Original Paper

Fatal Stings: Autopsy Findings in Scorpion Envenomation

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ABSTRACT

Scorpion envenomation represents one of the common clinical emergencies in various parts of India. There is considerable literature regarding clinical manifestations and management protocols; however only a few studies are available dealing with autopsy findings. One of the reasons is that mortality is not very high in scorpion envenomation as compared to snakebite envenomation.

The present study describes autopsy findings derived from five cases that were autopsied during a five year period. Common site for scorpion sting appears to be some peripheral part of the body, and the sting mark is usually one in number. In the present series, toxic myocarditis was suspected in 3 cases, while one person manifested cardiogenic shock, and the other had respiratory distress syndrome with multiorgan failure.

At autopsy, pulmonary oedema was noted in all the cases, and a state of circulatory failure in 3 cases. Correlation of gross and microscopic findings suggest that a state of shock occurs, resulting in circulatory failure and subsequent multiorgan failure.

Key Words: Scorpion sting; Autopsy; Pulmonary oedema

Introduction

Scorpion envenomation represents one of the common clinical emergencies in various parts of India, and is of immense importance because of its potential for causing serious clinical manifestations and subsequent fatality, especially in susceptible individuals and children. There is considerable literature regarding clinical manifestations and management protocols; however, only a few studies are available dealing with autopsy findings.[1-5] This is probably because mortality, while it is possible in scorpion envenomation, is not as high as in snakebite envenomation. It is important to have some information on autopsy findings related to scorpion sting deaths, as it not only helps to substantiate or refute clinical conclusions, but can also help in resolving confusion with other causes of death. In view of this, the present study was undertaken to evaluate autopsy findings in scorpion sting deaths, and determine characteristic features, if any.

Materials and Methods

This is an autopsy-based retrospective study of scorpion sting deaths conducted between the years 2006 to 2010 at the Dept of Forensic Medicine, Govt Medical College & Hospital, Nagpur. We examined all available files of inquest papers, autopsy reports, histopathology reports and case papers pertaining to deaths by scorpion envenomation. The information collected included age, sex, place of incident, medical attention received, probable clinical diagnosis, period of survival and autopsy findings.

Results

A total of 5 cases were autopsied during the selected five-year period, and their demographic profiles, nature of scorpion involved, period and place of incident, and clinical diagnosis are presented in **Table 1**. Characteristics features of sting marks are presented in **Table 2**.

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Table 1 Distribution of Cases According to Age, Sex and Stinging Incident

Case No.	Age (yrs) /Sex	Type of Scorpion	Month	Time of Incident	Place of Incident	Period of Survival	Clinical Diagnosis
1	9 /M	Red	March	22:00 hrs	Home	2.5 hrs	?Acute toxic myocarditis
2	2 /M	Black	September	18:00 hrs	Home	17.5 hrs	Cardiogenic shock with pulmonary oedema
3	13/F	Red	October	09:00 hrs	Jungle	30 hrs	?Myocarditis with pulmonary oedema
4	76/M	Red	October	14:00 hrs	Farm	174 hrs	Myocarditis with multiorgan failure
5	15/M	Red	October	11:00 hrs	Farm	23 hrs	RDS* with multiorgan failure

*RDS – respiratory distress syndrome

Table 2 Characteristic Features of Sting Marks

Characteristics of Sting Mark	Case 1	Case 2	Case 3	Case 4	Case 5
Site	Left foot	Right hand	Right hand	Right ring finger	Left great toe
Number	Single	Single	Single	Single	Single
Gross findings	Reddening and oedema	Extravasation of blood with inflammation	Reddening and oedema	Extravasation of blood with oedema	Extravasation of blood with inflammation
Microscopic findings	Congestion and mild acute inflammatory cell infiltrate	Congestion and haemorrhage	Congested blood vessels and acute inflammatory cell infiltrates	Haemorrhage with mixed inflammatory cell infiltrates	Congestion and haemorrhage

All the individuals were referred from rural areas adjoining Nagpur District. Specific autopsy findings of the cases are as follows:

Case 1:

Gross examination – brain and liver were congested; heart and pancreas were unremarkable; spleen, adrenals and intestine were normal, and kidneys showed haemorrhages in the cortex. Lungs were congested and oedematous.

Microscopic examination – brain, heart, spleen and adrenals were unremarkable; lungs were congested and oedematous; liver and pancreas were congested; kidneys showed cloudy change with congestion, and intestines showed congestion with lymphoid hyperplasia.

Case 2:

Gross examination – brain, liver and spleen were congested; heart, kidneys, adrenals and intestines appeared normal; lungs were congested and oedematous. Pancreas was unremarkable.

