A Rare Case of Scorpion-sting Induced Acute Dilated Cardiomyopathy that Responded Dramatically to Inotropes and L-Carnitine

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ABSTRACT

Introduction: Scorpion sting causing envenomation usually results in hypertensive crisis. A rare manifestation is acute dilated cardiomyopathy. Here we present a case where this was successfully managed.

Case Report: A 25 year old, previously healthy male with a history of scorpion sting 5 hours before coming to hospital, presented with pulmonary edema and hypotension. Patient was started on Dopamine, Dobutamine and Frusemide infusions and L-Carnitine. Initial ECG and Troponin T was suggestive of myocardial injury. First 2D-echocardiogram revealed dilatation of the LV with markedly reduced ejection fraction. Patient improved clinically on the 4th day and the inotropes were tapered off. A second 2D-Echocardiogram done on the 5th day recorded remarkable improvement with a normal LV size.

Conclusion: Scorpion sting may result in acute dilated cardiomyopathy in rare cases. Thus, in all cases of scorpion sting, observation in an ICU for one day is necessary. Supportive treatment with inotropes and L-Carnitine can successfully treat such a case.

Keywords: Scorpion-sting Induced Acute Dilated Cardiomyopathy

INTRODUCTION

Scorpion envenomation is an important health problem in tropical and sub-tropical regions especially during spring, summer and autumn.[1] Among 86 scorpion species in India *Mesobuthus tumulus* and *Heterometrus swammerdami* are important causes of stings that we see in hospitals.[2] *Mesobuthus tumulus* or Indian red scorpion commonly causes cardiovascular abnormalities.[3] Cardiovascular manifestations include hypertension crisis, hypotension and pulmonary edema. Pulmonary edema is caused by both increased capillary permeability and, rarely, cardiomyopathy.[4] Most of the reported cases of cardiomyopathy have showed systolic dysfunction. Acute dilated cardiomyopathy due to scorpion sting is rarely reported. We present a case of scorpion envenomation that presented as acute dilated cardiomyopathy which reversed with supportive treatment.

CASE REPORT

A 25 year old male presented with history of scorpion sting on his left middle finger around 11am. He came to the ICU by 4pm. On admission he had complaints of breathlessness and productive cough with pink frothy sputum which aggravated on lying down. His past history was not significant. He was farmer by occupation and used to do strenuous work without any discomfort in past years. He did not have any chest pain or chest discomfort. On examination he was profusely perspiring and restless. His BP was 70/50mmHg, pulse rate was 156/min and was regular, and he was tachypneic. SpO_2 was 94%.On auscultation, bilateral basal rales were present and S3 was audible. Liver was palpable 3 cm below costal margin. Neurological examination was normal.

He was put on Oxygen by face mask. Dopamine and Dobutamine infusions were started. Lasix infusion was given at 1mg/min. L-Carnitine was started at a dose of 500mg TDS. Injection Hydrocortisone was given at 50mg q8H for 48 hours. Antiplatelet drug and inj. Heparin was also started and I.V antibiotics were given.

Serial ECG monitoring showed:

On Admission - Sinus Tachycardia (150/min)

After 24 hours - T inversion in I,V_{3-6}; ST elevation V_{1-2}

After 48 hours - ST depression & T inversion in I, aVL, V_{2-6}

Importantly R wave was preserved in chest leads.

Troponin T (qualitative) was positive. Blood investigations showed mild anemia and leukocytosis, and mildly increased serum creatinine, SGOT and SGPT. Chest x-ray showed inhomogeneous opacities in bilateral basal and mid-lung regions. USG abdomen showed mild hepatomegaly. Initial 2D-Echocardiogram was done in 24 hours and showed Dilated LV, Global hypokinesia of the LV, Moderate MR, Mild PAH,& LVEF = 20-25%.

Patient's vitals improved after 72 hours and inotropes were gradually withdrawn. L-Carnitine tablets were continued. Lasix infusion was stopped and converted to 20mg q8H after 72 hours. Repeat 2D-Echocardiogram was done on the fifth day and showed Global hypokinesia of the LV, trace

