International Journal of Advances in Medicine
Sivakumar DK et al. Int J Adv Med. 2018 Apr;5(2):249-256
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: http://dx.doi.org/10.18203/2349-3933.ijam20180404

Original Research Article

Study of clinical profile and outcome of acute kidney injury in acute poisoning and envenomation

Siva Kumar D. K., Karthikeyan M.*

Department of Medicine, Institute of Internal Medicine, Madras Medical College, Tamil Nadu, India

Received: 06 January 2018
Accepted: 25 January 2018

***Correspondence:**
Dr. Karthikeyan M,
E-mail: drkarthikmanoharan@gmail.com

ABSTRACT

Background: Envenomation and poisonings can cause renal damage by number of mechanisms. Some of them may cause rhabdomyolysis or hemolysis, thereby leading pigment induced renal injury. Other contributory factors like shock, sepsis can also cause acute kidney injury (AKI). The study was done with the aim to evaluate the clinical profile and outcome of acute kidney injury in acute poisoning and envenomation and to find the relationship between early anti serum venom (ASV) administration and early presentation to tertiary care and outcome.
Methods: This prospective observational study carried out on 50 patients with history of envenomation and poisoning after meeting the requirements of inclusion criteria. History, examination findings and investigations results were collected and analysed.
Results: The incidence of AKI in envenomation and poisoning patients was 5.62%. Majority of the toxin induced AKI were due to the poisoning constitutes about 62%. Among them, paraquat (n=15) was the most common poison. snake bites were the commonest to cause AKI in the envenomation group (n=17). The average time between the event and arrival to hospital was 31 hours. Whereas in case of died patients, the average time between the event and arrival to hospital was about 59 hours. The mean time interval between poison consumption to ASV administration in recovered cases was 6.6 hours and in death cases it was 15 hours. Dialysis requirement was in about 43 (86%) patients. Of them 37 patients underwent hemodialysis (HD). 6 patients underwent peritoneal dialysis (PD). Total number of deaths in the study was 26 and the most common cause was respiratory failure (38.5%).
Conclusion: The present study suggests the most common cause of AKI in case of envenomation was snake bite and in case of toxin it was paraquat poisoning. Hence it is necessary to take initiative by the government to increase the facilities in primary health care centers to save the lives of the affected people and to impose restrictions on the availability of poisonous substances in the market.

Keywords: Acute kidney injury, Chemical poisoning, Snake venom

INTRODUCTION

Acute kidney injury (AKI) develops in many of the patients admitted with history of envenomation and poisoning. Multiple factors are in operation in the pathogenesis of renal dysfunction in these patients.

Snake envenomation is a significant public health problem in India.[1] Nephrotoxic snake bites, scorpion stings and wasp stings have been known to produce AKI in the group of envenomation. In snake envenomation, delayed presentation to health care, delayed administration of ASV and incomplete dosages are the main factors in predicting the outcome of AKI. AKI associated with bites and stings has a better prognosis than AKI associated with chemical poisoning. Among the poisons, Paraquat, rat killer paste, copper sulphate, hair dye, toxic chemicals and organophosphorus compounds

can cause renal failure.[4] In addition to the above, many poisonous plant substances can cause multiple organ dysfunction Syndrome in which renal failure is also one of the components.[5]

AKI can also develop in patients admitted with history of envenomation and poisoning because of sepsis and prerenal etiology. AKI is an important cause of morbidity and mortality in these groups of patients. AKI increase the incidence of infections in these groups of patients by catheter related sepsis and its attendant risks. In many patients, it can prolong the hospital stay and it causes a lot of economic burden on healthcare.

This study was undertaken to evaluate the clinical profile and outcome of acute kidney injury in acute poisoning and envenomation, to find the incidence of AKI in snake bites and poisons, to find the relationship between early anti serum venom (ASV) administration and early presentation to tertiary care and outcome.

METHODS

This prospective observational study was carried out in the Institute of Internal medicine, Madras medical college and Rajiv Gandhi Government General Hospital, Chennai, Tamil Nadu for a period of 6 months from February 2017 to July 2017.

A total number of 1210 patients admitted with history of poisoning and envenomation, were selected for the study. Of them 1051 patients were admitted with different forms of poisoning and 159 had history of envenomation. Total number of patients who developed AKI during the study period was 68. A total of 50 patients were selected according to the inclusion and exclusion criteria.

