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Letter to Editor

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Hyperthermia inducing posterior reversible encephalopathy syndrome

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Ahmed Yassin[1], Belal Aldabbour[1], Rafael Rodriguez[2,3], Xiang Fang[2,3]

[1] Department of Neurology, King Abdullah University Hospital, Jordan University of Science and Technology, Irbid, Jordan
[2] Department of Neurology, University of Texas Medical Branch, Galveston, Texas, United States of America
[3] Mitchell Center for Neurodegenerative Diseases, University of Texas Medical Branch, Galveston, Texas, United States of America

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Posterior reversible encephalopathy syndrome (PRES) is an increasingly recognized clinical and radiologic syndrome which presents with rapidly progressing symptoms that include headache, altered mental status, seizures, and visual disturbances.[1] It is most typically associated with hypertension, but it has also been reported in association with sepsis, preeclampsia, immunosuppressive agents, and autoimmune diseases.[1] Hyperthermia is not of the classical conditions associated with PRES, and review of literature reveals only one documented case of hyperthermia-induced PRES.[2] Here we report an atypical case in which PRES was induced by hyperthermia in a burn victim.

A 54-year-old Caucasian man was admitted to the burn unit with second- and third-degree burns that involved more than 70% of his body surface area after a gas tank explosion. On arrival, the patient was awake, alert, and able to communicate with his wife and the medical team through eye blinks and head nods. Brain computed tomography (CT) scan on admission was free from acute insults. The first week of his admission was complicated by fevers ranging between 38.1-38.9 °C. Blood cultures were positive for Pseudomonas (P.) aeruginosa. The infection responded to broad spectrum antibiotics, with fevers subsiding within three days, and the clinical course through the following week was uneventful.

In the third week of hospitalization, the fever recurred, spiking acutely to a maximum temperature of 41.6 °C. It was persistent and remained elevated in this range for 4 to 5 days during which, it was poorly responsive to antipyretics and cooling measures as well as to broad spectrum antibiotics. Meanwhile, the wife reported a change in the patient's mental status as

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Corresponding Author: Belal Aldabbour
Email: belal90md@gmail.com

his responses became delayed, and he stopped following visual cues. Neurologic examination revealed an awake and alert patient unable to follow commands and with absent blink to threat. No other focal deficits could be elicited through the limited neurological examination. There were no hypertensive episodes. Blood work revealed leukocytosis, and a thorough infectious workup, which included repetitive blood, urine, wound, and sputum cultures as well as chest X-rays and a transthoracic echocardiography was unrevealing.
A follow up head CT scan without contrast demonstrated bilateral temporo-occipital hypodensities, which were not noted on head CT at the time of admission. Magnetic resonance imaging (MRI) of brain with and without contrast was done. It revealed diffuse T2 and FLAIR hyperintensities in the bilateral occipital lobes as well as the splenium of corpus callosum, bilateral thalami, and mesial temporal lobes (Figure 1).

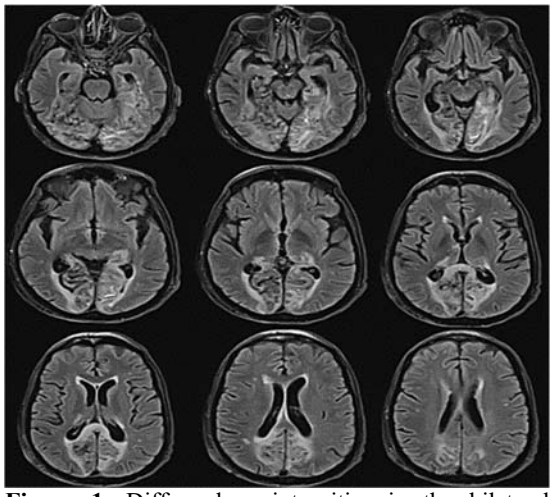

Figure 1. Diffuse hyperintensities in the bilateral occipital lobes, splenium of corpus callosum, bilateral thalami and mesial temporal lobes, apparent on FLAIR

Diffusion-weighted imaging (DWI)/apparent diffusion coefficient (ADC) demonstrated faint diffusion restriction mainly in the occipital lesions, while T1 pre and post contrast showed hyperintensity and enhancement patterns consistent with petechial hemorrhage (Figure 2). Magnetic resonance angiography (MRA) and venography (MRV) of the head and neck were unremarkable. Electroencephalography (EEG) showed generalized slowing, with no focal slowing or epileptiform discharges.

