
This work is not presented at any meeting.

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**Figure 1.** Magnetic resonance imaging of brain. T1-weighted (A, B, C) images showing hypointense lesions in the cerebellar vermis and isointense signals in cerebellar hemisphere and midbrain. T2-weighted (D, E, F) images showing hyperintense signals in cerebellar vermis, bilateral cerebellar hemisphere, and posterior-medial midbrain.

Serum electrolytes and coagulation profile were also normal. Liver function tests (aspartate transaminase 180 U/L [0-35 U/L], alanine transaminase 202 U/L [0-35 U/ L], serum bilirubin 3.4 mg/dL [0.3-1.2 mg/dL]), renal function tests (serum urea 74 mg/dL [8-20 mg/dL], serum creatinine 2.8 mg/dL [0.7-1.3 mg/dL]), thyroid function tests (thyroid-stimulating hormone 14  $\mu$ IU/mL [0.5-5.0  $\mu$ IU/mL], free thyroxine [FT3] .29 pg/mL [1.3-4.2 pg/mL], free thyroxine [FT4] .15 ng/mL [0.9-2.4 ng/ dL]), and anti-thyroid peroxidase antibody (anti-thyroid peroxidase Ab 1000 IU/mL [less than 35 IU/mL]) level were abnormal. Serum creatine kinase level was raised (350 IU/L [30-170 U/L]).

Peripheral blood smear for malarial parasite was negative. Blood culture was sterile. The electrocardiogram, chest X-ray, and 2-dimensional echocardiography were normal. Serology for venereal disease research laboratory test, human immunodeficiency virus, and viral hepatitis markers (HAV, HBV, and HCV) were negative. Lumbar puncture revealed normal opening cerebrospinal fluid (CSF) pressure (135 mm H<sub>2</sub>O) and CSF, analysis was normal (cells 3 cells/dL: all lymphocytes, proteins 32 mg/dL, and sugar 70 mg/dL). CSF gram stain, acidfast bacilli stain and tuberculosis polymerase chain reaction, and CSF viral markers for Japanese encephalitis and herpes simplex virus were negative. The brain magnetic resonance imaging done after 1 day revealed acute posteromedial midbrain and cerebellar infarcts (Figs 1, 2, and 3). Computed tomography angiography of neck and brain vessels was normal.

A diagnosis of heat stroke accompanied by acute infarcts was made. Patient was put under intensive care and managed with fluid resuscitation. Measures like rapid cooling therapy and antipyretic therapy were given to bring down the temperature. In view of hypothyroidism diagnosed biochemically, patient was started on thyroxine 100  $\mu$ g once a day. Two weeks later, the patient regained consciousness, but we observed slurring of speech and incoordination in all the 4 limbs. The patient's clinical and biochemical parameters improved over the next few days. Six months after rehabilitation, the patient continues to have scanning speech and limb ataxia although he can walk unsupported. ACUTE VERTEBROBASILAR TERRITORY INFARCTS DUE TO HEAT STROKE

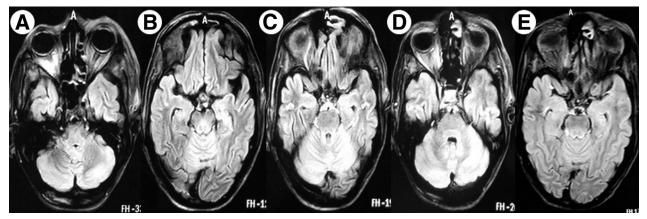
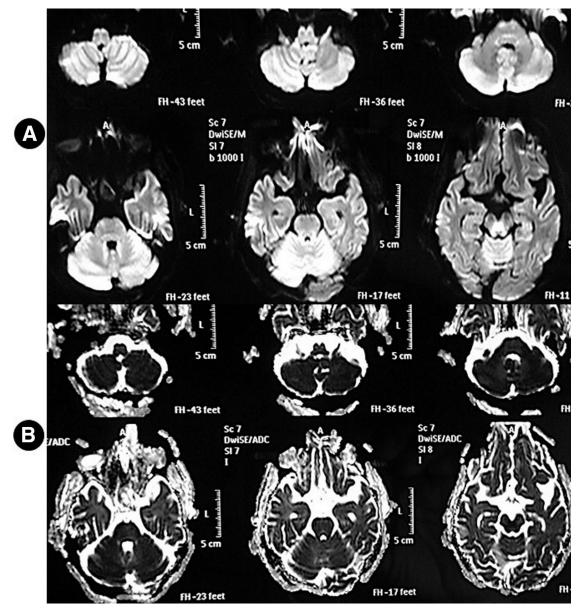