All patients admitted with history of envenomation and intake of poison was included in the study. Patients with diabetes mellitus, hypertension, prior renal disease and patients with history of chronic NSAIDS intake were excluded from the study.

At the time of admission, a detailed history was obtained from all the patients about the envenomation, time of bite, time of ASV administration, or about the mode of poisoning, amount of poison consumed, time interval between the event and admission in hospital and then, a clinical examination was performed at the bedside. Investigations included complete blood count, renal function test, liver function tests, electrolytes, whole blood clotting time, urine for hemoglobin/myoglobin and ultrasonogram of abdomen were done in all patients.

All the patients were managed according to the national snake bite management protocols.

In this study, the distribution of age, sex, changes and outcome in the patients with poisoning and envenomation and their correlation with incidence of AKI was studied.

Statistical analysis

The data was analysed using SPSS software. Pearsons correlation coefficient and p value were calculated to find the statistical significance. Variables were considered to be significant if p value <0.05. To compare three mean values, one-way ANOVA is applied followed by either Tukey's HSD post hoc tests for multiple pairwise comparisons. To compare two mean values, independent samples t-test was applied. To compare proportions between study and control groups, Chi-Square test was applied, if any expected cell frequency is less than five then Fisher's exact test was used. To analyze the data, SPSS (IBM SPSS Statistics for Windows, Version 22.0, Armonk, NY: IBM Corp. Released 2013) was used. Significance level is fixed as 5% (α=0.05).

RESULTS

Table 1: demographic data and clinical characteristics of the study participants.

Variables	Number of patients (n=50)	%
Age (in years)		
1 - 20	4	8.0
21 - 40	29	58.0
41 - 60	14	28.0
>60	3	6.0
Sex		
Male	39	78.0
Female	11	22.0
Bites/poisoning		
Bites	(n=19)	38.0
Snakes (species not known)	6	31.6
Snake (Russell's viper)	6	31.6
Snake (Saw scaled viper)	5	26.3
Unknown bite	1	5.3
Wasp sting	1	5.3
Poisoning	(n=31)	62.0
Cell oil	1	3.2
Copper sulphate	2	6.5
Crane killer	1	3.2
Ethylene glycol	1	3.2
OPC	1	3.2
Paraquat	15	48
Rat killer paste	6	19.4
Super vasmol	2	6.5
Unknown	1	3.2
Unknown tablets	1	3.2

A total of 50 patients were selected according to the inclusion and exclusion criteria. The incidence of AKI in envenomation and poisoning patients was 5.62%. Table 1 shows the demographic data and clinical characteristics of the study participants. Most of the AKI in poisoning and envenomation has occurred in the age group of 21-41

years. 39 (78%) patients were male and 11 (22%) were females. Majority of the toxin induced AKI were due to the poisoning. Poisoning constitutes about 62% of the AKI. Among them, paraquat (48.4%) was the most common poison. AKI due to envenomation comprised about 38% and snake bites were the commonest to cause AKI in the envenomation group.

Table 2 presents the outcome of AKI due to bites and poisoning. Out of 19 cases of envenomation, 6 cases with Russell's viper bites AKI, 4 recovered fully, whereas 2 had residual renal damage i.e., about 33%.

Among the 6 unknown snake bites causing AKI, only 3 recovered, whereas the other 3 died. Among the 5 saw scaled viper bites, all recovered fully, without any renal damage. Only one wasp sting with AKI was studied and that too fully recovered.

Among the poisoning with AKI, the predominant and the one with high death rate was paraquat poisoning. Among the 15 patients with paraquat ingestion with renal dysfunction, 11 patients (73.3%) patients died, only 26.7% patients recovered.

And among the 6 patients with rat killer paste poisoning with AKI, all 6 (100%) died. Among 2 patients with copper sulphate poisoning and AKI, both of them recovered. Among 2 patients with hair dye poisoning with AKI, one recovered and one died.

Table 2: Outcome of AKI due to bites and poisoning.

