CASE REPORT

Nephrotic syndrome after scorpion sting

Sangeetha Lakshmi Boju¹ · Hari Krishna Reddy Mogili¹ · R Ram¹ ·
Siva Kumar Vishnubotla¹

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Abstract Scorpion venom is a water soluble, antigenic and heterogeneous mixture. The venom is composed of varying concentration of neurotoxin, cardiotoxin, nephrotoxin, haemolytic toxin, phosphodiesterase, phospholipases, hyaluronidases, glycosaminoglycans, histamine, serotonin, and tryptophan and cytokine releasers. The reported incidence of scorpion sting in India is 0.6 %. Scorpion sting resulting in acute renal failure has been reported in the past, but not the nephrotic syndrome. We report a patient of nephrotic syndrome after scorpion sting. The lacunae in the present knowledge linking scorpion sting venom with nephrotic syndrome would only be replete with publications of similar reports.

Keywords Nephrotic syndrome · Scorpion sting · Minimal change disease

Introduction

Scorpion venom is a water soluble, antigenic and heterogeneous mixture. The venom is composed of varying concentration of neurotoxin, cardiotoxin, nephrotoxin,

haemolytic toxin, phosphodiesterase, phospholipases, hyaluronidases, glycosaminoglycans, histamine, serotonin, and tryptophan and cytokine releasers [1, 2]. The most potent toxin, however, is the neurotoxin. In India, 86 species of scorpions have been identified. *Mesobuthus tamulus* (the Indian red scorpion) and *Palmonusus gravimanus* are the common and most lethal species of scorpions in India with fatalities in adults and children [3]. The reported incidence of scorpion sting in India is 0.6 % [4]. Scorpion sting resulting in acute renal failure has been reported in the past, but not the nephrotic syndrome. We report a patient of nephrotic syndrome after scorpion sting.

Case report

A 49-year-old gentleman, non-diabetic and not a hypertensive, presented with history of scorpion sting over the fourth digit of the left hand. Patient had immediate intense local pain, swelling and redness. Within 1 h he had hypersalivation, sweating, vomiting and diarrhea. Patient was managed at a primary health care center for 48 h with local anesthetic infiltration and anxiolytics. The results of investigations done were: random blood glucose 117 mg/dL, serum creatinine 1.2 mg/dL, hemoglobin 14.5 g/dL and urine for albumin 1+. After 3 days, the patient complained insidious onset of abdominal distension, followed by pedal edema and facial puffiness. The symptoms worsened over next 1 week. There was no history of oliguria, dysuria and haematuria. Patient was managed at a tertiary care institute. The results of investigations done were: serum creatinine 2.9 mg/dL, total serum proteins 4.4 g/dL, serum albumin 1.4 g/dL, total cholesterol 347 mg/dL, hemoglobin 13.2 g/dL, urine examination albumin 4+, red blood cells 10–15/hpf, urine white blood

✉ R Ram
ram_5_1999@yahoo.com

Sangeetha Lakshmi Boju
Sangee_doctor@gmail.com

Hari Krishna Reddy Mogili
Hkr_mogili@gmail.com

Siva Kumar Vishnubotla
savskumar@yahoo.com

¹ Nephrology, Sri Venkateswara Institute of Medical Sciences, Tirupati, AP, India

cells 3–5/hpf, 24 h urine protein was 9.90 g. Ultrasound abdomen revealed: right kidney 10.7 cm and left kidney 10.2 cm. During the hospital stay the serum creatinine increased to 3.3 mg/dL. He was subjected to renal biopsy at that institute. Light microscopy on periodic acid Schiff, silver and trichrome stains revealed seven glomeruli, one of which was sclerotic. The glomeruli were normocellular, capillary loops were patent, and glomerular basement membrane revealed no spikes or double contours. No segmental sclerosis was observed. Tubules showed signs of acute injury. Interstitium and vessels were unremarkable. Immunofluorescence showed no fluorescence for any immunoglobulin and complement. The diagnosis was minimal change disease with acute tubular necrosis. The serum creatinine remained between 3.5 and 4.0 mg/dL for the next 2 weeks. Patient had worsening of pedal edema, facial puffiness and oliguria. He presented to our institute with breathlessness. Blood pressure was 140/90 mmHg, pulse was 110 bpm. Respiratory system examination revealed bilateral diffuse crackles and cardiovascular system examination left ventricular third heart sound. Serum creatinine was 7.0 mg/dL, blood urea was 148 mg/dL, total serum proteins 5.5 g/dL, serum albumin 2.5 g/dL, total cholesterol 237 mg/dL, triglycerides 182 mg/dL, hemoglobin 12.0 g/dL and 24 h protein 2872 mg. He was dialyzed for three sessions and subjected to renal biopsy. The renal biopsy revealed 14 glomeruli. There was no mesangial hyperplasia, glomerular basement membrane thickening or crescents. Tubules showed degenerated epithelium falling towards lumen, regenerating tubular epithelium with increased mitosis and hyperchromatic nuclei and periodic acid Schiff stained tubular casts. Interstitium and vessels were unremarkable. Immunofluorescence was negative for all immunoglobulins and complement.

