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Case Report

Atypical brain imaging findings associated with heat stroke: A patient with rhabdomyolysis and acute kidney injury: A case report ^{☆,☆☆,★,★★,†}

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ABSTRACT

Heat stroke is a serious medical condition that can cause multiple organ dysfunction, including central nervous system damage. The complications of heat stroke occur because of hypoperfusion, an inflammatory response, and thrombosis, resulting in variable imaging findings. This report describes a rare case of rapidly progressive heat stroke with rhabdomyolysis and acute kidney injury in a 53-year-old woman with atypical brain computed tomography and magnetic resonance imaging findings involving the bilateral cerebral cortex and deep gray matter but excluding the cerebellum. She had an increased diffusion-weighted imaging signal and a reduced apparent diffusion coefficient within the bilateral basal ganglia and cerebral cortex, which have not been reported previously. These findings indicate that cytotoxic edema is a potential mechanism of brain damage in individuals with heat stroke.

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Introduction

Heat stroke is a medical emergency that is characterized by an elevated core body temperature greater than 40°C with central nervous system (CNS) dysfunction and end-organ damage [1,2]. Neurologic abnormalities include memory loss, paral-

ysis, seizure, delirium, or coma [1–4]. The common complications of severe heat stroke include acute respiratory distress syndrome, disseminated intravascular coagulation (DIC), rhabdomyolysis, electrolyte disturbances, acute renal failure, and liver dysfunction [1,5]. There are a limited number of cases in the literature that describe the imaging findings of CNS abnormalities related to heat stroke. The common sites for

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[†] Authors' contribution: Byung Hoon Lee; design of the study, patient enrollment, data acquisition, data analysis/interpretation, and manuscript preparation.

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CNS abnormalities include the cerebellum, basal ganglia, cerebral cortex, brainstem, hippocampus, subcortical white matter, splenium, and external capsule [4]. This report describes a rare case of rapidly progressing heat stroke with rhabdomyolysis and acute kidney injury in a 53-year-old woman with atypical brain computed tomography (CT) and magnetic resonance imaging (MRI) imaging findings. To the best of our knowledge, this is the first case report describing unusual brain MRI findings and diffusion-weighted imaging (DWI) involving the bilateral cerebral cortex and deep gray matter.

Case presentation

A 53-year-old female patient was admitted to our emergency department with a 2-day history of altered mental status. The patient was found lying on the floor unconscious and was reported to have been sweating on the day before visiting our hospital. She had a history of alcohol abuse, and several empty alcohol bottles were found nearby. Since then, she had been externally cooled in her home for approximately 14 hours using an air conditioner before admission to our hospital. She did not have any comorbidities or a febrile illness separate from the heat injury. During the 2 days preceding her admission, the highest environmental temperature had been 36.0°C and it had rained. Upon arrival, the temperature of the patient was 38.0°C, blood pressure was 170/99 mmHg, and pulse rate was 137 beats/min. She was stuporous and only responded to painful stimuli. Laboratory test results showed elevated levels of creatine kinase (9676 units/L, normal: 0-170 units/L), serum creatine (4.05 mg/dL, normal: 0.50-0.90 mg/dL), and blood urea nitrogen (86.6 mg/dL, normal: 6-23 mg/dL). These results indicated rhabdomyolysis and acute kidney injury. She was treated with 4 L of intravenous fluid per day. Brain CT on admission showed multifocal low-density areas in the bilateral hippocampi and left cerebral cortex (Fig. 1). Brain MRI and magnetic resonance angiography were performed 5 hours after admission. DWI and the corresponding apparent diffusion coefficient (ADC) map revealed restricted diffusion in the cerebral cortex including the bilateral hippocampi and basal ganglia (Fig. 2). There was no significant subcortical white matter abnormality, and magnetic resonance angiography showed normal flow-related signal within the head and neck vessels. A day later, the patient showed a fixed and dilated pupil. Therefore, follow-up brain CT was performed that showed rapid progression of cerebral edema (Fig. 3). She was intubated and ventilated; however, she died 11 days later.

