

MRI findings in human brain after heat stroke

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The objective of this study is to explore the human brain after heat stroke by MRI technology. Study group consisted of 6 patients after heat stroke, with a score of 3 to 9 in Glasgow Coma Scale. The MRI studies including susceptibility weighted imaging were performed with a 1.5 T scanner. Some patients were followed up by the same MRI studies 2-3 times. Abnormal signals were detected at brain stem, cerebellar dentate nucleus, cerebellar peduncles, cerebellum, hippocampus, corona radiata, centrum ovale, et al. The lesions including micro hemorrhage, cytotoxic edema, vasogenic edema, hemorrhagic infarction, encephalitis. Heat stroke may cause multiple brain lesions such as ischemia, hemorrhage, infarction, inflammation and can produce remarkable symmetric lesions in the cerebellum.
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Heat stroke | MRI | Brain

1. Introduction

Heat stroke is characterized by an elevated core body temperature over 40°C and neurologic abnormalities including delirium, seizures, or coma [1, 2]. Potential immediate complications of severe heat stroke include shock, acute respiratory distress syndrome, acid-base or electrolyte disturbances, disseminated intravascular coagulation and rhabdomyolysis [1].

Heat-stroke is generally reported in case reports or small patients series. MRI findings include lesions in dentate nuclei [3], cerebellar hemispheres [4, 5, 6], cerebellar peduncles, midbrain, thalami [7], hippocampi [8], the splenium [3], temporo-occipital lobes [9]. Lee et al. [3] reported possible selective vulnerability of cerebellar neurons to heat injury.

2. Materials and methods

2.1 Patients

The study group included 6 patients (4 men, 2 women) after heat stroke, with a score of 3 to 9 in Glasgow Coma Scale. Their mean age was 67 years (range 43–90). All patients were admitted to the ICU with coma and fever (over 40°C) on hot summer days. Complications including rhabdomyolysis, acute renal failure, acid-base or electrolyte disturbances and disseminated intravascular coagulation were revealed by laboratory examinations to different degrees.

2.2 MRI Examinations

Initial MRI studies were collected within 2–5 days after heat stroke. MRI examinations were performed by a SIEMENS Avanto 1.5 T magnetic resonance scanner with a standard quadrature head coil. The MRI protocol included the standard structural sequences T1-weighted (TR450ms, TE15ms), T2-weighted (TR3000ms, TE100ms), diffusion-weighted, and FLAIR (TR8000ms, TE120ms). Imaging parameters of susceptibility-weighted imaging were as follows: TR 49ms, TE 40ms, slice thickness 2mm, 56 slices in a single slab. Some patients were followed up by the same MRI studies 2-3 times. Informed consent was taken from all family members of the patients and approval of the hospital ethics committee was obtained.

3. Results

Punctate hemorrhage was detected in brain stem of one patient after heat stroke with a score of 3 in Glasgow Coma Scale by susceptibility-weighted imaging [10]. Diffusion-weighted imaging revealed restricted water diffusion in the left centrum ovale (Fig. 1a~b). After 4 days, the hemorrhage of brain stem had no

significant change. However, abnormal signals in the left centrum ovale almost disappeared (Fig. 1c~d). 11 days later, encephalitis occurred in the left hippocampi (Fig. 1e~g). This patient died on the 25th day after heat stroke.

