Pediatric ischemic stroke following a scorpion sting

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Introduction

Stroke following scorpion stings is rare and potentially fatal. In its severe form, it involves life-threatening respiratory or cardiac damage; it may also cause the neurological severity of systemic manifestations.

Case report

We report the case of a 15-month-old male infant with febrile seizure, admitted in Intensive Care Unit 4 hours after being stung by a yellow scorpion.

He developed impaired consciousness, hemiplegia and respiratory distress followed by shock.

The chest radiograph was normal.

Echocardiographic revealed Severe LV dysfunction (LVEF <30%). Troponin was elevated.

The brain computed tomography showed bilateral subcortical white matter hypodensity. Axial T2 of brain magnetic resonance imaging (MRI) showed subcortical white matter hyperintensities.

The child was treated with scorpion antivenom, vasopressors.

He required mechanical ventilation for 5 days. The patient responded favorably to treatment after a 19-day hospital stay.

Complete resolution of hemiparesis during the follow period till the age of 4 was observed.

Comments

• **Scorpion envenomation** is a life-threatening condition in different regions of the world, particularly for pediatric populations. The condition is an endemic problem in the Middle East, South America, India, and Africa. **Buthus Occitanus Tunitanus** is a Saharan harmful species principally described in southern Tunisia, Algeria and Egypt.

• **children** exhibit more severe symptoms and have a considerably increased mortality rate compared with adults.

• **Neurological manifestations** are an indicator of the severity of scorpion sting.

• **In infants** blood brain barrier (BBB) is immature, venom toxin crosses the BBB and alters directly nerve cells. The neurotoxins partially inactivate depolarized axonal sodium channels, resulting in membrane hyperexcitability and repetitive uncontrolled axonal firing. Ultimately, excessive neuromuscular activity and autonomic dysfunction result.

• **The thermoregulatory disturbances** can be explained by a direct action of the scorpion venom on the central nervous system, or by massive liberation of cytokines IL-1α, IL-6, IL-10, and TNF-α.

• **The neurological manifestations of Buthus Occitanus Tunitanus venom** and histological abnormalities have been related to the high level of circulating catecholamines elicited by a direct effect of the venom on the sympathetic nervous system and adrenals. These severe neurological manifestations can be explained by either hypertensive encephalopathy, brain ischemia secondary to pulmonary edema and cardiac shock, or direct effect of the venom on the CNS.

Neurological ischemia can result from :

*Hypoxemia secondary to Pulmonary Edema and cardiogenic shock which would aggravate the central nervous system ischemia.

*Liberation of catecholamines in the adrenergic nerve terminals which causes hypertension leading to stroke and oedema (press syndrome).

*Prostaglandins and cytokines liberation (inflammatory response after envenomation) increase the systemic vascular resistance, decrease cerebral blood flow. IL-1, IL-2, IL-6 and TNF-α decrease the activity of natural anticoagulant mechanisms and cause disseminated intravascular coagulation.

Conclusion

This case illustrates a rare complication of **Buthus Occitanus Tunitanus** envenomation: **ischemic stroke**. The underlying mechanisms linking these cerebral accidents and scorpion venom were uncertain and multifactorial in origin (cerebral hypoperfusion secondary to hypotension, cardiogenic shock, consumption coagulopathy, spasm of the brain arteries, vasculitis, and cardiogenic brain embolism).

Early rapid consultation and treatment had improved our patient outcomes.