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.

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.[1] 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.[1-5] 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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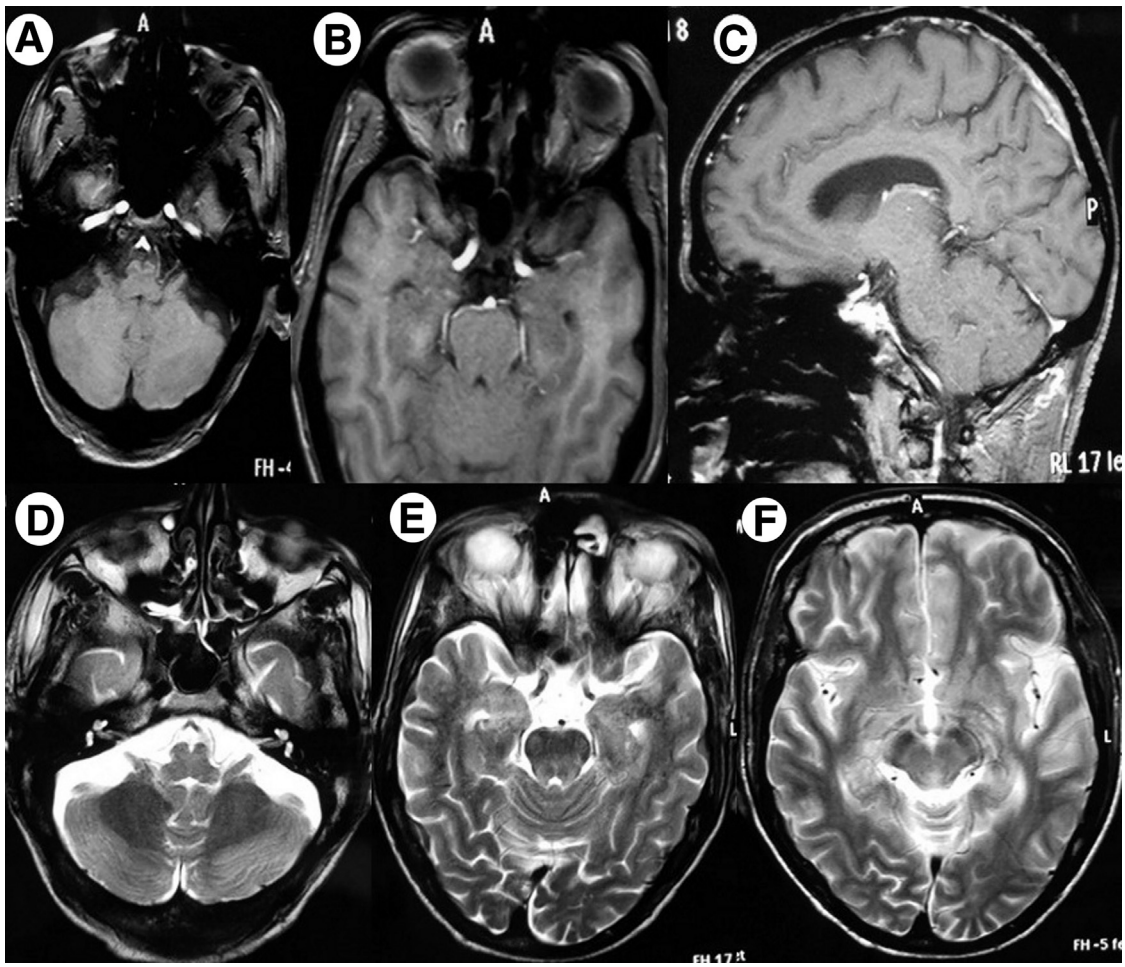

Figure 1. *Magnetic resonance imaging of brain. T1-weighted (A, B, C) images showing hypointense lesions in the cerebellar vermis and isointense signals in cerebellar hemisphere and midbrain. T2-weighted (D, E, F) images showing hyperintense signals in cerebellar vermis, bilateral cerebellar hemisphere, 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 μIU/mL [0.5-5.0 μIU/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_2O) and CSF, analysis was normal (cells 3 cells/dL: all lymphocytes, proteins 32 mg/dL, and sugar 70 mg/dL). CSF gram stain, acid-fast 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 μg 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.