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Anaphylaxis to scorpion antivenin and its management following envenomation by Indian red scorpion, *Mesobuthus tamulus*

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Abstract

Mesobuthus tamulus is an Indian red scorpion that is responsible for numerous cases of scorpion stings in the Indian subcontinent. Antivenin, vasodilators, and benzodiazepines are medications of choice in the treatment of scorpion bites. Adverse reactions such as anaphylaxis to antivenin have been infrequently described in the literature. We, herein, present a case of a 42-year-old man stung by Indian red scorpion while gardening at home in India, who presented with extreme pain at the sting site and signs of cardiotoxicity. He was treated with scorpion antivenin and vasodilators but developed anaphylaxis to antivenin. We discuss management strategies. Anaphylaxis to antivenin should be on the differential during management of scorpion bites because classical signs of anaphylaxis may be absent.

Keywords: Anaphylaxis, antivenin, corticosteroids, envenomation, *Mesobuthus tamulus*, scorpion

Introduction

The estimated number of scorpion stings is 1.2 million/year with 0.27% (3250) deaths; children having the highest mortality.[1] The majority of the cases are nonlethal with localized pain and minimal systemic involvement. Scorpion envenomation with high morbidity and mortality is usually due to either excessive autonomic activity and cardiovascular toxic effects or neuromuscular toxic effects. It is a major public health problem in the parts of Central and South America, North Africa, the Middle East, and South Asia. Antivenin is the specific treatment for scorpion envenomation combined with supportive measures including vasodilators in patients with cardiovascular toxic effects and benzodiazepines when there is neuromuscular involvement.[2] Although rare, severe hypersensitivity reactions including anaphylaxis to scorpion antivenin (SAV) are possible. We describe our management of a case of severe anaphylaxis to antivenin.

Case Report

A 42-year-old male was stung by a red scorpion to his right index finger while gardening. He was hospitalized an hour later because of severe pain at the sting site. The patient had no known allergies except to dust. He was a nonsmoker, nondiabetic, and normotensive. On examination, the patient was anxious, extremities were cold, noticed to have profuse sweating [Figure 1], pulse was weak and 66 beats/min, and blood pressure of 150/94 mm Hg. Oxygen saturation was 98% on ambient air. Heart sounds were muffled with no murmur and gallops. His management included cardiorespiratory monitoring, antivenin (6 vials [60 ml] of Haffkine Biopharma monovalent antivenin diluted in 100 ml of normal saline over 30 min by

intravenous route), and prazosin (500- μ g orally repeated every 3 h).[3] Electrocardiogram on arrival showed ST-T changes [Figure 2] and at the end of 1-h after admission he developed left bundle branch block [Figure 3]. Investigations showed blood sugar level - 7.5 mmol, CPK-MB - 128.20 IU/L (normal 0–20 IU/L). About 2 h after admission he had emesis, complained of giddiness, breathlessness, tightness in chest, tickling sensation in throat, and uneasiness. Blood pressure dropped to 60 mm Hg, heart rate 133/min [Figure 4], bronchospasm evident by wheezing; he developed edematous face and swelling of eyelids [Figure 1], and oxygen saturation dropped to 88%. He was given nasal oxygen, intravenous 500 mg of methyl prednisolone, and fluid resuscitation to stabilize his hemodynamics. He improved within 30 min; extremities became warm, and his vital signs returned to normal.

He did not require any additional doses of antivenin. He became symptom free. He was observed overnight, tolerating enteral diet and was able to ambulate without pain. He was subsequently discharged.

Discussion

Mesobuthus tamulus (Indian red scorpion) venom (α toxins) delays the closing of neuronal sodium channels stimulating autonomic centers: Sympathetic and parasympathetic, leading to “autonomic storm.”[4] This also causes a massive release of endogenous catecholamines: Epinephrine and norepinephrine.[5] Circulating endogenous catecholamine itself acts as prophylaxis against anaphylaxis because of which anaphylaxis to SAV is rarely seen or absent.[6,7] Venom-induced anaphylaxis could be one of the differentials but its presentation nearly 3 h after the bite makes it very unlikely. Furthermore, the first dose effect of prazosin can cause severe hypotension but the accompanying bronchospasm and edema makes anaphylaxis more plausible.