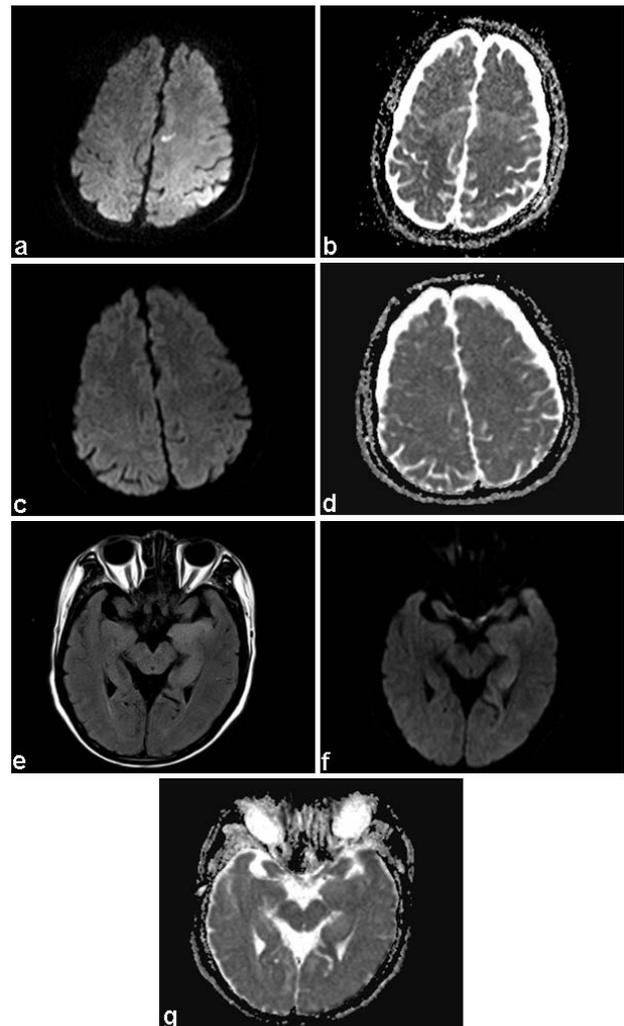


Fig.1 MRI images obtained in a 64-year-old man after heat stroke with a score of 3 in GCS. DWI (a) and ADC (b) maps show restricted water diffusion in the left centrum ovale; After 4 days, restricted water diffusion almost disappear (c~d); 11 days later, T2FLAIR (e), DWI (f) and ADC (g) maps show mild hyperintensity in the left hippocampi.

Remarkable symmetric lesions were found in cerebellar peduncles of one patient in the 2th day after heat stroke with a score of 8 in Glasgow Coma Scale. T2WI, DWI and ADC maps showed symmetric hyperintensity in cerebellar peduncles [11]. The symmetric hyperintensity gradually weakened on T2WI and returned to almost normal on DWI in the 37th day after heat stroke.

Conflict of interest: No conflicts declared.

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Diffusion-weighted imaging revealed symmetrical restricted water diffusion in the bilateral dentate nuclei [11] for one patient after heat stroke with a score of 3 in Glasgow Coma Scale. In addition, restricted water diffusion (Fig. 2a~b) and punctate hemorrhage [10] were detected in right corona radiata. Death occurred to the patient in the 5th day after heat stroke.

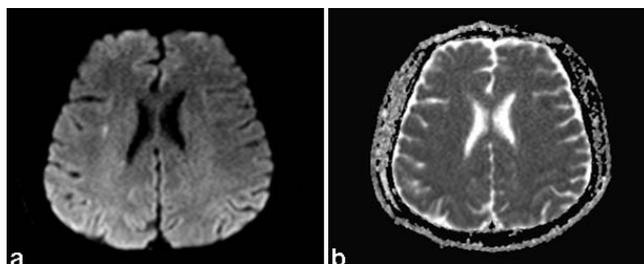


Fig.2 MRI images obtained in a 43-year-old man after heat stroke with a score of 3 in GCS. DWI and ADC maps show restricted diffusion in the right corona radiata (a~b).

Acute massive cerebral infarction of the right hemisphere (Fig. 3a~c) occurred in one patient after heat stroke with a score of 6 in Glasgow Coma Scale. And punctate hemorrhage was detected in right frontal lobe [10]. The patient became a plant man and died about 3 months later.

