

# ORIGINAL ARTICLE

---

## A STUDY ON THE INCIDENCE AND ETIOLOGY OF ACUTE KIDNEY INJURY IN HEMOTOXIC SNAKE ENVENOMATION CASES IN A TERTIARY CARE CENTRE

B. Purushottam Rao<sup>1</sup>, P. Siva Teja<sup>2</sup>, V. V. Satyanarayana<sup>3</sup>

### HOW TO CITE THIS ARTICLE:

B. Purushottam Rao, P. Siva Teja, V. V. Satyanarayana. "A Study on the Incidence and Etiology of Acute Kidney Injury in Hemotoxic Snake Envenomation Cases in a Tertiary Care Centre". Journal of Evidence based Medicine and Healthcare; Volume 2, Issue 9, March 02, 2015; Page: 1312-1316.

**ABSTRACT: INTRODUCTION:** Snake bite envenomation is a frequently encountered problem in tropical countries like India, especially in the rural areas of south India. Most of the victims are farmers. The World Health Organization has estimated that nearly 1,25,000 deaths occur among 2,50,000 poisonous snake bites world-wide, of which India accounts for 10,000 deaths. Acute Kidney Injury (AKI) is an important consequence of a snake bite and its proper supportive management after the anti-venom administration is of utmost importance, for a good patient outcome. **AIM AND OBJECTIVES:** To assess the incidence of Acute Kidney Injury in patients with hemotoxic snake envenomation. To assess the etiological factors involved in Acute Kidney Injury on snake bite victims. **METHODOLOGY:** This prospective study was carried out at King George Hospital, Visakhapatnam, Andhra Pradesh. It is a tertiary care government hospital, attached to Andhra Medical College, Visakhapatnam. **STUDY DESIGN:** This was a prospective and descriptive type of study. **RESULTS:** A total of 214 cases of hemotoxic snake bite were included in this study, who were admitted in the hospital from September 2012 to August 2014. The incidence of renal failure in hemotoxic snake bite is 23.3%. The probable etiology of AKI in snake bite victims is DIC in 48% of cases, direct nephrotoxicity in 24% of cases. **CONCLUSION:** This study concludes that Acute Kidney Injury occurs in 23.3% of cases of hemotoxic snake bite. DIC is the most common etiologic factor for AKI in snake bite followed by direct nephrotoxicity, hypotension, capillary leak syndrome and sepsis.

**KEYWORDS:** Envenomation,, Hemotoxic, Disseminated intravascular coagulation, Acute kidney injury.

**INTRODUCTION:** Snake bite envenomation is a frequently encountered problem in tropical countries like India, especially in the rural areas of South India. Most of the victims are farmers. Over 2,000 species of snakes are known world-wide, of which around 400 are poisonous. These snakes belong to the families, elapidae, viperidae, hydrophidae and colubridae.<sup>[1]</sup> The World Health Organization has estimated that nearly 1,25,000 deaths occur among 2,50,000 poisonous snake bites world-wide every year, of which India accounts for 10,000 deaths.<sup>[2,3]</sup> The involvement of predominantly young, healthy and the working population in rural areas which are compounded by poverty and the lack of access to health care services in these areas, signify the social and economic impact of this problem.

Acute Kidney Injury is mainly observed following bites by the snakes which belong to the viperidae group and it is seen less with sea snake bites and the bites of snakes of the colubridae group. Most of the Indians are victims of Russell's viper or *Echis carinatus*, bites, which cause

# ORIGINAL ARTICLE

---

AKI.<sup>[4,5]</sup> The only specific treatment of a poisonous snake bite is antivenom. Serotherapy must be initiated as early as possible to save the life and to avert complications, such as renal failure. A proper supportive management after the anti-venom administration is of utmost importance for a good patient outcome. Tubular necrosis and cortical necrosis are the main causes of AKI.<sup>[6]</sup> The AKI which occurs after snake bite is usually reversible, but if acute cortical necrosis occurs, it may lead to an incomplete recovery. The main cause of this unacceptable incidence of snake bite fatalities is that people try out all kinds of "bizarre remedies" initially, instead of going to the nearest hospital. The main object of this study is to highlight the incidence and etiology of acute kidney injury among the patients with hemotoxic snake envenomation.

**PATIENTS AND METHODOLOGY:** All patients admitted to the medical wards of King George Hospital, Visakhapatnam, Andhra Pradesh, with history of snake bite from September 2012 to August 2014 were included in this study. The study was a prospective one. The patients of both sexes of all age groups excluding the paediatric group were included in this study.

