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Original Article

Cardiology Section

Elevated Cardiac Troponin (cTnI) Levels Correlate with the Clinical and Echocardiographic Evidences of Severe Myocarditis in Scorpion Sting Envenomation

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ABSTRACT

Objective: This study was to evaluate the predictive value of the cardiac troponins in scorpion sting myocarditis at a tertiary care hospital in Raichur, (Karnataka state) India.

Methods: A total of 84 consecutive patients were prospectively studied. The data included the demographics, the time of presentation to the hospital, the clinical features, the cardiac troponin levels and the echocardiographic findings.

Results: 12 patients with only local symptoms had troponin levels of less than 0.01µg/L. 12 patients with local and systemic symptoms without an echocardiac evidence of myocarditis had troponin values of 0.01 to 0.11 µg/L. 60 patients with an echocardiographic evidence of myocarditis had troponin levels of above 0.11 µg/L. 6 patients with severe myocarditis who required ventilator support or which led to death had troponin values which were higher than 10 µg/L.

Conclusions: High cardiac troponin levels predict myocarditis in scorpion sting envenomation and they can be a useful tool in guiding the therapy early.

Key Words: Scorpion, Myocarditis, Envenomation, Echocardiography, Troponins

INTRODUCTION

Scorpion sting envenomation with systemic involvement is a life threatening medical emergency. It is a frequent event in the tropical, sub-tropical and the temperate zones of the world and it poses a public health problem in certain parts of India [1]. Mesobuthus tumulus or the Indian red scorpion is the most toxic scorpion species in India, which is abundantly found in western Maharashtra, northern Karnataka, Andhra Pradesh, Saurashtra and Tamilnadu [2,3]. The clinical manifestations of scorpion sting envenomation are vomiting, sweating, cold extremities, pulmonary oedema and death [4,5]. Cardiopulmonary complications, mainly pulmonary oedema and shock, are the leading causes of death [6-8].

Echocardiography is an excellent tool in evaluating various parameters of the cardiac functions. It has been used to document myocarditis in scorpion sting envenomation [9-12]. Left ventricular systolic dysfunction is the dominant finding and left ventricular dilatation and regional wall motion abnormalities have been described infrequently [9]. However, its routine use is limited by its non-availability.

Earlier studies had shown elevated creatine phosphokinase levels which had suggested an increased skeletal muscle activity and signs of cardiac and or skeletal muscle injuries [13-15]. Cardiac troponins (cTnI) have become important diagnostic and prognostic tools in acute coronary syndromes. The levels of the cTnIs increase in certain non coronary conditions also. The importance of the cTnI levels in scorpion sting myocarditis was reported by Meki et al., [15]. This study was intended at evaluating the levels of the cTnI correlation with the echocardiographic findings of myocarditis.

MATERIALS AND METHODS

This prospective study was conducted at Raichur in northern Karnataka, a part of India, during the period from April 2009 to March 2012 (3 years). 3 centres with tertiary care facilities (Rajiv Gandhi Super Speciality Hospital, Navodaya Medical College and Hospital, and Shivam Hospital and Research Centre) participated in the study. All the patients presented to the emergency department with a history of scorpion sting envenomation, the presence of sting marks and with the sting or the scorpion seen in the vicinity of the victim by a bystander. In a total, 84 patients were studied during the 3 year period. An informed consent was taken from the relatives of the patients. The details of the clinical features and the treatment which was given at the referring centre was noted from the referral letters. All the clinical details which included the blood pressure, heart rate, chest findings and the temperature of the extremities were recorded at the arrival to the emergency department and at hourly intervals on a pretested standard chart.

The diagnosis of various systemic involvements was done, based on the clinical manifestations and investigations. Myocarditis was diagnosed if the patient had tachycardia, muffled heart sounds, a gallop rhythm, ECG changes (a low amplitude, ST segment changes and/or the presence of arrhythmias), a low ejection fraction on echocardiography and elevated cTnI values (>0.11 µg/L).

Echocardiography: All the patients underwent echocardiography within 6 hours of their presentation to the emergency department. The echocardiography was done with a Sonosite M Turbo Ultrasound system by using the standard views and protocol. A parenteral sedation was used in paediatric patients to maximize their cooperation during the study. The main parameters which

were evaluated were the Left Ventricular Ejection Fraction (LVEF) which was assessed by the M mode, regional or global hypokinaesia, the RV function and valvular regurgitation.

Cardiac Troponin estimation: cTnI was assessed in all the patients between 1 to 6 hours of their presentation, irrespective of the type of presentation, by using VIDAS TROPONIN I Ultra (TNIU). It is an automated quantitative test which is used for the determination of human cardiac troponin I in human serum by using an enzyme-linked fluorescent assay technique. The measurement values of the VIDAS Troponin I Ultra kit range from 0.01to 30 µg/L. A value of more than 0.11 µg/L is considered to be significant.