Bites and poisoning	Outcome							
	Recovered		Partially recovered		Death		Total	
Bite	**N**	**%**	**N**	**%**	**N**	**%**	**N**	**%**
Snakes (species not known)	3	50.0	0	0.0	3	50.0	6	100.0
Snake (Russell's viper)	4	66.7	2	33.3	0	0.0	6	100.0
Snake (Saw scaled viper)	5	100.0	0	0.0	0	0.0	5	100.0
Unknown bite	1	100.0	0	0.0	0	0.0	1	100.0
Wasp sting	1	100.0	0	0.0	0	0.0	1	100.0
Total	14	73.7	2	10.5	3	15.8	19	100.0
Poisoning								
Cell oil	0	0.0	0	0.0	1	100.0	1	100.0
Copper sulphate	2	100.0	0	0.0	0	0.0	2	100.0
Crane killer	0	0.0	0	0.0	1	100.0	1	100.0
Ethylene glycol	1	100.0	0	0.0	0	0.0	1	100.0
OPC	0	0.0	0	0.0	1	100.0	1	100.0
Paraquat	4	26.7	0	0.0	11	73.3	15	100.0
Rat killer paste	0	0.0	0	0.0	6	100.0	6	100.0
Super vasmol	1	50.0	0	0.0	1	50.0	2	100.0
Unknown	0	0.0	0	0.0	1	100.0	1	100.0
Unknown tablets	0	0.0	0	0.0	1	100.0	1	100.0
Total	8	25.8	0	0.0	23	74.2	31	100.0

Table 3: Relationship between time of arrival to hospital and outcome.

Outcome	N	Mean time interval btn the event and arrival to hospital	Std error	P value
Recovered	22	31.18	6.768	0.086
Partially recovered	2	65.00	55.000	
Death	26	59.65	10.098	
Total	50	47.34	6.509	

Table 4: Relationship between ASV administration time and outcome.

Outcome	N	Mean time interval btn the event and arrival to hospital	Std error	P value
Recovered	12	6.64	1.77	0.05
Partially recovered	2	4.00	1.00	
Death	3	15.00	3.768	
Total	17	0	1.734	

Among the patients with toxin induced AKI who recovered from the illness, the average time between the event and arrival to hospital was 31 hours. Whereas those who succumbed in AKI with envenomation or poisoning, the average time between the event and arrival to hospital was about 59 hours (Table 3).

The mean time interval between poison consumption to ASV administration was 15 hours in case of died patients whereas I recovered patients it was 6.36 hours and partially recovered patients it was 4 hours (Table 4).

Table 5: Relationship between quantity of paraquat poisoning and outcome.

Paraquat quantity	Outcome							
	Recovered		Partially recovered		Death		Total	
	N	%	N	%	N	%	N	%
≤20 ml	0	0.0	0	0.0	1	100.0	1	100.0
21-50 ml	3	42.9	0	0.0	4	57.1	7	100.0
>50 ml	0	0.0	0	0.0	3	100.0	3	100.0
Unknown quantity	1	25.0	0	0.0	3	75.0	4	100.0
Total	4	26.7	0	0.0	11	73.3	15	100.0

In our study, paraquat poisoning was the most common cause of toxin induced AKI. Among those who consumed paraquat, greater the volume, worse is the prognosis. None of the patients, who consumed more than 50 ml survived. In patients who have ingested between 21 to 50 ml, 4 out of 7 died (57%). This shows the high case fatality rate of paraquat poisoning (Table 5).

In our study, all the patients who developed hypotension (14) during their course of toxin induced AKI, none of them survived. In those patients, who did not develop hypotension during their course of illness, about 66% recovered and about 33% died. P value was found to be <0.01 and the difference in this relationship was found to be statistically significant.

Table 6: Relationship between hypotension and outcome.

Hypotension	Outcome								P value
	Recovered		Partially recovered		Death		Total		
	N	%	N	%	N	%	N	%	
Yes	0	0.0	0	0.0	14	100.0	14	100.0	<0.001
No	22	61.1	2	5.6	12	33.3	36	100.0	
Total	22	44.0	2	4.0	26	52.0	50	100.0	

Table 7: Relationship between peak creatinine and outcome.

