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Original Research Paper

General Medicine

CARDIOMYOPATHY IN CASE OF SCORPION BITE

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KEYWORDS :

INTRODUCTION

Scorpion stings are really painful but rarely harmful. Scorpion envenomation is potentially fatal in many regions worldwide.The sting effect depends on the size of the victim, the season, the age of the offender and the delivery dose of the scorpion [1,2]

It is generally observed as an important emergency health problem in tropical and subtropical countries especially in spring, summer and autumn seasons. [3]. Scorpion envenoming may cause multiorgan insuffificiency, neurotoxicity and cardiotoxicity in the affected people and it may be deadly, especially for children. Among 86 scorpion species in India Mesobuthus tumulus and Heterometrus swammerdami are important causes of stings that we see in hospitals [4]. Mesobuthus tumulus or Indian red scorpion commonly causes cardiovascular abnormalities [5] .Not only scorpion-related cardiomyopathy but also increased pulmonary capillary permeability sourced from excessive stimulation of alpha receptors are advocated to be the underlying cause of pulmonary edema following scorpion envenomation.[6,7] We present here a case report of a 18 year old female with the clinical appearances of intense myocardial dead tissue related with left ventricular dysfunction and pulmonary edema following scorpion bite.

CASE REPORT

18 year old female resident of Bhokardan Jalna suffered a scorpion bite on right great toe. Within 5 hours she presented with complains of breathlessness, palpitation, dizziness and profuse sweating. She had complain of severe pain at site of bite. She had no significant past history and no other predisposing cardiac risk factor. On examination patient was Conscious oriented. She looked sick, sweaty, pale and dyspnic.

Pulse 140/min blood pressure was 90/60 mm HG, saturation was 80% on room air. Jvp was raised. There were no bleeding tendencies and local tenderness was present . S1, S2 Normal and there was no murmur. There were Crepitation in bilateral lower zones. She was put on Oxygen by face mask and started on higher antibiotics. Dopamine and Noradrenaline infusions were started. Lasix infusion was given at 1mg/min .Injection Hydrocortisone was given at 50mg q8H for 48 hours.

Hemogram showed predominnat leukocytosis of 25,000 Liver and renal function were within normal limit.
ECG showed sinus tachycardia
NT PRO BNP : 25000
ABG PH 7.3; pCo2 35; pO2 60; Bicarbonate 18.3; Serum lactate 2.9
2decho : EF 20%
Global LV hypokinesia with severely compromised LV systolic dysfunction

Chest x ray: Fluffy shadows in bilateral lower lung fields suggestive of pulmonary edema.

After 48hrs Condition Improved.
With cardiologist opinion patient discharged on tablet lasilactone and carvidilol.

