International Journal of Medical and Health Research
ISSN: 2454-9142
www.medicalsciencejournal.com
Volume 4; Issue 1; January 2018; Page No. 79-83

Incidence of acute kidney injury and its associated factors among snake bite patients in rural area of Salem district

[1] Dr. Suresh Kanna Singaram, [2] Dr. Elanchezhian JA, [3] Dr. Balamurugan Subbiah, [4] Dr. Shankar Radhakrishnan
[1] Professor, Department of Medicine, Government Mohan Kumaramangalam Medical College, Salem, Tamil Nadu, India
[2] Associate Professor, Department of Medicine, Government Mohan Kumaramangalam Medical College, Salem, Tamil Nadu, India
[3] Consultant Physician, Sri Narayani Hospital and Research Center, Sri Puram, Tamil Nadu, India
[4] Associate Professor, Department of Preventive Medicine, VMKVMC, Salem, Tamil Nadu, India
[*]Corresponding Author: Dr. Elanchezhian JA

Abstract
Background: In India Agriculture being one of the major occupations, snake bite is a familiar occupational hazard of farmers, plantation workers and others, resulting in tens of thousands of deaths each year and innumerable cases of chronic physical handicap. India accounts for about 40 to 50,000 deaths per year due to snake bite. Among the four species Russell's viper is the major cause of snakebite cases in India leading to increased morbidity and mortality mainly due to acute kidney injury (AKI).An incidence of renal involvement with snakebite envenomation was reported between 1.4–28% in various series of studies.
Aim: To assess the incidence and factors associated with development of acute kidney injury among the snake bite patients.
Methodology: A prospective study was conducted at Government Mohan Kumaramangalam Medical College,Salem for a period of one year between April 2015 and March 2016. A total of 131 snake bite patients were included in the study based on fulfilling the inclusion criteria. A detailed history taking related to type of snake and time of bite including the presenting symptoms were enquired and recorded and a complete physical examination was conducted in each case and laboratory investigations related to kidney and liver functions along with bleeding disorders was done for all patients. All the patients were followed either till discharge or death. The kidney injury was graded according to AKIN classification.
Results: The incidence of acute kidney injury among the snake bite patients was 32.3% with varying stage ranging from 1 to 3 among them stage 1 was found to be more common(16%) and stage 3 was reported in 3.8% of the patients. Cellulitis feature was found to be more common among the patients who had developed AKI and the incidence of cellulitis increases as the staging of AKI increases. Similarly ptosis, regional lymphadenopathy, respiratory failure, septicaemia and DIC increases as the severity of AKI increases. The increase in age, increase in the ASV vials used and longer duration between the bite and needle time were found to have a significant association with increase in the severity of AKI. Among 131 patients 21 patients required dialysis either in the form of hemodialysis or peritoneal dialysis and 18 patients died and among them 13 had AKI and in that 4 of them had stage 3 AKI.
Conclusion: Snake envenomation leading to acute kidney injury is a major health issue due to delayed referral to appropriate care centre. Early referral to specified care centre and prompt administration of ASV can reduce mortality and morbidity due to snake bite leading on to AKI.

Keywords: snake bite, acute kidney injury, cellulitis, anti-snake venom

Introduction

In tropical countries snake bite is one among the commonest cause for morbidity and mortality. Worldwide report had shown that at least 4,25,000 envenoming and 25,000 deaths occur each year due to snakebite. These figures may be as high as 25,41,000 envenoming and more than 1,00,000 deaths, envenoming occurs in one in every four snakebite cases [1]. In India Agriculture being one of the major occupations, snake bite is a familiar occupational hazard of farmers, plantation workers and others, resulting in tens of thousands of deaths each year and innumerable cases of chronic physical handicap [2]. India accounts for about 40 to 50,000 deaths per year due to snake bite [3].

As of today total identified species of snakes are about 2500 - 3000 and among them only 500 are considered to be venomous [4]. There are two important families of venomous snakes in Southeast Asia one is Elapidaewhich have a short permanently erect fangs and it includes the cobras, king cobra, kraits, coral snakes and the sea snakes and the second one is Viperidaewhich have long fangs and are normally folded up against the upper jaw but when the snake strikes it gets erected. Viperiae is further classified into two groups the typical vipers (Viperinae) and the pit vipers (Crotalinae). In India the most common venomous snakes are the Indian cobra (*Najanaja*), the common krait (*Bungaruscaeruleus*), the Russell's viper (*Daboiarusselii*) and the saw-scaled viper (*Echiscarinatus*) [5, 6].

