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Case Report

Glomerulonephritis Following Russell's Viper Bite – 1st Case Report in Bangladesh

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Abstract

Russell's viper bite causing coagulopathy, hypotension, DIC, rhabdomyolysis and acute kidney injury. Nephropathy usually is caused by RV having hemotoxic or myotoxic venom components. Multiple mechanism causes renal pathologic changes including tubular necrosis, cortical necrosis, interstitial nephritis, and glomerulonephritis. But above all glomerulonephritis is very rare. We report a case of Russell's viper bite who died of renal failure due to focal segmental proliferative glomerulonephritis admitted int the Rajshahi Medical College Hospital, Rajshahi, Bangladesh.

Keywords: Snake bite, Russell's viper, Glomerulonephritis.

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Introduction

Venomous snakebite is an important public health problem in Bangladesh. A nation-wide community-based survey in Bangladesh done sometime before recorded approximately 700,000 snake bites/year with about 6,000 deaths.[1] Nephropathy usually is caused by the bites of the snakes having hemotoxic or myotoxic venom components. These snakes are vipers (Russell's viper, saw-scaled viper, hump-nosed pit viper), Greater black krait and sea-snake. In Bangladesh Russell's viper was reported to be present initially in Rajshahi and Khulna division later on the geographical range is expanding.[2] Renal pathologic changes following snakebite are mostly tubular necrosis, cortical necrosis and interstitial nephritis. Glomerulonephritis is very rare. Hemodynamic alterations caused by vasoactive mediators, cytokines and direct nephrotoxicity account significantly for the development of nephropathy. Haemorrhage, hypotension, disseminated intravascular coagulation, intravascular hemolysis and rhabdomyolysis enhance renal ischemia leading to renal failure.[3-6] We report a case of Russell's viper bite who died of renal failure due to focal segmental proliferative glomerulonephritis admitted in Rajshahi Medical College Hospital, Rajshahi, Bangladesh.

Case Report:

A 28-yr-old male farmer of Chapainawabganj was bitten by a snake over lateral malleolus of left foot on 01.06.2016 while he was walking through paddy field at evening. The description of snake was consistent

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with Russell's viper. Then he put a firm ligature at mid-thigh level and he was taken to local 'kabiraj' after 1-2 hours of bite. The 'kabiraj' incised his leg at multiple siteswith a sharp blade. There was no history of subconjunctival haemorrhage, GI bleeding or epistaxis. After 2-3 hours of bite the leg was gradually swollen and the patient had several times vomiting.

On admission: he was found to anxious looking, not anaemic, non-icteric, pulse-90/min, BP- 110/70 mmHg, temperature 100.6^0F, respiratory rate - 20/min, left leg and foot was swollen. There was small bleeding from the bite site. No other bleeding point was seen. Urine output was 300 ml in previous 12 hours. 20 minutes whole blood clotting test (20 WBCT) was positive. All systemic examination was unremarkable.

Investigations profile:

Date	S. Creatinine	Urine R/E	Electrolytes	Hb	WBC	CPK	Platelet count	RBS	IPD
04/06/16	6.1 mg/dl	RBC – Plenty		10 mg/dl	12000/cmm	500	130000/cmm		
08/06/16	5.5 mg/dl		Na -133.5 mmol/l, K - 5.4 mmol/l			600			
11/06/16	7.7 mg/dl		Na -130 mmol/l K- 4.55 mmol/l						
13/06/16	8.4 mg/dl		Na -130 mmol/l, K - 6.0 mmol/l			500			1st IPD done
14/06/16	7.0 mg/dl							5.1 mmol/l	
16/06/16	8.5 mg/dl		Na -129.6 K - 6.2						2nd IPD done
30/06/16	5.6 mg/dl					300			

After admission the patient had received 20 vials of polyvalent antivenom (made by VINS Bio Products Limited, India). After receiving antivenom, 20 min WBCT became negative. Two peritoneal dialysis were done due to persistently high creatinine level. But his renal status remained static up to a certain level. The patient was advised to go home with weekly follow up schedule.

Follow up	S. creatinine
07-7-2016	3.7 mg/dl
14-7-2016	3.2 mg/dl
22-7-2016	3.4 mg/dl

Average urine output- 2 L/day. As creatinine level was persistently high despite adequate support, renal biopsy was performed which showed focal segmental proliferative glomerulonephritis (Figure).