Microscopic examination – brain was congested and oedematous; myocardium and intestines showed congestion. Kidneys showed cloudy change with focal tubular

necrosis. Adrenals and pancreas were unremarkable; lungs were congested and oedematous with mononuclear inflammatory cell infiltrates in the interstitium; liver showed mild mononuclear inflammatory infiltration in the periportal region, and spleen showed congestion and haemorrhages.

Case 3:
Gross examination – brain, liver and spleen were congested; heart, adrenals, intestines and pancreas appeared normal; lungs were congested and oedematous, and kidneys showed haemorrhages in the cortex.

Microscopic examination – brain and adrenals were unremarkable; myocardium, liver, spleen, kidneys, intestines and pancreas appeared congested; and lungs were oedematous.

Case 4:
Gross examination – brain, liver, spleen, and intestines were congested; lungs were congested and oedematous; heart showed eccentric atherosclerosis in coronary arteries; kidneys and adrenals showed haemorrhages in the cortex; and pancreas showed multiple haemorrhages.

Microscopic examination – brain was unremarkable; spleen and intestines were congested. Sections from myocardium showed features of acute myocardial infarction. Lungs were oedematous with alveolar haemorrhages; liver showed hepatocyte necrosis around central vein with mild portal inflammation composed of lymphocytes and polymorphonuclear infiltrates; kidneys showed normal glomeruli with focal tubular necrosis, interstitium showed neutrophilic and lymphocytic infiltration; adrenals showed haemorrhage, necrosis and focal lymphocytic infiltrate; and pancreas showed features of acute pancreatitis with haemorrhage, and necrosis with focal lymphocytic infiltrates.

Case 5:
Gross examination – brain and liver were congested; heart, spleen, kidneys, adrenals, intestines and pancreas appeared normal; and lungs were congested and oedematous.

Microscopic examination – brain, myocardium and adrenals were unremarkable; liver, spleen, kidneys, intestines and pancreas were congested; and lungs were congested and oedematous.

Discussion

Among 86 species of scorpions present in India, *Mesobuthus tamulus* (Indian red scorpion) and *Heterometrus swammerdami* (formerly *Palmaneus gravimanus*; Indian black scorpion) are of medical importance.[6,7] Among the five cases of the present study, four deaths were due to Indian red scorpion and one was due to Indian black scorpion. The Indian red scorpion is predominantly present in Rayalaseema in Andhra Pradesh, Bellary in Karnataka, Chennai and Madurai in Tamil Nadu, Thane, Raigad, Ratnagiri, Marathwada and Western parts of Maharashtra, as well as in parts of Puducherry, Saurashtra, Uttar Pradesh and Bihar. The Indian black scorpion is seen in Kerala, and Vidharbha and Marathwada regions of India. The Indian red scorpion is the most lethal amongst all venomous species of scorpions, while the Indian black scorpion is less poisonous.[8,9]

The common sites for scorpion sting are usually the peripheral parts of the body such as hand and foot. Local envenoming causes pain, erythema, and swelling.[10] Venom is deposited skin-deep to subcutaneous tissues, and almost complete absorption of the venom from the sting site occurs in 7-8 hours. 70% of the maximum concentration of venom in the blood is generally reached within 15 minutes, and the time needed to reach maximum venom blood concentration is 101±8 minutes in experimental animals.[9] In the present series, a sting mark was identified with reddening and oedema in 2 cases (40%), while local haemorrhage was noted in 3 cases (60%). Microscopic examination showed congestion in 4 cases (80%), haemorrhage in 3 cases (60%), and inflammatory cell infiltrates in 3 cases (60%). The inflammatory cells were identified at the sting site as early as 2.5 hours after the stinging incident.

Fatalities from scorpion stings are generally rare, and usually involve young children.[11] In India, the reported incidence of scorpion sting is 0.6%.[9] Local symptoms are the commonest manifestations observed following a stinging incident, though various systemic manifestations are also reported.[7] Death is largely attributed to pulmonary oedema and circulatory failure.[1] However, fatality due to intracerebral haemorrhage has also been reported.[6] Temporal events in systemic envenoming usually develop in two stages. A cholinergic phase is characterized by vomiting, sweating, hypersalivation, priapism, bradycardia and hypotension. This stage is followed by an adrenergic phase consisting of hypertension, tachycardia and

cardiac failure. Cranial nerves and neuromuscular junctions may also be affected. Respiratory failure can be precipitated and is multifactorial.[10]