Outcome	N	Mean (mg/dl)	Std. error	P-value
Baseline creatinine				
Recovered	22	2.14	0.393	0.139
Partially recovered	2	5.40	4.000	
Death	26	2.97	0.502	
Total	50	2.70	0.344	
Peak creatinine				
Recovered	22	5.00	0.541	0.002
Partially recovered	2	11.80	3.00	
Death	26	4.72	0.495	
Total	50	5.13	0.408	

Table 7 presents the relationship between laboratory parameters and outcome. Mean baseline creatinine value in 50 patients was 2.70±0.344. The difference in mean baseline creatinine value between recovered, partially recovered and death were not significant (p=0.139). The mean peak creatinine value in 50 patients was 5.13±0.408. But the difference between mean peak creatinine value between recovered, partially recovered and death was statistically significant (p=0.002).

There was no significant correlation between the initial deranged whole blood clotting time and the outcome of AKI (p=0.09). Among those patients who recovered (22), around 52% had a prolonged whole blood clotting time. Among those patients who died (26), around 39% had a prolonged whole blood clotting time (Table 8).

In our study, pigmenturia was seen in around 8% of patients. Among the 50 AKI patients studied, hemoglobinuria was found in 3 patients and myoglobinuria was found in 1 patient. Dialysis requirement was in about 43 (86%) patients. Of them 37 patients underwent hemodialysis (HD). 6 patients, underwent peritoneal dialysis (PD). The rest 7 (14%) dialysis was not done.

Table 10 presents the reasons of death in study participants. Total number of deaths was 26. The most common cause of death in our study was respiratory failure in 10 (38.5%) patients.

The next common cause of death was shock and presence of multi organ dysfunction syndrome (MODS), in about 8 patients (about 30.8%). Followed by toxic hepatitis in 6 patients, and cardiac arrest and DIC each in one case.

Hypotension was found to have a worst impact on the outcome of AKI and the relationship was found to be statistically significant.

Table 8: Relationship between initial WBCT and outcome.

Initial WBCT	Outcome								P value
	Recovered		Partially recovered		Death		Total		
	N	%	N	%	N	%	N	%	
Prolonged	12	52.2	2	8.7	9	39.1	23	100.0	0.094
Within normal	10	37.0	0	0.0	17	63.0	27	100.0	
Total	22	44.0	2	4.0	26	52.0	50	100.0	

Table 9: Presence of urine Hb/Mb in toxin induced AKI.

Urine test	Number of patients (n=50)	%
Negative	46	92.0
Hb +ve	3	6.0
Mb +ve	1	2.0
Dialysis done		
Yes	43	86
No	7	14
Type of dialysis	N=43	
HD	37	86.0
PD	6	14.0

Table 10: Cause of death in toxin induced AKI.

Cause of death	Number of patients (n=26)	%
Sudden cardiac arrest	1	3.8
Respiratory failure	10	38.5
Toxic hepatitis/MODS	6	23.1
Shock/MODS	8	30.8
DIC	1	3.8

Table 11: Discharge creatinine among the different types of AKI.

Type of renal failure	N	Mean discharge creatinine(mg/dl)	Std. error	P-Value
Oliguric	13	2.47	0.525	0.029
Non - oliguric	10	1.16	0.050	

The mean discharge creatinine in oliguric AKI in our study was 2.47 mg/dl and the mean discharge creatinine in non-oliguric AKI was found to be 1.16 mg/dl (Table 11).

DISCUSSION

A total of 50 cases of AKI in envenomation and poisoning were taken in the study. About 114 cases of snake bite occurred during the study period. Of them 19 cases of AKI were included in the study. Of them 17 cases were due to snake bite. In our study, Russell's viper was the most common species causing AKI, followed by Saw scaled viper. 16 out of 17 bites occurred in males. Similar observations were also made by Chugh and Pinho et al.[6,7] This shows males are at high risk for snake bite envenomation because of their occupational risk.[8] Almost all patients, who were studied did not have any prior renal disease or other comorbidities.