Among these four species Russell's viper is the major cause of snakebite cases in India leading to increased morbidity and mortality mainly due to acute kidney injury (AKI).[7]An incidence of renal involvement with snakebite envenomation was reported between 1.4–28% in various series of studies [8],

and the incidence of ARF was reported as 10–32% [9-11]. Among the multifactorial causation for the pathogenesis of snakebite‐induced AKI (SAKI), elevated oxidative and carbonyl stress (CS) are the leading cause.Oxidative stress (OS) results in the modification of protein either directly through the oxidation of aminoacid residues by reactive oxygen species (ROS) or indirectly by an increased generation of reactive carbonyl species [12, 13].

Previous studies had shown a greater susceptibility to sAKI with increasing age [14, 15] and it had also quoted the risk factors for developing AKI in snake bite which included age <12 years, time from hospitalization to antivenom treatment >2 h, time from snakebite to receiving antivenom>2 h, longer duration from snakebite to hospital arrival, cellulitis, regional lymphadenopathy, hypotension, higher total bilirubin level, lower hemoglobin level, intravascular hemolysis, incoagulable blood on 20-min whole blood clotting test (20WBCT), prolonged bleeding time, prolonged prothrombin time (PT), hemorrhagic manifestations, serum creatine kinase >2000 IU/L, dark or brown urine color, albuminuria, and longer length of hospitalization [16-19].

Tubular necrosis and cortical necrosis are the most common pathological lesions in Saki [20]. The AKI which occurs after snake bite is usually reversible, but if acute cortical necrosis occurs, it may lead to an incomplete recovery. Acute kidney injury though considered as an important complication of snake bite, proper supportive management along with the antivenom administration is of utmost importance for a good patient outcome.

Aim

To assess the incidence and factors associated with development of acute kidney injury among the snake bite patients.

Methodology

A prospective study was conducted at Government Mohan Kumaramangalam Medical College, Salem for a period of one year between April 2015 and March 2016. A total of 131 snake bite patients were included in the study based on fulfilling the inclusion criteria. The study was conducted after getting the clearance from the institutional ethical committee and the informed consent either from the patients or the guardian of the patient. The inclusion and exclusion criteria of our study is as follows

Inclusion Criteria

1. History of Snake bite with signs of Envenomation
2. Progressive elevation of serum creatinine >0.3mg/dl from baseline, a percentage increase in the serum creatinine concentration of >50% or oliguria of less than 0.5ml/kg/hr for more than 6hrs.
3. Age is more than 18 years.

Exclusion Criteria

1. Patients with Pre-existing Renal Diseases with history of Snake bite.
2. Extreme age groups - age more than 80 years
3. Patients with contracted kidneys with normal Renal Parameters with history of Snake bite
4. Exposure to nephrotoxic drugs

A detailed history taking related to type of snake and time of bite including the presenting symptoms were enquired and recorded and a complete physical examination was conducted in each case. The laboratory investigations included haemoglobin, 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 normalised ratio (INR), urine microscopy, urine albumin, kidney and liver function tests and serum electrolytes. The radiological investigations included X-ray of the chest and ultrasonography of the abdomen.

All the patients received the tetanus toxoid. Anti-Snake Venom (ASV) was administered in a dose of 5 vials (50ml) in the mild cases, in a dose of 5-10 vials (50-100ml) in the moderate cases and in a dose of 10-20 vials (100-200ml) 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. Renal replacement therapy (either peritoneal or haemodialysis, depending upon the availability of the resources), was given to 21 patients. All the patients were followed either till discharge or death. The kidney injury was graded according to AKIN classification.

Table 1: The AKIN classification/staging system of acute kidney injury [21].

Stage	SCr	UO
1	↑ SCr ≥26.5 µmol/L (≥0.3 mg/dL) or ↑SCr ≥150 a 200% (1.5 a 2×)	<0.5 mL/kg/h (>6 h)
2	↑ SCr >200 a 300% (>2 a 3×)	<0.5 mL/kg/h (>12 h)
3[b]	↑ SCr >300% (>3×) or if baseline SCr ≥353.6 µmol/L (≥4 mg/dL) ↑SCr ≥44.2 µmol/L (≥0.5 mg/dL)	<0.3 mL/kg/h (24 h) oranuria (12 h)

[a] SCr, serum creatinine; UO, urine output.

