

## **Scorpion bite, a sting to the heart and to coronaries resulting in Kounis syndrome**

Sir,

In the very interesting paper published in IJCCM,<sup>[1]</sup> the authors reported on a 14-year-old-boy who was bitten by scorpion and developed chest pain, vomiting,

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loose stools, cold sweating, raised jugular venous pressure, tachycardia and hypotension. Laboratory findings showed increased troponin T, proBNP and CPKMB, bilateral lung fluffy shadows, reduced ejection fraction, and ST segment elevation in II, III, aVF, V3, V4, V5, and V6, and T-wave inversion in I, aVL, V1 and V2 leads compatible with acute myocardial injury and pulmonary oedema. This case raises the following important questions on pathophysiology, treatment, and differential diagnosis of scorpion envenomation:

- Centruroides and parabuthus scorpions are associated primarily with neuromuscular toxicity, whereas androctonus, buthus, and mesobuthus scorpion envenomation is associated with cardiovascular involvement, resulting from hyperstimulation of autonomic centers release of catecholamines and vasoactive peptides. Since venom specific immunoglobulin E (IgEs) have been identified by using skin tests and IgE immunoblots and mast cell degranulation and histamine release are also involved in scorpion venom-induced-inflammatory pain, hypersensitivity cardiovascular involvement is also present<sup>[2]</sup>
- Myocardial infarction has been reported following scorpion envenomation on some occasions.<sup>[3,4]</sup> Scorpion envenomation may result in immediate late and delayed hypersensitivity reactions in susceptible individuals. Cardiovascular toxicity implies venom amount-dependent action with progressing effects, while hypersensitivity means inflammation causing either Kounis hypersensitivity-associated acute coronary syndrome or hypersensitivity myocarditis, which are not venom amount dependent and may arise at any time during or soon after envenomation. It seems likely that the described patient developed coronary vasospasm manifesting as Type I variant of Kounis syndrome progressing to myocardial injury.<sup>[5]</sup> Hypersensitivity or toxic myocarditis could not be confirmed because neither coronary angiography nor magnetic resonance imaging was available. Coronary angiography is normal in myocarditis while in Kounis syndrome may reveal the coronary spasm. Magnetic resonance imaging shows subendocardial concentration of gadolinium in Kounis syndrome while in myocarditis the gadolinium is concentrated in subepicardial areas<sup>[5]</sup>
- Several studies have shown no significant difference in patients on steroids and in steroid-free patients in terms of mortality and intensive care unit length of stay. The hydrocortisone hemisuccinate regimen has limited effect in critically ill envenomated children and, therefore, it should not be recommended.<sup>[6]</sup>

Antivenom combined with prazosin and dobutamine is indicated for cardiovascular effects and benzodiazepines for neuromuscular involvement. Adrenaline and fluid resuscitation – not glucocorticoids, antihistamines, or mast-cell stabilizers – are the mainstays of treatment for scorpion venom-induced anaphylaxis. However, administration of antihistamines and mast cell stabilizers, but not hydrocortisone, could be used as a supplementary treatment of life-threatening scorpion envenomation. The described patient developed a pulmonary edema and received corticosteroids but not prazosin. Alpha receptor stimulation plays an important role in the pathogenesis of pulmonary edema. The use of prazosin, a postsynaptic alpha blocker, is recommended for significant sympathetic symptoms. Cardiogenic pulmonary edema with arterial hypertension responds favorably to prazosin. Oral prazosin is fast acting, easily available, and highly effective. Corticosteroids are beneficial in interstitial noncardiogenic pulmonary edema.

Scorpion envenomation is a complex condition, which requires rigorous action, meticulous treatment and careful follow-up.

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