MRI and MRV features of a patient with Heat Stroke: a case report

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Case report

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Abstract

Background Heat stroke (HS) is a critical illness that can cause multiple organ dysfunction including damage to the central nervous system (CNS), which can be life-threatening in severe cases. The brain image lesions of HS patient with CNS damage has been rarely reported before and usually variable in different cases, causing confusing to doctors when encounter these patients in the clinic. Cerebral venous thrombosis (CVT) is a rare cause of stroke that mostly affects young people and children. The pathogenesis of brain damage caused by HS is complex, CVT may be involved in the pathogenesis of HS with CNS damage. In this manuscript, we report a case of HS with CVT with symmetrical lesions in the bilateral putamen, posterior limb of internal capsule, external capsule, insula lobe, and subcortical white matter inside the brain. Case presentation We introduced a 48-year-old man who suffered from HS in the hot summer. At the time of admission, he showed high body temperature, coma and shock. Later, he had laboratory evidence of rhabdomyolysis syndrome, acute kidney and liver damage, electrolyte imbalance, acid-base balance disorders, and high D-dimer levels. After several days of anti-shock treatment, his level of consciousness has improved but his vision has declined. The cerebral magnetic resonance imaging (MRI) showed symmetrical lesions of the bilateral posterior limb of internal capsule, putamen, external capsule and insula, and subcortical white matter, and cerebral magnetic resonance venography (MRV) showed the formation of deep cerebral venous thrombosis (DCVT). Therefore, the anti-coagulation treatment was given to patient. After timely clinical intervention, the symptom of the patient was gradually improved. Conclusions The case shows that HS can cause CVT. Therefore, we believe that when we need to identify the cerebral MRI findings of HS, early MRV can greatly help the diagnosis of the disease, and can effectively improve the prognosis.

Background

Heat stroke (HS) is a systemic inflammatory response syndrome (SIRS) whose pathophysiological process is similar to severe sepsis. The main clinical symptoms are core body temperature greater than 40 °C, multiple organ dysfunction including damage to the central nervous system (CNS). CNS abnormalities include inattention, memory loss, paralysis, convulsions, coma, and so on. Multiple organ dysfunction syndrome can occur in critically ill patients. Common complications of heat stroke are acute respiratory distress syndrome (ARDS), disseminated intravascular coagulation (DIC), shock, rhabdomyolysis, acid-base or electrolyte disorders, renal failure, cerebral edema and liver dysfunction[1, 2]. At present, the cerebral image changes of HS have been rarely reported and the mechanism of CNS damage caused by HS is not fully understood. In addition, which parts of the brain that are more susceptible to damage caused by HS remain unclear.

Cerebral venous thrombosis (CVT) is a rare form of cerebrovascular disease. Young people and children are the main patient groups of CVT. Due to the age of onset, and different cause of CVT, its clinical manifestations are diverse. The common clinical manifestations of CVT include high intracranial pressure symptoms (headache, papilledema, and vomiting), focal symptoms and encephalopathy-like symptoms. Although the encephalopathy-like symptoms are rare, however, most of the symptoms are
serious, manifested by epilepsy, mental disorders, confusion and coma[3]. There are many different risk factors of CVT for example: pregnancy/puerperium, oral contraceptive use, dehydration, cancer and infections. The most common brain parenchymal lesion of CVT is intracerebral infarction and hemorrhage[4], followed by focal cerebral edema, such as lesions in the thalamus and basal ganglia caused by obstruction of the deep venous system[5].

In this article, we report a case of HS with CVT with symmetrical lesions in both sides of the basal ganglia inside the brain.

