Original Paper

Snakebite Envenomation – Clinical Profile, Anti-snake Venom Therapy and Complications

Sreekrishnan TP*, Ajith V**, Sabareesh B*, Bharath Prasad*, Ajith Kumar J*, Naveen Mohan*, Priya R Menon*, Pillay VV***, Gireesh Kumar KP*

ABSTRACT

Snakebite is an environmental and occupational hazard in India. Due to varying clinical presentations, a study on snakebite cases was undertaken with a syndromic approach as described in World Health Organization (WHO) guidelines to verify the outcome and response to anti-snake venom (ASV). The aim was to standardize snakebite treatment in order to help manage these patients more effectively to reduce morbidity and mortality.

A series of 54 patients of snake envenomation were observed and medically intervened.

History, clinical examination, 20 minutes whole blood clotting time (WBCT), serial monitoring of prothrombin time (PT), international normalized ratio (INR), and renal function tests (RFT) were monitored. Forty two (77.8%) patients had signs of local envenomation, 24 (44%), 3 (5%), 2 (3%) and 2 (3%) patients had haemotoxic, extending signs of local envenomation, neurotoxic and combined reaction, respectively. Only 8 (14.8%) had 20 min WBCT positive compared to 18 (33.3%) positive for PT/INR at 0 hour, even though 31 (57%) had signs of envenomation.

ASV was given to 31 patients, 8 developed anaphylaxis, and 2 had anaphylatic shock, all of whom recovered with standard treatment. Five patients had previous atopy/ asthma, and were premedicated with antihistamine and steroids, they did not show any reaction to ASV.

This study shows that a syndromic approach is an effective tool for managing snake envenomation. Serial monitoring of PT/INR (4–6 hourly) appears to be a better option than bleeding time (BT) or clotting time (CT). We did not encounter ASV-induced anaphylaxis very frequently.

Key Words: Snakebite; Anti-snake venom; ASV; Anaphylaxis; Whole blood clotting time; WBCT; Renal function tests; RFT; Bleeding time; BT; Clotting time; CT; Prothrombin time; PT; International normalized ratio; INR

INTRODUCTION

Snakebite is an environmental health hazard in rural and urban areas of many South East Asian countries including India. The epidemiology of snakebites in these regions is not adequately studied, a primary reason for which is that snakebite victims are treated by traditional healers. According to the most conservative estimates, at least 81,000 snake envenoming and 11,000 fatalities occur in India each year, making it the most heavily affected country in the world.[1]

The risk of envenomation after bites by venomous snakes varies with species but is on an average only about 50%. Bites in which fangs pierce the skin but there are no features of envenomation are known as dry bites.

Even though there are a large number of venomous and non-venomous snakes in India, specific treatment by way of anti-snake venom (ASV) is available only for the "Big Four": common cobra, common krait, saw-scaled viper and Russell's viper. So identification of snakes is not very important in managing snakebite victims. Evolutionary

*Dept of Emergency Medicine, Amrita Institute of Medical Sciences & Research, Cochin.
**(*Author for correspondence*): Dept of Emergency Medicine, Amrita Institute of Medical Sciences & Research, Cochin, Kerala. E-mail: ajith.v123@gmail.com.
***Poison Control Centre, Amrita Institute of Medical Sciences & Research, Cochin, Kerala.

mimicry of venomous by non-venomous species further complicates accurate identification. For example, in Kerala, in several cases the harmless snake, Indian wolf snake (*Lycodon aulicus*) is confused with the venomous common krait (*Bungarus caeruleus*). However, a syndromic approach can help differentiate between venomous and non-venomous bite, or even a dry bite.[2,3]

Some people who are bitten or suspect or imagine that they have been bitten by snakes, may develop quite striking clinical features even when no venom has even been injected. This may range from tingling sensations, stiffness of the limbs, and vomiting to vasovagal shock and collapse. One should understand that the syndromes (**Table 1**) are more important than recognizing the snake because some snakebites are dry bites, some are non-venomous, and some are really venomous, and if the clinical features correspond to any of the major syndrome groups, only then will there be response to ASV. However, it is important to note that the venom in different related species will have some cross-relationship and these may also respond to treatment with ASV.

