*Case Report*

# Posterior Reversible Encephalopathy Syndrome in a Child Stung by *Androctonus mauretanicus* Scorpion

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Posterior reversible encephalopathy syndrome (PRES) after scorpion sting was very rarely reported in literature. This is an authenticated report of PRES occurring, in a 3-year-old previously healthy girl, as a complication of the Moroccan *Androctonus mauretanicus* sting. According to the available and recent data, we attempt to discuss the potential mechanisms leading to this neurologic disorder and to determine the possible cause–effect relationship between scorpion venom and its development. **Key Words:** *Androctonus mauretanicus*—child—posterior reversible encephalopathy syndrome—scorpion sting.


Posterior reversible encephalopathy syndrome (PRES) was initially described by Hinchey in 1996 as a clinicoradiologic entity.[1-3] Even if its clinical picture, including cephalagias, altered mental status, visual disturbance, and seizures,[1-3] is aspecific, the radiologic findings are characteristic by the revealing of focal vasogenic edema mostly developed in the subcortical white matter and preferentially in the parieto-occipital areas of the brain.[2-5]

Although numerous PRES-related risk factors have been identified in many studies such as hypertension, pre-eclampsia, eclampsia, chemotoxic agents, and renal injuries,[3-6] the pathophysiological process leading to this condition remains unclear and controversial.[2-5]

Also, reports of PRES after scorpion sting are infrequent and amount to only a recent and unique observation where the Brazilian *Tityus bahiensis* envenomation was incriminated.[7] The second case in literature is described here.

## Case History

A 3-year-old previously healthy girl was stung on the right foot by a black scorpion. An hour and a half later, she was admitted to our hospital with vomiting and impaired consciousness. The physical examination found a confused, with a Glasgow Coma Score of 14, and polypneic (respiratory rate of 36 breaths per minute) girl. She also had cold extremities, her pulse was 160 bpm and her blood pressure was 160/90 mm Hg.

During the first hours of her admission, this child appeared to have labile blood pressure marked by episodes of hypertension (the maximal peak was 170/110) alternating with those of hypotension. Then her blood pressure has decreased significantly and rapidly to the point at which she became unconscious and presented a severe cardiogenic shock. So, dobutamine was promptly started at 20 μg/kg/minute, and she was intubated and ventilated under continuous sedation. Despite these measures and the high doses of dobutamine, the blood

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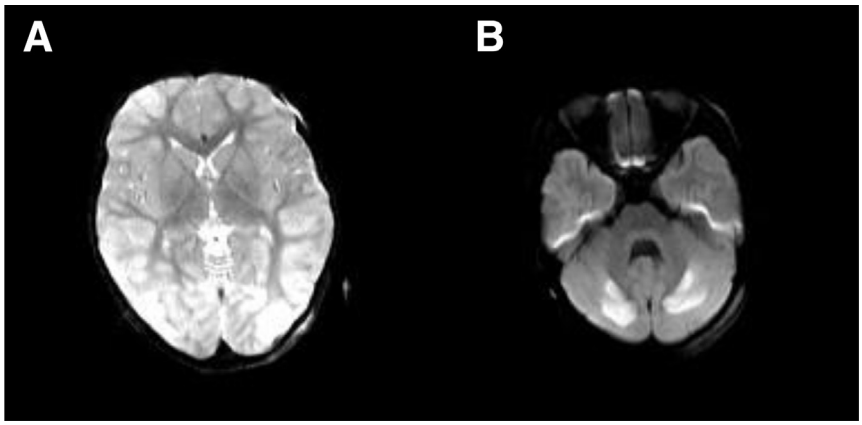


**Figure 1.** *(A): Areas of hyperintensities on axial T2 of brain magnetic resonance imaging (MRI) involving, in a symmetrical and diffuse way, the subcortical white matter in parieto-occipital lobes. (B) Axial MRI image (fluid-attenuated inversion recovery) shows a hyperintense white matter in cerebellar regions.*

pressure lowered progressively. At that time, norepinephrine was added, therefore, the hemodynamic stabilization was accomplished. Initial laboratory findings revealed hyperleukocytosis (19 700 cells/mm$^3$), thrombocytopenia (78 000 platelets/mm$^3$), and high level of blood glucose. A cardiogenic pulmonary edema was also observed in chest radiograph.

Within 48 hours of her hospitalization, the respiratory and hemodynamic states showed considerable improvement, which enabled us to wean her off the ventilator and hemodynamic supports. Then, she was extubated but her Glasgow Coma Scale score was 12/15. Five hours later, she developed partial tonic seizures (right limbs and face). These seizures were refractory to midazolam and diazepam, therefore, phenobarbital infusion and reintubation were required. The levels of blood electrolytes were within the normal ranges.

At this point, a computed tomographic scan of the brain was performed and was normal. A cerebral magnetic resonance imaging was carried out showing subcortical, diffuse, and symmetrical hypersintensity in T2 and fluid-attenuated inversion recovery sequences, particularly well pronounced in the parieto-occipital and cerebellar regions, which is compatible with the radiologic pattern of "PRES" (Fig 1).

The girl showed visible clinical improvement within 1 week, so she was extubated. On her discharge after 20 days, a low visual acuity and right arm monoparesis were found. Six months later, a progressive neurologic recovery was noted (Fig 2).

## Comments

Scorpionism is endemic and represents a real public health problem in Morocco particularly in southern regions.[8,9] It is commonly caused by *Androctonus mauretanicus*, also labeled as black scorpion, which belongs to Buthidae family and is the most dangerous arthropod for human beings in Morocco.[8,9] Its envenomation occurs generally in the summer period and usually results in severe and serious complications, especially, among children.[8,9]

In fact, it is well known that *A mauretanicus* venom is a complex and heterogenous mixture of peptides, which mainly contains neurotoxins.[9,10] Then the envenomed patient can develop a wide spectrum of systemic symptoms as a consequence of sympathetic and parasympathetic overstimulation.[10,11] Even more, the heart–lung failure is the most severe and life-threatening complication resulting from both this venom-induced autonomic storm (indirect effect) and a toxic myocarditis (direct effect).[10,11] However, the central nervous system impairment after severe envenomation by *A mauretanicus* can uncommonly occur in our context. As an illustration, there were 2 cases of acute ischemic strokes after *A mauretanicus* sting, which were recently reported in Morocco, and the underlying mechanisms linking these cerebral accidents and scorpion venom were uncertain and multifactorial in origin (cerebral hypoperfusion secondary to cardiogenic shock, consumption coagulopathy, spasm of the brain arteries, vasculitis, and cardiogenic brain embolism).[10,12,13] Moreover, this case, to our best knowledge, is the first authenticated case describing a PRES as a toxic encephalopathy after *A mauretanicus* sting.[7,14]

Since its first description, PRES has witnessed an increasing interest over the past 15 years, particularly in childhood.[15-19] Clinically, seizures are the most common clinical manifestation found in children.[17-19] Also, the brain magnetic resonance imaging features were atypical in pediatric population (approximately in 80% of cases in Chen study).[17]

Whereas several authors have tried to draw up an exhaustive list of PRES-related etiologies and propose a myriad of hypotheses,[3-6,17] its pathophysiological process remains unclear and intriguing.[6,14,20] Above all, the vasogenic edema, typically seen in PRES is more likely because of the blood–brain barrier disruption, which may be probably induced by 2 mechanisms: hyperperfusion and/or endothelial dysfunction.[14,15]