CASE REPORT

Diffusion-weighted Magnetic Resonance Imaging of a Severe Heat Stroke Patient Complicated with Severe Cerebellar Ataxia

Ryusuke Ookura¹, Yoshihiko Shiro², Tomoko Takai¹, Miyuki Okamoto² and Masaaki Ogata¹

Abstract

A 78-year-old woman was admitted for severe heat stroke with brain damage. She was unconscious on arrival at the emergency room. Her armpit temperature was 42.0°C and blood pressure was 76/25 mmHg. She was rapidly cooled and given intensive treatment. On magnetic resonance imaging, T1- and T2-weighted images showed no significant signals, but diffusion-weighted images demonstrated localized symmetrical injuries of the cerebellum and thalami. She gradually became conscious, but severe cerebellar ataxia persisted.

Key words: heat stroke, cerebellar ataxia, thalamus, superior cerebellar peduncle, magnetic resonance imaging

(Inter Med 48: 1105-1108, 2009)
(DOI: 10.2169/internalmedicine.48.2030)

Introduction

It is known that the brain stem and cerebellum are susceptible to severe heat stroke. We present a case of severe heat stroke with brain damage, whose magnetic resonance imaging (MRI) showed interesting images of brain injury.

Case Report

A 78-year-old woman was witnessed to have collapsed in a graveyard at 0:30 pm on a sunny hot day in August. The outside air temperature was 32.2°C, the relative moisture was 63% and the average speed of wind was 5.7 m/s, according to the weather news. She was brought by ambulance to the emergency room at 1:00 PM. We obtained her medical history from her “handbook for handicapped people”, which presented that she was blind in the right eye because of past cranial operation and had hypertension and diabetes mellitus.

On arrival, her armpit temperature was 42.0°C, blood pressure was 76/25 mmHg, and pulse rate was 120/min, regular. SpO₂ was 99% under 5 L O₂ inhalation. She was comatose. Her arms and legs were rigid bilaterally. She had contact burns on the right side of waist and right thigh, and the medial side of left knee. Blood test showed high CPK level (469 IU/L). Peripheral white blood cell count, platelet count and CRP were respectively 5,160/μL, 16.7×10⁴/μL, 0.0 mg/dL. Chest radiograph, electrocardiography and brain computed tomography (CT) scanning showed no abnormal findings.

She was diagnosed as severe heat stroke and cooled initially by rapid infusion of 1,000 mL of 4°C solution, and ice pack on her head, armpits and inguinal regions. Just after admission, her urinary bladder temperature was decreased to 38.1°C. Then her body was cooled by a cooling blanket until the monitored urinary bladder temperature was kept under 38°C. Infusion of 400 mL glycerol per day was started for the purpose of preventing brain edema. The burns presented only the redness of the skin on arrival, however, rapidly formed blisters. Later, the burns advanced to the third degree as debridement was required. Blood test at 5 pm on the day showed that platelet count remarkably decreased to 5.4×10⁴/μL. We started continuous intravenous infusion of heparin by 10,000 unit/day for disseminated intravascular coagulation (DIC) syndrome. On day 3, she remained un-
conscious. The platelet count decreased to 1.9×10⁴/μL and bleeding tendency was recognized. We stopped the infusion of heparin and started the transfusion of fresh frozen plasma (FFP) to supply the coagulation factors. The following brain CT scanning showed no abnormalities. Electroencephalography (EEG) revealed that bilaterally symmetrical low voltage α-wave with excessive slow wave.

From day 5, her consciousness was gradually improved and she was able to respond to orders. Physical examination showed scanning speech, dysmetria, intention tremor and severe truncal ataxia. On the other hand, eyeball movements were intact and no signs of paralysis were found in her extremities. The platelet count was elevated to 8.5×10⁴/μL and bleeding tendency was diminished. On day 7, magnetic resonance imaging (MRI) showed high intensity signals in the cerebellum, midbrain and bilateral thalami only on diffusion-weighted image (DWI) with TR/TE, 4,999/118; 5.5 mm slice thickness/1 mm gap; b factor 1,000 s/mm² (Fig. 1). No signals were observed on T1- and T2-weighted images (Fig. 2). On day 21, however, the signal intensity on the cerebellum was lowered and high intensity signals on the midbrain and thalami almost vanished from MR images (Fig. 3). On day 38 when she was transferred to another hospital for skin grafting for burns, she still had significant cerebellar dysfunction.

**Discussion**

It is known that severe heat stroke causes many kinds of neurological dysfunctions. Several case reports presented cerebellar dysfunctions caused by heat stroke (1-5). Lee et al reported that there may be selective vulnerability of cerebellar neurons to heat injury (6). Some reports showed a case of heat-stroke complicated with progressive cerebellar atrophy after months (1, 3, 7). Therefore, cerebellar dysfunctions caused by heat stroke should be followed up for a long time.

Concerning the present case, the complication of secondary brain damage was expected on her arrival at the emergency room. We cooled her immediately and aggressively used glycerol for brain protection. In the clinical course, both CT scan and MRI did not show any asymmetrical ischemic lesions or edema in the brain. MRI, however, showed the symmetrical lesions localized in the cerebellum, midbrain and thalami on DWI. These lesions were thought to be caused directly by heat injury.

Bazille et al reported a case of heat stroke whose MRI
showed no abnormal signals on T1- and T2-weighted images but high intensity signals in the central tegmentum on DWI (8). They concluded that the region of high intensity signals corresponded to the superior cerebellar peduncles (SCP) and the injury of the region resulted in cerebellar deafferentation. In the present case, the highest intensity signals appeared from the anterior region of cerebellum to the red nuclei via the tegmentum and the posterior midbrain. Only DWI demonstrated the abnormalities related to the brain damage by heat stroke. The areas of the high intensity signals were regarded as corresponding to the SCP running which is thought to be one of the most vulnerable regions to heat (8).

Additionally, Purkinje cells in the cerebellar cortex are also known as vulnerable to heat (1, 2, 5, 8). Krainer reported that almost complete loss of Purkinje cells was revealed in the histopathological examinations of autopsy in a case of heat stroke (9). The afferent tract from Purkinje cells changes the neurons in dentate nucleus and enters the tegmentum of midbrain through SCP, and divides into two tracts, the cerebellorubral tract which ends to red nucleus, and the cerebellothalamic tract which ends to ventrolateral nucleus in thalamus. Both tracts are composed of axon fibers from the dentate nucleus. In our case, it was supposed that the diffused high intensity signals in the cerebellum on DWI demonstrated the injury of the axon fibers in the two tracts from Purkinje cells, and that the injury of Purkinje cells was related with the clinical symptoms such as severe ataxia.

Electroencephalogram detected diffuse slow wave abnormality in the present case. Gandjour reported a case of heat stroke with alpha-coma in EEG (10). However, the significance of EEG in heat stroke was not determined because few reports discuss the abnormalities in EEG.

Heat stroke can cause some serious systemic complications such as rhabdomyolysis, DIC syndrome and multiple organ failure. In addition, it can cause various types of brain damage, which include neurological dysfunctions primarily related to heat and the ones related to circulatory insufficiency and so on. Therefore, it is necessary to always carefully pay attention to such brain damage as well as systemic complications in the diagnosis and treatment for heat stroke.

References

7. Yaqub BA, Daif AK, Panayiotopoulos CP. Pancerebellar syndrome

© 2009 The Japanese Society of Internal Medicine
http://www.naika.or.jp/imindex.html