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A Case of Reversible Acute Myocarditis Due to Scorpion Sting in South Saudi Arabia

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Introduction

Globally, the annual estimated incidence rate of scorpion sting is 1.2 million which lead to 3250 deaths with case fatality rate of 0.27% (1). Scorpion sting is sizable medical problem that was well documented in Middle East (2). In epidemiological study conducted in Saudi Arabia from period of 1993 through 1997 BY Al Asmari et al., the incidence rate of scorpion sting was 9 cases/10,000 annually. Al Medina- Al Munawra region was the highest prevalence ratio followed with A L - Baha, Hail, Bisha, and Riyadh with average of 33.33%, 15.32%, 10.78%, 9.16% and 1, 6% consecutively. In study the age younger than 15 years represented around 34 % of critical cases and the local manifestations were predominant and around 24% was admitted. Only one patient died due to pulmonary edema. The most common identified scorpion species were Leiurus quinquestriatus, Androctonus crassicauda, and Apistobuthus pterygocercus (3). In Saudi Arabia, the pediatric patients had approximately mortality rate of 5% and antecedent to the antivenom era the main cause of death was shock and pulmonary edema (4). After considering the anti-scorpion antivenom protocol as mainstay of management in the scorpion sting, the mortality rate decreased, though the pulmonary edema and cardiac manifestations persist to be the main cause of death (2, 5).

The clinical presentations of scorpion sting depend on the amount of injected venom, age of patients and the duration of time between the sting and administration of antivenom (6). Scorpion neurotoxins are accountable to induce autonomic storm through enormous release of adrenergic and cholinergic neurotransmitters. This catecholamine flare-up lead to severe vasoconstriction and later to impaired left ventricular (LV) function which contribute to pulmonary edema and cardiogenic shock (7). Here we present a case report of a 2-year-old male patient who developed myocarditis following scorpion envenomation, who was successfully treated with scorpion polyvalent antivenom.

Case Report

2 years old, saudi, male, presented in Al Bashair Hospital with history of 2 scorpions stings in the right hand. Upon arrivalto emergency department, he was irritable, persistently vomiting, shortness of breath and high blood pressure (BP) for his age. Patient received 5 vials of scorpion polyvalent antivenom, with repeated dose of 5 vials after 40 minutes. Patient was pretreated with 0.2 mg intravenous atropine and 20 mg intravenous hydrocortisone.

Patient had severe respiratory distress for which he was intubated and referred as live saving to the Maternity and Children Hospital (MCH) in Bisha, Asir region of province of Saudi Arabia. Upon

arrival to Pediatric Intensive Care Unit (PICU), his vital signs were heart rate (HR) 170 beats per minute, BP 126/88 mmHg, Temperature 37.3°C, respiratory rate (RR) 26 breaths per minute, oxygen saturation (SpO_2) 94%, and random blood sugar (RBS) 109 mg/dl.

The mechanical ventilation setting as follow:SIMV/PC mode; FiO2= 80%; VR= 20/min; PiP= 10 cmH_2O; PEEP= 5 cmH_2O; IT 0.5sec. His arterial blood gases were as follow: pH=7.25; pCO_2= 38mmHg; pO_2=77.5 cm H2O; HCO_3=16.4mmol/L; lactate =2.0 mmol/L Base (Ecf) = -9.8 mmol/L.

The clinical examination revealed edema in the right hand. Glasgow come scale(GCS) Eyes 3 Motor 3 Verbal 1 total score=7/15, pupils constricted, not response to light. Cardiopulmonary examinations showed equal air entry bilaterally with cardiac assessment S1 and S2 heart sounds normal, with systolic murmur grade 2/6 on apex area, no S3 or S4 heart sound heard. No gallop rhythm or pericardial rub. His capillary refill time was <2 sec. Other physical examinations were normal. Electrocardiogram (ECG) showed sinus tachycardia, diffuse ST -depression in leads V3, V4, V5, V6 (Figure 1).

