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Management of acute pulmonary edema due to scorpion sting

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INTRODUCTION

Scorpion sting is an important medical emergency in tropical countries especially in rural areas. Bare feet walking young children, in early darkness are at high risk of getting stung and they represent about half of the envenomations .The vast majority of serious reactions occur in less than 10 years of age. Acute pulmonary edema is one of the leading cause of death due to scorpion sting and therefore early recognition and adequate treatment can save precious lives.[1,2]

PROPERTIES

Among the various species of scorpion identified, only 25 species are of medical importance and two main species found in India are Palmaneus Garvimanus (Big black scorpion) and Mesobuthus tamulus (An Indian red scorpion) (Fig 1). The venom containing glands are present in the 'teleson ', the last tail segment. During the day they retreat in crevices of dwelling, come out in the night and there fore most stings are during night. The venom is composed of various phospholipases, phosphodiesterases, cardiotoxin, neurotoxins, nephrotoxins, histamine, serotonin.

Voltage dependant ion channels are altered by the venom. Alpha (of Buthus sp.) and beta (of Centruoides sp.) toxins act on sodium channel at presynaptic nerve terminals. Scyllatoxin, charybdotoxin of Leiurus species and Tityus toxin act by inhibiting calcium dependant potassium channels. This leads to autonomic storm.[1,2]

PATHOPHYSIOLOGY

Major manifestations are due to the massive release of catecholamines due to alpha receptor stimulation which lead to autonomic storm and cardiac damage due to ischemia and arrhythmia. It has been found that alpha receptor stimulation can lead to suppression of insulin secretion which leads to hyperglycemia, hyperkalemia, fatty acid accumulation which can damage the heart. The venom also causes increased angiotensin II levels, secondary to activation of RAA system, which can lead to hypertension. Acute pulmonary edema in scorpion sting is mainly due to cardiac failure due to above mechanisms. It can also be due to non cardiogenic factors such as abundant micro thrombi and increased alveocapillary membrane permeability (Fig 2).[1,2]

CLINICAL FEATURES

Can have varied clinical features in the form of autonomic storm characterised by cold peripheries, excessive sweating, vomiting, mydriasis, priapism.[1,2]

Serious life threatening features are encephalopathy, cardiac failure and pulmonary edema. Tachycardia and hypertension can be

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seen within 4 hours of bite due to catecholamines. Some times hypotension and bradycardia can occur with in 1-2 hours of sting due to cholinergic activity. Later hypotension is due to cardiac failure and also due to fluid loss due to excessive sweating and vomiting. In severe cases patient can develop manifestation of DIC and septic inflammatory response syndrome, MODS.[1,2,3]

The diagnosis of pulmonary edema is suspected by the presence of tachypnea, cough, inspiratory retraction of intercostal spaces and by the presence of lung crackles on auscultation of one or both lungs. Tachypnea or intractable cough at admission could mean pulmonary edema in evolution. Close monitoring is indeed vital to detect and treat pulmonary edema.[1,2]

INVESTIGATIONS

1. The diagnosis is confirmed by the presence of arterial hypoxemia and alveolar infiltrates over one or both lungs on the chest roentgenogram. Some times x-ray may not show evidence of pulmonary edema.
2. Electrocardiographic changes frequently seen are peaked T waves in V2-6, ST segment elevation in leads I, aVL, increased QR duration (ventricular activation time) and LVH by voltage criteria. Low voltage complexes throughout the record and left anterior hemiblock indicate poor prognosis.[2,3]
3. Echocardiography can show poor left ventricular contractility, ejection fraction.[2,3]
4. Blood investigation can reveal elevated CPK-MB levels.
5. Some recent studies have shown that in patients with pulmonary edema there was an increase in total plasma proteins and hemoglobin concentrations because of the transfer of hypotonic fluid from the intravascular compartment into the lung interstitium when compared to those with out pulmonary edema.[4]

MANAGEMENT

Management of acute pulmonary edema needs proper intensive care monitoring and appropriate medications.

GENERAL MANAGEMENT

1. Establish airway, breathing, and circulation. In patients with pulmonary edema we need to intubate and give ventilatory support to improve oxygenation and to provide adequate positive end expiratory pressure . Give oxygen support in less severe cases.
2. Administer intravenous fluids adequately to maintain circulation.
3. Monitor pulse, blood pressure, respiratory rate, Spo_2.Use invasive monitoring of blood pressure if needed especially in very sick patients.
4. Give proper muscle relaxants like benzodiazepines for severe muscle spasms.[2,5,6]