RESULTS

84 patients who were admitted to the emergency department were studied during the study period. A majority were below 12 years [Table/Fig-1] 12 patients were above 18 years (14.2%), 24 patients were between 12 and 18 years (28.5%) and 48 were below 12 years (57.1%)]. 12 patients had only local symptoms (14.2%). Echocardiography revealed decreased LVEF values in 60 patients (71.4%). A majority had LVEF values of less than 40%. (50 patients i.e., 83.3%). A severe LV dysfunction was noted in 20 patients (33.3%). No patient had significant valvular regurgitation. RV dysfunction was noted in half of the patients who had the LV dysfunction (30 patients). The patients with the RV dysfunction had severe hypotension (systolic BP of less than 90 mmHg). Among the severe LV dysfunction group, 4 patients required ventilator support and 2 patients died due to refractory shock and multi-organ dysfunction.

The cardiac troponin levels were normal in 12 patients with only local symptoms (less than 0.01 µg/L). 12 patients with some systemic manifestations but without an evidence of myocarditis on echocardiography had cTnI values between 0.01 and 0.11 µg/L. These patients were observed for 24 to 48 hours and discharged in a stable state. 60 patients with an echocardiographic evidence of myocarditis had high troponin levels (mean -2.4 µg/L, range -0.4 to 12 µg/L). 6 patients with severe myocarditis who required ventilator support or which led to death, had values of > 10 µg/L. The elevated troponin levels correlated well with an evidence of myocarditis (LVEF <50%, r= -0.41, P<0.05). A majority of the patients with myocarditis were below 12 years of age (45 patients). Normal troponin levels were noted in all the patients who did not have an evidence of myocarditis on echocardiography.

Age	< 12 years	48 (57.1%)
	12 to 18 years	24 (28.5%)
	>18 years	12 (14.2%)
Symptoms	Local only	12 (14.2%)
	Local & systemic	72 (85.7%)
ECHO findings	LVEF <50%	60 (71.4%)
	LVEF 50-40%	10 (16.6%)
	LVEF 40-30%	30 (50%)
	LVEF <30%	20 (33.3%)
	RV dysfunction	30 (50%)
Cardiac Troponin	<0.01 µg/L	12 (14.2%)
	0.01 to 0.11 µg/L	12 (14.2%)
	>0.11 µg/L	60 (71.4%)
	>10 µg/L	6 (7.1%)
LVEF (%) and Troponin(µg/L)	LVEF 50-40	1.2±0.2
	LVEF 40-30	2.1±0.4
	LVEF <30	6.5±1.9

[Table/Fig-1]: Clinical and Investigation findings

DISCUSSION

The Indian red scorpion (Mesobuthus tumulus) venom is a potent sodium channel activator [12], which results in the stimulation of the autonomic nervous system, which in turn leads to the sudden release of endogenous catecholamines into the circulation [3]. The venom initially leads to a transient cholinergic phase, followed by sustained adrenergic hyperactivity, which is a venom dose dependent phenomenon [16]. The clinical manifestations depend upon the dose of the venom, the age of the patients, the season of the sting and the time lapse between the sting and the hospitalization. The myocarditis which was documented in 60 patients (71.4%) in this study, was higher than that which was reported by Pol et al., (20%) [17]. This may be due to the early use of echocardiography and cardiac troponins (a majority of the symptomatic patients underwent echocardiography and cTnI at the emergency department itself on their arrival). This was similar to that which was reported by Bouaziz et al., [18]. Various studies have reported different mortality rates after scorpion sting envenomation [19-22]. The low mortality (2 patients- 2.3%) in this study was due to the early diagnosis and the early use of ianotropics and prazosin in the intensive cardiac care unit after the echocardiographic assessment by the cardiologist.

Our study revealed a 71.4% incidence of myocarditis based on echocardiography. A majority of the patients had LVEF values of less than 40%. The patients with clinically severe manifestations had LVEF values of less than 30%. A predominant finding was LV global hypokinesia. No significant valvular regurgitation was noted, which indicated primary muscle dysfunction as the cause of the pulmonary oedema. RV dysfunction contributes to hypotension in the significant patients. These patients will be benefitted by the early use of vasopressors and ianotropics.

cTnI assessment has become gold standard in the assessment of acute coronary syndromes. cTnI may be elevated in non-ischaemic conditions. Its measurement and benefits in scorpion sting envenomation has been reported by Meki AR et al., [15]. In their study on 41 children, elevated cTnI levels were noted in 17 severe cases and 14 moderate cases. The level was normal in 10 children with mild symptoms. cTnI showed 100% specificity and sensitivity for the diagnosis of myocardial injury in relation to the Echo finding in the envenomed victims. In severe cases, cTnI was found to be positively correlated with LVEF. Based on their findings, they had concluded that cTnI was a specific marker for the diagnosis of myocardial injury in scorpion envenomation. In our study also, all the 60 patients with myocarditis had elevated cTnI levels, which further confirmed the findings of Meki AR et al., [15]. cTnI was markedly elevated in severe myocarditis. 24 patients who had normal levels of cTnI and normal echocardiographic findings were discharged in 24 to 48 hours. The normal levels of cTnI in patients without an evidence of myocarditis is an important indicator, as it will help the clinicians when echocardiography is not available.