Fig. 1 Complete effacement of foot processes

Electron microscopy revealed the foot process effacement, vacuolization, and microvillous transformation of epithelial cells and increased density of cytoskeleton (Fig. 1). With the diagnosis of minimal change disease and acute tubular necrosis, the patient was continued on dialysis. Urine output improved after three more weeks of dialysis. Serum creatinine stabilized to 2.0 mg/dL after 8 weeks of scorpion sting. Other investigations after 8 weeks were: 24 h urine protein 1376 mg, total serum protein 5.5 g/dL, serum albumin 2.4 mg/dL, total cholesterol 225 mg/dL and triglycerides 185 mg/dL. As the proteinuria persisted even after 8 weeks, the patient was initiated on tablet prednisolone 2 mg/kg on alternate days. After 12 weeks, the serum creatinine was 1.8 mg/dL and 24 h urine protein was 900 mg. The prednisolone was tapered and stopped. At last follow-up, 24 weeks after the scorpion sting the serum creatinine and 24 h urine protein were 2.0 mg/dL and 1090 mg (Fig. 2).

Discussion

The kidneys and liver are the main routes of excretion from the human body. The kidneys have the largest concentration of venom. This appears to be due to two reasons, rapid redistribution of the venom from the blood to the kidneys and slow removal from the kidney [5].

Scorpion stings can result in acute renal failure by multitude of factors. Direct venom toxicity to the tissues due to high molecular weight substances like hyaluronidase and sphingomyelinase is reported, potassium channel toxins may mediate vascular contraction by causing smooth muscle depolarization and hence afferent arteriolar vasoconstriction, systemic inflammatory response syndrome, complement activation, myocarditis and adrenergic mediated vasoconstriction may all reduce renal blood flow [6]. In addition haemoglobinuria-related pigment nephropathy [7], haemolytic uraemic syndrome [8] and acute interstitial nephritis [9] are also reported as causes of acute renal failure.

The pathogenesis of nephrotic syndrome following scorpion sting is not known. Due to the action of an unrecognized substance similar to the pore forming peptides of *Buthus martensii* may damage glomerular basement membrane [6].

The diagnosis of nephrotic syndrome is indisputable in this patient. The renal biopsy, done twice, suggested the diagnosis of minimal change disease and acute tubular necrosis. The temporal relation with the scorpion sting and the nephrotic syndrome is clear cut. The lacunae in the present knowledge linking scorpion sting venom with

Fig. 2 Panel chart—clinical events

nephrotic syndrome would only be replete with publications of similar reports.

Compliance with ethical standards

Conflict of interest All authors have nothing to disclose and no conflicts of interest.

Informed consent The consent of the patient is taken.

References

1. Ramachandran LK, Agarwal OP, Achyuthan KE, Chaudhury L, Vedasiromani JR, Ganguly DK. Fractionation and biological activities of venoms of the Indian scorpions *Buthus tamulus* and *Heterometrus bengalensis*. Indian J Biochem Biophys. 1986;23:355–8.
2. Ismail M, Abd-Elsalam MA. Are the toxicological effects of scorpion envenomation related to tissue venom concentration? Toxicon. 1988;26:233–56.
3. Bawaskar HS, Bawaskar PH. Scorpion sting: a study of the clinical manifestations and treatment regimes. Curr Sci. 2008;95:1337–40.
4. Chippaux JP, Goyffon M. Epidemiology of scorpionism: a global appraisal. Acta Trop. 2008;107:71–9.
5. de Sousa Alves R, do Nascimento NR, Barbosa PS, et al. Renal effect and vascular reactivity induced by *Tityus serrulatus* venom. Toxicon. 2005;46:271–6.
6. Viswanathan S, Prabhu C. Scorpion sting nephropathy. NDT Plus. 2011;4:376–82.
7. Chadha JS, Leviav A. Hemolysis, renal failure, and local necrosis following scorpion sting. JAMA. 1979;241:1038.
8. Valavi MJ, Ansari A. Hemolytic uremic syndrome following *Hemiscorpius lepturus* (scorpion) sting. Ind J Nephrol. 2008;18:166–8.
9. Gmar-Bouraoui S, Aloui S, Ben Dhia N, et al. Scorpion sting and acute interstitial nephropathy: apropos of 1 case. Med Trop (Mars). 2000;60:305–6.