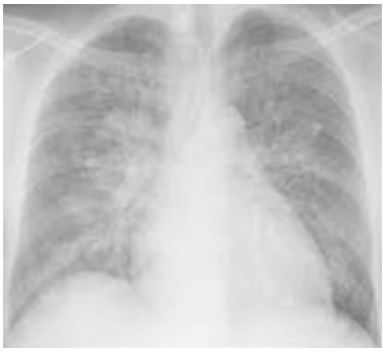

Figure 1. Chest X-ray Showing Moderate To Severe Pulmonary Edema

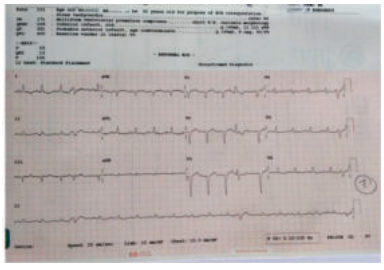

Figure 2. Electrocardiography With Sinus Tachycardia

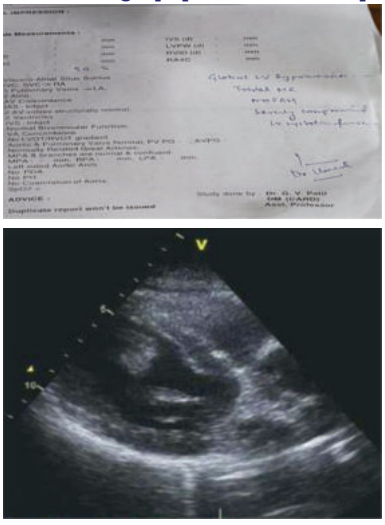

Figure 3 And 4 : 2 DECHO Showing Global LV Hypokinesia

DISCUSSION

Scorpion venom contains a neurotoxin, haemolysins, agglutinins, haemorrhagins, leucocytolysins, coagulins, ferments, lecithin and chlolesterin.[8] The venom produces both local as well as systemic reactions. Local reactions consist of itching, edema and ecchymoses with buring pain.[9] The cardiovascular manifestations comprise successively of giddiness, bradycardia, a fall of body temperature; restlessness and tachycardia; and finally pulmonary edema.[10] The scorpion venom stimulates the peripheral sympathetic nerve endings and release of catecholamines from the adrenal medula (directly as well as through parasympathetic stimulation)[11]. Thus the venom is a powerful arrhythmogenic agent. These actions of the venom are inhibited by atropine, propranolol and phentolamine.[12] 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.

Especially children are at greater risk than adults. This can be explained with the fact that small children or infants are hemodynamically be more unstable and vulnerable than adults. In addition small children and infants are at high risk in term of morbidity and mortality. [13,14]. Moreover since they are not able to protect themselves against the environmental dangers and since their awareness level is lesser than adults their morbidity and mortality rates can go significantly higher. For these reasons these risk groups can get stinged on multiple parts of their body by scorpions. Having a lower weight than the other humans around, has created an extra risk for this case.

Yarom et al. studied the scorpion venom effects on the cardiovascular system and they found that the mortality in scorpion sting envenomation is primarily due to toxic effects of venom on myocardium. The resulting cardiac failure and pulmonary edema are attributed to venom induced cardiorespiratory dysfunction. Catecholamine overstimulation can cause coronary microvascular spasm leading to myocardial perfusion derangement [15]. Asmaekhattabi et al. developed a consensus on classification of clinical consequences of scorpion sting. They included four classes. Grade-I envenomation-local manifestations, caused by a "dry" sting, sting with no venom injection. Grade II minor systemic manifestations attributed to autonomic storm like tachycardia, sweating, fever and vomiting without life threatening. Grade III-major systemic manifestations (mainly of circulatory and respiratory system) and Grade IV –lethal envenomation [16]. Bahloul et al. have explained that both hemodynamic mechanism and vascular permeability mechanisms will lead to pulmonary edema following scorpion sting envenomation [17]. Suresh et al. found out that the mean levels of Pro BNP found to be high in children with scorpion sting induced myocarditis [18].

Treatment of pulmonary edema due to scorpion envenomation follows the same principles as those applied for the treatment of cardiogenic pulmonary edema in general: this begins with oxygen supplementation targeting an oxygen saturation of 92% or more, by oxygen mask, continuous positive airway pressure, noninvasive ventilation, or conventional mechanical ventilation. Dobutamine effectively improves hemodynamic parameters and may reduce mortality in severe scorpion envenomation. Prazocin, an alpha adrenergic blocker, was found to add a significant benifit to the outcomes of scorpion sting management. In a review from west India, the mortality rates in the pre-prazocin era (1961-1983) were ranging from 25% to 30%. Later on with prazocin therapy the mortality rate was reduced significantly to less than 1%. Prazocin was then considered as an antidote to scorpion venom [19,20]

CONCLUSION

Scorpion venom has potent cardiotoxic effect though rare but life threatening. Catecholamine storm appears to be the mechanism of cardiovascular toxicity. Any age group can develop cardiac complication irrespective of previous health condition hence serial ECG monitoring must be done for early detection of cardiac complication. Positive inotropic agents and alpha and beta blocker agents with supportive therapy are recommended in the treatment.

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Consent

Written informed consent was taken from the patient for publication of this case report and accompanying images.

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