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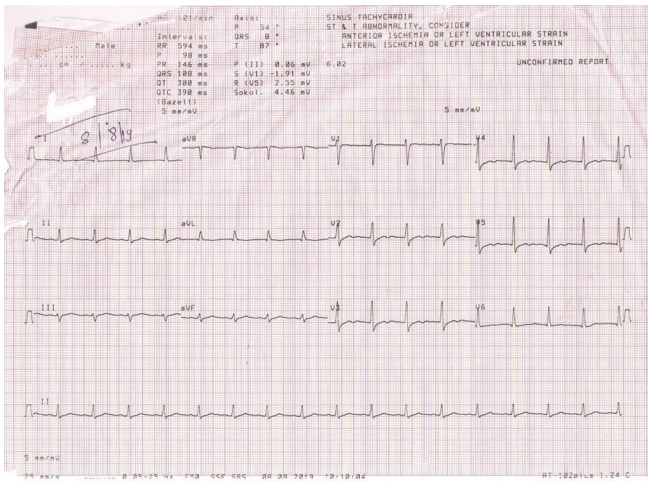
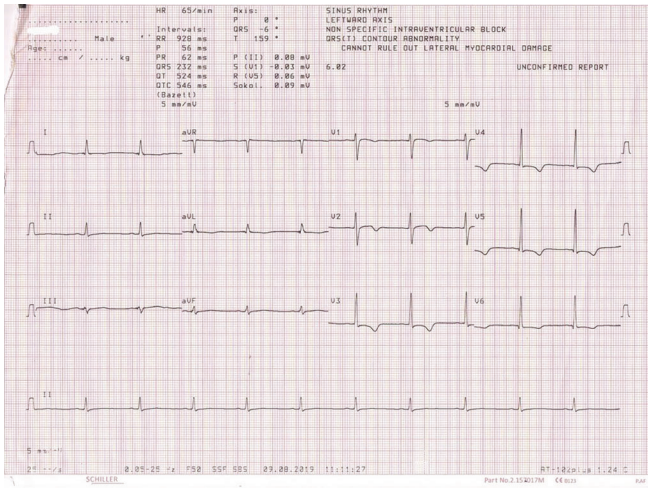
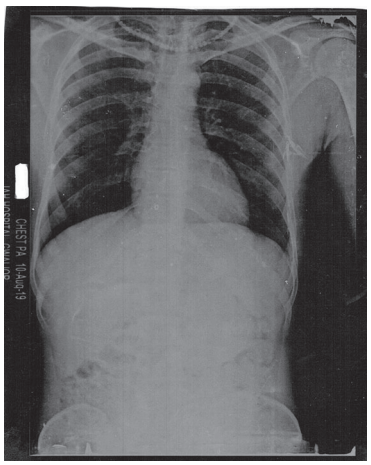


Figute-1: ECG after 24 hours



Figute-2: ECG after 48 hours



Figute-3: Chest X-Ray

MR, mild PAH, and LVEF = 35-40%. LV dimensions were within normal limits. Patient was shifted to ward and later discharged after a total hospitalization of 10 days.

DISCUSSION

90% of scorpion stings result in local envenomation only.[3] Average mortality rate is proposed to be only 0.27%.[7] Scorpion venom is a complex mixture of neurotoxins, cardiotoxins, nephrotoxins, Hemolysin, hyaluronidases, and phospholipases.[3] Almost complete amount of venom reaches blood within 7-8 hours of sting. Our patient presented in acute pulmonary edema and cardiogenic shock, which was a grade-3 envenomation, after 5 hours of sting.

Various mechanisms of scorpion sting induced cardiotoxicity have been proposed. They include:

- Acute coronary spasm causing myocardial ischemia
- Direct cardiotoxic effect of the venom
- Anaphylactic shock caused by toxin
- Excess catecholamine release causing myocardial stunning and dysfunction analogous to Takotsubo cardiomyopathy.[5]

Cardiotoxicity occurs in two stages. Early vascular phase is due to profound vasoconstriction due to catecholamine release which acutely increases LV afterload. It is followed by a myocardial phase which results in myocardial stunning and systolic dysfunction. RV dysfunction has also been reported in scorpion sting cases.

Even though acute systolic dysfunction of ventricles have been reported, acute dilation of ventricles has rarely been reported. Such acute dilation of ventricles may be due to catecholamine induced microvascular spasm and ischemia similar to that in Takotsubo cardiomyopathy. Miranda et al reported a case of 7 year old boy with severe heart failure following scorpion envenomation in which the cardiac MRI showed apical ballooning of LV as in Takotsubo cardiomyopathy.[8] Microvascular ischemia with normal coronaries is the main pathophysiology of such 'stress cardiomyopathy' and positive response to ionotropes and supportive treatment have been seen.[9]

Similar to such observations, our patient also responded dramatically to inotropes and L-Carnitine. While the role of inotropes in scorpion sting is well established, the role of L-Carnitine has been less well studied. L-Carnitine may have supportive role in recovery of myocardium and requires further studies.[10] The role of ACE inhibitors is controversial as scorpion venom is itself proposed to inhibit Angiotensin converting enzyme, leading to Bradykinin accumulation and resultant pulmonary edema.[3] Bawaskar et al first proposed the role of alpha blockers(Prazosin) in treating scorpion sting.[3] It was not used in our case as the patient presented in shock.

CONCLUSION

Cardiomyopathy following scorpion sting, though rare, is a serious complication. Our patient presented as an even lesser reported case of acute dilation of left ventricle with severe LV dysfunction. He responded well to inotropes and L-Carnitine. Thus, in order to prevent catastrophic results, all patients of scorpion sting should be monitored in ICU with continuous ECG monitoring. 2D-Echocardiogram should be considered in all patients with grade 2 envenomation and above.

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