Specimen: Renal tissue

Gross Description:

Specimen was received in formalin with proper patient's identification and consists of singe core of renal tissue, measuring 10 mm in length. Embedded such in one block.

Microscopic description (H&E and PAS stains)

Sections reveal single core of partially autolysed renal tissue and contain 10 glomeruli, Four glomeruli reveal mild segmental mesangial cell hyperplasia with increase of mesangial matrix. The remaining glomeruli are normal. Capillary basement membrane thickness appears normal. Renal tubules and blood vessels are unremarkable. There are foci of interstitial round cell infiltration.

Histologic Diagnosis: Kidney (Biopsy): Focal segmental proliferative glomerulonephritis.

Figure: Renal biopsy report (photograph of the histology was not available as we could not find the slide after several attempts from the lab)

Then patient was given pulse therapy with Inj. Methylprednisolone (1g intravenous daily for 3 days) and Inj. Cyclophosphamide 750mg/m^2 monthly for 2 months but the condition of the patient gradually deteriorated both in clinically and laboratories parameter with gradually increasing creatinine level. Patient took discharge on request from hospital and went home and he was requested to get hospitalization again. Patient died after 2 months of bite at home due to complications of kidney failure.

Discussion

In Rajshahi Medical College Hospital, many of the patients developed AKI following Russell's viper bite. Most of the patients of AKI following Russell's viper improved with dialysis and other supportive therapy. As this patient did not respond with supportive therapy including dialysis rather deteriorating, we did renal biopsy. Histopathology report revealed segmental mesangial cell hyperplasia with increase of mesangial matrix. There are foci of interstitial round cell infiltration consistent with focal segmental proliferative glomerulonephritis (FSPGN). We started immunosuppressive pulse therapy with Inj. Cyclophasphomide and Inj. Methylprednisolone in the Nephrology department of RMCH but did not respond as clinical as well as laboratories parameter didn't improve and the patient died due to complications of renal failure at his home.

This is the first patient of fatal FSPGN reported from Bangladesh following Russell's viper bite.

Acute renal failure or acute kidney injury (AKI) is the leading cause of death in hemotoxic snake like Russell's viper bites. The classic renal lesions described in viper envenomation are acute tubular necrosis (ATN) followed by diffuse or patchy cortical necrosis[7]. Acute interstitial nephritis and glomerunephritis are very rare.

The cause of renal lesions are 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.[3-6]

Acute cortical necrosis is a devastating, fortunately less common complication of a hemotoxic snake bite. 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.*[7] 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.[8] The treatment and outcomes in acute interstitial nephritis are varied; spontaneous recovery as well as

progression to chronic kidney disease has been documented.

Glomerulonephritis following a snakebite is a contentious issue. There are a few reports of histologically proven crescentic as well as immune complex-mediated proliferative glomerulonephritis[7,9] Glomerulonephritis is a potentially fatal complication of hemotoxic snakebite; it should be actively sought in patients with persistent renal failure following hemotoxic snakebite.

Sitprija and Boonpucknavig [4,5] described two patients with crescentic glomerulonephritis after Russell's viper bites in Thailand. In another study of 38 patients bitten by the green pit viper or *Russell's viper*, the authors observed thickening of the mesangial areas and mild mesangial proliferation in most of their patients, and diffuse glomerular hypercellularity (ascribed to marked mesangial proliferation) in two patients. Other glomerular changes observed are ballooning of capillaries, endothelial swelling, mesangiolysis and splitting of the glomerular basement membrane; however, the significance of these is difficult to ascertain[10] Immunofluorescence microscopy showed IgM, C3, and fibrin deposits.

Proliferative glomerutonephritis due to toxic action of the venom was reported by Seedat et al.[11] Sant and Purandare[12-14] from the K.E.M. Hospital, a general hospital in Bombay, India have reported toxic proliferative glomerulitis in patients as well as experimental animals following viperine snake envenomation.

Conclusion

Glomerular changes are usually benign, and an immunological mechanism has been implicated in their pathogenesis[5]. In our patient glomerulonephritis was severe, the glomerular changes were presumably due to the multiple cause though not fully evaluated. Glomerulonephritis following Russell's viper bite is very rare but, in our case, histologically we found focal segmental proliferative glomerulonephritis. So, its various complications should be kept in mind while treating a patient of Russell's viper bite.

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