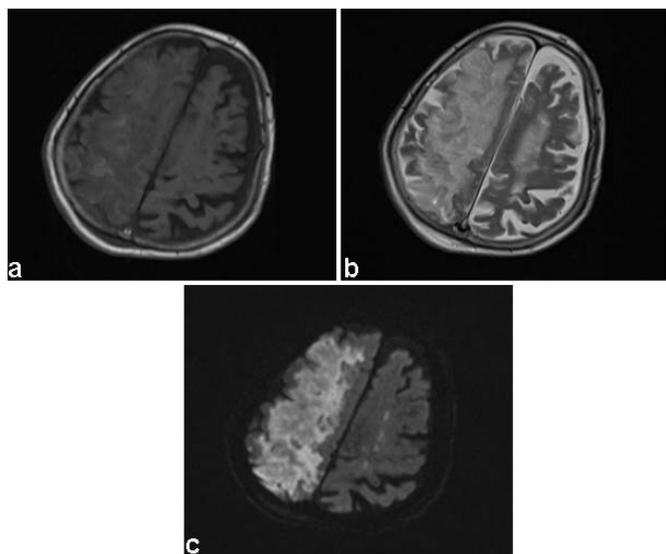


Fig.3 MRI images obtained in a 90-year-old man after heat stroke with a score of 6 in GCS. T1WI (a), T2WI (b) and DWI (c) images show acute massive cerebral infarction of the right hemisphere.

T2-weighted and FLAIR imaging revealed mild symmetric hyperintensity in the cerebellar hemispheres for one patient after heat stroke with a score of 9 in Glasgow Coma Scale [11]. For another patient after heat stroke with a score of 8 in Glasgow Coma Scale, acute punctate cerebral infarction of the right cerebellum was detected (Fig. 4a~c). The two patients gradually recovered and cerebellar dysfunction remained.

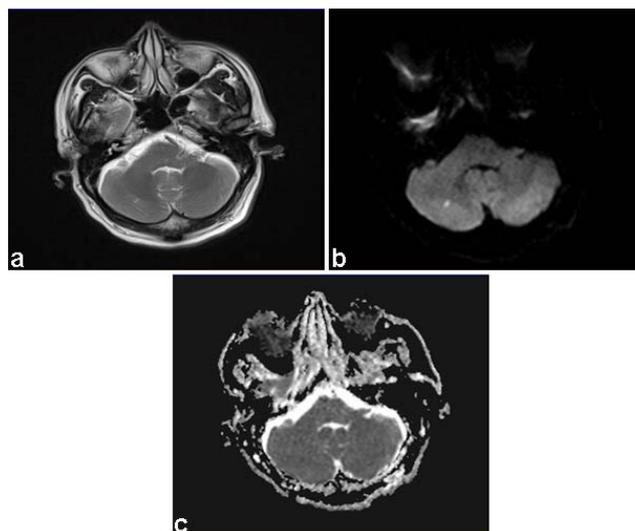


Fig.4 MRI images obtained in a 65-year-old man after heat stroke with a score of 8 in GCS. T2WI (a), DWI (b) and ADC (c) maps show acute punctate cerebral infarction in the right cerebellum.

4. Discussion

Heat stroke is a life-threatening condition characterized by severe hyperthermia associated with central nervous system abnormalities (including delirium, seizures, or coma) [12]. Bazille C et al. [13] confirmed the selective vulnerability of Purkinje cells to heat-induced injury and revealed the neuropathology of heat stroke was severe diffuse loss of Purkinje cells associated with heat shock protein 70 expression by Bergmann glia. A cytotoxic and/or excitotoxic mechanism has been suggested [12], possibly of ischemic nature [3] resulting from hypoperfusion induced by vascular endothelial damage which usually accompanies heat-stroke [14]. In addition, findings associated with cerebellar efferent pathways have been reported [3, 7, 13, 15].

MRI findings include lesions in dentate nuclei, cerebellar hemispheres, cerebellar peduncles [3-7]. Our results are in agreement with previous studies. Symmetrical abnormal signals were found in dentate nuclei, cerebellar hemispheres or cerebellar peduncles for 3 patients after heat stroke. In this study, punctate hemorrhages were detected in brain stem, corona radiata and frontal lobe by susceptibility weighted imaging. The suggested explanation for intracerebral hemorrhage with heat stroke is concomitant complication of disseminated intravascular coagulation. In addition, pneumonia complicated with heat stroke has been reported [13]. Unlike previous studies, encephalitis occurred to one patient. The suggested explanation is the role of the systemic inflammatory response caused by heat stroke [14].

5. Conclusions

In summary, heat stroke may cause multiple brain lesions such as ischemia, hemorrhage, infarction, inflammation and can produce remarkable symmetric lesions in the cerebellum. The MRI studies including diffusion weighted and susceptibility weighted imaging play an important role in the diagnosis and evaluation the prognosis for heat stroke.

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