Snake venom consists of various enzymes, including zinc metalloproteinase haemorrhagicants and procoagulant enzymes. These enzymes stimulate blood clotting with formation of fibrin which can lead to disseminated intravascular coagulopathy or consumption coagulopathy. Prothrombin time (PT) is a simple test by which we can detect the defect in extrinsic pathway of coagulation cascade. PT will be prolonged if the plasma levels of any of the requisite factors is below 10% of normal, and it is relatively more sensitive and can be prolonged even before the onset of clinical manifestations, whereas 20 minutes whole blood clotting time (20 WBCT) is generally significantly prolonged only in severe deficiency of the various coagulation factors. Therefore PT is a better blood investigation than 20 WBCT in making an early diagnosis of haematoxic snake envenomation.

Jamrus et al in their study showed that PT with INR can be an alternative test for evaluation of coagulopathy in green pit viper bitten patients with potentially improved inter-laboratory standardisation.[4] Another study in a similar geographical area in India showed PT is a superior coagulation parameter which can detect coagulopathy earlier.[5]

The mainstay of management of snakebite envenomation is anti-snake venom (ASV), which is highly effective, though it can cause adverse reactions including anaphylaxis rarely. ASV used in India is produced from hyperimmunised equine serum which is then refined, purified and concentrated against the venoms of the "Big Four." ASV should be used in snakebite cases with features of systemic or local envenomation. Patients with symptomatic coagulopathy have to be supported with blood products, and patients with neurological features should be treated with atropine, neostigmine, mechanical ventilation and ASV.

Table 1: Snakebite syndromes

Syndrome	Clinical Features	Species	Management
Syndrome 1	Local envenomation with bleeding or clotting disturbances	Viper sp	ASV
Syndrome 2	Local envenomation with bleeding or clotting disturbances, shock, acute kidney injury	Russells viper, Saw-scaled viper	ASV
Syndrome 3	Local envenoming (swelling, etc) with paralysis	Cobra, King cobra	Atropine + Neostigmine + Mechanical ventilation + ASV
Syndrome 4	Paralysis with minimal or no local envenoming Bitten on land while sleeping on the ground	Krait	Atropine + Neostigmine + Mechanical ventilation + ASV
Syndrome 5	Paralysis with dark brown urine & acute kidney injury: Bitten on land (with bleeding/clotting disturbance) Bitten on land while sleeping indoors	Russell's viper Krait	ASV Atropine + Neostigmine+ Mechanical ventilation + ASV

Ten vials (100 ml) of polyvalent ASV is the initial quantity of ASV required. Usually more than 20% of cases will develop either early (within few hours) or late (5 days or more) allergic reactions following antivenom administration. Features of early anaphylactic reactions (10-180 min) range from itching, uricaria, dry cough, fever, nausea, vomiting, abdominal colic, diarrhoea, tachycardia to severe life threatening anaphylaxis – hypotension, bronchospasm, angioedema and even death. Pyrogenic reaction (1-2 hr) presents with chills, fever, and hypotension. Late serum sickness-type reaction (1-12 days) manifests as fever, nausea, vomiting, diarrhoea, itching, urticaria, arthralgia, myalgia, lymphadenopathy, mononeuritis multiplex, periarticular swellings, immune complex nephritis and rarely encephalopathy. Most of these reactions can be treated with antihistamines and steroids with/without adrenaline.[6] The International Collaborative Study of Severe Anaphylaxis has observed that the risk of anaphylaxis in a hospital population was highest for anti-snake venom.[7]

The purpose of this study was to find out the usefulness and efficacy of a syndromic approach towards snakebite management, and to know the clinical importance of PT/INR and its status over other coagulation parameters, as well as the effectiveness of ASV, and to assess the incidence of ASV induced anaphylaxis.

MATERIALS AND METHODS

A retrospective study was conducted on 54 patients who reported to the department of Emergency Medicine in a tertiary care hospital in Cochin from 2008 April to 2011 March with a history of snakebite. Diagnosis was based on snakebite reported by the patient or his bystanders and the clinical presentation.

Detailed history and clinical examination were done initially, followed by serial 20 min WBCT from the time of presentation to ER and for the next 6 hrs, serial monitoring of PT/INR at time of presentation to ER and 6th hourly, single breath count, renal function tests and blood counts. The patients were followed up later to look for any complications such as DIC, cellulitis, renal failure or ARDS. ASV was administered to the patients either presenting with clinical features of systemic or severe local envenomation, or laboratory findings suggestive of envenomation as per WHO guidelines for snakebite.[6] Reassessment was done after 6 hours following completion of initial administration of ASV, and further need of ASV was considered accordingly. Maximum dose of ASV given in any case was 35 vials. Patients were also monitored for development of anaphylaxis following ASV administration. Statistical analysis were done using chi-square method

RESULTS

A total of 54 patients were analysed in this study. All the cases in this study were admitted in the Dept of Emergency Medicine and managed by trained emergency medicine physicians. There was a male preponderance (65%) in the study which was similar to earlier studies.[8,9] There were 6 paediatric cases in the study. Bite mark was evident in all cases. Out of the total number of cases, 23 bites occurred in the left lower limb, 20 in the right lower limb, 6 in the right upper limb, 4 in the left upper limb, and 1 on the face. Signs of local envenomation were evident in 42 (77.8%) cases.