Echocardiogram (ECHO) reported mild LV dilatation, mild mitral regurgitation, small central jet at least 1/3 of the left atrium, jet area of 10% of left atrium (LA) area, peak gradient (PG) 30mmHg, ejection fraction (EF) 57%, fraction shortening (FS) 28%, no pericardial effusion (Figure 2). Chest X-ray was normal and the endotracheal tube was in the place (Figure 3). His laboratory results showed elevation of cardiac enzymes: creatine phosphokinase (CPK), creatine phosphokinase – myoglobin binding isoenzyme-MB (CPK-MB) and cardiac troponine I (cTnI), and coagulation profile (Table 1). The patient received a total of 4 doses of scorpion polyvalent antivenom as recommended by local toxicology center according to the Saudi Ministry of Health (MOH) protocol. Over 24 hours, there was gradually improvement in patient clinical status. Patient was weaned and extubated and his cardiac enzymes decreased to normal.

After 3 days, He was stepped down fromPICU to pediatric ward. His repeated echocardiogram showed mild LV dilatation LVDd 36.5mm (Z score +2.23), LVDs 23.4mm(z score + 2.14), mild mitral regurgitation, small central jet PG = 22.9 mmHg,normal LV function EF 54.9 %. He was discharged with followed up after 2weeks. Echocardiogram was repeated at follow up visit in 2 weeks and showed a normal EF 78.5 %, no mitral regurgitation, normal LV size and no pericardial effusion (Table 2).

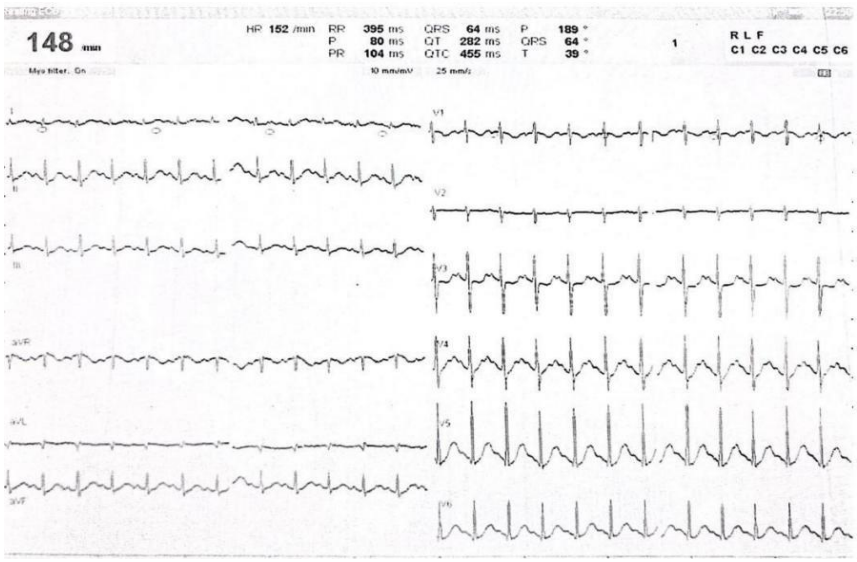

Figure 1: Electrocardiogram (ECG) of the patient during admission showing sinus tachycardia, diffuse repolarization abnormalities.