Discussion

Heat stroke is a life-threatening, multisystem disorder characterized by severe hyperthermia and is associated with CNS abnormalities and end-organ damage [1,2,6]. Common initial neurologic symptoms include memory loss, paralysis, seizure, delirium, impaired consciousness, or coma [1–4]. In addition, patients with heat stroke present with rhabdomyolysis, acute renal insufficiency, DIC, intestinal dysfunction,

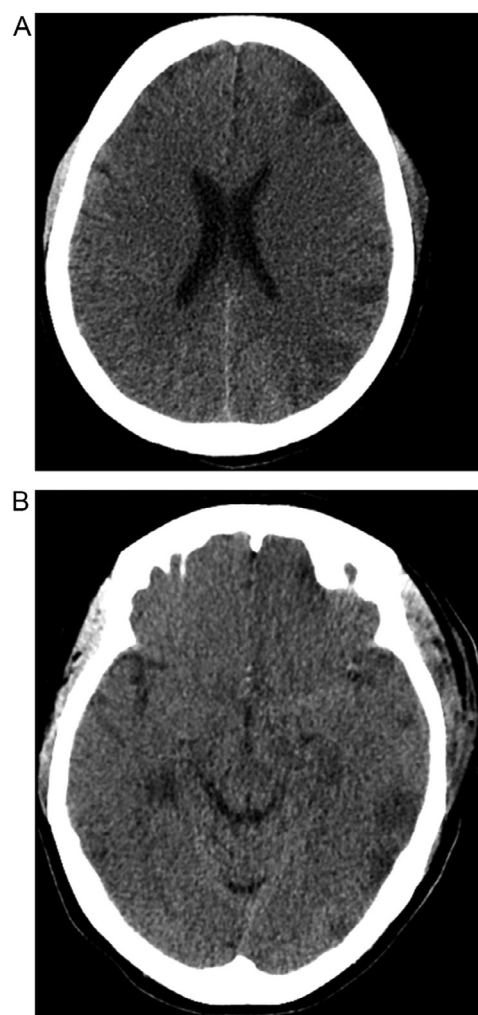


Fig. 1 – A 53-year-old woman with a history of altered mental status. The initial noncontrast brain CT image shows decreased cortical gray matter attenuation in the left cerebral cortex (A) and bilateral hippocampi (B).

and liver dysfunction [1,5]. Consistent with patients in previous reports, this patient presented with unconsciousness, rhabdomyolysis, and acute kidney injury.

Previously, case reports and small patient series have reported brain MRI findings in patients with heat stroke [7–10]. In addition, the mechanism of CNS abnormalities caused by heat stroke is multifactorial, and thus, cerebral imaging findings vary. There are several proposed mechanisms of CNS abnormalities caused by heat stroke.

Initially during heat stress, there is predominant peripheral vasodilatation that facilitates heat loss through the skin. This is accompanied by compensatory vasoconstriction of the splanchnic and renal vasculature that prevents functional hypovolemia. As a result of the failure of splanchnic vasoconstriction and decreased mean arterial pressure, cerebral blood flow decreases, resulting in cerebral ischemia [10]. Restricted diffusion because of cytotoxic edema is one of the common MRI findings associated with cerebral ischemia. Severe hypoxic-ischemic brain damage in adults primarily affects