In the present series, clinically toxic myocarditis was suspected in 3 cases, while one person manifested cardiogenic shock, and the other had respiratory distress syndrome (RDS) with multiorgan failure. Scorpion venom causes alterations in cardiorespiratory system leading to circulatory failure, pulmonary oedema and death. While analyzing the autopsy findings, it was noted that brain was either congested or unremarkable in 4 cases (80%), while cerebral oedema was noted in 1 case (20%). Heart was found normal in 4 cases (80%), and in 1 case (20%) there was acute myocardial infarction. Lungs were congested and oedematous in all the cases, while haemorrhage in the alveoli was noted in 1 case (20%). In 3 cases (60%), liver was normal, while in 1 case (20%), there was mild mononuclear inflammatory infiltration in the periportal region, and in 1 case (20%), hepatocyte necrosis around the central vein with mild portal inflammation composed of lymphocytes and polymorphonuclear infiltration was identified. Spleen was normal in 4 cases (80%), and in 1 case (20%) there was haemorrhage. Kidneys were normal in 2 cases (40%), while in 3 cases (60%) there were changes varying from cloudy degeneration to tubular necrosis and neutrophilic and lymphocytic infiltration in the interstitium. In 4 cases (80%), the adrenal glands were normal, and in 1 case (20%) there was haemorrhage, necrosis and focal lymphocytic infiltration. Pancreas was normal in 4 cases (80%), and in 1 case (20%), there were signs of acute haemorrhagic pancreatitis. Intestines were normal in all the cases, except for lymphoid hyperplasia in one case.

Though the number of cases examined during the study was small, the data suggest certain characteristic features. In the present study, we have observed pulmonary oedema in all the cases. Correlation of gross and microscopic findings at autopsy suggests that a state of shock occurs, resulting in circulatory failure and subsequent multiorgan failure. The multiorgan failure is characterized by changes in hormonal environment with a massive release of counter-regulatory hormones, such as catecholamines, glucagon, cortisol, angiotensin-II, and decreased levels of insulin and increased blood glucose level.[12]

With regard to the central nervous system, most of the symptoms are due to either release of catecholamines from adrenal glands, or release of acetylcholine from postganglionic parasympathetic neurons. In the present series, we noted haemorrhage, necrosis and focal lymphocytic infiltrates in adrenal glands in one case, and the survival period of the deceased was about 174 hours. Autopsy features indicated "exhausted" adrenal glands. Acute renal failure may develop in scorpion stings, and may be due to hypotension or venom-related renal oedema that causes decreased glomerular filtration rate and urinary flow.[12] Hepatic injury or hepatic failure may also occur, and is clinically characterized by increased levels of aspartate aminotransferase (AST) and alanine aminotransferase (ALT).[12] Hepatic injury can be ascribed to sustained state of hypotension and hypoxia as indicated by centrilobular hepatocyte necrosis at autopsy.

The gross and microscopic findings of heart have been described by Gueron and Yaron (1970).[4] They observed degeneration of muscle fibres, focal necrosis, interstitial oedema and cellularity with maximum involvement of the papillary muscles and subendocardial areas. Cupo et al (1994) had observed isolated focal myocytolysis and countless contraction bands with mononuclear infiltrates close to areas of myocytolysis.[1] In the present series, we observed features of acute myocardial infarction in one case. This may have been caused by hypoxia due to positive inotropic effects of catecholamines on the myocardium. In our case, the deceased displayed eccentric atherosclerotic changes, and microscopic findings suggestive of acute myocardial infarction. Therefore, individuals with known history of ischaemic heart disease would logically constitute a vulnerable group, and need more attention from the treating physician.

Experimental studies have shown that the injection of whole venom and purified toxins from the venom of scorpions can cause increased gastric and pancreatic injuries as well as disorders of intestinal motility.[12] Bartholomew (1970) has described 30 cases of scorpion sting, and amongst them 24 had developed acute pancreatitis.[11] According to him, a rise in serum amylase, which occurred in 24 (80%) cases, was found as early as one hour after the sting. In nine (30%) of the patients with hyperamylasaemia there was no abdominal pain. Transaminase studies showed a raised serum glutamic pyruvic transaminase (SGPT) in five cases. No case of hyperbilirubinaemia, occurred. It is known in Trinidad that acute pancreatitis is a common complication of the sting of *T. trinitatis*. Waterman (1938), as cited by Barthlomew (1970), described two cases of acute oedematous

pancreatitis, two of haemorrhagic pancreatitis, and 12 of pancreatic pseudocysts, all of which were found at laparotomy following stings by *T. trinitatis*.[11] In this series, we noted acute haemorrhagic pancreatitis in one case, and it was due to red scorpion sting. It is probable that venom-induced injury to beta cells of the pancreas may cause inhibition of insulin secretion and play a role in the development of hyperglycaemia. Based on animal experiments, Murthy and Hase (1994) postulated that insulin has a primary metabolic role in preventing and reversing the cardiovascular, haemodynamic, and neurological manifestations and pulmonary oedema induced by scorpion envenoming.[13] This premise needs further elucidation.

In conclusion, it can be stated that the pathophysiology of scorpion envenomation is a complex one, and has so far not been completely elucidated. Of late, the role of venom and immune response in triggering the release of inflammatory mediators that are largely mediated by cytokines is being reviewed.[12] This is expected to help in understanding the complexities of scorpion envenomation, and formulating better treatment options.

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