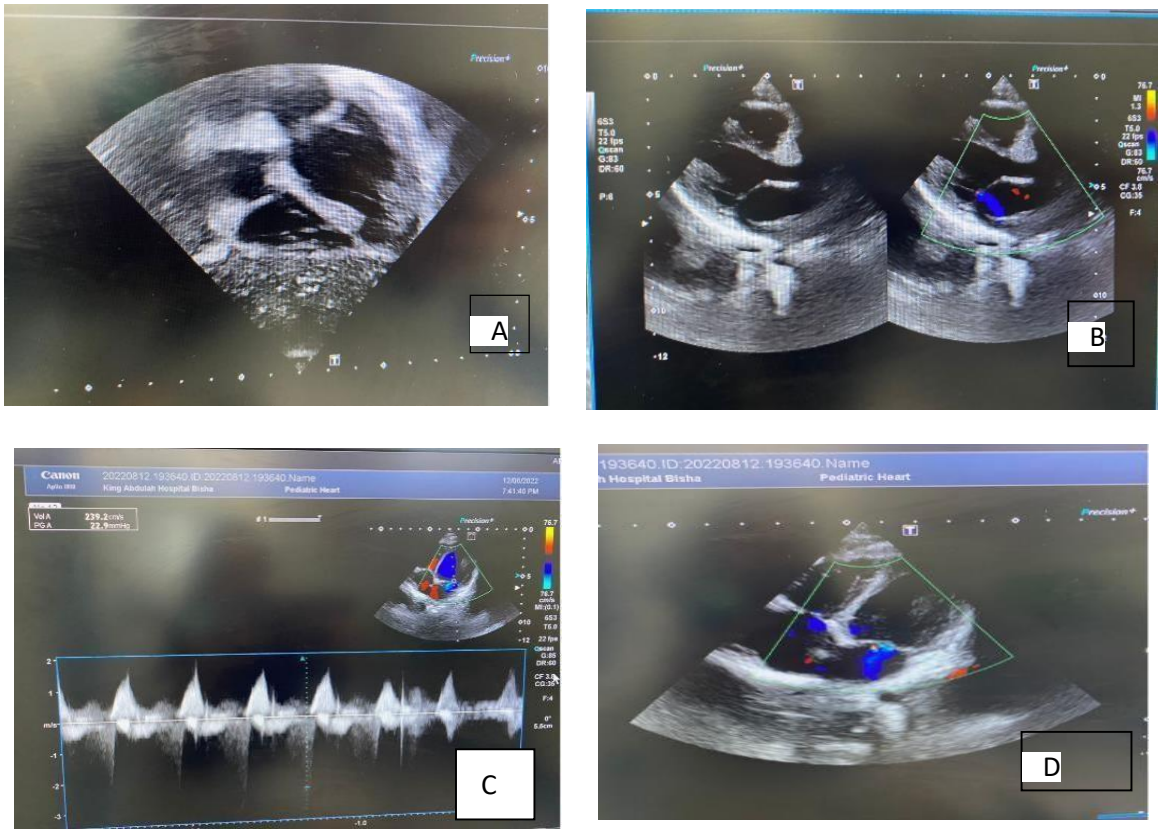

Figure 2: A) Subcostal view B) Long axis parasternal view C) Mitral valve regurgitation Doppler D) 4 chambers view. All show LV dilatation, mitral regurgitation.

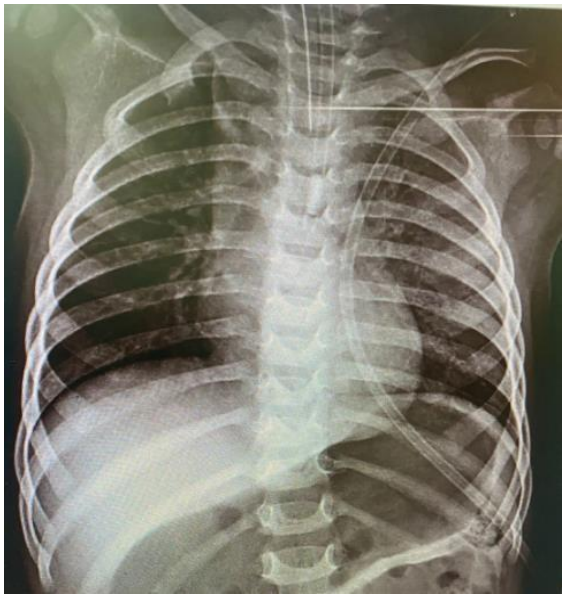

Figure 3: Chest X-ray of the patient during admission showing reported as normal.

