WILDERNESS & ENVIRONMENTAL MEDICINE, **27,** 302–306 (2016)

BRIEF REPORT

Acute Interstitial Nephritis Following Snake Envenomation: A Single-Center Experience

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Objectives.—To identify the clinical and histopathological characteristics of patients who develop acute interstitial nephritis (AIN) following snake envenomation.

Methods.—A retrospective analysis of patients diagnosed with snake envenomation-induced AIN from October 2013 to November 2014.

Results.—After snake envenomation, 88 patients developed acute kidney injury (AKI). Biopsies were performed on 7 patients due to nonrecovery of kidney function. Among these, 5 patients had AIN. Thus, AIN accounted for 5.7% of snakebite-related acute kidney injury. All patients had severe envenomation at presentation and had prolonged renal failure. Kidney biopsy found a mixed infiltrate composed of predominantly lymphocytes, with variable proportions of other cells including eosinophils neutrophils and plasma cells. The response rate to corticosteroids was 80%.

Conclusions.—AIN after snake bite is not uncommon. AIN needs to be considered in patients with persistent renal failure after snake envenomation. Identifying this complication is of utmost importance because of the potentially reversible nature.

Key words: Renal failure, viper bite, tubulo-interstitial inflammation

Introduction

Snakebite is an important occupational hazard in countries of Southeast Asia, especially India. The most medically significant snake species in India include Russell's viper *(Daboia russelii)* and saw-scaled viper (*Echis carinatus*). Acute kidney injury (AKI) is the leading cause of death in *D russelii* envenoming in India.[1] In addition to increased morbidity and mortality, it can lead to progressive kidney failure in the long term.[2] Acute tubular necrosis (ATN) and cortical necrosis account for the majority of renal lesions after viper bites.[3,4] Information on acute interstitial nephritis (AIN) after envenomation is limited. So far only 1 case series and a few single case reports have been published, including 1 case report from our group.[5] Due to paucity of data, the optimal management strategies and long-term outcomes of snake venom–induced AIN is not known. Here we report our experience and treatment outcomes of AIN after presumed *D russelii* envenomation.

Materials and Methods

We identified the total number of patients admitted with snake envenomation-related AKI between October 2013 and November 2014 from hospital records. AKI was diagnosed according to Kidney Disease Improving Global Outcomes 2012 criteria. The details of the patients who underwent kidney biopsy were collected from the renal biopsy registers maintained in the Departments of Nephrology and Pathology at our institution. A total of 88 patients were admitted during the period. As per hospital policy, kidney biopsies are performed if the serum creatinine remains greater than 2 mg/dL 4 weeks post-envenomation. Seven patients with snake envenomation and AKI underwent kidney biopsy during this period. Five patients were reported to have acute interstitial nephritis. The discharge summaries and case records during the hospital stay were retrieved and a chart review was performed using a predefined data collection proforma. Two pathologists who were blind to clinical data reviewed the biopsy slides. Follow-up information was collected from outpatient records.

Results

During this period, 88 patients were admitted with AKI after snake envenomation. Among the 7 patients who underwent kidney biopsy, 5 patients (4 males and 1 female) were reported to have AIN. AIN accounted for 5.7% of snake envenomation-related AKI.

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All patients sustained bites in lower limbs. Species identification was not possible in 2 patients, and the rest were presumed to be due to *D russelii* (as reported by the patients). Fang marks were visible in all patients. Three patients sustained bites during farming-related activities. All patients developed severe local reaction with systemic envenomation and had evidence of disseminated intravascular coagulation at presentation. After admission, all patients were started on prophylactic broad spectrum antibiotics after taking blood and local site cultures. Polyvalent snake antivenom (*Naja naja* [cobra], *E carinatus* [saw-scaled viper], *Bungarus caeruleus* [common Krait], *Daboia russellii* [Russell's viper]) was administered to 4 patients within 6 hours of envenomation. One patient received antivenom from the referring hospital; further doses were not given as the whole blood clotting time had already normalized at the time of admission. Patients 1, 3, and 4 had minor infusion reactions (chills and rigor), which were treated by slowing the antivenom infusion, intravenous hydrocortisone, and antihistamines. The reactions were not severe enough to warrant discontinuation of further antivenom doses. Oliguric renal failure was documented within 24 to 72 hours after the bite; all patients were supported with haemodialysis. Urine sediment was unremarkable at the time of admission. The time frame for recovery of disseminated intravascular coagulation varied from 5 to 16 days. Antibiotics were stopped by

Table 1. Clinical and biochemical characteristics of patients with snakebite-induced AIN

Parameter	*Patient 1*	*Patient 2*	*Patient 3*	*Patient 4*	*Patient 5*
Age (y)	40	22	29	50	32
Sex	Male	Male	Male	Female	Male
Occupation	Farmer	Student	Farmer	Housewife	Farmer
Snake species	*D russelii*	*D russelii*	Unidentified	*D russelii*	Unidentified
Creatinine (mg/dL) at presentation	4.0	6.8	12.0	3.2	2.0
Nadir platelet count	50,000	15,000	18,000	58,000	27,000
PT INR	2.5	3.0	2.5	2.0	1.7
Resolution of DIC	5 days	16 days	7 days	10 days	10 days
Total dose of SAV received*	21 vials	35 vials	Treated elsewhere, details not available	10 vials	50 vials
Antibiotics received	Vancomycin ceftriaxone	Piperacillin-Tazobactum	Vancomycin, piperacillin-tazobactum, meropenam	Ceftriaxone cloxacillin	Piperacillin-Tazobactum
Other drugs received†	Metoclopramide frusemide	Famotidine metoclopramide	Pantoprazole metoclopramide lorazepam	Famotidine amlodipine ondansetron	Pantoprazole metoclopramide
Bite to biopsy time (days)	33	30	46	32	30
Renal function at the time of biopsy	On dialysis	On dialysis	On dialysis	3.4 mg/dL	On dialysis
Response to steroids	Yes	Yes	No	Yes	Partial
Duration of follow-up	>1 year	>1 year	Lost after 3 months	9 months	5 months
Creatinine on last follow-up (mg/dL)	1.2	0.9	10	1.3	1.6

AIN, acute interstitial nephritis; PT-INR, prothrombin time and international normalized ratio; DIC, disseminated intravascular coagulation; SAV, snake antivenom.

* All 4 patients received 70 to 100 mL (7−10 vials) of polyvalent ASV diluted in 100 mL saline over 6 hours followed by 30−50 mL every 6 hours until whole blood clotting time <20 minutes.

† Prescribed according to patient requirements as per the discretion of treating physician.