ORIGINAL STUDY

The Clinical Management of Scorpion Stings in Children

El Amin E. O.

Muscat Region, Ministry of Health Muscat, Sultanate of Oman

Abstract:

Management of scorpion envenomation in children is a difficult problem⁽¹⁾. The envenomation syndrome is not very well understood in humans⁽²⁻⁴⁾although there has been extensive work on animal envenomation⁽⁵⁻⁷⁾. This is an attempt to bring together the two experiences in order to produce an acceptable management protocol for envenomation in children. This protocol is based on the management of more than 2,500 cases over a period of seven years in Saudi Arabia where the mortality was about five per cent at the start and zero at the end. The management protocol adopted there in 1992 was a result of serious work on clinical data from Ministry of Health hospitals and laboratory work in the pharmacology department of the faculty of Pharmacy, King Saud University.

Pathophysiology of Scorpion Envenomation:

There are more than 350 species of scorpion with about fifteen being dangerous⁽⁸⁾. Of these fifteen, *Leiurus quinquestratus* and *Androctonus crassicauda* together with other *Buthus spp*. are prevalent in the Middle East. *Tityus serrulatus* and *Centruriodes spp*. are encountered in Central and South America. *Buthus tamulus* and *Palamneus spp*. are found in India.

The usual site of the sting is the lower limb, followed by the upper limb but the venom establishes itself within minutes all over the body⁽⁹⁾. The scorpion venom is a polypeptide that is capable, at molecular levels, of altering the sodium channel mechanisms. This subsequently leads to sustained depolarisation of the cell membrane. The brunt of its effect takes place in the autonomic nervous system leading to massive discharges from both the sympathetic and parasympathetic branches. This is sometimes referred to as an autonomic storm in the body⁽³⁾. The effects are seen all over the body, but are more serious on the cardiovascular and the respiratory systems.

Address for correspondence: **Dr. Eisa Osman El Amin**, MBBS, DTCh, FRCP Senior Consultant Pediatrician Khoula Hospital, P.O. Box 3067, PC 112 Muscat, Sultanate of Oman E-mail: eosman@omantel.net.om

Effects on the cardiovascular system:

Arrhythmia:

Outpouring of catecholamines into the circulation directly affects the heart leading to arrhythmia. Sinus tachycardia is most common although ventricular ectopics also may occur. Increased parasympathetic tone can result in bradycardia. Temporary myocardial hypoxia reflects on some ECG changes such as Twave and ST segment alterations⁽¹⁰⁾. Echocardiography will show some degree of reduction in the ejection fraction ⁽¹¹⁾.

Hypertension:

Hypertension is common in envenomated children due to several factors: (1) release of catecholamines from adrenal glands and postganglionic nerve endings⁽¹²⁾. (2) Catecholamine actions on alpha–adrenergic receptors increasing the peripheral resistance⁽¹³⁾. (3) The catecholamine actions on beta-adrenergic receptors either increasing the cardiac contractility or releasing renin from the kidneys⁽¹⁴⁾.

Hypotension:

Hypotension is usually short–lived and is rarely profound. It can occur alone or alternating with hypertension. It is mainly due to a low cardiac output and is usually accompanied by bradycardia.

Pulmonary Oedema:

This is the most serious complication of envenomation and is the usual cause of death in this syndrome. It occurs both with and without hypertension. The mechanisms causing it include (1) Acute arterial hypertension resulting in left ventricular failure⁽¹⁵⁾. (2) Impaired left ventricular filling due to severe sinus tachycardia⁽¹⁶⁾. (3) Myocardial damage attributable to sympathomimetic overstimulation⁽¹⁷⁾. (4) Increased venous return due to contraction of smooth muscles in the venous bed⁽¹⁸⁾. (5) Joint vasopressor effects and kinin release in the lungs that increases the vascular permeability - a form of pulmonary leak syndrome⁽¹⁹⁾. Some patients experience periodic breathing and apnoea, which is believed to be due to vagal stimulation⁽²⁰⁾.

Other body systems are also affected and patients can develop convulsions, laryngeal spasms and some muscle contractions for brief periods⁽²¹⁾. Secretions from salivary and lachrymal glands are increased. Intestinal motility is increased. Animal studies have shown some degree of hyperglycaemia, hypocalcemia and electrolyte disturbances⁽²²⁾. This is not the case in human envenomation and very few patients will show mild hyperglycemia or hypocalcaemia⁽²¹⁾. Clotting mechanisms are normal although envenomation with some Indian species has been shown to cause some derangement⁽²³⁾.

Clinical Presentation:

The table shows the salient clinical features encountered in 400 patients representing part of an ongoing study of scorpion stings in Saudi Arabia. These features can be divided into local and general features.

Local:

The site of the sting is usually identified on the skin by the presence of a single puncture mark with or without erythema. Parasthesia is usually demonstrable at that site. Local pain is excruciating immediately after the sting and gradually eases. The pain may radiate centrally and cause intense axillary or groin pain. The stinger of the scorpion is very strong and does not break in the skin but it can make an incision if the stung person moves at that time.

