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Renal Complications Following Snake Bite Envenomation

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Abstract

Snakebite is a common medical emergency and occupational hazards particularly in tropical areas. Viper bites are more common than other poisonous snakebites in humans. The World Health Organization has estimated that there are approximately 1,25,000 deaths among 2,50,000 poisonous snake bites worldwide every year, of which India accounts for 10,000 deaths. Acute kidney Injury (AKI) is an important consequence of snakebite and its proper supportive management after the anti-venom administration is of utmost importance, for a good patient outcome. Renal involvement following snakebite envenomation has not been explored in Saudi Arabia. This is a retrospective study which was carried out at Aseer Central Hospital, a tertiary and referral hospital located in the southern part of Saudi Arabia where envenomation with snake is common medical health problem. The aim of the study is to determine the frequency of renal involvement following envenomation with snake and to determine the risk factors that contribute to the development of such complications. 134 cases of snakebites were identified during the 5-year period (100 male patients and 34 females). The mean age was 30 ± 19 y. Hematuria was seen in 19.7% and mild proteinuria was observed in 39.4%of the cases. Acute Kidney Injury (AKI) occurred in 17 cases (12.7%) and supportive renal replacement therapy was required in 4 cases. The cause of renal failure was based mainly on clinical ground: 6 cases due to pre-renal (volume loss due to bleeding), 10 due to tubulo-interstitial lesions and one case had heavy proteinuria and hematuria and glomerulonephritis was suspected. Full recovery was seen in 15 cases (88.2%) and partial recovery with permanent damage was observed in 2 cases (11.8%) after follow up for 24 months. The most important risk factors of development of AKI are older age, duration of symptoms before treatment initiated i.e. the late presentation to hospital and the abnormal coagulation in the form of disseminated intravascular coagulation. In conclusion, this study concludes that acute kidney injury occurs in 12.7% of the victims of snakebite and hemodialysis and a supportive treatment appear to be the mainstay of the therapy in the cases which are complicated by renal failure.

Keywords: Snakebite • AKI • Renal damage • Dialysis

Introduction

Snakebite is a common health problem in tropics [1].The true incidence is not well documented and it varies from one region to another. Venous snakes belong to four families: Atractaspidae, Elapidae, Viperidae and Hydrophiadae. Different types of Russel viper are commonly found in southern part of Saudi Arabia [1,2]. Viper bites are more common than other poisonous snakebites in humans. The World Health Organization has estimated that there is approximately 1,25,000 deaths among 2,50,000 poisonous snake bites worldwide every year, of which India accounts for 10,000 deaths [3]. Systemic effect of venom includes coagulopathy, neurological abnormalities, musculo-skeletal defect and renal failure. Acute Kidney Injury (AKI) is an important consequence of snakebite and its proper supportive management after the anti-venom administration is of utmost importance, for a good patient outcome [4]. The incidence of snakebite is not well studied in Saudi Arabia and only few reports were published regarding the general features of snakebite. Alsadon et al, have published a report reviewing the number of cases of snakebite over four years, and found 14,679 cases of snakebites were reported during the four-year study period, with a higher prevalence in males (80%) in their productive age. Most patients were within the age group between 25 and 44 followed by 44 to 64 years. The majority of snakebite affected inhabitants were reported from farms of the rural areas, commonly during night hours of spring and summer seasons when snakes are very active. Only 36 (0.24%) patients out of 14,679 were reported dead and 14,643 (99.63%) were discharged after the treatment [5]. Although, renal involvement is a well-known presentation only case reports were published from Saudi Arabia [5,6].

This is a retrospective study, which was carried out at Aseer Central Hospital, a tertiary and referral hospital located in the southern part of Saudi Arabia where envenomation with snake is common medical health problem. The aim of the study is to

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determine the frequency of renal involvement following envenomation with snake and to determine the risk factors that contribute to the development of such complications.

Regardless of taking action and sheath with a dextrose arrangement and utilizing iodized oil to slow the polymerization of N-butyl-2-cyanoacrylate, cementing happened quickly and care was expected to forestall capture of the catheter in the ureter. For this situation weaken povidone-iodine sclerosant demonstrated simply briefly useful because of mechanical disturbance of the fistula.

Material and Methods

All cases admitted due to snakebite between 2015 and 2019 were included. Medical records of all cases were reviewed for demographical data, time of admission after bitten by snake, clinical presentation and all laboratory data on admission, during hospitalization and at time of discharge were recorded. Treatments given were also assessed and any information regarding the type of snake was obtained. A clinical history taking and a complete physical examination were done in each case. The laboratory investigations included serum creatinine, blood urea nitrogen, electrolytes, hemoglobin, total and differential leucocyte counts, platelet counts, red cell counts, bleeding and clotting times, the coagulation profile which included the prothrombin time, the activated partial thromboplastin time and the International Normalized Ratio (INR), urine microscopy, urine albumin, liver function tests and 24 h urine collection for protein. The radiological investigations included X-ray of the chest and ultrasonography of the abdomen.