Out of the 54 cases, 24 patients had haemotoxic envenomation, 3 patients had extending signs of local envenomation, 2 had purely neurotoxic envenomation, and 2 patients had combined neurotoxic and haemotoxic envenomation.

A total of 8 (14.8%) patients had positive 20 min WBCT. Serial 20 min WBCT did not show any significant change. Initially, INR was elevated in 18 (33.3%) patients and serial monitoring 4th hourly of PT/INR revealed elevation in 8 (14.8%) more patients (**Fig 1).** Among the former who had PT/INR positive at 0 hr, all needed blood product transfusions along with ASV as they went into DIC.

Forty-two patients had local envenomation, 12 (22%) patients did not have progressive extension of pain and these patients did not receive ASV. Sixteen (30%) patients with PT/INR positive at 0 hr had signs of extending local envenomation and needed ASV. Two patients with purely

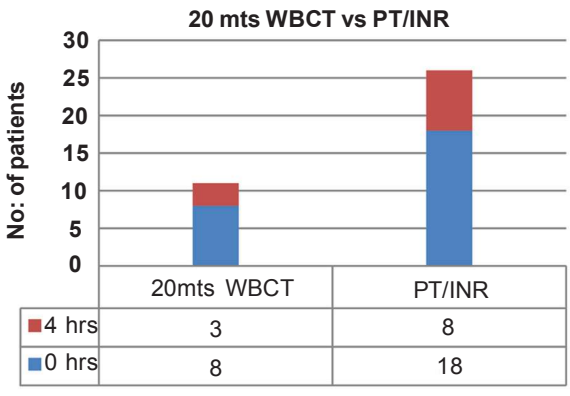

Fig 1

neurotoxic features at 0 hr had signs of extending local envenomation and were treated with atropine, neostigmine and ASV. These patients had signs of respiratory distress and were effectively treated with mechanical ventilation. Two patients had combined haemotoxic, neurotoxic and local envenomation signs and were treated effectively with ASV, atropine, neostigmine and blood products. Five patients who had PT/INR positive at 4 hr also had local signs of envenomation initially. Three patients had extending signs of local envenomation with lymphadenitis, and were treated with ASV alone. A statistical comparison between PT/INR and WBCT at the time of presentation to check for the ability of each test to detect envenomation earliest, showed a significant p value of <0.001 in case of PT/INR. The syndromic approach adopted in this study is highlighted in **Table 2**.

Renal function test (creatinine) was abnormal in 13 (24.1%) patients and all of them required haemodialysis. Anti-snake venom was administered in 31 patients (57.4%). Ten vials were required in 7 (12.96%) patients, 20 vials in 9 (16.6%) patients, 30 vials in 12 (22.22%) patients, and 31–35 vials in 3 (5.6%) patients. Of 31 patients who received ASV, 8 patients developed anaphylaxis, and 2 developed anaphylactic shock, all of whom recovered with standard treatment. Five patients had previous atopy/asthma, and were pre-medicated prior to ASV administration with antihistamine(chlopheniramine maleate) and steroid (hydrocortisone), and these patients did not show any reaction to ASV.

Blood and blood products (fresh frozen plasma, cryoprecipitate) were administered in the case of 16 (29.6%) patients. All developed local reaction and 15 patients had elevated PT/INR at 0 hr.

Of 54 patients, 29 (53.7%) had no complications, 18 patients had coagulopathy,16 had cellulitis, 13 had renal failure, 8 had acute respiratory distress syndrome, 1 patient had toxic myocarditis (patient with haemotoxic envenomation), 1 patient had acute thrombotic stroke, and 1 patient had acute respiratory failure.

Final Outcome

1. Forty-five patients (83.3%) were discharged without any disabilities.
2. Two patients were discharged with disability with hypoxic encephalopathy.
3. Seven patients died due to multi-organ dysfunction; mortality rate was 13%.