Figure 2. Fluid-attenuated inversion recovery images (A-E) showing hyperintensity in cerebellar vermis, bilateral cerebellar hemisphere, and posterior-medial midbrain.



**Figure 3.** Diffusion-weighted images (A) showing restriction in cerebellar vermis, bilateral cerebellar hemisphere, and posterior-medial midbrain with corresponding fall in apparent diffusion coefficient map value (B).

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## Discussion

Heat stroke may produce acute or remote neurologic complications. Acute neurologic manifestations may range from minor symptoms like irritability, irrational behavior, hallucinations, downbeat nystagmus, and opsoclonus to severe features like cranial nerve abnormalities, seizures, cerebellar dysfunction, and coma.<sup>1,2,4,6,7,9</sup> As reported by Lin et al,<sup>8</sup> neurologic deficit after heat stroke may persist even after 3 years in the form of permanent cerebellar dysfunction (cerebellar atrophy) and transverse myelopathy. The cognitive dysfunction may range from mild attention and memory deficit to profound dementia.<sup>2</sup>

The patient was diagnosed with heat stroke due to history of prolonged exposure to high temperature, followed by hyperpyrexia, altered sensorium, and multiple organ dysfunction. Other possible diagnoses such as viral encephalitis, thrombotic thrombocytopenic purpura, and malaria were also considered but ruled out by appropriate investigations. Possibility of thrombotic thrombocytopenic purpura was ruled out because our patient did not have signs of thrombocytopenic purpura and microangiopathic hemolytic anemia as it is characterized by pentad of fever, renal dysfunction, thrombocytopenic purpura, microangiopathic hemolytic anemia, and neurologic manifestations. Other causes of stroke like cardioembolic, thrombophilias, and familial were ruled out with appropriate investigations. Possibilities of extrapontine myelinolysis or Wernicke encephalopathy were also ruled out.

The pathophysiology of neurologic involvement includes increased intracranial pressure and autonomic dysfunction, which leads to cerebral hypoperfusion and ischemia. Cytokines may have an important role in pathogenesis of heat stroke manifestations.<sup>4</sup> Abnormal coagulation may increase the risk of intracranial hemorrhage.<sup>3</sup> The thermal injury to brain is directly proportional to the duration of high temperature. The cerebellar dysfunction in association with heat stroke has been reviewed by various authors. Cerebellar dysfunction in heat stroke is because of high susceptibility of Purkinje cells to metabolic stress related to hyperthermia as evidenced by Malamud et al and Bazille et al.<sup>5,10</sup> They found severe reduction in the number of Purkinje cells and fibers along with swelling, pyknosis, or disintegration of the remaining cells on histopathologic examination of cerebellar atrophy after heat stroke.<sup>9</sup>

Acute infarcts are a rare complication of heat stroke. Our patient might be the first case report of acute vertebrobasilar territory infarcts due to heat stroke who had persistent neurologic features in the form of cerebellar speech and limb ataxia. To conclude, acute infarcts due to heat stroke are very rare and worsen the prognosis of heat stroke. Early and prompt management of heat stroke can limit the neurologic complications. This might be the first case report of acute vertebrobasilar territory infarcts due to heat stroke.

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