Scorpion Sting-induced Pulmonary Edema*

**Scintigraphic Evidence of Cardiac Dysfunction**

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We report two cases of pulmonary edema due to scorpion sting. Radionuclide ventriculography (MUGA scan) showed localized cardiac dysfunction. The cardiac injury induced by the scorpion venom persisted for a prolonged period.

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Scorpion sting evokes severe cardiovascular response, mainly pulmonary edema. The etiology and pathogenesis of these changes are not clear. We report two cases of pulmonary edema due to scorpion sting. In both cases, localized cardiac dysfunction was demonstrated by MUGA scan. Primary pulmonary injury was also found in the first patient.

**CASE REPORT**

**CASE 1**

A 28-year-old soldier was referred to the emergency room with nausea and vomiting two hours after being stung on his thigh by a yellow scorpion. His past medical history was negative.

On admission, he was somnolent with profuse sweating. The physical examination was normal except for tachycardia of 120/min. The sting on the leg was not seen. Laboratory tests revealed WBC count of 19,000 with a shift to the left. Hematocrit was 53 percent; electrolyte levels were normal. The ECG showed sinus tachycardia. He was treated with 1,500 ml of normal saline solution and four ampules of antiserum. Two hours later he developed severe dyspnea. Moist rales were heard over both lungs. Blood gas samples showed severe hypoxia: Po$_2$, 36 mm Hg; PCO$_2$, 32 mm Hg; pH, 7.4; creatinine phosphokinase (CPK) was elevated to 512 units, serum aspartate aminotransferase - 76 units, lactic dehydrogenase (LDH)- 419 units. Chest x-ray film revealed white lungs (Fig 1). The ECG showed ST-T changes in the inferolateral wall. Fluid administration was stopped and the patient received a CPAP mask with high oxygen flow; four additional ampules of antiserum, morphine, furosemide, digoxin and high dosage of steroids. Following this therapy, a Swan-Ganz catheter was inserted and showed a wedge pressure of 12 mm Hg and pulmonary arterial pressure of 23/16 mm Hg. Pulmonary artery oxygen saturation was 58 percent. Radionuclide ventriculography performed with a mobile gamma camera (MUGA scan) showed decreased contraction of the septum and the apical wall. Left ventricular contractility was reduced with ejection fraction of 27 percent. The right ventricle was enlarged with reduced function: EF - 24 percent (Fig 2,3).

According to these data, we concluded that we were dealing with cardiac dysfunction. The normal wedge pressure reflected the effectiveness of the medical therapy or the possibility of capillary leak syndrome with adult respiratory distress syndrome (ARDS). Over the following three days there was dramatic improvement, the dyspnea disappeared, the chest x-ray film showed clearing, and the Po$_2$ level rose to 85 mm Hg on room air. The ECG returned to its normal pattern. MUGA scan on the patient's discharge showed improved regional wall motion with left ventricular ejection fraction of 60 percent and right ventricular ejection fraction of 41 percent. Six months later there was still apical hypokinesis with left ventricular ejection fraction of 56 percent. The right ventricle was still enlarged with an ejection fraction of 48 percent. On follow-up examination, the patient is asymptomatic.

**CASE 2**

A ten-year-old boy was stung by a scorpion and referred to our emergency room with irritability and salivation. Physical examination revealed: blood pressure of 105/70 mm Hg, heart rate of 150/min, clear lungs and priapism. He was treated with atropine and diazepam and three ampules of antiserum. No fluids were administered. A few hours later he developed shortness of breath and both physical examination and chest x-ray film were compatible with pulmonary edema. An ECG showed diffuse ST-T changes, especially in the anterolateral wall. The CPK level was elevated: 614 µ; SCOT, 60µ; LDH, 440 µ. He improved rapidly with therapy with oxygen and intravenous furosemide. Two days later, radionuclide ventriculography revealed reduced global LV function (EF = 41 percent) with hypokinesis of the anteroseptal and apical walls.