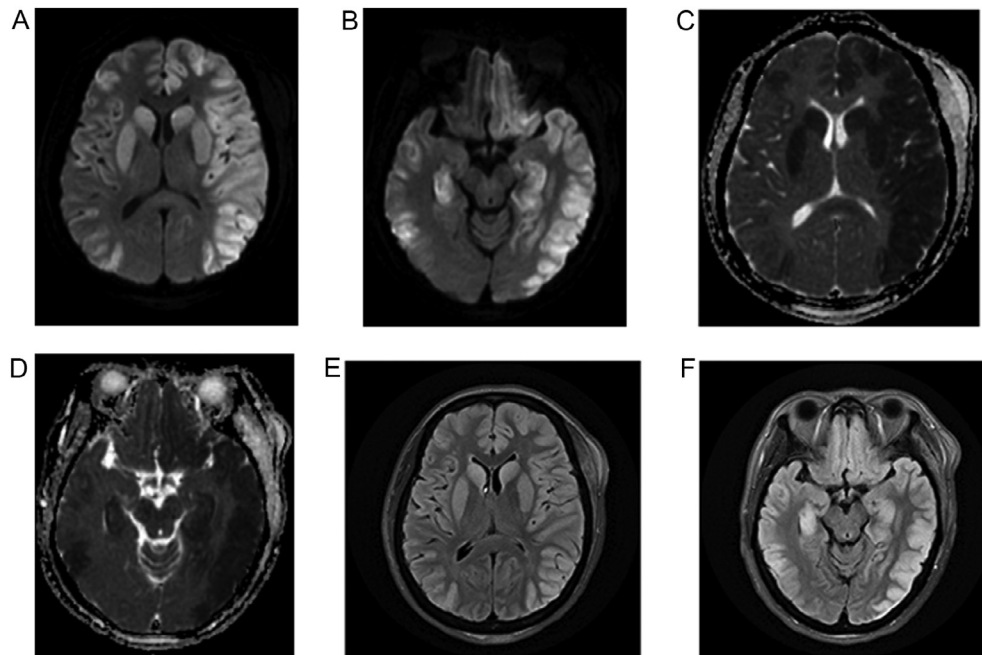


Fig. 2 – Diffusion-weighted imaging (DWI) (A and B) and the corresponding apparent diffusion coefficient (ADC) map (C and D) images taken 5 hours after admission show restricted water diffusion in the basal ganglia and the cerebral cortex (A and C), and the hippocampi (B and D). The signals are hyperintense on FLAIR (E and F).



Fig. 3 – A follow-up brain CT image taken the following day shows rapid progression of her cerebral edema.

gray matter structures such as the basal ganglia, thalami, cerebral cortex, cerebellum, and hippocampi [11]. MRI findings in the present case showed increased signal intensities on T2-weighted imaging and fluid-attenuated inversion recovery (FLAIR) that were hyperintense on DWI and hypointense on the corresponding ADC maps within the bilateral basal ganglia and cerebral cortex including the hippocampi. Therefore, the suggested explanation of CNS involvement in this patient with heat stroke was hypoxic-ischemic brain injury because of circulatory insufficiency.

Alternatively, the heat may have a direct toxic effect on the CNS. Purkinje cells located in the cerebellar cortex are susceptible to direct thermal injury [12]. Thus, the cerebellum is particularly vulnerable to heat stress. Several studies demonstrated MRI abnormalities in the cerebellum [9,10,12–14]. However, there was no abnormal signal intensity or atrophy in the cerebellum in the present case on MRI.

Excessive release of inflammatory cytokines causes a breakdown of the blood-brain barrier and blood-cerebrospinal fluid barrier, leading to vasogenic edema and CNS injury [14,15]. Typical MRI findings associated with vasogenic edema include hyperintense T2 and FLAIR signals, which do not show restricted diffusion. In this case, all brain lesions on DWI showed restricted diffusion, suggesting there was no area of vasogenic edema in the brain parenchyma.

Heat stroke causes DIC by reducing protein C, protein S, antithrombin III levels, and changing the vascular endothelium [1]. DIC can cause intracerebral hemorrhage, thrombosis, and minor infarction, resulting in small-vessel ischemic damage [16]. White matter signal abnormalities on FLAIR are typically observed in small-vessel disease and may be secondary to small-vessel occlusion from DIC [14]. Several studies demonstrated white matter signal abnormalities on T2-weighted imaging and FLAIR during heat stroke [4,14,17,18]. However, there was no abnormal signal intensity in the cerebral white matter and evidence of hemorrhage in the present case.

McNamee et al reported abnormal signal intensity in the pons in a heat stroke patient with central pontine myelinolysis [19]. However, there was no abnormal signal intensity in the brainstem in the present case.

In summary, this case demonstrated increased DWI signal and reduced ADC within the bilateral basal ganglia and

cerebral cortex, which has not been reported before. These findings implicate cytotoxic edema as a potential mechanism of brain damage during heat stroke.