[b] Stage 3 also includes patients requiring RRT independent of the stage (defined by SCr and/or UO) they are in at the moment they initiate RRT.

All the data were entered and analysed using SPSS version 21. Mean and standard deviation was derived for all the parametric variables and chi-square test was used to derive association between two variables and regression analysis was done between the dependant and independent variables. P <.05 was considered as statistically significant.

Results

The age and sex wise distribution of the study subjects is shown in table 2. It is seen from the table that majority of the study subjects are in the age group between 30 and 50 years and the male: female ratio was 2.01: 1. The minimum age of the study subjects was 18 years and the maximum age was 80 years with a mean age of 38 years. Among the study subjects the maximum number of them had the bite in right foot followed by left foot and the other sites of bite were right and left hand (table 3). Most of the subjects (48%) had the bite to

needle time with less than 2 hrs and the maximum time for the bite to needle time was 50 hrs with a mean time of 3.5 hrs (table 4). Of the various clinical manifestations of the study subjects cellulitis was found to be the most common manifestation followed by regional lymphadenopathy. Oliguria was present in 52% of the subjects, DIC and septicaemia was seen in 12.2% and 9% respectively and respiratory failure was seen in 6% for whom ventilator support was given (table 5). The incidence of acute kidney injury among the snake bite patients was 32.3% with varying stage ranging from 1 to 3 among them stage 1 was found to be more common (16%) and stage 3 was reported in 3.8% of the patients (table 6). Logistic regression analysis between acute kidney injury and the other clinical and laboratory manifestations among the study subjects was shown in table 7. Cellulitis feature was found to be more common among the patients who had developed AKI and the incidence of cellulitis increases as the staging of AKI increases. Similarly ptosis, regional lymphadenopathy, respiratory failure, septicaemia and DIC increases as the severity of AKI increases. The increase in age, increase in the ASV vials used and longer duration between the bite and needle time were found to have a significant association with increase in the severity of AKI. Among 131 patients 21 patients required dialysis either in the form of hemodialysis or peritoneal dialysis and 18 patients died and among them 13 had AKI and in that 4 of them had stage 3 AKI.

Table 2: Age and sex wise distribution of the study subjects

Age group	Male	Female	Total	P value
18 – 30	16 (21.3%)	9 (16%)	25 (19%)	0.716
31 – 40	22 (29.3%)	16 (28.5%)	38 (29%)	
41 – 50	14 (18.6%)	15 (26.7%)	29 (22.1%)	
51 – 60	13 (17.3%)	11 (19.6%)	24 (18.3%)	
61 – 70	7 (9.3%)	4 (7.1%)	11 (8.3%)	
>70	3 (4%)	1 (1.7%)	4 (3%)	
Total	75 (100%)	56 (100%)	131 (100%)	
Mean ± SD	38.4 ± 6.8	37.3 ± 7.2	37.9 ± 6.6	

Table 3: Distribution of the study subjects based on the site of bite

Site of bite	Frequency	Percentage
Left foot	37	28.2%
Right foot	58	44.2%
Left hand	16	12.2%
Right hand	20	15.2%
Total	131	100%

Table 4: Distribution of the study subjects based on the bite to needle time

Bite to needle time	Frequency	Percentage
<2 hrs	63	48%
2 – 5 hrs	28	21.3%
5 – 10 hrs	14	10.6%
10 – 20 hrs	11	8.3%
20 – 30 hrs	8	6.1%
30 – 40 hrs	3	2.2%
>40 hrs	4	3%
Total	131	100%

Table 5: Distribution of the study subjects based on the clinical manifestations at the time of presentation

Clinical presentation	Frequency	Percentage
Cellulitis	98	74.8%
Hypotension	11	8.3%
Regional lymphadenopathy	76	58%
Ptosis	24	18.3%
Respiratory failure	8	6.1%
Oliguria	68	51.9%
DIC	16	12.2%
Septicaemia	12	9.1%

Table 6: Distribution of the study subjects based on the staging of acute kidney injury

AKI	Frequency	Percentage
Stage 0	88	67.1%
Stage 1	21	16%
Stage 2	17	12.9%
Stage 3	5	3.8%
Total	131	100%

Table 7: Logistic regression analysis between acute kidney injury and the other clinical and laboratory manifestations among the study subjects