**DISCUSSION**

The clinical presentation of scorpion sting is manifested by nausea, vomiting, profuse sweating, irritability, salivation, priapism, urinary retention, hyperperistalsis and goose flesh; CNS involvement includes convulsions and coma. The cardiovascular manifestations of scorpion sting are hypertension and various arrhythmias. Severe dyspnea due to pulmonary edema has been reported in 25 to 35 percent of those patients with severe scorpion sting. Shock developed in up to 40 percent of these patients. ECG pattern includes ST-T changes and early myocardial infarction-like pattern.

![Figure 1. Chest x-ray film during pulmonary edema.](https://journal.publications.chestnet.org/pfaccess.ashx?url=/data/journals/chest/21614/ on 06/21/2017)

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Hemodynamic studies on dogs, performed by Gueron et al., showed that there is an increase in blood pressure and left ventricular end diastolic pressure four minutes after scorpion venom injection. Cardiac hemodynamic and scintigraphic assessment of scorpion sting in humans has not been reported. Necropsy of fatal cases showed marked changes, especially in the papillary muscles and in the subendocardial tissue. These included diffuse or focal myocarditis and muscle necrosis.13

These pathologic and hemodynamic abnormalities point clearly to myocardial failure as the cause of pulmonary edema. The pathogenesis of cardiovascular manifestations is unclear. One theory is that scorpion venom causes release of adrenal and noradrenal from neurons, ganglia cells and adrenals. The clinical picture and pathologic findings are similar to those described in cases of severe pheochromocytoma or catecholamine overdose.44 The urinary catecholamine levels are also elevated in patients with severe scorpion sting.1 The relative role of alpha- and beta-adrenergic receptor activation is unknown. Another pathogenic mechanism is direct myocardial injury by the venom, probably through reduction of Na-K-ATPase levels. The cardiac damage is usually rapidly reversible clinically and pathologically.8

Lung injury due to scorpion venom was described in animals only. Rossi et al.7 injected Brazilian scorpion venom into rats and found intra-alveolar hemorrhage, perivascular edema, interstitial infiltrate of mononuclear cells, eosinophils and mast cells. The electron microscopic changes include severe structural damage in the pneumocytes, intracellular vesicles and endothelial swelling, changes that are similar to those described in epinephrine overdosage.

The mortality rate of scorpion sting has been reduced within the last years from 27 to 3.9 percent.4 Mortality usually occurs during the first 24 h. Most of the fatalities were found among children and were associated with cardiac failure.7 There is close correlation between the time interval to initiation of treatment with antivenom and the clinical severity of the sting.4,10

Treatment of severe scorpion sting consists mainly of specific antivenom. Administration of anticholinergic drugs or alpha- or beta-blocking agents depends on the specific symptoms and signs.11

These two patients represent severe scorpion sting with cardiopulmonary involvement. The cardiac manifestations include tachycardia, diffuse ST-T changes, elevation of CPK levels, pulmonary edema and reduced left and right ventricular function with abnormal regional wall motion according to the MUGA scan. To our knowledge, this is the first report in the literature where hemodynamic studies by Swan-Ganz catheter and MUGA scan were performed. In contrast to the finding described in the literature that the myocardial damage is rapidly reversible, in our first case the ejection fraction was reduced over a prolonged period, and in the second case, it was still reduced two days after the scorpion sting. In both cases, there was hypokinesia of the anteroseptal and apical walls, suggesting local injury or necrosis. In the first case, primary pulmonary injury was probably the major determinant of the pulmonary edema, in view of the normal wedge pressure.