LABORATORY	ADMISION	6 HOURS	12 HOURS	24 HOURS	4 DAYS DISCHARGE
CK (U/L)	NA	NA	NA	232	181
CK MB (U/L)	54	97	142	49	27
CPK/CPK-MB RATIO				21%	14%
TROPONINE (ng/ml)	1.66	0.75	0.44	0.38	0.22
LDH (U/L)		385	580	354	
ALT (U/L)	33	30.4	30.3	26.3	
AST (U/L)		79	100		
AMYLASA (U/L)	194	167	92		
LIPASE (U/L)	61	13	NA		
GLUCOSE (mmol/L)	7.78	4.61	4.27	4.32	

Table 1: Laboratory chemistry summary

LV dimension/ LV function		Admission		Follow up 2 weeks
		Z score		Z score
BSA m^2	0.54			
LVDd (mm)	36.5	+2.23	26.6	-1.40
LVDs (mm)	23.4	+2.14	14.6	-2.04
EF (%)	54.9		78.5	
FS (%)	26.3		45.1	

Table 2: Left ventricular dimensions and Left ventricular function during admission and follow up according to z score for body surface area.

Discussion

Yarom et al. studied the scorpion venom effects on the cardiovascular system and they found that the deaths in scorpion sting envenomation are initially due to the toxic effects of venom on the myocardium. Heart failure and pulmonary edema are because of the venom effect that can stimulate cardiorespiratory dysfunction. They come to the end that catecholamine hypersecretion can cause coronary microvascular spasms leading to myocardial perfusion detriment (4) (5).

The clinical presentation is diversely related to the locations of the sting, quantity of venom inoculated, age of the patient, etc. A consensus on the classification of clinical consequences of scorpion sting developed by Khattabi et al. in 2011, included four classes: Class I: envenomation-local manifestations, sting with no venom injection ("dry sting"). Class II: minor systemic manifestations attributed to autonomic storms like tachycardia, sweating, fever, and vomiting without life-threatening. Class III major systemic manifestations, mainly of the circulatory and respiratory system, and Class IV lethal envenomation (5) (6).

This consensus is helpful to categorize the severity of the patients according to the clinical findings. Many studies have shown the serum concentrations of CPK, cTnI, and natriuretic peptides after scorpion envenomation increased. The CPK-MB activity, arise in children with scorpion sting envenomation specific and highly sensitive in the cases of myocardial injury following scorpion stings (5) (7). Gueron et al. studied the echocardiographic findings and radionuclide angiograms in children with a scorpion sting. The echocardiogram findings were involvement in the contractility and low EF with severe scorpion sting envenomation, maybe due to the myocardial ischemia induced by catecholamines (8).

Prakash et al. studied 21 children with myocarditis and ECG changes were seen in 19.04%. X-ray chest changes were seen in 33.3% of cases and 100% showed varied abnormal ECHO findings as hypokinesia of the left ventricle, decreased LVEF, myocarditis, pulmonary arterial hypertension, and grade I tricuspid regurgitation. (5). The scorpion venom is a water-soluble antigenic complex which contains a neurotoxin, cardiotoxin, nephrotoxin, hemolysins, phosphodi esterases, phospholipases, hyaluronidases, histamine, and other chemicals (9). The primary target of scorpion venom is voltage-dependent ion channels. The cardiovascular toxic effect of the venom causing toxic myocarditis is the reduction of Na K-ATPase, and adrenergic myocarditis is by releasing adrenaline and noradrenaline from neurons, ganglia, and adrenals, in this way increasing myocardial oxygen demand by the direct inotropic and chronotropic effect on the compromised myocardial blood supply (9).

In the clinical of envenomation, it has been proposed that the interaction between the quantity of venom introduced in the body of the patient and the distribution volume should intervene in a critical threshold of scorpion toxin plasma concentration. In this case, systemic vasoconstrictor effects induce an increase in the systemic arterial pressure and the LV-filling pressure and decreased cardiac output. The early phase of cardiac dysfunction, as well called the "vascular phase", is continued by severe cardiomyopathy, involving both ventricles, and is reversible in days to weeks (10). The venom can cause myocardial damage by several pathogenetic mechanisms: -Myocardial ischemia by coronary spasm: Release of vasoactive, inflammatory, and thrombogenic peptides and amine constituents (histamine, serotonin, bradykinin, leukotrienes, thromboxane), which act on the coronary vasculature and induce coronary artery vasospasm and facilitate platelet aggregation as well as thrombosis (11).