Case Presentation

On a hot afternoon in July, a 48-year-old man developed symptoms of nausea, vomiting, headache, chest tightness and shortness of breath while working outdoors for two hours in a southern city of China. The outside temperature at this time was 35 °C. It is worth note that the patient lived in the north part of China and traveled to the southern part when he was sick. After 15 hours, he was in a coma and was transferred to the emergency department of the hospital. The patient was in good health before the onset of the disease. On arrival, temperature of the patient was 40.2 °C, blood pressure was 75/40 mmHg, pulse rate was 100 beats/min. His blood oxygen saturation was 95% under balloon assisted ventilation. Laboratory tests of the patient suggested rhabdomyolysis syndrome, acute kidney injury, hepatic disfunction, hyperkalemia, and metabolic acidosis. The levels of D-dimer in the serum of the patient was elevated (1022 ug/L, normal range 0–232 ug/L). Therefore, the patient was diagnosed as HS. He was immediately treated with ice blanket cooling, plasma exchange, and ventilator assisted ventilation et al. Brain CT scans, performed on the 3rd day, showed symmetrical low-density lesions in the bilateral basal ganglia. On day 7, the state of consciousness improved, but his sight was unclear. And eye examination was normal. Brain magnetic resonance imaging (MRI) was performed 8 days after admission. The cerebral MRI revealed slight hyperintensity signal in the bilateral putamen on diffusion-weighted imaging (DWI) sequence and bilateral symmetrical hypointensity in the middle of putamen, and hyperintensity around them on the apparent diffusion coefficient (ADC), fluid-attenuated inversion recovery (FLAIR) and T2 weight imaging (T2WI) sequence. The lesions showed hyperintensity in the middle of bilateral putamen, and hypointensity around them on T1 weight imaging (T1WI) sequence (Fig. 1A-E). Inferior sagittal sinus, straight sinus and Galen vein were not shown on cerebral magnetic resonance venography (MRV) performed on the 12th day. The superior sagittal sinus is poorly developed on MRV as well (Fig. 1F). Intravenous treatment of mannitol, subcutaneous injection of low molecular weight heparin calcium (5000IU, two times/day) was initiated in order to reduce high intracranial pressure and treat CVT. The cerebrospinal fluid (CSF) examination done on day 17 showed elevated levels of protein (1.87 g/L, normal range 0.15–0.45 g/L) and immunoglobulin G (267.0 mg/L, normal range: 0–34.0 mg/L). The CSF pressure was 210 mmH2O (normal range 80–180 mmH2O). Susceptibility-weighted imaging (SWI) obtained on the same day suggested bilateral hemosiderin deposition or hemorrhagic foci in the basal ganglia (Fig. 2). Follow-up MRI obtained 25 days after admission showed symmetrical abnormal signals in the bilateral posterior limb of internal capsule, putamen, external capsule and insula lobe. They were
hypointense on T1WI and hyperintense on T2WI, FLAIR and ADC. They were not limited by diffusion on DWI. Strip and dot-like signals which were isointense and slightly hypointense on T1WI and hypointense on T2WI can be seen in the lesions. DWI revealed bilateral hyperintensity on frontal lobe and occipital lobe (Fig. 3A1–E3). The distal superior sagittal sinus is undeveloped on the follow-up MRV. Inferior sagittal sinus, straight sinus and vein of Galen can be shown on it (Fig. 4). On day 28, MRI after gadolinium administration revealed bilateral hyperintensity of the basal ganglia, and the range of lesions was reduced compared with MRI obtained on day 25 (Fig. 5). The patient was discharged with blurred vision on the 38th day.

**Discussion And Conclusions**

Severe HS can be life-threatening, and nearly 30% of survivors suffering from permanent neurological sequelae[1]. The damage of the HS to the CNS is caused by a variety of factors. The heat itself is directly toxic to brain cells (such as Purkinje cells of the cerebellum)[6]. Excessive secretion of cytokines such as interleukin-1 can disrupt the blood-brain barrier, which in turn leads to vasogenic edema[7]. It has been reported that lesions in a HS patient show high intensity on DWI and hypointensity on ADC may indicate cell-derived edema rather than vasogenic edema[8]. DIC can cause intracerebral hemorrhage, and microthrombus derived from DIC can cause small vessel ischemic injury. Incomplete circulatory function can lead to cerebral ischemia and hypoxia injury. Metabolic disorders can cause myelin to dissolve[9]. In addition, recent studies have shown that excitotoxic injury may also be involved in the pathogenesis of heat stroke. Li J and his coworkers found that the NAA/Cr (N-acetyl aspartate/creatine) value was low on magnetic resonance spectroscopy imaging of HS patients[10].