DISCUSSION

Snakebite is a neglected health hazard in tropical countries. In India the burden of morbidity and mortality due to snakebite is very high. Management of snakebite is not streamlined, even though the World Health Organization (WHO) has laid down guidelines for management of snakebite for South East Asia.

This study was aimed at providing a systematic approach in identifying, investigating and treating snakebites. All the cases included in this study were admitted under the Dept of Emergency Medicine and were managed by trained emergency medicine physicians. The protocol followed was to observe the patients and have their blood samples tested for up to 24 hours after a bite. The study emphasises the demographic patterns observed in similar studies on snakebite from South India, with regard to male predominance and site of bite.[8,9] Available literature suggests that only 5–10% of snakebite patients develop severe envenoming.[10–12]

Table 2: Syndromic approach of the study

	Syndrome	Number of Patients	Treatment	Outcome
1	Local envenomation - extending	3 (5%)	ASV	
2	Bleeding tendencies +/- local envenomation	PT/INR at 0 hr – 16 (30%) PT/INR at 4 hr - 8 (15%)	ASV + blood (15) ASV	6 deaths, 1 disability 1 death, 1 disability
3	Neurological signs +/- local envenomation	2 (3%)	Neostigmine, atropine, ASV, ventilation	Both patients survived
4	Bleeding tendencies + Neurological signs +/- local envenomation	2 (3%) (PT/INR at 0 hr + NT)	Neostigmine, atropine, ASV 2 – blood	Both patients survived

Identifying and managing this small group promptly is an important challenge. This needs continuous bedside monitoring to reassure the patient and also for constant reassessment of his clinical condition. Most patients with snakebite are unable to identify the bitten snake species, either because of their ignorance or poor visibility at night. Treating such patients on the basis of snake species would understandably be a difficult task. That is where the role of a syndromic approach for treating snakebite assumes importance.

In this study, syndromes were broadly divided into 4 main categories:

1. Local envenomation
2. Bleeding tendencies +/- local envenomation
3. Neurological signs +/- local envenomation
4. Bleeding tendencies + neurological signs +/- local envenomation

Management based on this syndromic approach yielded very good results. Local envenomation is one of the earliest sign of envenomation. 80-95% of viperid and 25-70% of elapid venoms contain enzymes, which include digestive hydrolases, proteinases, which results in damage of the vascular permeability leading to oedema, blistering, bruising and necrosis. Peptidases and hyaluronidase promote spread of venom. Elapid venom in addition contains acetylcholinesterases, which results in neuromuscular weakness, while viperid venom contains procoagulants which results in disseminated intravascular coagulation.

All these indicate that persisting pain, which extends proximally causing lymphadenitis after a bite can serve as a warning sign that some proteinacious substance (venom) has been infiltrated. We also noticed that non-venomous snakes also produce local pain, but this type of pain subsides over a period of time. Bleeding from the bite site and IV cannulation sites is also an early sign of haemotoxic envenomation. Features of drowsiness at the time of presentation may also suggest an early neurotoxic envenomation.

In our study, we managed all cases of snakebite without identifying the snake. We discovered that those patients with local signs of envenomation, which is progressive in nature and producing lymphadenitis should be managed with high suspicion of envenomation. Most patients with local signs of envenomation have high chance of developing coagulopathy, which need management with ASV and blood products including cryoprecipitate and FFP.

Haemotoxic syndrome was the commonest snakebite syndrome followed by neurotoxic and mixed envenomation. These results are very similar to other reports from different part of India.[13,14]

Neurotoxic envenomation was identified by drowsiness/ptosis/deterioration of single breath count monitored half hourly for the first 6 hrs, and then hourly for the next 18 hrs. Patients displaying features of neurotoxic envenomation needed management with neostigmine, atropine, ASV and mechanical ventilation to pre-empt the possibility of respiratory depression.