Severe anaphylaxis to SAV was encountered in our case in spite of “autonomic storm”.[4] Antivenin is composed of venom-specific F (ab')₂ fragments of immunoglobulin G (IgG) purified from equine plasma that has been immunized with one or more scorpion venoms. It binds and neutralizes venom toxins, facilitating redistribution away from target tissues, and elimination from the body. There are no absolute contraindications to antivenin treatment, but patients with the previous history of reactions to equine serum in the past and those with a strong history of atopic diseases (especially severe asthma) are at high risk of severe reactions and should, therefore, be given antivenin only if they have signs of systemic envenoming. Anaphylactic reaction to antivenin is majorly due to complement activation by IgG aggregates or residual Fc fragments or direct stimulation of mast cells or basophils by antivenin protein.[8]

In our patient itching and urticaria; premonitory signs and manifestations of severe anaphylaxis were masked due to high levels of circulating catecholamines. Neutralization of circulating venom by SAV prevents delayed closing of neuronal sodium channels and inhibition of catecholamine release; moreover, effects of catecholamines are blunted by excessive release of known biochemical mediators of anaphylaxis. In view of the preexisting catecholamine-induced myocardial injury[9] and the already circulating endogenous catecholamines, epinephrine was not used as the first line of treatment. Epinephrine evokes ventricular arrhythmia and aggravates autonomic storm. In such situation, steroids could be the first line of treatment as they augment the action of already circulating catecholamine and counter the anaphylaxis.[10]

To date, there have not been any randomized, placebo-controlled trials for medications in anaphylaxis and their sequence of use in acute emergencies. The evidence for the use of epinephrine comes mostly from observational studies and the studies of anaphylaxis in animal models. Due to ethical considerations, placebo-controlled trials with epinephrine have never been performed. The use of H1 antihistamines and glucocorticoids has been due to extrapolation of their uses in the management of urticaria and acute asthma, respectively.

Conclusion

Though rare, anaphylaxis to antivenin should be on the differential during management of scorpion bites because classical signs of anaphylaxis may be absent. Early and adequate resuscitation with fluids, steroids, and bronchodilators will avoid life threatening distributive shock. As antivenin is a protein, there will always be a risk of anaphylaxis. Emergency medications (epinephrine, glucocorticoids, H1 antihistamines, bronchodilators) and airway management equipment (endotracheal tube and bag mask) should always be

handy for such situations.

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Conflicts of interest There are no conflicts of interest.

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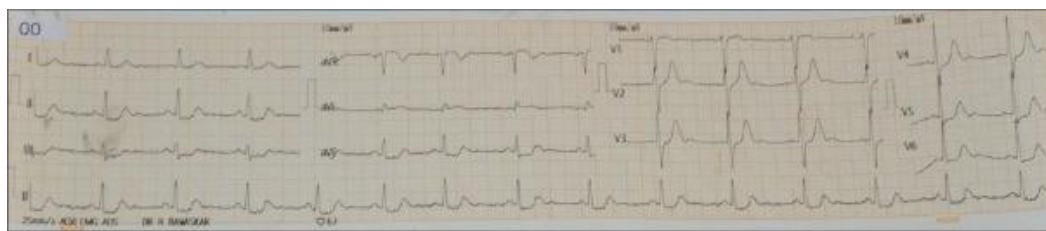
Figures and Tables

Figure 1



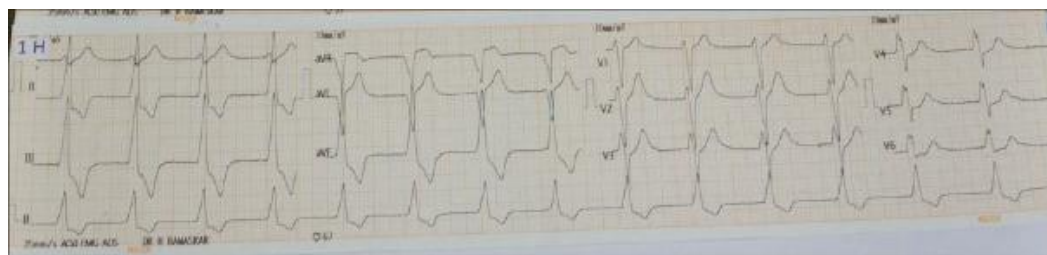
Profuse sweating and puffiness of face

Figure 2



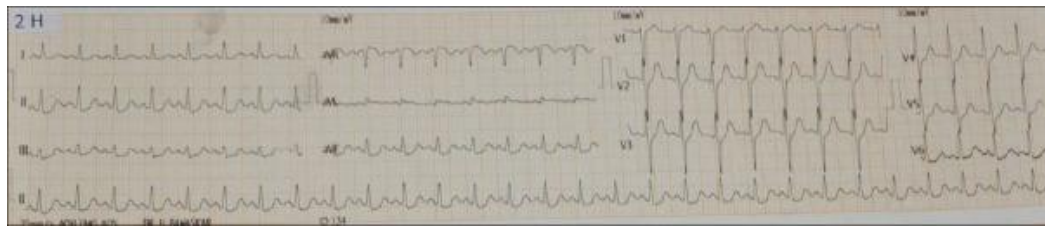
Tented T V2-V6 ST depression in II, III, and AVF

Figure 3



Left bundle branch block

Figure 4



Heart rate 133/min, ST elevated in AVL

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