Inclusion criteria

1. A definitive history of snake bite.

2. The clinical picture was consistent with that of a snake bite, such as the presence of fang marks, cellulitis, and coagulopathy.

3. The presence of an acute kidney injury which is defined as an abrupt (within 48 hours), absolute increase in the serum creatinine concentration of 0.3 mg/dL from the baseline value which was measured at admission, either in our hospital or elsewhere, after the snake bite, before referral to our hospital, or a percentage increase in the serum creatinine concentration of 0.50 percent above the baseline, or oliguria of less than 0.5 mL/kg per hour for more than six hours, or serum creatinine of more than 1.5 mg/dL or oliguria (urine output of less than 400 mL/day).

Exclusion criteria

1. The patients with a pre-existent renal disease (Serum creatinine of >1.5 mg/dL prior to the snakebite or ultrasonography of the abdomen, which was suggestive of bilateral small kidneys, loss of the corticomedullary differentiation, obstructive nephropathy and other renal pathologies).

2. Diagnosed cases of hypertension or diabetes mellitus.

3. Exposure to nephrotoxic drugs or toxins.

All patients received the tetanus toxoid. Anti-Snake Venom (ASV) was administered in a dose of 5 vials (50 ml) in the mild cases, in a dose of 5-10 vials (50-100 ml) in the moderate cases and in a dose of 10-20 vials (100-200 ml) as an intravenous infusion in a drip for over 30 minutes. Antibiotics and diuretics were administered, as indicated. Transfusions of blood and blood products were given to the indicated patients. Hemodialysis was initiated in four patients. The patients were followed up until they were discharged or until they died.

Statistical analysis

Data was analyzed using SPSS, Student t-test and Chi-square was used to compare those who developed renal involvement from those who did not.

Results

134 cases of snake bites were identified during the 5-year period (100 male patients and 34 females). The mean age was 30 ± 19. Table 1 shows the common symptoms and signs at presentation. Laboratory findings are shown in Table 2. Hematuria was seen in 19.7% of the cases and mild proteinuria was observed in 39.4% of the cases (Table 3). Renal impairment (AKI) occurred in 17 cases (12.7%) and supportive renal replacement therapy was required in 4 cases. The cause of renal failure was based mainly on clinical ground: 6 cases due to pre-renal (volume loss secondary to bleeding), 10 due to tubulo-interstitial lesions and one case had heavy proteinuria and hematuria and glomerulonephritis was suspected. Kidney biopsy was not performed because of the bleeding tendencies that some patients had and the rapid recovery in majority of the cases. Full recovery was seen in 15 cases (88.2%) and partial recovery with permanent damage was observed in 2 cases (11.8%) after follow up for 24 months. The two cases continue to be under follow up with average serum creatinine of 2.5 mg/dl. Both referred for kidney biopsies but patients refused. Risk factors for development of AKI was analyzed (Table 4) and it indicates that the most important risk factors of development of AKI are older age, duration of symptoms before treatment initiated i.e. the late presentation to hospital and the abnormal coagulation in the form of disseminated intravascular coagulation.

Table 1. Common symptoms and signs of snakebite seen in the study group (134 cases) M=100 (74.6%); F=24 (25.4%).

Symptoms and Signs	n (%)
Symptoms	
Local pain	134 (100)
Local swelling	128 (97)
Vomiting	38 (28.8)
Bleeding	24 (18.2)
Headache	8 (6.1)
Signs	
Local tenderness	102 (77.3)
Local redness	48 (36.4)
Local blisters	14 (10.6)
Local lymphadenitis	10 (7.6)
Local abscess	8 (6.1)

Table 2. Laboratory findings of 134 cases of snakebites.

Tests and Laboratory findings	n (%)
Leukocytosis, WBC >10 × 109/L	64 (48.5)
Thrombocytopenia, platelets<100 × 109/L	24 (18.2)
Prolonged PT, >3 sec. above control	48 (36.4)
Prolonged APTT, >10 sec. above control	30 (22.7)
Low fibrinogen,<2.0 g/L	18 (13.6)
High FDP, >40 mg/L	18 (13.6)
Elevated SGOT (AST), >30 U/L	24 (18.2)
Elevated SGPT (ALT), >30 U/L	8 (6.0)
Elevated CPK, >200 U/L	22 (16.7)
Elevated LDH, >450 U/L	14 (10.6)
Elevated bilirubin, >20 µmol/L	10 (7.6)
Mild albuminuria (Dipstick)	52 (39.4)
Hematuria, >10 RBC/HPF	26 (19.7)
Granular cast in urine	24 (18.2)

Table 3. Renal involvement following snakebite.

	n (%)
Acute Kidney Injury (AKI)	17 (12.7)
Dialysis	4 Patients (Duration 9.8 ± 4.1 days)
Causes of renal impairment (Clinical diagnosis)	
Pre-renal	6 (35.3%)
Tubular-interstitial	10 (39)
Suspected glomerulonephritis	1 (5.8)
Outcome of Renal impairment	
Full recovery	15 (88.2)
Partial recovery	2 (11.8)

Table 4. Risk factors of AKI following snake bite.