The clinical and radiological presentation was consistent with PRES due to hyperthermia secondary to burn-induced hypermetabolic state. High fever persisted over the following days, then responded gradually to antipyretic measures, and subsided after two weeks, at which point the patient was able to track hand motion and followed command. Follow-up brain MRI was not done as the patient suffered further complications that ultimately resulted in multiorgan failure and death within few weeks.

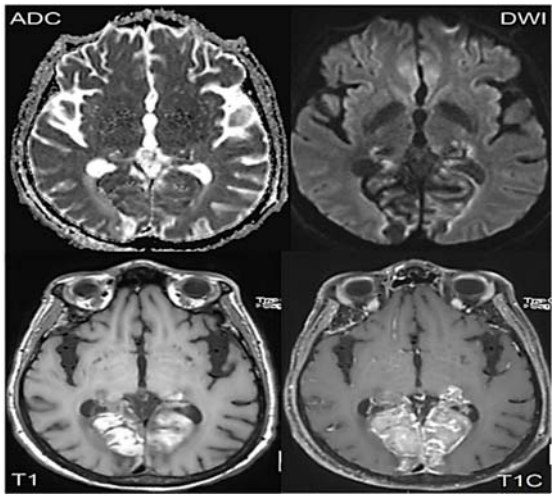

Figure 2. Diffusion-weighted imaging (DWI)/apparent diffusion coefficient (ADC) demonstrating faint diffusion restriction, while T1 pre and post contrast shows hyperintensity and enhancement patterns consistent with petechial hemorrhage

Hyperthermia can lead to vasogenic cerebral edema through several mechanisms such as increasing the permeability of blood-brain barrier, inducing alterations in regional cerebral blood flow, or disrupting autoregulatory mechanisms and cerebral pressure-flow autoregulation.[2-4] The cerebellum and Purkinje cells appear to be most vulnerable to hyperthermic insult and permanent injury; while structures in the limbic system, brainstem, basal ganglia, and cortex are also liable for heat injury.[2,4] In the case reported by Tan et al., permanent cerebellar atrophy was noted.[2] In our case, however, the cerebellum was apparently spared during the acute stage while remaining structures in the posterior circulation were affected in keeping with neuroimaging findings of PRES.[1] A more longitudinal follow-up was not possible in our case due to the unfortunate death of the patient.

Neuroimaging in sepsis-associated encephalopathy (SAE) may be normal in some cases, or it may reveal nonspecific findings such

as multiple strokes or white matter lesions in the centrum semiovale,[5] also, the lack of identifiable pathological organisms and the unresponsiveness to empiric antibiotics point away from SAE. Other possible causes of PRES are less likely to have contributed to our patient's picture. Extensive cultures and investigations for a septic source were repeatedly negative. The severity and delayed onset of hyperthermia were uncharacteristic of sepsis and more likely due to hypermetabolic state. In addition, the initial septic episode due to P. aeruginosa was relatively brief and responded well to antibiotics. Moreover, the latency of more than two weeks until the development of PRES is unusual in sepsis-induced PRES. Uncontrolled hypertension and drugs as possible contributing factors were excluded upon review of the medical chart.

Our case represents the second report in the literature of PRES precipitated by severe hyperthermia. Early diagnosis and aggressive treatment of hyperthermia may prevent the development of this complication in patients with heat exposure or help in mitigating its sequelae.

Conflict of Interests

The authors declare no conflict of interest in this study.

Acknowledgments

None.

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