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Asymmetric Septal Hypertrophy in a 41-Year-old Woman with Noonan’s Syndrome*

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A 41-year-old woman had Noonan’s syndrome. Her heart was complicated by asymmetric septal hypertrophy, hypertrophy of the left ventricular free wall, severe pulmonary stenosis, and right ventricular hypertension. On autopsy, a quantitative histologic analysis of the heart revealed that the area of disarray was limited both to the ventricular septum and the left ventricular free wall as in a normal heart. This is not typical of hypertrophic cardiomypathy because the extent of disarray is high in most cases of hypertrophic cardiomyopathy. Some form of hypertrophic cardiomypathy, however, seemed to be present in this patient because right ventricular pressure overload did not affect the left ventricular free wall. To clarify the relation between hypertrophic cardiomyopathy and Noonan’s syndrome, quantitative histologic analysis is necessary.

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Pulmonary stenosis (PS) and asymmetric septal hypertrophy (ASH) are sometimes found in patients with Noonan’s syndrome. Generally, ASH in Noonan’s syndrome is considered to be the result of combined hypertrophic cardiomyopathy (HCM). However, secondary ASH frequently followed marked right ventricular hypertension that is found in PS or mitral stenosis. In such cases, the extent of disarray in the ventricular septum, which is diffuse in HCM, is as minimal as that found in normal hearts. At autopsy, we carried out a quantitative analysis of disarray of the heart in a patient with Noonan’s syndrome coexisting with PS and ASH.

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CASE REPORT

A 41-year-old woman was admitted to our hospital because of syncopal attacks. Her history included a normal delivery, mild cyanosis, and a systolic heart murmur at birth. She had experienced palpitations, chest discomfort on exertion, and occasional syncopal attacks for several years before hospital admission. She had no history of hypertension or family history of HCM. Her height was 141 cm and her weight was 45 kg. Her pulse was 84/min and irregular, and her blood pressure was 120/80 mm Hg. She had clubbed fingers and cyanotic nail beds. Typical features of Noonan’s syndrome were present. A grade 3/6 systolic ejection murmur and wide splitting of the second heart sound were present. The cardiothoracic ratio was 66 percent. The electrocardiogram showed an abnormal frontal QRS axis (−145°) rSR in V1, rs in V5, and QS in V6. Multifocal premature ventricular contractions and frequent ventricular tachycardia were also observed. Laboratory examination revealed marked polycythemia (red blood cell count, 10,140,000/cu mm) and a normal 46,XX karyotype. The echocardiogram (Fig 1) revealed ASH (septum/posterior wall = 28/13 = 2.2), systolic anterior motion (SAM) of the anterior leaflet of the mitral valve, and semicircular-like movement of the aortic valve. Cardiac catheterization revealed a pressure gradient of 130 mm Hg across the pulmonary valve and 41 percent right-to-left shunt at the atrial level. A right ventriculogram showed a thickened and dome-like pulmonary valve and marked dilatation of the main pulmonary artery. No pressure gradient was recorded at the left ventricular outflow tract. She died of septic complication of acute cholecystitis 11 months after her first hospital admission.

Autopsy revealed a markedly hypertrophic heart (650 g) with normal coronary arteries. The right ventricular wall was 12 mm thick, the ventricular septum was 28 mm thick, and the left ventricular posterior wall was 16 mm thick (Fig 2). In general, the left ventricular free wall on autopsy is thicker than that detected on echocardiogram. The heart was cut transversely from base to apex serially at 1-cm intervals. The slices were sectioned in a plane perpendicular to the long axis of the left ventricle at 25-μm thickness by a microtome. Hematoxylin-eosin-stained tissue sections were directly enlarged 50 times (50×50 in the area) on a large sheet of white paper using a projector. Areas of disarray were traced and automatically measured using an image analyzer (VIP-21, Olympus Optical Co., Ltd). Quantitative histologic analysis revealed that the area of disorganization of myocardial fibers was less than 5 percent both in the ventricular septum and in the left ventricular free wall (normal range of disarray by our method: 0 to 10 percent). Interstitial fibrosis was observed in the right ventricular free wall and on the right side of the ventricular septum (Fig 2).

FIGURE 1. Echocardiogram shows asymmetric septal hypertrophy and systolic anterior motion of the anterior leaflet of the mitral valve (arrows). IVS indicates interventricular septum.