

Fatal scorpion envenomation: a case report

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Dear Editor,

Scorpion envenomation, also known as scorpionism, is a major cause of morbidity and mortality in certain tropical and subtropical areas of the world [1, 2]. Of the approximately 160 scorpion species found in Brazil, *Tityus serrulatus* accounts for the most severe accidents [3]. *Tityus serrulatus* envenomation is characterised by local pain, autonomic storm, and cardiotoxicity that can be fatal. Death occurs secondarily to cardiovascular dysfunction that may result in cardiogenic shock [3, 4].

CASE PRESENTATION

A previously healthy 19-year-old woman was transferred from a small hospital to our emergency department. Approximately five hours before admission, she had experienced severe pain on the right shoulder while getting dressed; she had felt something moving inside her shirt but did not visually detect the agent. This incident was followed by nausea, vomiting, non-productive cough, shortness of breath, and bilateral paraesthesia of the upper extremities. She had no significant prior history and no predisposing cardiac risk factors. She was living in a house under repair that had building material in various rooms and was located in a region with an elevated incidence of scorpionism. Upon presentation to the hospital, the patient exhibited diaphoresis, blood pressure of 116/95 mm Hg, a respiratory rate of 40 breaths per minute, oxygen saturation of 68% while breathing ambient air, and a temperature of 37.1°C.

A notable finding of lung auscultation was coarse bilateral crackles. Skin examination showed a mild right supraclavicular papule and oedema. The patient was peripherally cool with prolonged capillary refill (> 4 s). The remaining general examinations produced normal findings. Notable laboratory test results included haemoglobin of 16.8 g dL⁻¹, a white blood cell count of 29,340 mm⁻³, glucose of 289 mg dL⁻¹, serum potassium of 3.6 mmol L⁻¹, creatinine of 1.3 mEq L⁻¹, lactate of 7 mmol L⁻¹ (reference range < 2 mmol L⁻¹), pH of 7.22, pCO₂ of 24.4 mm Hg (3.3 kPa), pO₂ of 58 mm Hg (7.7 kPa), bicarbonate of 10.4 mEq L⁻¹, base excess of -16.7 mmol, oxygen saturation of 83.9%, CK of 385 U L⁻¹ (reference range < 198 U L⁻¹), troponin of 416 ng dL⁻¹ (reference range < 0.04 ng dL⁻¹), and proBNP of 344 pg mL⁻¹ (reference range < 100 pg mL⁻¹). Platelets count were 302 × 10⁹ L⁻¹, activated partial thromboplastin time (APTT) 41 s, and international normalised ratio (INR) 1.39. An electrocardiogram revealed sinus tachycardia and some premature ventricular contractions. A chest X-ray showed a normal-size heart and pulmonary vascular congestion. A transthoracic echocardiogram was requested to assess heart function; this assessment revealed akinesis of the basal and mid left ventricular (LV) segments, significant LV dysfunction, and moderate mitral valve insufficiency. Noninvasive mechanical ventilation, furosemide, and morphine sulphate were started. Because the patient lived in a region with a high

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incidence of scorpionism and had a clinical picture that was extremely characteristic of scorpion envenomation, scorpion anti-venom was administered intravenously.

The patient was transferred to the intensive care unit. After three hours on noninvasive mechanical ventilation, she exhibited cardiopulmonary arrest due to pulseless electrical activity (PEA). Cardiopulmonary resuscitation was immediately commenced, and the airway was secured with an endotracheal tube. Return of spontaneous circulation (ROSC) was achieved in two minutes, but the patient developed hypotension, which was managed by norepinephrine infusion. Because of persistent hypotension despite the initiation of aggressive intravenous volume repletion and vasopressor therapy, a pulmonary artery catheter was introduced. Right heart catheterisation revealed a cardiac index (CI) of $0.9 \text{ L min}^{-1} \text{ m}^{-2}$, mean pulmonary capillary wedge pressure of 19 mm Hg, and venous oxygen saturation (SvO_2) of 40%. Dobutamine ($0.5 \mu\text{g kg}^{-1} \text{ min}^{-1}$) was commenced. A few hours later, the patient exhibited cardiopulmonary arrest again due to PEA, with ROSC two minutes into resuscitation. During day 2, she developed worsening shock, and her norepinephrine and dobutamine doses were progressively increased. Vasopressin, epinephrine, and amrinone were subsequently added. The patient had another episode of PEA; ROSC occurred after less

than a minute of cardiopulmonary resuscitation. On day 3, the patient was receiving norepinephrine ($2 \mu\text{g kg}^{-1} \text{ min}^{-1}$), dobutamine ($20 \mu\text{g kg}^{-1} \text{ min}^{-1}$), vasopressin (0.04 IU h^{-1}), epinephrine ($0.1 \mu\text{g kg}^{-1} \text{ min}^{-1}$), and milrinone ($750 \mu\text{g kg}^{-1} \text{ min}^{-1}$). At that point, lactate was 21 mmol L^{-1} , SvO_2 was 89%, CI was $1.5 \text{ L min}^{-1} \text{ m}^{-2}$, the $\text{PaO}_2/\text{FiO}_2$ ratio was 160, and ProBNP was $22.280 \text{ pg mL}^{-1}$. In addition, platelets count were $97 \times 10^9 \text{ L}^{-1}$ and INR 6.68. A mobile inferior cava vein thrombus was seen on an ultrasound (Figure 1). The decision was made to insert an intra-aortic balloon pump (IABP) for management of the patient's persistent hypotension in the context of reduced ejection fraction. Although this intervention was successful, the patient remained persistently hypotensive and exhibited another PEA cardiopulmonary arrest, after which there was no ROSC after cardiopulmonary resuscitation.