REFERENCES

- [1] Bouchama A, Knochel JP. Heat stroke. *N Engl J Med* 2002;346(25):1978–88 PubMed:12075060. doi:10.1056/NEJMra011089.
- [2] Guerrero WR, Varghese S, Savitz S, Wu TC. Heat stress presenting with encephalopathy and MRI findings of diffuse cerebral injury and hemorrhage. *BMC Neurol* 2013;13:63 PubMed:23773322. doi:10.1186/1471-2377-13-63.
- [3] Yeo TP. Heat stroke: a comprehensive review. *AACN Clin Issues* 2004;15(2):280–93 PubMed:15461044.
- [4] Cao L, Wang J, Gao Y, Liang Y, Yan J, Zhang Y, et al. Magnetic resonance imaging and magnetic resonance venography features in heat stroke: a case report. *BMC Neurol* 2019;19(1):133 PubMed:31215399. doi:10.1186/s12883-019-1363-x.
- [5] Yang M, Li Z, Zhao Y, Zhou F, Zhang Y, Gao J, et al. Outcome and risk factors associated with extent of central nervous system injury due to exertional heat stroke. *Medicine (Baltimore)* 2017;96(44):e8417 PubMed:29095276. doi:10.1097/MD.00000000000008417.
- [6] Koh YH. Heat stroke with status epilepticus secondary to posterior reversible encephalopathy syndrome (PRES). *Case Rep Crit Care* 2018;2018:3597474 PubMed:29984005. doi:10.1155/2018/3597474.
- [7] Lee JS, Choi JC, Kang SY, Kang JH, Park JK. Heat stroke: increased signal intensity in the bilateral cerebellar dentate nuclei and splenium on diffusion-weighted MR imaging. *AJNR Am J Neuroradiol* 2009;30(4):E58 PubMed:19179428. doi:10.3174/ajnr.A1432.
- [8] Murcia-Gubianas C, Valls-Masot L, Rognoni-Amrein G. Brain magnetic resonance imaging in heat stroke. *Med Intensiva* 2012;36(7):526 PubMed:21676501. doi:10.1016/j.medin.2011.05.003.
- [9] Ookura R, Shiro Y, Takai T, Okamoto M, Ogata M. Diffusion-weighted magnetic resonance imaging of a severe heat stroke patient complicated with severe cerebellar ataxia. *Intern Med* 2009;48(12):1105–8 PubMed:19525609. doi:10.2169/internalmedicine.48.2030.
- [10] McLaughlin CT, Kane AG, Auber AE. MR imaging of heat stroke: external capsule and thalamic T1 shortening and cerebellar injury. *AJNR Am J Neuroradiol* 2003;24(7):1372–5 PubMed:12917130.
- [11] Huang BY, Castillo M. Hypoxic-ischemic brain injury: imaging findings from birth to adulthood. *Radiographics* 2008;28(2):417–39 quiz 617 PubMed:18349449. doi:10.1148/rg.282075066.
- [12] Muccio CF, De Blasio E, Venditto M, Esposito G, Tassi R, Cerase A. Heat-stroke in an epileptic patient treated by topiramate: follow-up by magnetic resonance imaging including diffusion-weighted imaging with apparent diffusion coefficient measure. *Clin Neurol Neurosurg* 2013;115(8):1558–60 PubMed:23411048. doi:10.1016/j.clineuro.2013.01.005.
- [13] Albukrek D, Bakon M, Moran DS, Faibel M, Epstein Y. Heat-stroke-induced cerebellar atrophy: clinical course, CT and MRI findings. *Neuroradiology* 1997;39(3):195–7 PubMed:9106293. doi:10.1007/s002340050392.
- [14] Mahajan S, Schucany WG. Symmetric bilateral caudate, hippocampal, cerebellar, and subcortical white matter MRI abnormalities in an adult patient with heat stroke. *Proc (Bayl Univ Med Cent)* 2008;21(4):433–6 PubMed:18982090. doi:10.1080/08998280.2008.11928446.
- [15] Cannon JG. Inflammatory cytokines in nonpathological states. *News Physiol Sci* 2000;15:298–303 PubMed:11390930.
- [16] Sonkar SK, Soni D, Sonkar GK. Heat stroke presented with disseminated intravascular coagulation and bilateral intracerebral bleed. *BMJ Case Rep* 2012;2012:1–3 PubMed:23087280. doi:10.1136/bcr-2012-007027.
- [17] Biary N, Madkour MM, Sharif H. Post-heatstroke parkinsonism and cerebellar dysfunction. *Clin Neurol Neurosurg* 1995;97(1):55–7 PubMed:7788975.
- [18] Fuse A, Yamashiro K, Oji Y, Furuya T, Noda K, Hattori N, et al. Reversible focal cerebral cortical lesions in a patient with heat stroke. *Intern Med* 2013;52(3):377–80 PubMed:23370749. doi:10.2169/internalmedicine.52.7645.
- [19] McNamee T, Forsythe S, Wollmann R, Ndukwu IM. Central pontine myelinolysis in a patient with classic heat stroke. *Arch Neurol* 1997;54(8):935–6 PubMed:9267966. doi:10.1001/archneur.1997.00550200005002.