Clinical manifestations	AKI stage 0 (n=88)	AKI stage 1 (n=21)	AKI stage 2 (n=17)	AKI stage 3 (n=5)	P value
Cellulitis (n=98)	55 (62.5%)	21 (100%)	17 (100%)	5 (100%)	<.0001
Hypotension (n=11)	2 (2.2%)	4 (19%)	4 (23.5%)	1 (20%)	0.01
Regional lymphadenopathy (n=76)	37 (42%)	19 (90%)	15 (88.2%)	5 (100%)	<.001
Ptosis (n=24)	6 (6.8%)	5 (23.8%)	8 (47%)	5 (100%)	<.001
Respiratory failure (n=8)	0	1 (4.7%)	3 (17.6%)	4 (80%)	<.001
Oliguria (n=68)	25 (28.4%)	21 (100%)	17 (100%)	5 (100%)	<.001
DIC (n=16)	0	1 (4.7%)	10 (58.8%)	5 (100%)	<.001
Septicaemia (n=12)	0	1 (4.7%)	7 (33.3%)	4 (80%)	<.001
Mean number of ASV vials used	12	18	26	34	<.001
Mean age	34.2	37.6	47.6	49.8	<.001
Mean bite to needle time (hrs)	1.8	8.8	10.4	20.8	<.001
Death (n=18)	5 (5.6%)	3 (14.2%)	6 (35.2%)	4 (80%)	<.001
Male (n=75)	48 (54.5%)	16 (76%)	8 (47%)	3 (60%)	0.0817
Female (n=56)	41 (46.5%)	4 (19%)	9 (52.9%)	2 (40%)	

Discussion

Snakebites have the highest incidence in Asia and they represent an important health problem. The exact pathogenesis of AKI following snake bites, is not well established. However, a number of factors contribute to it, like bleeding, hypotension, circulatory collapse, intravascular haemolysis,

disseminated intravascular coagulation, microangiopathic haemolytic anaemia and the direct nephrotoxicity of venom [22]. Tubular necrosis (53.6%) and cortical necrosis (24.3%) are the main causes of acute renal failure [20]. Acute interstitial nephritis has also been described [23]. Males are affected more often than the females, as they constitute the working majority who are actively engaged in farming and other outdoor activities. Our findings were concurrent with those of earlier studies [24, 25]. In our study, predominantly, the younger population was involved (20-40 years of age), probably due to their more ambulant nature.[26,27] A study which was done by Bhat *et al*., had also noted that 80% of the cases occurred in this age group[28]. In our study, most of the victims who developed were bitten on the lower limbs (72.4%). A similar observation was reported in a study which was done by Viramani *et al*. [26] Majority of the snake bites occurred between 6 am to 10 pm, i.e., during working hours in the field. As expected, the snake bites more commonly involving lower limbs. The incidence of AKI in the present study was 32.1% which was found to be relatively high when compared to the other studies similar studies from India [29-31]; whereas the prevalence of acute renal failure in snake bite in studies from Nigeria, Israel, Thailand, and South-east Anatolia has been found to be 1-10%, 6.2%, 5%, and 8%, respectively [32-34]. A study done by Tushar B. Patil, at Nagpur found the incidence of AKI among snakebite patients as 22% [35].

Almost all of the patients with AKI had local cellulitis, indicating the vasculotoxic nature of envenomation. In viperine bites, the earliest symptom is development of pain and swelling due to cellulitis, which can spread over whole extremity, and can also lead to compartment syndrome threatening the viability of the limb or its part [36]. This can have important consequences if it leads to loss of digits due to ischemia and gangrene. The other common symptoms were oliguria (51.9%), edema, which are consequences of renal failure in this patients. 8.3%patients had hypotension, and other bleeding manifestations were present in 12.2% patients. Similar figures have been reported previously also [37].

Cellulitis was one of the independent risk factors which was related to the development of AKI in our study. The earliest symptoms which are seen in the patients of viper bite are pain and swelling at the bitten part. Regional lymphadenopathy was another significant independent factor which contributed to the development of AKI. Like cellulitis, gangrene at the bite area and regional lymphadenopathy can be bedside indicators of the amount of toxin which is released by the snake bite.