We report a case involving a young and healthy woman who had an episode of intense pain on her right shoulder when she was getting dressed; subsequently, she exhibited signs of sympathetic system overstimulation such as diaphoresis, tachycardia, tachypnoea, hyperlactataemia, and hyperglycaemia; cholinergic syndrome with nausea and vomiting; and acute pulmonary oedema. Although the scorpion was not seen by the victim, the typical clinical manifestations presented by the patient, who lived in a house under repair that was

loaded with building material and located in the north-western part of the state of São Paulo, Brazil, where there is a high prevalence of scorpionism, strongly suggest a diagnosis of scorpion envenomation. Notably, between the years of 2008 and 2014, almost half a million people in Brazil had scorpion accidents, many of whom were in São Paulo State; 581 of these individuals died. Moreover, in this region, there were 1200 cases of scorpion envenomation in 2016, an annual incidence of 78 new cases per 100,000 people [5]. Although most stings cause only local effects, our patient had heart failure that rapidly led to cardiogenic shock, manifested by low CI, impaired end-organ perfusion, and laboratory markers of cardiac distress. *Tityus serrulatus* venom has many constituents, including toxins that bind to sodium channels in cell membranes, causing prolonged depolarisation and the abundant release of epinephrine, norepinephrine, and acetylcholine from the sympathetic and parasympathetic nervous systems. Cardiac failure and cardiogenic shock are the most feared complications of scorpionism; these complications result from the massive release of catecholamines, myocardial damage induced by the venom, or myocardial ischaemia [6-10]. In the described case, cardiogenic shock was treated with supportive therapy, including fluids, vasopressors, inotropes, mechanical ventilation, and an IABP. An IABP is the most widely used device for me-



FIGURE 1. Ultrasound demonstrating a mobile inferior cava vein thrombus

chanical support of cardiogenic shock. It reduces systolic afterload and augments diastolic perfusion pressure, resulting in improvement in cardiac output and coronary blood flow. Because an IABP produces an extremely limited increase in cardiac output delivery, extracorporeal membrane oxygenation (ECMO) and mechanical LV assist devices (LVADs) have emerged as important strategies when cardiogenic shock is refractory to initial therapy [11]. Our patient initially presented with low SvO₂ and high lactate levels, which are common findings for most patients presenting with shock. As the patient deteriorated, SvO₂ and lactate were both extremely elevated, a combination observed in advanced stages of shock, which is typically associated with poor outcomes. Mixed venous saturation (SvO₂) obtained from the main pulmonary artery and SvO₂ determined from the superior vena cava were established as parameters to assess the imbalance between oxygen supply and demand.

A decrease in SvO₂ generally implies inadequate oxygen delivery but can also be secondary to increased oxygen demand. In contrast, high SvO₂ can result from reduced oxygen extraction by the cells in microcirculatory shunting or mitochondrial dysfunction [12]. In fact, various studies have demonstrated associations between degree of mitochondrial impairment and organ dysfunction, clinical severity, and poor outcomes [13]. Moreover, many experimental studies have confirmed the relationship between hyperlactataemia and increased morbidity and mortality in the critically ill [14]. Although tissue hypoxia leading to anaerobic metabolism is one of the causes of high lactate concentration in the blood, hyperlactataemia in the critically ill is mostly due to stimulation of β₂ adrenergic receptors by the surge of endogenous catecholamines that occurs in these patients. Epinephrine stimulates β₂ adrenergic receptors that increase the activity of Na⁺/K⁺ ATPase, which accelerates glycolysis, resulting in increased lactate production [15]. We believe that increased glycolysis

secondary to autonomic storm and the use of epinephrine as an inotrope were important contributing factors to our patient's hyperlactataemia.

The patient also presented a coagulopathy manifested by low platelets, increased APTT and INR, and an inferior cava vein mobile thrombus. In fact, coagulopathy associated with scorpion envenomation has been reported by several authors and can be explained by catecholamine release, systemic inflammatory response, and complement system activation [16].

In conclusion, this case highlights that severe scorpion envenomation can cause intense pain across the affected area; in most severe cases, such pain is followed by excessive autonomic activity and cardiovascular toxicity manifested by heart failure and cardiogenic shock. Scorpion envenomation is a cause of morbidity and mortality and a public health problem in certain parts of the world. Countries with a high prevalence of scorpion envenomation should implement specific policies to prevent scorpion accidents. Emergency physicians and intensivists should be vigilant for the development of severe complications in victims of scorpionism.

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