

## Heat stroke induced cerebellar dysfunction: A “forgotten syndrome”

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Received: August 28, 2013 Revised: September 26, 2013

Accepted: November 2, 2013

Published online: November 16, 2013

### Abstract

We report a case of heat stroke induced acute cerebellar dysfunction, a rare neurological disease characterized by gross cerebellar dysfunction with no acute radiographic changes, in a 61 years old ship captain presenting with slurred speech and gait ataxia. A systematic review of the literature on heat stroke induced cerebellar dysfunction was performed, with a focus on investigations, treatment and outcomes. After review of the literature and detailed patient investigation it was concluded that this patient suffered heat stroke at a temperature less than that quoted in the literature.

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**Key words:** Heat stroke; Cerebellar syndrome; Ataxic hemiparesis; Hyperthermia; Cerebellar atrophy

**Core tip:** Heat stroke induced cerebellar damage is a

rare and challenging neurological problem. The cerebellum is vulnerable to high temperature which may cause irreversible cell damage with permanent disability. Thorough evaluation with neuroimaging and laboratory investigations are required to exclude alternative diagnosis.

Kosgallana AD, Mallik S, Patel V, Beran RG. Heat stroke induced cerebellar dysfunction: A “forgotten syndrome”. *World J Clin Cases* 2013; 1(8): 260-261 Available from: URL: <http://www.wjgnet.com/2307-8960/full/v1/i8/260.htm> DOI: <http://dx.doi.org/10.12998/wjcc.v1.i8.260>

### INTRODUCTION

Heat stroke induced cerebellar damage is a rare and challenging neurological problem. The cerebellum is vulnerable to high temperature which may cause irreversible cell damage with permanent disability. Thorough evaluation with neuroimaging and laboratory investigations are required to exclude alternative diagnosis.

### CASE REPORT

A previously well 61 years old man presented to Bahrain hospital with acute cerebellar dysfunction and was subsequently transferred to Australia. He was the captain of an oil tanker, sailing from Mozambique to Bahrain, when found by his crew, lying on his bed, awake but unresponsive, except blinking his eyes and moving his head, having failed to respond to several calls. The ship's log recorded the cabin's air conditioner non-functioning for several days, with temperature reaching 38 °C. He reported feeling unwell with reduced oral intake in the preceding days with bridge officers questioning dehydration. On presentation, he was semiconscious, (Glasgow Coma scale: 11/15), febrile (38 °C), stable vital signs and severe dehydration. Pupils were equal and reacting to light. Doll's eye

manoeuvre was normal. He had slurred dysarthria, bilaterally flaccid limbs, up-going toes and a maculopapular truncal rash. Provisional diagnosis favoured heat stroke, rather than vascular stroke or viral/post viral illness. He was transferred from Bahrain to Sydney, after initial resuscitation. Arriving to Liverpool, he was alert and oriented, responsive to commands but cerebellar dysarthria made speech unintelligible. Cranial nerve examination was normal with no nystagmus and motor function revealed normal tone, 5/5 power, brisk, symmetrical reflexes but positive Babinski sign bilaterally. Sensation was unimpaired. Cerebellar examination was grossly abnormal, with severe dysarthria, dysmetria, disdiadochokinesis, heel to shin ataxia and ataxic gait, requiring two people to assist ambulation. Routine biochemistry and haematology were normal. Lumbar puncture revealed marginally raised protein (0.64 g/L, normal range 0.15-0.45 g/L) with normal cells and negative viral polymerase chain reaction.

Vasculitic screen, paraneoplastic antibodies and heavy metal screen were normal. Infectious aetiologies were excluded, with negative serology for HIV, *tropheryma whipplei*, hepatitis and other viral pathogens. Imaging, including computed tomography (CT) brain, CT Circle of Willis, magnetic resonance imaging (MRI) and MRI spectroscopy were normal. The patient made very slow progress and was transferred for rehabilitation.

## DISCUSSION

Heat adversely affects almost all organ systems, with the central nervous system (CNS) particularly vulnerable and, within the CNS, the cerebellum is most susceptible<sup>[1]</sup>. Hyperthermia may cause organ failure, unless managed aggressively in the acute setting, especially if the core body temperature exceeds 40 °C<sup>[1,2]</sup>. Such elevated temperature was not reported in this case but that does not exclude it. Although the mechanism is not fully understood, several theoretical and experimental models have been described. A recent report claims that hyperthermia increases heat shock protein synthesis and cytokine activation, with a sepsis like reaction disrupting the blood brain barrier, causing vasogenic oedema and cell death<sup>[2]</sup>. This theory is supported by animal models where inhibition of the cytokine pathways inhibits cell damage from heat stroke<sup>[2,3]</sup>. Heat is directly toxic to cerebellar purkinje cells, which have the highest concentration of heat shock protein in order to counteract increased sensitivity<sup>[3]</sup>. A post mortem series in the early 1950's showed that the cerebellum is particularly vulnerable to heat damage. This was confirmed by recent radiological studies<sup>[4,5]</sup>. The presentation

is variable, ranging from coma to tetraparesis. Post mortem studies have shown swelling of purkinje cells and cell death<sup>[3]</sup>, with the duration of hyperthermia correlating with the extent of cell death<sup>[6]</sup>. A case report identified a diffuse pattern of heat related brain injury, involving the subcortical white matter and hippocampus in addition to the cerebellum<sup>[7]</sup>.

Despite the air conditioner malfunctioning for some days, in the present case, the duration of exposure to hyperthermia was not defined. Heat stroke induced cerebellar atrophy, as per CT and MRI, generally involves both the vermis and cerebellar hemispheres, with cerebral hemispheres being spared in almost all reported cases.

Similar changes are seen in other degenerative, drug induced and paraneoplastic diseases<sup>[5,6]</sup>. Patients with marked cerebellar dysfunction, may be radiologically normal with cerebellar atrophy appearing months or years later on CT or MRI scans<sup>[5]</sup>. This patient had normal investigations and has been clinically stable over the last month, suggestive of non-progressive cerebellar damage. He was transferred for rehabilitation and will be followed with imaging to determine future cerebellar atrophy. While the literature describes hyperthermia involving temperatures in excess of 40 °C<sup>[1,2]</sup> such elevated temperatures were not recorded in this patient, yet other alternative diagnoses were excluded with detailed investigations, thereby suggesting cerebellar damage may occur with lower temperatures than previously described.

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**P- Reviewers:** Lu KC, Schencking M **S- Editor:** Ma YJ  
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