CONCLUSION

Scorpion sting envenomation is a life threatening medical emergency. Severe myocarditis is an important cause of morbidity

and mortality in this situation. The early use of echocardiography and cTnI can identify the subgroup of patients who require early aggressive therapy. Echocardiography is not available routinely and in such situations, the cardiac troponin estimation can guide an early therapy and it can indicate the prognosis. An early discharge may be feasible if the cTnI values and echocardiography are normal. Further studies are needed to confirm our findings.

REFERENCES

[1] Santhanakrishna BR, Ranganathan G, Ananthasubramanian P. The cardiovascular manifestations of scorpion stings in children. *Indian Paediatr* 1977; 14:353-56.

[2] Mahadevan S. Scorpion sting. *Indian Paediatr*.2000; 37: 504-13.

[3] Ismail M. The scorpion-envenoming syndrome. *Toxicon* .1995: 3: 825-58.

[4] Sofer S, Gueron M. Vasodilators and hypertensive encephalopathy following scorpion envenomation in children. *Chest* .1990; 97: 118-20.

[5] Bawaskar HS, Bawaskar PH. The cardiovascular manifestations of scorpion sting in India (A review of 34 children). *Ann Trop Paediatr.* 1991; 11: 381-87.

[6] Santhanakrishnan BR, Balagopal Raju V. The management of scorpion sting in children. Trop Med Hyg. 1974; 77: 133-35.

[7] Biswal N, Murmu Uday C, Mathai B, Bakachander J, Srinivasan S. The management of scorpion sting envenomation. *Paediatrics Today.* 1999; 2(4): 420-26.

[8] Murthy KRK, Vakil AE, Yeolekar RE. Insulin administration reversed the metabolic and the echocardiographic changes in acute myocarditis which were induced by the Indian red scorpion (B. tumulus) venom in experimental dogs. *Ind Heart J* .1990; 42: 35-37.

[9] Abrough F, Ayari M, Nouria S. Assessment of the left ventricular function in severe scorpion envenomation – a combined hemodynamic and ECHO-doppler study. *Inten Care Med*. 1995; 21: 629-35.

[10] Rajashekhar D, Moha A. Clinical and echocardiographic findings in patients with myocardial toxicity which is caused by scorpion sting. *National Medical Journal of India.* 2004;17:307-9.

[11] Amaral CFS, Lopes JA, Magalhaes RA, de Rezende NA. Electrocardiographic, enzymatic and echocardiographic evidence of the myocardial damage after Titys serrulatus poisoning. *Amer J Cardiol* .1991;67:655-57.

[12] Gueron M, Margulis G, Sofer S. Echocardiographic and radionu clide angiographic observations following scorpion envenomation by Leiurus Quinquestriatus.*Toxicon.* 1990;28:1005-9.

[13] Sofer S, Shahak E, Solnim A, Gueron M. Myocardial injury without heart failure following envenomation by the scorpion L quinquestriatus in children. *Toxicology.* 1991;3:383-85.

[14] Mehmet B, Aydin E, Ilyas Y, Vuslat B, Metin K, Fuat G. Scorpion sting envenomation in children in southeast Turkey. *Wilderness Environ Med*. 2009; 20; 118-24.

[15] Meki AR, Mohamed ZM, Mohey El-deen HM. Significance of the assessment of serum cardiac Troponin-I and intereukin-8 in scorpion envenomation in children. *Toxicon* .2003;41:129-37.

[16] Bawaskar HS, Bawaskar PH. Management of the cardiovascular manifestations which are caused by poisoning due to the bite of the Indian red scorpion (Mesobuthus tumulus). *Br Heart J.* 1992; 68: 478-80.

[17] Pol R, Vanaki R, Pol M. The clinical profile and the efficacy of prazosin in scorpion sting envenomation in children in north Karnataka (India). *Journal of Clinical and Diagnostic Research*. 2011; 5: 456-58.

[18] Bouaziz M, Bahloul M, Hergafi L, Kallel H, Chaari L, Hamida CB et al., The factors which are associated with the pulmonary edema in severe scorpion sting patients: a multivariate analysis of 428 cases. *Clin Toxicol (Phil)*. 2006; 44: 293-300.

[19] Biswal N, Bashir RA, Murmu Uday C, Mathai B, Balachander J, Srinivasan S. The outcome of scorpion sting envenomation after a protocol guided therapy. *Indian J Paediatr*. 2006; 73: 577-82.

[20] Bawaskar HS, Bawaskar PH. Prazosin in the management of the cardiovascular manifestations of scorpion sting envenomation. *Lancet* .1986; 1: 510-11.

[21] Bawaskar HS, Bawaskar PH. Indian red scorpion envenomation. *Indian J Paediatr* .1998; 65: 383-91.

[22] Prasad R, Mishra OP, Pandey N, Singh TB. Scorpion sting envenomation in children: the factors which affect the outcome. *Indian J Paediatr* .2011; 78(5): 544-48.

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