In our study, 100% patients who suffered from AKI, developed bleeding manifestations, which was higher when compared to the study conducted by Chugh K.S (60-65%) [38]. The mortality rate in our study was 13.7 % (18 patients) and among AKI patients it was 30.2%. This was almost in par with other studies which were conducted in India (22-50%) [39]. The patients who had recovered from AKI had a shorter bite to needle time, as was observed by Sharma *et al*. [31] The mortality due to snake bite can be prevented by intervention at various levels, which include early transfer of the patient to a primary health care facility, where ASV should be administered at the earliest. The high risk patients should be identified early and referred to higher canter. Patient's fluid status should be optimised, with early detection and treatment of coagulopathy. Further, renal replacement therapy should be initiated at the earliest to prevent serious consequences of uremia.

Conclusion

The present study concludes that the incidence ofAKI among the snake bite patients was 32.1%. The clinical manifestations such as cellulitis, lymphadenopathy, oliguria and bleeding manifestations were having a significant association among patients with AKI. The ASV therapy time, bite to renal insufficiency time and coagulation abnormalities were the major prognostic factors predicting the final outcomes. The overall mortality of snake bite induced acute renal failure is 30.2%. Snake envenomation leading to acute kidney injury is a major health issue due to delayed referral to appropriate care centre. Early referral to specified care centre and prompt administration of ASV can reduce mortality and morbidity due to snake bite leading on to AKI.

References

1. Kasturiratne A, Wickremasinghe AR, de Silva N, Gunawardena NK, Pathmeswaran A, Premaratn, R, *et al*. The global burden of snakebite: a literature analysis and modelling based on regional estimates of envenoming and deaths. PLoS Med. 2008; 5(11):e218.
2. Warrell DA. WHO/SEARO Guidelines for the clinical management of snakebite in the Southeast Asian Region. SE Asian J Trop Med Pub Hlth. 1999; 30:1-85.
3. Warrell DA. Injuries, envenoming, poisoning, and allergic reactions caused by animal. In: Warrell DA, Cox TN, Firth JD, Benz J Jr, editors. Oxford Textbook of Medicine. Oxford: Oxford University Press, 2003, 923-45.
4. Al-Homrany MA. Acute renal failure due to snake-bite: clinical aspects. Saudi J Kidney Dis Transpl. 1998; 9(3):285-289.
5. Simpson ID, Norris RL. Snakes of medical importancein India: is the concept of the "Big 4" still relevant and useful? Wilderness Environ Med. 2007; 18(1):2-9.
6. Saini RK, Sharma S, Singh S, Pathania NS. Snake bite poisoning: a preliminary report. J Assoc Physicians India. 1984; 32(2):195-197.
7. Chugh KS. Snake‐bite‐induced acute renal failure in India. Kidney Int. 1989; 35:891-907.
8. Jha V, Chugh KS. Community‐acquired acute kidney injury inAsia. Semin Nephrol. 2008; 28:330-47.
9. Pinho FM, Yu L, Burdmann EA. Snakebite‐induced acute kidneyinjury in Latin America. Semin Nephrol. 2008; 28:354-62.
10. Kanjanabuch T, Sitprija V. Snakebite nephrotoxicity in Asia. SeminNephrol. 2008;28:363-72
11. Burdmann EA, Jha V, Sitprija V. Acute kidney injury in the tropics.Comprehensive Clinical Nephrology. 4th ed., Ch. 67. St. Louis, Missouri: Saunders Elsevier, 2011, 813-20.
12. Mukhopadhyay S, Ghosh A, Kar M. Methylglyoxal increase inuremia with special reference to snakebite‐mediated acute renalfailure. Clin Chim Acta. 2008; 391:13-7.
13. Miyata T, van Ypersele de Strihou C, Kurokawa K,

