

Severe envenomation by *Hottentotta tamulus* (white scorpion) presenting with cardiogenic shock

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Introduction

First report on *Hottentotta tamulus* (white scorpion) as a medically important species of scorpion was published in 2013¹. *Hottentotta tamulus* was subsequently reported as a cause of severe envenomation, almost all cases being reported from the Jaffna peninsula of Sri Lanka². We report a six-year-old child with severe envenomation following *Hottentotta tamulus* envenomation as the first reported case from the Mannar district. Furthermore, cardiogenic shock has not previously been reported in a Sri Lankan child.

Case report

A 6-year-old boy was transferred to the District General Hospital (DGH) Mannar following a witnessed white scorpion bite over his right foot. At presentation, he was restless, agitated, and had involuntary movements of the limbs and body and abnormal behaviour.

On arrival at the local hospital, he was breathing spontaneously with good effort. However, he had cold peripheries and low volume pulses. Sequential blood pressures (BPs) were 120/80 mmHg, 100/90 mmHg and 90/60 mmHg; sequential oxygen saturations (SpO₂) were 99%, 86% and 62%. He was managed with intramuscular (IM) adrenaline, intravenous (IV) hydrocortisone and IV saline bolus (20 ml/kg). However, he continued to have haemodynamic instability progressing to hypotension (BP 60/40 mmHg) and non-palpable pulses. He was transferred to the DGH Mannar.

In DGH Mannar, he had cold peripheries, prolonged capillary refill time (>6 seconds), non-palpable pulses, an undetectable SpO₂ and the BP was 60/40 mmHg. He was given IM adrenaline (0.15ml of 1:1000 bolus), IV hydrocortisone (60mg), IV chlorpheniramine (5mg) and a saline bolus (20ml/kg). There was poor response; another 3 boluses of IM adrenaline were given followed by an IV adrenaline infusion. One more saline bolus was given (20 ml/kg). Still, response was poor with BP un-recordable, cold peripheries, no improvement of pulse volume and unrecordable SpO₂. IV adrenaline infusion (1µg/kg/min) and IV dopamine 10µg/ kg/ min) were started. SpO₂ was

still unrecordable. Child was intubated. Just after intubation, he went into cardiac arrest and needed cardiorespiratory resuscitation. Heart beat returned but SpO₂ was not more than 60%. Chest x-ray showed pulmonary oedema. Electrocardiogram (ECG) showed sinus tachycardia. 2D echocardiogram revealed myocarditis with severe left ventricular dysfunction; cardiac troponin I titre was 2.26 ng/ml. He had excessive salivation, tachypnoea and sweating. Arterial blood gases revealed severe metabolic acidosis and hypoxaemia (pH 7.04, HCO₃ 10 mEq/l, pO₂ 39 mmHg).

Antidote prazosin 0.25mg was administered through a nasogastric tube as scorpion-specific anti-venom and IV prazosin were unavailable. BP started to increase and pulse volumes improved. BP was 115/67 mmHg while on 2 inotropes while giving frusemide to control pulmonary oedema. Oxygen saturation picked up to 95%.

Discussion

Case reports of *Hottentotta tamulus* envenomation in Sri Lanka mainly originated from the Jaffna peninsula^{2,3}. Scorpion envenomation can lead to significant systemic manifestations. This boy presented with agitation, involuntary movements and abnormal behaviour following a scorpion bite. These symptoms are consistent with the neurotoxic effects of scorpion venom, which can result in autonomic dysfunction. Presence of vomiting, excessive salivation, tachypnoea, and sweating further supports the involvement of the autonomic nervous system⁴. Patient's initial presentation at the local hospital showed haemodynamic instability with cold peripheries and low-volume pulses, despite adequate oxygenation, suggesting a toxic reaction leading to cardiovascular compromise⁵. Tachyarrhythmia can lead to further haemodynamic instability, especially in the context of toxin-induced myocardial dysfunction⁶.

In this child, ECG showed sinus tachycardia and 2D echocardiogram revealed myocarditis with severe left ventricular dysfunction. Scorpion venom induces a significant surge in hormones epinephrine and norepinephrine. Initially, this leads to tachycardia, high BP, and increased agitation. As cardiac function deteriorates, respiratory distress, pulmonary oedema, and hypotension can follow. Children are especially susceptible to these effects⁷.

Management initially included treatment for anaphylaxis with IM adrenaline. However; the patient's condition deteriorated, with worsening hypotension and non-palpable pulses. The presence of severe metabolic acidosis (pH 7.04) and hypoxaemia indicated significant systemic involvement, potentially exacerbated by decreased cardiac output. Progression to cardiorespiratory arrest in this patient underscores the severity. Successful resuscitation and subsequent management with intubation, prazosin,

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and frusemide highlight the critical care interventions required.

The absence of specific antivenom and IV prazosin posed significant challenges in managing this case, illustrating the resource limitations faced by rural healthcare settings⁸. Despite these limitations, patient's condition stabilized with available treatment and his clinical status improved markedly, as evidenced by normalized BP and good pulse volume. The rapid improvement following administration of oral prazosin highlights its role in managing scorpion envenomation when specific antivenom is not available.

Parental consent

Written informed consent was obtained from the parents for publication of this case report.

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