Acute interstitial nephritis following viper bite: a rare association

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Introduction

Snake bite is a common cause of morbidity and mortality in rural India, accounting for an annual age-standardized mortality rate of 4.1/100 000 to 5.4/100 000, depending on the geographic location [1]. Renal failure is common following a viperid bite, the majority of the patients develop acute tubular necrosis and a lesser number may develop cortical necrosis. Renal lesions apart from acute tubular necrosis and cortical necrosis are not well characterized in hemotoxic snake envenomation. Acute interstitial nephritis in the setting of hemotoxic envenomation is rare.

Case report

A 40-year-old farmer was admitted with a history of a Russell’s viper bite on the dorsum of his right foot. He developed a local reaction followed by oliguric renal failure within 24 h. At the time of admission his platelet count was 50 × 10⁹/L (50 000/µL), PT-INR was 3, activated partial thromboplastin time was >100 s and serum creatinine was 353.6 µmol/L (4.0 mg/dL). There was no evidence of hemolysis or rhabdomyolysis. He was started on polyvalent antivenom venom and parenteral antibiotics (ceftriaxone and vancomycin) and was initiated on hemodialysis. Hematological parameters stabilized within 5 days of admission, and antibiotics were discontinued by Day 10 as the cellulitis resolved. The patient remained oliguric and, as the renal failure was persisting, a renal biopsy was done on Day 33 following the snake bite.

Light microscopy revealed 10 glomeruli, normal morphology, moderate to marked interstitial inflammatory infiltrate composed of predominant lymphocytes and scattered eosinophils (Figures 1 and 2). There was tubular atrophy amounting to 30% of the core.

Immunofluorescence microscopy showed 2+ C1 q deposits in the mesangium. Other complement components and immunoglobulins were absent.

The patient was started on prednisolone 1 mg/kg/day, which was followed by good diuresis. Serum creatinine declined to 265.2 µmol/L (3.0 mg/dL) at the end of the first week. At the end of the second week serum creatinine declined to 79.56 µmol/L (0.9 mg/dL). Corticosteroids were continued for another 2 weeks and then stopped.

Discussion

Renal failure is the leading cause of death in hemotoxic snake bites. Nationwide estimates of the prevalence of renal failure in hemotoxic snake bites are not available; figures ranging from 13 to 44% have been reported [2, 3]. The actual estimates might be much higher since it is a common practice among rural people to approach traditional healers and snake charmers rather than hospitals following a snake bite.

The classic renal lesions described in viprid envenomation are acute tubular necrosis (ATN) followed by diffuse or patchy cortical necrosis. The cause of acute tubular necrosis is thought to be multifactorial; it includes direct nephrotoxicity of the venom, hypotension, pigment-induced nephropathy secondary to hemoglobinuria and myoglobinuria, disseminated intravascular coagulation, hemodynamic alterations and cell injury induced by the release of proinflammatory cytokines and complement. Data regarding the renal ultra-structure are sparse; the ultra-structural pattern is different from that of acute tubular necrosis due to other causes; the changes include endothelial swelling, tubular epithelial necrosis and shedding, acellular segments of the tubular basement membrane and an interstitial infiltrate composed of mast cells, eosinophils, lymphocytes and plasma cells suggestive of reversible intravascular coagulation as the primary insult, tubular damage being secondary to ischemia [2].

Acute cortical necrosis is a devastating, fortunately less common complication of a hemotoxic snake bite; it is the second commonest cause of cortical necrosis in India. In diffuse cortical necrosis, all parts of the cortex except a narrow subcapsular zone remain ischemic. It is considered to be the result of a severe form of underlying disseminated intravascular coagulation. Chug et al. [2] reported fibrinoid necrosis and occlusive thrombosis in arteries and arterioles in ~20% of cases.

Literature regarding acute interstitial nephritis in the setting of hemotoxic snake bite is sparse; only four case reports and one case series comprising five patients have been published [4]. The exact cause of acute interstitial nephritis in viper envenomation is not clear; it is presumed to be an allergic response to the venom. Both lymphocyte predominant and eosinophil predominant infiltrates have been described previously. In the case series by Golay et al., 4/5 of the patients had eosinophil-predominant interstitial infiltrates; the possibility of a superadded...
drug-induced insult could not be confidently ruled out. The treatment and outcomes in Acute Interstitial Nephritis (AIN) are varied; spontaneous recovery as well as progression to chronic kidney disease has been documented (Figure 2).

Other renal lesions are not well characterized in viperine bites. Glomerulonephritis following a snake bite is a contentious issue. There have been a few reports of histologically proven crescentic as well as immune complex-mediated proliferative glomerulonephritis [2, 5].

Even though our patient was prescribed beta lactam antibiotics in the first week, there was no sign of improvement in renal function even after 3 weeks of stopping all potential drugs that could have caused AIN; hence, we believe that it would be the snake venom that is responsible for AIN. Our case also had glomerular C1q deposits, a finding that has not been described previously.

Interstitial nephritis is a rare but potentially reversible complication of hemotoxic snake bite; it should be actively sought in patients with persistent renal failure following hemotoxic snake bite. Our case highlights the effectiveness of corticosteroid therapy in interstitial nephritis induced by snake bite.

Conflict of interest statement. None declared.

References

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