Direct cardiotoxic effect of the venomcausing toxic myocarditis by two mechanism the reduction of Na-K-ATPase and the adrenergic myocarditis by releasing adrenaline and noradrenaline from neurons, ganglia, and adrenals, thereby increasing myocardial oxygen demand by a direct inotropic and chronotropic effect on already compromised myocardial blood supply (11). -Anaphylactic reaction: Release of allergenic proteins causes anaphylactic shock leading to hypotension with vasodilation and decreased intravascular volume with reduced myocardial perfusion (11). Scorpion venom inhibits angiotensin- converting enzyme (ACE), resulting in accumulation of bradykinin, which is implicated in the development of pulmonary edema (11). According to the consensus on classification on clinical findings by Khattabiet al., our patient presented signs of the direct cardiotoxic effect ofthe venom as grade II to III with manifestations attributed to autonomic storms like tachycardia, sweating, fever and vomiting and grade III major systemic manifestations, mainly cardiac manifestations as myocarditis (6).

The laboratory findings in our patient, were similar to one studied in 32 children in Israel, with scorpion envenomation without heart failure, with high total CPK level, elevate CPK MB level, and CPK-MB/ CPK ratio exceeding 6%, concluded that the CPK- MB activity is specific and highly sensitive in detecting myocardial damage in children following scorpion envenomation (12). The cTnI showed 100% specificity and sensitivity for the diagnosis of myocardial injury concerning ECHO findings in the envenomed victims. In severe cases, cTnI was positively correlated with IL-8. The IL-8 may be involved in the pathogenesis of myocardial injury of scorpion envenomation, while negatively correlated with % FS and LVEF. Abdel Raheem et al. concluded that cTnI is a specific marker for the diagnosis of myocardial injury in scorpion envenomation while other biochemical markers did not show such specificity. Both cTnI and IL-8 may be useful to forecast the fatal outcome of scorpion envenomation (13).

Chakoun-Walha et al. reported in patients with moderate scorpion envenomation with positive T wave, high values of troponin suggest the presence of cardiac dysfunction (14). In our patient the cTnI was high from the beginning correlated with the ECHO and ECG findings. Echocardiography is an excellent tool to evaluate various parameters of cardiac function available in our emergency. It has been used to document myocarditis in scorpion sting envenomation, being the LV systolic dysfunction the dominant finding (15). Based on echocardiography patients with normal LVEF can be observed in general wards and safely discharged. Most of the patients with mild to moderate LV dysfunction can be managed in a minor intensive care unit and patients with severe LV dysfunction should be observed in PICU for the requirement of inotropic support (16).

The successful treatment of scorpion envenomation is mainly attributed to symptomatic treatment. Scorpion sting envenomation-associated myocarditis, acute heart failure, and pulmonary edema need appropriate supportive care. The typical intensive care treatment for acute pulmonary edema and cardiogenic shock is appropriate supportive measures and often includes oxygenation, ventilation, use of inotropic support, and vasodilators (5) (17). The rapid reversibility of cardiac dysfunction together with normalization of the enzymatic levels, ECG, and ECHO, indicate the occurrence of an acute myocardial lesion without the association of coronary disease (17).

Conclusion

Scorpion venom can have a different clinical grade according to the type of scorpion, amountof venom, age of the patient, however the potent cardiotoxic effect which can lead to myocardial dysfunction. It is advisable to get thorough evaluation including cardiac evaluation in children with scorpion sting

envenomation. The cTnI is a bed side patient investigation to confirm the diagnostic of myocarditis. The ECHO is very useful in identifying patients with LV dysfunction after scorpion sting envenomation and it early use in emergency is feasible.

The Saudi MOH protocol for management scorpion sting myocarditis is effectively, with low morbidity and mortality leading to a satisfactory outcome.

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