Bleeding from the bite site and IV cannulation site, microscopic haematuria, gum bleeding, petechiae or epistaxis are the earliest signs of haemotoxic envenomation. Serial monitoring of 20 min WBCT and PT/INR were done at the time of arrival of the patient to ED and serially monitored 4th hrly if the initial results were within normal limits. It has been observed that for 20 min WBCT to become positive at least 80% of the coagulation parameters need to get deranged, whereas PT/INR was able to pick up coagulopathy even if 10% parameters are deranged.[5] Hence PT/INR is more likely to help in identifying coagulopathy earlier, and this should be undertaken wherever the facility is available. Prothrombin time (PT) is the time it takes plasma to clot after addition of tissue factor. PT test evaluates the extrinsic and common pathways of the coagulation cascade. The normal range for a healthy person is 0.8–1.2. A high international normalized ratio (INR) indicates a higher risk of bleeding. Increased consumption of coagulation factors (in disseminated intravascular coagulation) may prolong the PT. Cases of positive PT/INR or those with signs of bleeding manifestations were managed with ASV (20 vials) and blood products. In such cases PT/INR was repeated after 6 hrs to check for correction of coagulopathy. This 6-hr repetition was considered necessary because the liver takes 6 hrs to generate new clotting factors. So it is possible to get a wrong misleading result if it is repeated before 6 hrs.

The syndromic approach that we evolved from this institutional study is detailed in **Table 3**.

In this study, local signs were the most common clinical presentation that we came across which resulted in complications such as cellulitis. Investigations into the oral bacterial flora of various snakes and their venom have revealed a mixture of both aerobic and anaerobic bacterial species.[15,16] Infection with these microbes should be

Table 3: Snakebite syndromes evolved in this study

	Syndrome	Assessment	Treatment
1	Local envenomation alone	Extending local sign +/- lymphadenitis	ASV
2	Bleeding tendencies +/- local envenomation	PT/INR at 0 hr (+) PT/INR at 4 hr (+)	ASV + blood products ASV
3	Neurological signs +/- local envenomation	Drowsiness/ ptosis/deterioration of single breath count monitored half hourly for first 6 hrs and then hourly for the next 18 hrs	Neostigmine + atropine + ASV, ventilation
4	Bleeding tendencies + neurological signs +/- local envenomation	PTINR at 0 hr + NT drowsiness/ptosis/deterioration of single breath count monitored half hourly for first 6 hrs and then hourly for the next 18 hrs	Neostigmine+ atropine +ASV + blood products + ventilation

managed with appropriate antibiotics and ASV. The next most common complication encountered was acute renal failure, and was attributable to tubular damage by venom, haemoglobinuria, rhabdomyolysis, hypotension and renal microthrombi formation causing acute tubular necrosis, which was reversible.[17,18]

The rationale for the use of ASV is well-defined; doses required in different envenomation situations vary greatly and are subject to severity grade. All patients who needed ASV were initially managed with 10 vials of ASV and further increased as needed.[19,20]

Respiratory failure is the most important cause of morbidity and mortality in victims of neurotoxic snakebite. Along with anticholinergics and ASV, cardiorespiratory support with mechanical ventilation remains the corner stone for the management of neurotoxic snake envenomation.[21,22]

Patients who presented with local signs of envenomation along with initial 0-hour PT/INR elevation needed more aggressive management with ASV and blood products. These patients were managed with 20–30 vials of ASV initially itself. Early institution of ASV in indicated cases is beneficial in preventing complications due to snake envenomation. But it has its own adverse effects which range from mild anaphylaxis in the form of urticaria, palpitations,and bronchospasm to full-blown anaphylactic shock. However, in our experience, anaphylaxis due to ASV is a rare phenomenon and even if it occurs can be easily managed with adrenaline, steroids and antihistamines. Pre-medication with steroids and antihistamines has helped prevent anaphylaxis even in patients with history of atopy / asthma.

Limitations: Neurotoxic envenomation cases were managed with mechanical ventilation but the number of cases of neurotoxic envenomation were relatively less in this study. As this is an ongoing study, we propose to shed more light on this topic in future.

CONCLUSION

From our study we concluded that a syndromic approach is an effective regimen for managing snake envenomation without identifying the snake. Local signs of envenomation are an earliest sign of envenomation and these cases can deteriorate rapidly. Serial monitoring of PT/INR (4^{th} hrly) appears to be a better option than 20 min WBCT in identifying haemotoxic snakebite. These cases have to be managed with ASV and blood products. Neurological and local signs assessment was done half hourly for the first 6 hrs and then hourly for the next 18 hrs Respiratory failure is the most important cause of morbidity and mortality in victims of neurotoxic snake bite. Along with anticholinergics and ASV, cardiorespiratory support with mechanical ventilation is essential for the management of neurotoxic snake envenomation. All cases of snake envenomation, identified by progressive extension of local signs or positive PT/INR or neurological signs (drowsiness/ptosis/deterioration of single breath count) needed treatment with ASV +/- blood products +/- neostigmine/mechanical ventilation. ASV induced anaphylaxis is quite rare, and even if it occurs it can be easily managed with standard protocol. Pre-medication with steroids and antihistamines can prevent anaphylaxis due to ASV.