SPECIFIC MANAGEMENT

Major drug used in scorpion sting envenomation is Prazosin. Prazosin is a selective alpha-1 adrenergic receptor blocker. It dilates veins and arterioles, there by reducing pre-load and left ventricular impedance without rise in heart rate and rennin secretion. It also inhibits sympathetic outflow in central nervous system. It enhances insulin secretion, which is inhibited by venom action. It has also been found useful even in cases with hypotension. Prazosin is available as 1 mg tablet. The dose recommended is 30 microgram/kg/dose. In myocardial dysfunction and acute pulmonary edema one dose of prazosin is given and then start inotropic and vasodilatory support.[2,5,6]

In children with pulmonary edema with or without hypertension, management should be directed towards relieving afterload without compromising preload. The use of diuretics to minimize the volume overload seems a reasonable measure only when renal water excretion is impaired. For this far better options are good inotropic agent such as dobutamine, which increases myocardial contractility and cause systemic vasodilation due to beta receptor stimulation. The usual dosage used is 5-15 microgm/kg/min. To reduce afterload sodium nitroprusside (SNP), an arterial vasodilator, can be used in situations with hypertension. The recommended dosage is 0.3-5 micro gm/kg/min. Monitor blood pressure and other vitals carefully as it can cause sudden hypotension. The drug has to be protected from light by using black covering to infusion set, to prevent degradation. The metabolic product is cyanide, which is converted to thiocyanate in the liver and removed by kidneys. [2,6]

Nitroglycerine which is a venodilator can be used at the dose of 1-5 micro gm/kg/min. Used in situations associated with hyotension and normal blood pressure.[6]

Once signs of recovery are imminent taper vasodilators and inotropes and 1 hour before stopping vasodilators switch to oral prazosin Q 6 hourly for next 24 hours. Monitor vitals. Discharge as general condition improves.[2,6] (Fig 3). Recently Insulin (0.1-0.2U/kg/day) has been recommended for the treatment of scorpion sting as it increases glycogen content of heart, stabilizes lysosomal membranes, stimulate surfactant synthesis which can counter the development of pulmonary complications.[6] Serotherapy has not much role in management.[7] Control hypertension.