Factors	AKI	Non-AKI	P-value
Age	35.06 ± 18.25	21.12 ± 16.2	0.023
Duration of symptoms	11.4 ± 18.9	5.8 ± 8.73	0.006
Presence of bleeding	41.20%	13.70%	0.001
Oliguria	23.50%	9%	0.005
Anuria	11.80%	0	0.012
Jaundice	11.80%	0	0.013
Hemoglobin	10.9 ± 4.2	13.0 ± 2.5	0.011
Platelets	121.8 ± 71.8	222.2 ± 114.2	0.003
PT	67.5 ± 20	30.7 ± 15	0.025
PTT	117.5 ± 50	59 ± 30	0.024
SGPT	184.7 ± 80	29.9 ± 10	0.017
SGOT	132.2 ± 70	39.9 ± 20	0.022
Indirect bilirubin	3.2 ± 2.5	0.8 ± 0.5	0.001
LDH	1009.71 ± 100	370.11 ± 90	0.005

Discussion

Over 2000 species of snakes are known world-wide; however, only 400 of these are venomous [5]. These snakes belong to four families: elapidae, viperidae, hydrophidae, and colubridae [5]. Renal involvement has been reported following bites from members of the latter three families, including the Russell's viper, *Echis carinatus* [7-9]. The most important of all renal syndromes is AKI which has been reported with varying frequency in different studies [10-13]. Although most of the venomous snakes are known to cause AKI, the vast majority of these cases are due to viper bites [10]. Data on the precise incidence in different geographical regions is lacking but obviously varies with the distribution of the snakes. In India, the incidence of AKI is reported to be 13%-32% following *Echis carinatus* or Russell's viper bites [10]. In Nigeria, the reported incidence rate for AKI after *Echis carinatus* bite is 1% and following puff adder bite is 10% [9,10]; In Israel it is 6.2% following Palestinian viper bite [11], in Thailand it is 5% following Russell's viper and sea snake bite [12] and in ceylon the reported incidence of AKI following bite of unidentified vipers is 27% [12].

Renal involvement following snake bite in Saudi Arabia is not well documented.

Hematuria and proteinuria are the commonest renal abnormalities found in 20%-70% of the reported cases [13,14]. Oliguria or anuria may develop from within a few hours, to as late as 96 hours, after the bite [15].

Pathological studies showed that Tubulointerstitial lesions, predominantly tubular necrosis, are observed in 60%-80% of patients who develop AKI following snake bite [14-18].

AKI is an established risk factor for the development of CKD in the long-term and limited data exist regarding the long-term outcomes of patients who develop AKI following envenomation. Priyamvadaone et al. observed that one third of their patients developed adverse renal outcomes on long-term follow-up [17]. Herath et al. reported that 37%of patients who sustain AKI following envenomation develop CKD by the end of 1 year [18]. These patients were old, and the majority had comorbidities like hypertension and diabetes, which are independent risk factors for CKD. Waikhom et al. [19] reported that 41% of patients who sustain envenomation develop persistent renal abnormalities in the long-term. The patients who developed adverse renal outcomes in this study were older and had a lower GFR at the time of hospital discharge. Advanced age and severe renal failure are established risk factors for CKD progression [18-20]. During snakebite envenomation, three mechanisms may cause AKI: An immunological reaction (anaphylaxis, action of immune complexes).

Inflammatory syndrome and hemodynamic disorders, directly related to the intensity of the patient's innate defense response and bleeding and direct toxic action of the venom on the kidney parenchyma [21]. Renal pathologic changes include tubular necrosis, cortical necrosis, interstitial nephritis, glomerulonephritis, and vasculitis. Hemodynamic alterations caused by vasoactive mediators, cytokines, and direct nephrotoxicity account significantly for the development of nephropathy. Hemorrhage, hypotension, Disseminated Intravascular Coagulation (DIC), intravascular hemolysis, and rhabdomyolysis enhance renal ischemia leading to AKI [20,21].

In conclusion, renal manifestations among patients with snake envenomation included reduce urine volume, renal tenderness, proteinuria, hematuria, electrolytes abnormalities, and AKI were common and significant complications. The factors associated with AKI included bites from snakes of the family Viperidae or presenting the clinical syndrome of Viperidae envenoming, duration from bite to receipt of antivenom ≥ 2 h, leukocytosis, overt DIC, rhabdomyolysis, hyponatremia and presence of microscopic hematuria.

Conclusion

Our findings support the hypothesis of multifactorial involvement in the pathogenesis of AKI After sustaining Viperidae bites, a significant number of patients with normal renal function developed proximal and distal renal tubular dysfunction. These findings might help clinicians to provide optimal management of patients who are at risk for the development of AKI in order to reduce the incidence of AKI in tropical countries.

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