The mean time between the bite and ASV administration in those who have recovered was 6.64 hours and those who partially recovered were about 4 hours and those who died were 15 hours. This clearly shows that earlier the ASV administration, less the complications and better the outcome. The mean time between the snake bite and arrival to hospital was about 2.4 days. But in those patients who died because of snake bite with AKI, the mean time was increased to about 4.6 days. This is about double the average time in the study. This shows a timely referral is very important in managing AKI in snake bite. These observations were in accordance with the findings of Vikrant et al. In his study, mean duration of arrival at

hospital was 3.4 days.[9] Kalantri et al noted mean bite to hospital time of 6.5±10.3 hours.[10]

In our study out of 17 AKI in snake bites, hypotension occurred in only 3 patients and all three patients expired. This shows hypotension has an adverse impact on the survival of these patients. This might be due to severe blood loss due to extended whole blood clotting time caused by snake venom in patients.[11] Hence whole blood clotting time serves as a good screening test for envenomation and starting of ASV. None of the patients had pigmenturia.

In our study of 17 patients, 16 patients were treated with renal replacement therapy. One patient died before starting dialysis. The average cycles of hemodialysis requirement in snake bite was about 4.37 cycles. 3 patients expired because of snake bite AKI. In all these bites, the species of snake was not known. Out of the 6 bites caused by Russell's viper, 2 recovered only partially. The biopsy findings in these two patients were thrombotic micro angiopathy in one patient and cortical necrosis in another patient. Cortical necrosis in renal biopsy indicates poor prognosis and it is classically produced by the bite of Russell's viper. Similar findings were done by Vikrant et al.[9] Delay in administration of ASV might be the reason for the development of renal lesions.[12]

In this study, out of 19 patients with bite, wasp sting bite was seen in one patient and presented with renal failure with MODS. There was no hypotension and his WBCT was normal. His urine was positive for myoglobin. He required only one cycle of HD and he recovered. Unknown bite in one patient presented with renal failure that is non-oliguric. There was no hemodynamic instability and whole blood clotting screen was normal. His urine was negative for pigments and he recovered without renal replacement therapy.

In our study, 15 cases of paraquat poisoning with AKI was studied. This constitutes about 48.4% of the AKI in the poisoning group. Sex distribution showed 10 cases in males and 5 cases in females. Most of them were in the age group of 20-40 years. Except one patient, none of them had prior comorbidities or renal disease. The average time between the paraquat ingestion and arrival to MMC was about 35 hours. Hypotension was present in 2 patients and both of them expired. Whole blood clotting time was normal in all patients. None of them had pigmenturia. The technique of preemptive charcoal hemoperfusion was not adopted in our study. This is because most of the patients presented very late after the onset of renal failure. All of them were treated with hemodialysis. According to Cavalli et al, the survival rate in paraquat poisoning can be increased to 50% by hemoperfusion.[13] Out of the 15 patients with paraquat ingestion with AKI, 11 patients (73%) died. Our study once again proves the high case fatality rate of paraquat ingestion as reported in earlier studies.[14] Only 4 patients (26%) survived in our study. The most common cause of death in paraquat poisoning was respiratory failure in our study. We tried cyclophosphamide and dexamethasone in our patients to prevent lung injury, inspite of that many patients developed respiratory complications. But in a study by Banupriya et al, the main cause of mortality was due to MODS.[15]

In the present study, rat killer paste poisoning with AKI constituted about 6 (19%) of the cases among the poisoning group. Sex distribution showed a male predominance of 83% (5 out of 6 cases). None of them had prior comorbidities or renal disease. All the 6 patients of rat killer paste poison with AKI died in our study (100% mortality rate). This shows presence of AKI in rat killer paste poisoning may carry a worst prognosis. Out of 6 patients, 4 patients had hypotension (66%). All 6 patients had a deranged coagulation profile during the clinical course. Pigmenturia was present in none of the patients. Out of 6 patients, 4 were supported with renal replacement therapy. Peritoneal dialysis was done in 3 patients and hemodialysis was done in 1 patient. Common cause of death in rat killer paste poison with AKI in our study was toxic hepatitis, leading to fulminant liver failure with MODS. Liver transplantation was not done for our patients with liver failure. These observations were almost similar to the studies of Jegan et al.[16]

The next common causes of AKI in poisoning group were copper sulphate, super vasmol poisoning, cell oil poisoning, ethylene glycol, OPC, crane killer poisoning and unknown poison. Multiple factors were responsible for AKI in these poisons. Pigmenturia was found in copper sulphate poisoning and unknown poisoning. Many patients were hemodynamically unstable and multiple organ dysfunction were also present.