Baynes JW. Alterations in nonenzymatic biochemistry in uremia: Origin andsignificance of carbonyl stress in long‑term uremic complications. Kidney Int. 1999; 55:389-99.
14. Silveira PV, Nishioka S, De A. South American rattlesnake bite in a Brazilianteaching hospital. Clinical and epidemiological study of 87 cases, withanalysis of factors predictive of renal failure. Trans R Soc Trop Med Hyg.1992; 86:562-4.
15. NishiokaSde A, Silveira PV. A clinical and epidemiologic study of 292 cases oflance-headed viper bite in a Brazilian teaching hospital. Am J Trop Med Hyg.1992; 47:805-10.
16. Pinho FM, Zanetta DM, Burdmann EA. Acute renal failure after Crotalusdurissussnakebite: a prospective survey on 100 patients. Kidney Int. 2005; 67:659-67.
17. Athappan G, Balaji MV, Navaneethan U, Thirumalikolundusubramanian P. Acute renal failure in snake envenomation: a large prospective study. Saudi J Kidney Dis Transpl. 2008; 19:404-10.
18. Paul J, Dasgupta S. Early prediction of acute kidney injury by clinicalfeatures of snakebite patients at the time of hospital admission. N Am J Med Sci. 2012; 4:216-20.
19. Dharod MV, Patil TB, Deshpande AS, Gulhane RV, Patil MB, Bansod YV. Clinical predictors of acute kidney injury following snake bite envenomation. N Am J Med Sci. 2013; 5:594-9.
20. Mittal BV. Acute renal failure following poisonous snake bite. J Postgrad Med. 1994; 40(3):123-26.
21. Mehta RL, Kellum JA, Shah SV. Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. Crit Care. 2007; 11:R31
22. Kohli HS, Sakhuja V. Snake bites and acute renal failure. Saudi J Kidney Dis Transpl. 2003; 14(2):165-76.
23. Sitprija V, Suvanpha R, Pochanugool C, Chusil S, Tungsanga K. Acute interstitial nephritis in snake bite. Am J Trop Med Hyg. 1982; 31:408-10.
24. Punde DP. Management of snake-bite in rural Maharashtra: a 10- year experience. Natl Med J India. 2005; 18:71-75.
25. Hati AK, Mandal M, De MK, Mukherjee H, Hati RN. The epidemiology of snake bites in the district of Burdwan, West Bengal. J Indian Med Assoc. 1992; 90(6):145-47.
26. Viramani SK, Dutt OP. A profile of snake bite poisoning in Jammu region. J Indian Med Assoc. 1987; 85:132 -34.
27. Sharma SK, Chappuis F, Jha N, Bovier PA, Loutan L, Koirala S. The impact of snake bites and the determinants of the fatal outcomes in southeastern Nepal. Am. J Trop. Med. Hyg. 2004; 71(2):234-38.
28. Bhat RN. Viperine snake bite poisoning in Jammu. J Indian Med Assoc. 1974; 63:383-92.
29. Basu J, Majumdar G, Dutta A, Sengupta SK, Kundu B, Dass S, Neelakantan C, *et al*. Acute renal failure following snake bite (viper). J Assoc Physicians India. 1977; 25(12):883-890.
30. Athappan G, Balaji MV, Navaneethan U, Thirumalikolundusubramanian P. Acute renal failure in snake envenomation: a large prospective study. Saudi J Kidney Dis Transpl. 2008; 19(3):404-410.
31. Sharma N, Chauhan S, Faruqi S, Bhat P, Varma S. Snake envenomation in a north Indian hospital. Emerg Med J. 2005; 22(2):118-120.
32. Efrati P, Reif L. Clinical and pathological observations of sixty-five cases of viper bite in Israel. Am J Trop Med Hyg. 1953; 2(6):1085-1108.
33. Sitprija V, Boonpucknavig V. Snake venoms and nephrotoxicity, In: eds Lee CY. Berlin, Springer Verlag. Snake Venoms 1979, 1951-1953
34. Danis R, Ozmen S, Celen MK, Akin D, Ayaz C, Yazanel O. Snakebite-induced acute kidney injury: data from Southeast Anatolia. Ren Fail. 2008; 30(1):51-55.
35. Tushar B, Patila B, Yogendra V, Bansoda, Mangesh B Patil. Snake Bite Induced Acute Renal Failure: A Study of Clinical Profile and Predictors of Poor Outcome.World J Nephrol Urol. 2012; 1(2-3):59-65.
36. Ahmed SM, Ahmed M, Nadeem A, Mahajan J, Choudhary A, Pal J. Emergency treatment of a snake bite: Pearls from literature. J Emerg Trauma Shock. 2008; 1:97-105.
37. Mahmood K, Naqvi IH, Talib A, Salkeen S, Abbasi B, Akhter T, *et al*. Clinical course and outcome of snake envenomation at a hospital in Karachi. Singapore Med J. 2010; 51:300.
38. Chugh KS, Pal Y, Chakravarty RN, Datta BN, Mehta R, Sakhuja V, *et al*. Acute renal failure following poisonous snake bite. Am J Kidney Dis. 1984; 4:30-8.
39. Athappan G, Balaji MV, Navaneethan U, Thirumalikolundusubramanian P. Acute Renal Failure in Snake Envenomation: A Large Prospective Study. Saudi J Kidney Dis Transpl. 2008; 19:404-10.