Figure 1

Figure 2: Pathophysiology of Myocardial Failure in Scorpion Sting

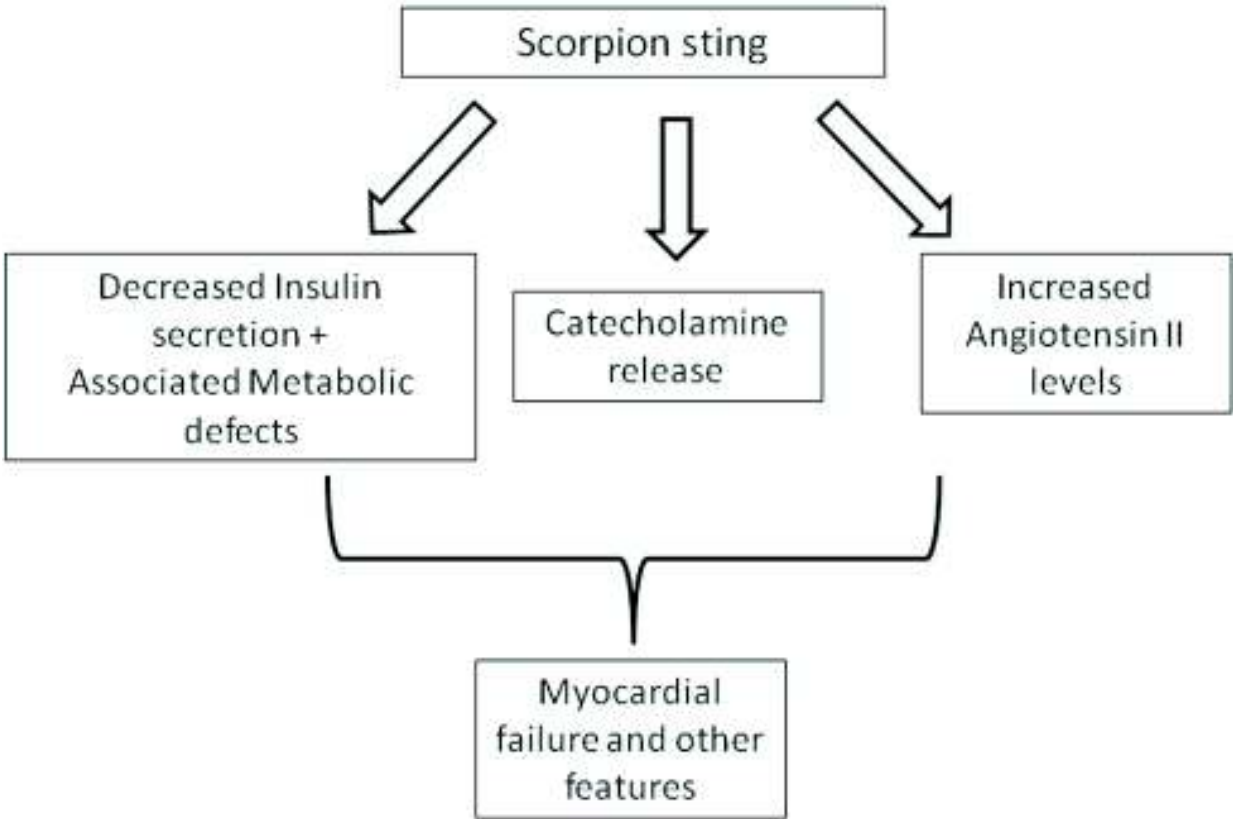
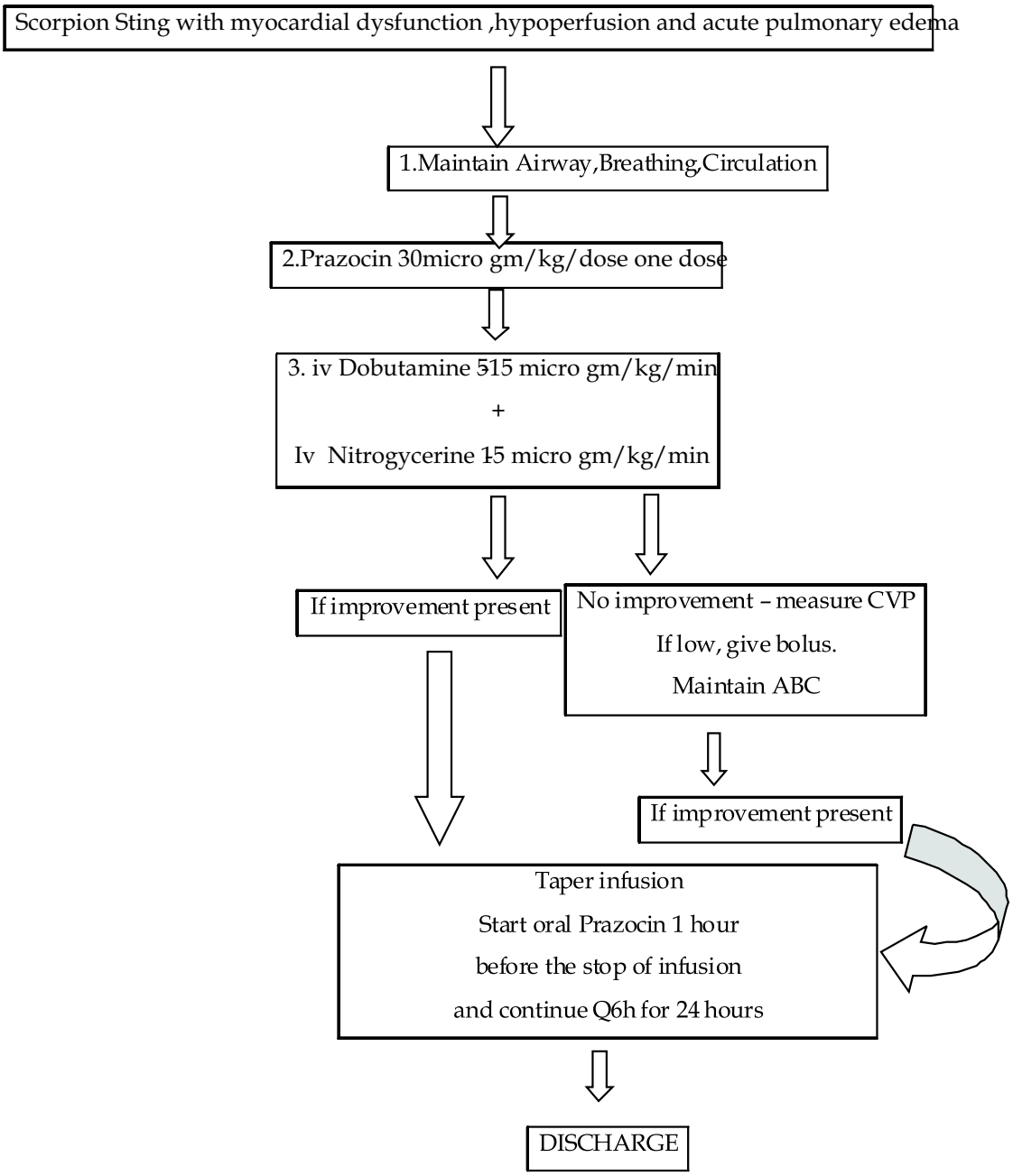


Fig 3. Management protocol of Scorpion Sting



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