In the present study, 2 cases of copper sulphate poisoning with AKI were studied. Both of them presented with anemia and non-oliguric type of renal failure. There was evidence for intravascular hemolysis. SGOT/SGOT was mildly increased in both the cases. In one patient, anemia was severe enough to cause transfusion of 2 unit of packed RBCs. These observations are in agreement with the studies of Naqvi.[4]

In our study, 2 cases of supervasmol poisoning with AKI were studied. Among them, one patient died presented with shock and multi organ dysfunction was documented in the form of increased SGOT/SGPT, AKI, and respiratory failure. Similar findings were also noted by Ramulu in his study on 31 patients of alleged hair dye ingestion.[17]

In our study, one case of cell oil poisoning with AKI was studied. He was hemodynamically unstable at the time of presentation. He also had a multi organ dysfunction with transaminitis and respiratory failure and he succumbed to his illness before starting renal replacement therapy.

In this study, 2 cases of pesticide poisoning with AKI were studied, one due to organophosphorus compound and other due to crane killer poisoning. Both of these patients had hypotension and peritoneal dialysis was done for the renal failure. Inspite of the supportive measures, both the patients expired. The exact cause of renal failure in our patients are not known, as we didn't do biopsy in both of these patients. There have been case reports of acute renal failure after inhalation of organophosphates in the literature and the suspected mechanism is renal tubular injury.[18] But in general, acute kidney injury following organophosphorus compounds ingestion is extremely rare, until there are other causes like hypotension and sepsis, because of prolonged mechanical ventilation. Similarly, crane killer poisoning (Carbamates) causing AKI and death is also a rare event.

In our study, one patient with ethylene glycol poisoning with AKI was studied. The exact quantity of the poison was not known.

His coagulation profile and blood pressure was normal. SGOT/SGPT was increased. He had renal failure, for which one cycle of HD was done and recovered with supportive measures. These findings supports the statement that even a small amount of ethylene glycol will have toxic effects on the kidney.[19]

The mortality rate in the present study was 52% which was higher than the previous studies.[20,21] This increased incidence of higher mortality rate can be attributed to late presentation of patients in the hospital.

Strengths

- Since the study included both poisoning and envenomation patients, it gives a holistic picture of patients, with toxin induced acute kidney injury.
- Strict protocols were followed for both snake bite and poisoning management, according to the national and international guidelines.

Limitations

- Since the study period is only 6 months and only 50 cases of AKI have been studied, the study may not be representative of the entire spectrum of toxin induced AKI.
- Renal biopsy was not included in the study, since it was done routinely for majority of patients at our centre.
- Among the poisons, the study has been dominated by paraquat and rat-killer paste. Hence the outcome and other results, may not be uniformly applicable to the entire poisoning induced AKI.
- Preemptive charcoal hemoperfusion, was not attempted in paraquat poisoning in our study, since many patients presented very late after the onset of renal failure.

Recommendations

- National snake bite protocols should be followed in each and every case of snake bite.
- Primary care physicians should be trained in snake bite management and first aid care for poisoning patients.
- A uniform referral protocols should be formulated and to be followed at the primary health care level.
- People living in the rural areas should be educated about the ill effects of snake bite and the need to present to a nearby health centre, immediately after a snake bite.
- Herbicides like paraquat and rat killer paste, which has a very high case fatality rate, should be banned and suitable alternatives to be discovered.
- Stress management clinics and counseling centres to be set up in all tertiary care hospitals, to counsel people who have difficulty in coping up with the family and day-to-day stress.

CONCLUSION

The results of the study demonstrated that most of the poisoning cases associated with AKI were involved in the age group of 40-60 years particularly of males due to their risks involved in their occupation. Snake bite and paraquat poisoning contributes to the most common causes of incidence of AKI. Hence it is essential to take the initiatives by the government to provide ASV and necessary facilities even in primary health care centers in all areas to prevent deaths caused by snake bite and to impose restrictions on the availability of poisonous substances in the market.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the institutional ethics committee