For many years, only a few scattered cases have been reported in the literature to document the imaging findings of the central nervous system. Brain lesions caused by HS are usually symmetrical in imaging examination. Summarizing the MRI reported in the literature, the lesions are mainly distributed in the cerebellum, thalamus, basal ganglia, cerebral cortex, brainstem, hippocampus, subcortical white matter, external capsule and splenium. The lesions of the cerebellum are mainly concentrated in cerebellar cortex, superior cerebellar peduncle, vermis of cerebellum, corpora dentatum. Besides, the caudate nucleus of basal ganglia are high-incidence areas [6, 7, 9, 11-18]. However, our case presents symmetrical lesions of both sides of the posterior limb of internal capsule, putamen, external capsule and insula lobe and subcortical white matter, which has not been reported before. Due to the selective vulnerability of cerebellar neurons and Purkinje cells to thermal damage[14, 19], HS is prone to damage the cerebellum. Many HS patients show cerebellar symptoms such as ataxia[20]. However, no abnormal signals of the cerebellum were found on the MRI of our patient. Although some studies have found cerebellar atrophy delays on radiographic images[21].

It is reported in the literature that brain damage caused by HS is hyperintense lesions in the combination of the following imaging sequences: T2WI and FLAIR[9, 11], DWI and FLAIR[12], T1WI and T2WI. Sometimes these anomalous signals are limited to DWI[13]. However, the lesions in our case showed high and low mixed signals in the above sequences. Sometimes lesions can be enhanced on contrast-
enhanced examination. It has been reported that cerebral lesions of HS patient show punctiform hemorrhage on SWI[22].

Shock is one of the numerous risk factors for CVT[23]. Dentali F et al. reported that the sensitivity of D-dimer in CVT was 94%[24]. The deep venous system consists of straight sinus, vein of Galen and internal cerebral veins. MRI of deep cerebral venous thrombosis (DCVT) can often observe bilateral thalamic lesions involving the basal ganglia[25]. At the time of admission, the patient suffered a shock and his D-dimer level was increased. Due to bilateral basal ganglia lesions, we prescribed MRV examination and found abnormalities. It is rare in previous literatures to mention MRV changes in patients with HS, so this case deserves to be reported. Interestingly, the patient's lesions were mainly located in the bilateral basal ganglia without thalamic, which is different from the common lesions of DCVT. Two previous studies have shown that low molecular weight heparin is more suitable for the treatment of uncomplicated CVT than unfractionated heparin[26, 27]. In general, CVT can achieve a good prognosis after treatment[28]. In a study by Arauz A et al., approximately 90% of patients with obstructed cerebral veins were recanalized[29]. After the patient was treated with low molecular weight heparin for 13 days, the second MRV performed on the 25th day showed recanalization of the obstructed cerebral veins. On day 38, the patient was discharged with blurred vision, and Modified Rankin Scale Score was 2.

The mechanism of brain damage caused by HS is complex and diverse, and its cerebral imaging changes are various. We report a patient with high and low mixed signals of bilateral posterior limb of internal capsule—putamen—external capsule and insula, and subcortical white matter on MRI, and his MRV showed the formation of CVT. After treatment, his obstructed cerebral veins were recanalized. We believe that when the cerebral MRI findings of HS patient need to be identified, MRV and other related tests should be performed, and timely treatment can improve the prognosis.

Abbreviations

HS: Heat stroke; CNS: Central nervous system; CVT: Cerebral venous thrombosis; MRI: Magnetic resonance imaging; MRV: Magnetic resonance venography; SIRS: Systemic inflammatory response syndrome; ARDS: Acute respiratory distress syndrome; DIC: Disseminated intravascular coagulation; DWI: Diffusion-weighted imaging; ADC: Apparent diffusion coefficient; FLAIR: Fluid-attenuated inversion recovery; T2WI: T2 weight imaging; T1WI: T1 weight imaging; CSF: cerebrospinal fluid; SWI: Susceptibility-weighted imaging; NAA/Cr: N-acetyl aspartate/creatine; DCVT: Deep cerebral venous thrombosis

Declarations

Ethics approval and consent to participate

This study was approved by the Ethics Committee from The First Hospital of Jilin University, China.
Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Availability of data and materials

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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Authors’ contributions

LZC drafted the manuscript; JW, YXG, YML and JHY collected the patient information; MQZ and JFC did manuscript editing and data interpretation. All authors read and approved the final manuscript.