General:

Soon after being stung the body becomes cold and clammy and there may be shivering. The core body temperature generally remains normal; occasionally it falls and sometimes it increases. There is increased secretion from the mouth, nose and eyes. Nausea and retching are common and patients occasionally vomit. Irritability and agitation are common features and patients can hurt themselves during that stage. Priapism occurs in some cases and so does urine retention.

Cardio-respiratory:

Sinus tachycardia occurs in most patients; some develop bradycardia. Hypertension occurs more than hypotension but the two can alternate. Both are short lived. Tachypnoea is common and it is very important to monitor this because an increase may signal the start of pulmonary oedema. Pulmonary oedema is the most serious complication of envenomation although it occurs in less than 20% of cases. It occurs alone or in combination with shock and death is very common with the combination⁽²⁴⁾.

Investigations:

Some degree of leucocytosis can follow envenomation in children. Mild hyperglycaemia, hypocalcaemia and hyponatraemia might be encountered in some patients but these have never been serious management issues⁽²¹⁾. Coagulation is usually normal. Serum amylase is sometimes elevated. Creati-

nine kinase and lactic dehydrogenase are always high in the severe case⁽¹¹⁾.

ECG will reveal sinus tachycardia in most patients. Severely affected ones will have T-wave and ST segment changes of subepicardial hypoxia. In the occasional patient Q- waves can appear ⁽¹⁰⁾. Echocardiography of the severely affected patient will show some degree of left ventricular hypokinesia and decreased ejection fraction⁽¹¹⁾. Almost all patients return to normal within 72-96 hours.

Management:

Local Pain:

Local infiltration with xylocaine 0.5% can be used to ease the pain but cutaneous application of xylocaine provides less relief. Crepe bandage can be applied tightly enough to block the lymphatics and delay the spread of venom. Tourniquets should not be used and there is no place for skin incisions.

The Antivenom:

A lot of controversy has risen over the usage of antivenom ⁽²⁵⁻²⁹⁾. Neutralisation animal studies and our clinical experience suggest that it should be given. Five to ten ml of the commercially available Pasteur scorpion antivenom⁽³⁰⁾ are needed to neutralise the venom injected by either *Leiurus* or *Androctonus spps*. One has to resort to wisdom and local experience in order to decide on the need for antivenom: If it is readily available and affordable it is better to be given to all sting victims as quickly as possible. In other settings it has to be given at the start of symptoms. Asymptomatic patients should be observed for at least six hours. It is probably not needed in asymptomatic child stung more than 48 hours previously.

Agitation and restlessness:

Chloropromazine and promethazine given at a dose of 0.5 mg/kg, repeated if necessary, are suitable for agitated and restless patients. Chloral hydrate was not enough to sedate our patients. Phenobarbitone and morphine potentiate the effects of the venom

Fluid Management:

After years of trials we conclude that envenomated children handle fluids very badly. They should be kept slightly dehydrated and plasma should not be used for resuscitation⁽²⁷⁾. Shock is treated if the blood pressure falls to lower than one-third of normal and/or the skin/rectal temperature gradient is more than 5°C. Saline 0.18%-0.45% is given at 10 ml/kg over 30 minutes for resuscitation. This is followed by the same solution at 2.5 ml/kg/hr until recovery from shock and then maintained on one-third of the allowance for their age. In seriously ill patients it is better to monitor fluid intake by the use of a central venous pressure line kept at 8 cm water.

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Hypertension:

Treatment should aim at reduction of the afterload on the heart. Because hypertension is usually transient a short-acting hypotensive is desirable. Nifedipine and hydralazine are suitable agents although captopril (an ACE inhibitor) has been used with good results(31).

Pulmonary Oedema:

This is the most serious complication and all attempts should be made to prevent it, the fluid management detailed above being geared towards that. However, pulmonary oedema can ensue at any time in spite of the restriction of fluids and even with hypotension. In most of cases it is associated with high or normal blood pressure. Patients should be observed for breathlessness and desaturation and a baseline chest x-ray is desirable in symptomatic patients. If pulmonary oedema ensues the patient should be shifted to ICU for further management. The threshold for mechanical ventilation should be low because these patients can maintain normal blood gases to the last minute and then suddenly collapse as a result of exhaustion. Intravenous furosemide is a good adjunct to management. They usually need ventilatory support for one or two days after which the outcome

Miscellaneous:

 Some patients may develop convulsions and diazepam is then a good choice.

 Biochemical or clinical hypocalcaemia can be treated with calcium infusion.

 Atropine should be avoided and used only for severe bradycardia of less than 60min.

 Priapism is not a sign of danger and does not need any special attention.

 Antitetanus is usually not indicated unless dictated by the circumstances of the sting.

Conclusion:

The syndrome of scorpion sting is complex and variable. Its management is sometimes difficult and seriously envenomed patients need ICU care. This protocol is well tried in Saudi Arabia where mortality due to stings has been eliminated. It is always desirable to use antivenom raised from the local species but in its absence the commercially available polyvalent antivenom should be used at the correct dose.

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