REFERENCES

1. Mohapatra B, Warrell DA, Suraweera W, Bhatia P, Dhingra N, Jotkar RM, et al. Million Death Study Collaborators. Snakebite mortality in India: a nationally representative mortality survey. PLoS Negl Trop Dis. 2011;5(4):e1018.
2. Rachaiah NM, Jayappagowda LA, Siddabyrappa HB, Bharath VK. Unusual case of acute renal failure following multiple wasp stings. N Am J Med Sci. 2012;4:104-6.
3. Deshpande PR, Ahsan Farooq KK, Bairy M, Prabhu RA. Acute renal failure and/or rhabdomyolysis due to multiple bee stings: A retrospective study. N Am J Med Sci. 2013;5:235-9.
4. Naqvi R. Acute kidney injury from different poisonous substances. World J Nephrol. 2017;6(3):162-7.
5. Smal KK, Sahu HK, Kar MK, Palit SK, Kar BC, Sahu CS. Yellow oleander (cerbera thevetia)

poisoning with jaundice and renal failure. J Assoc Physicians India. 1989;37:232-3.
6. Chugh KS. Snake-bite-induced acute renal failure in India. Kidney Int. 1989;35:891–907.
7. Pinho FM, Zanetta DM, Burdmann EA. Acute renal failure after Crotalus durissus snakebite: a prospective survey on 100 patients. Kidney Int. 2005;67:659-67.
8. Warrel DA. World Health Organisation; WHO Library Cataloguing-in-Publication data (NLM classification: WD 410). Guidelines for the management of snake-bites. 2010:1-150.
9. Vikrant S, Jaryal A, Parashar A. Clinicopathological spectrum of snake bite-induced acute kidney injury from India. World J Nephrol. 2017;6(3):150-61.
10. Kalantri S, Singh A, Joshi R, Malamba S, Ho C, Ezoua J, et al. Clinical predictors of in-hospital mortality in patients with snake bite: A retrospective study from a rural hospital in central India. Trop Med Int Health. 2006;11:22-30.
11. 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.
12. Chugh KS, Pal Y, Chakravarty RN, Datta BN, Mehta R, Sakhuja V, Mandal AK, Sommers SC. Acute renal failure following poisonous snakebite. Am J Kidney Dis. 1984;4:30-8.
13. Cavalli RD, Fletcher K. An effective treatment for paraquat poisoning. In: Biochemical mechanisms of paraquat toxicity. Author AP, editor. New York: Academic Press; 1977: 213-34.
14. Singh S, Bambery P, Chaudhry D, Makharia G, Kakkar N, Singh D. Fatal paraquat poisoning: report of two cases. J Assoc Phys Ind. 1999;47(8):831-2.
15. Banupriya M, Mahendrakumar K, Karthik PS, Kannan R. A study on clinical profile of paraquat poisoning in a tertiary care hospital. Int J Adv Med. 2017;4:1088-91.
16. Jegan A. Study of clinical profile and outcome of AKI in acute poisoning and envenomation. Ddissertation submitted to Madras Medical College, Tamil Nadu, India. 2014:1-60.
17. Ramulu P, Rao PA, Swaroop KK, Marx PK, Devi CVR. A prospective study on clinical profile and incidence of acute kidney injury due to hair dye poisoning. Int J Res Med Sci. 2016;4:5277-82.
18. Zafar R, Munawar K, Nasrullah A, Haq S, Ghazanfar H, Sheikh AB, Khan AY. Acute Renal Failure due to Organophosphate Poisoning: A Case Report. Cureus. 2017;9(7):e1523.
19. Tadokoro M, Ozono Y, Hara K, Taguchi T, Harada T, Ideguchi M, Senju M. A case of acute renal failure due to ethylene glycol intoxication. Nihon Jinzo Gakkai Shi. 1995;37(6):353-6.
20. Chan YC, Fung HT, Lee CK, Tsui SH, Ngan HK, Sy MY, et al. A prospective epidemiological study of acute poisoning in Hong Kong. Hong Kong J Emerg Med. 2005;12:156-61.
21. Kumar SV, Venkateswarlu B, Sasikala M, Kumar GV. A study on poisoning cases in a tertiary care hospital. J Nat Sci Biol Med. 2010;1:35-9.

Cite this article as: Sivakumar DK, Karthikeyan M. Study of clinical profile and outcome of acute kidney injury in acute poisoning and envenomation. Int J Adv Med 2018;5:249-56.