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**Table 1**

Table 1. Timeline of Brain Imaging
| Day | Brain CT/MRI Findings                                                                 | Brain MRV Findings |
|-----|--------------------------------------------------------------------------------------|-------------------|
| 3   | Symmetrical low-density lesions in the bilateral basal ganglia on CT.                 | ND                |
| 8   | Slight hyperintensity in the bilateral putamen on DWI. Bilateral symmetrical hypointensity in the middle of putamen, and hyperintensity around them on the ADC, FLAIR and T2WI. Hyperintensity in the middle of bilateral putamen, and hypointensity around them on T1WI. | ND                |
| 12  | ND                                                                                   | Inferior sagittal sinus, straight sinus and Galen vein were not shown on MRV. The superior sagittal sinus is poorly developed on MRV. |
| 17  | Bilateral hemosiderin deposition or hemorrhagic foci in the basal ganglia on SWI.    | ND                |
| 25  | Symmetrical abnormal signals in the bilateral posterior limb of internal capsule, putamen, external capsule and insula lobe. They were hypointense on T1WI and hyperintense on T2WI, FLAIR and ADC. They were not limited by diffusion on DWI. Strip and dot-like signals which were isointense and slightly hypointense on T1WI and hypointense on T2WI can be seen in the lesions. Bilateral hyperintensity on frontal lobe and occipital lobe on DWI. | ND                |
| 25  | ND                                                                                   | The distal superior sagittal sinus is undeveloped on MRV. Inferior sagittal sinus, straight sinus and vein of Galen can be shown on it. |
| 28  | Abnormal enhancement within the bilateral basal ganglia on MRI after gadolinium administration. | ND                |

“ND” represents “Not Done”

**Figures**
Figure 1

A-F. Brain MRI was performed 8 days after admission. The cerebral MRI revealed slight hyperintensity signal in the bilateral putamen on (A) DWI sequence and bilateral symmetrical hypointensity in the middle of putamen, and hyperintensity around them on the (B) ADC, (C) FLAIR and (D) T2WI sequence. The lesions showed hyperintensity in the middle of bilateral putamina, and hypointensity around them on (E) T1WI sequence. (F) Inferior sagittal sinus, straight sinus and Galen vein were not shown on MRV performed on the 12th day. The superior sagittal sinus is poorly developed on MRV.
Figure 2

SWI obtained on the same day suggested bilateral hemosiderin deposition or hemorrhagic foci in the basal ganglia
Follow-up MRI obtained 25 days after admission showed symmetrical abnormal signals in the bilateral posterior limb of internal capsule, putamen, external capsule and insula lobe. They were hypointense on (A1, white arrows) T1WI and hyperintense on (B1, white arrows) T2WI, (C) FLAIR and (D) ADC. They are not limited by diffusion on (E1) DWI. Strip and dot-like signals which were isointense and slightly
hypointense on (A1–2, black arrows) T1WI and hypointense on (B1–2, black arrows) T2WI can be seen in the lesions. (E2–3) DWI revealed bilateral hyperintensity on frontal lobe and occipital lobe.

Figure 4

The distal superior sagittal sinus is undeveloped on the follow-up MRV. Inferior sagittal sinus, straight sinus and vein of Galen can be shown on it.
On day 28, MRI after gadolinium administration revealed abnormal enhancement within the bilateral basal ganglia, and the range of lesions were reduced compared with MRI obtained on day 25.

**Figure 5**

On day 28, MRI after gadolinium administration revealed abnormal enhancement within the bilateral basal ganglia, and the range of lesions were reduced compared with MRI obtained on day 25.

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