REFERENCES

1. Kasturiratne A. Wickremasinghe AR, de Silva N, Gunawardena NK, Pathmeswaran A, et al. The global burden of snakebite: a literature analysis and modelling based on regional estimates of envenoming and deaths. 2008. In: Winkel K (ed). *PLOS Medicine* 5 (11): e218. doi:10.1371/journal.pmed.0050218. PMC2577696. PMID18986210. Retrieved 2009.06.24).
2. Simpson ID, Norris RL. Snakes of medical importance in India: Is the concept of the "Big 4" still relevant and useful? *Wilderness Environ Med* 2007;18(1): 2–9.
3. Joseph JK, Simpson ID, Menon NCS, Jose MP, Kulkarni KJ, Raghavendra GB, Warrell DA. First authenticated cases of life-threatening envenoming by the hump-nosed pit viper (*Hypnale hypnale*) in India. *Trans Royal Soc Trop Med Hyg* 2007;101(1): 85–90.
4. Pongpita J, Limpawittayakula P, Juntiang J, Akkawat B, Rojnuckarin P. The role of prothrombin time (PT) in evaluating green pit viper (*Cryptelytrops sp*) bitten patients. *Trans Royal Soc Trop Med Hyg* 2012;106(7):415–418.
5. Gireesh Kumar KP. Hemotoxic snake envenomation – prothrombin time better predictor of mortality. *Amrita J Med* 2011;7(2): 24–27.
6. Warrell DA. WHO/SEARO Guidelines for the Clinical Management of Snakebite in South-East Asia Region; 2010. New Delhi: World Health Organization.
7. International Collaborative Study of Severe Anaphylaxis. *Pharmacoepidemiol Drug Saf* 2003;12(3):195–202.
8. Sharma SK, Khanal B, Pokhrel P, Khan A, Koirala S. Snakebite - reappraisal of the situation in Eastern Nepal. *Toxicon* 2003; 41:285–289.
9. Brunda G, Sashidhar RB. Epidemiological profile of snake-bite cases from Andhra Pradesh using immunoanalytical approach. *Indian J Med Res* 2007;125:661–668.
10. Isbister GK, Currie BJ. Suspected snakebite: one year prospective study of emergency department presentations. *Emerg Med* 2003;15:160–169.
11. Hughes A. Observation of snakebite victims: Is twelve hours still necessary? *Emerg Med* 2003;15:511–517.
12. Mead HJ, Jelinek GA. Suspected snakebite in children: a study of 156 patients over 10 years. *Med J Aust* 1996;164:467–470.
13. Punde DP. Management of snake-bite in rural Maharashtra: a 10-year experience. *Natl Med J India* 2005;18:71–75.
14. Adhisivam B, Mahadevan S. Snakebite envenomation in India: a rural medical emergency. *Indian Pediatr* 2006;43:553–554.
15. Goldstein EJ, Citron DM, Gonzalez H, Russell FE, Finegold SM. Bacteriology of rattlesnake venom and implications for therapy. *J Infect Dis* 1979;140:818–821.
16. Lam KK, et al. A cross-sectional survey of snake oral bacterial flora from Hong Kong, SAR, China. *Emerg Med J* 2011;28: 107–114.
17. Mahasandana S, Rungruxsirivorn Y, Chantarangkul V. Clinical manifestations of bleeding following Russell's viper and Green pit viper bites in adults. *Southeast Asian J Trop Med Public Health* 1980;11:285–293.
18. Danis R, Ozmen S, Celen MK, Akin D, Ayaz C, Yazanel O. Snakebite-induced acute kidney injury: data from Southeast Anatolia. *Renal Failure* 2008;30:51–55.
19. Sharma N, Chauhan S, Faruqi S, Bhat P, Varma S. Snake envenomation in a north Indian hospital. *Emerg Med J* 2005;22:118–20.
20. Bawaskar HS. Snake venoms and antivenoms: critical supply issues. *J Assoc Physicians India* 2004;52:14–17.
21. Mehta SS, Kelkar PN, Parikh NN. Respiratory failure after snake bite poisoning successfully treated with prolonged artificial ventilation. *Indian J Anaesth* 1968;16:273–275.
22. Bhattacharya P, Chakraborty A. Neurotoxic snake bite with respiratory failure. *Indian J Critical Care Med* 2007;11(3):161–164.