## **INCLUSION CRITERIA:**

1. Patients with history of snake bite with cellulitis either with bleeding diathesis or with prolonged clotting time or both.
2. Patients with history of snake bite with oliguria irrespective of the number of days since time of bite.
3. AKI was defined as an abrupt (within 48 hrs), absolute increase in the serum creatinine concentration of  $> 0.3\text{mg/dl}$  from the base line value which was measured after admission or else-where, after the snake bite, or a percentage increase in the serum creatinine concentration of  $> 50\%$  above the baseline, or oliguria of  $< 0.5\text{ ml/kg/hr}$  for  $> 6\text{ hrs}$  or serum creatinine  $> 1.5\text{ mg/dl}$  or oliguria (urine output  $< 400\text{ ml/day}$ ).<sup>[7]</sup>

## **EXCLUSION CRITERIA:**

1. Patients with history of snake bite with any neurological problems such as ptosis, respiratory failure etc.
2. All patients were subjected to ultrasonography of abdomen and were excluded if it showed bilateral small kidneys or obstructive nephropathy or loss of corticomedullary differentiation or any other significant renal pathology.
3. They were also excluded if they had previous records suggesting serum creatinine  $> 1.5\text{mg/dl}$  or if they were exposed to nephrotoxic drugs.

A clinical history taking and a complete physical examination were done in each case. The laboratory investigations included hemoglobin, total and differential leucocyte counts, platelet counts, red cell counts, peripheral smear for fragmented RBCs, fibrinogen, reticulocyte count, 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, ultrasonography of the abdomen and CT scan brain whenever needed.

## ORIGINAL ARTICLE

---

All patients received the tetanus toxoid. A 20 minute whole blood clotting test is performed to determine if any coagulopathy is present and 10 vials of ASV is administered, over 1 hr. After 6 hrs, a further coagulation test is performed & another 10 vials of ASV should be administered in the event of continued coagulation defect. Antibiotics and diuretics were administered, as indicated. Transfusion of blood and blood products were given to the indicated patients. Renal replacement therapy (either peritoneal or hemodialysis, depending upon the availability of resources) was given to 22 patients. The patients were followed up till they were discharged or till they died.

**OBSERVATION AND RESULTS:** A total of 214 cases of hemotoxic snake bite were included in this study, who were admitted to medical wards of King George Hospital during the period from September 2012 to August 2014.

The incidence of Acute Kidney Injury in hemotoxic snake bite is 23.3% and it has been provided in table I. In our study the sex wise distribution of the study subjects was clarified, based on whether they had AKI, and among males it was 34 (22.6%) and among females it was 16 (25%). It is provided on table II.

The distribution of probable etiology of AKI has been shown on table III. Out of 50 cases of AKI, 24 patients (48%) had DIC, 12 cases (24%) had direct nephrotoxicity.

**DISCUSSION:** Of the 214 patients included in this study with hemotoxic snake envenomation, AKI occurred in 50 patients. The exact pathogenesis of AKI following snake bite is not well established. However, a number of factors contribute to it, like bleeding, hypotension, circulatory collapse, intravascular hemolysis, disseminated intravascular coagulation, microangiopathic hemolytic anemia and the direct nephrotoxicity of venom.<sup>[8]</sup>

In this study, the incidence of AKI in snake bite is found to be 23.4% (50/214). A study in PGI Chandigarh by Dr. Chugh et al 1984, it was 28.6%. A study in Sardarjang Hospital in New Delhi by Banerjee and Siddiqui 1976, it was observed as 16.9%.<sup>[9,10]</sup>

The study by verma et al 1982, Mather and Raja Rathnam 1987, it was 13-32%. A study from JIPMER by Vijeth, Dutta 1997 it was observed as 32.5% (B). A study in Seth G.S. Medical College by Mitral B.V. 1994 it was 16.2%. Patil et al, noted that AKI developed in 20.48% cases of snake bite, whereas Ali et al., observed that 17% cases of viper bite were complicated by AKI.<sup>[11,12]</sup> So, the incidence of renal failure in our hospital in this study was 23.3% and when compared to other studies was neither very high nor low.

A study on Chen JB et al 1997, Factor contributing to acute renal failure following viperine bite are hemorrhage, hypotension, DIC, hemolysis and Rhabdomyolysis. Intravascular hemolysis and DIC contributed to the development of Acute Kidney Injury.<sup>[13]</sup> DIC is the important cause of renal dysfunction in most cases of ARF.<sup>[14]</sup> Coagulation and bleeding abnormalities are strongly associated with development of AKI in patients of snake bite. Sharma et al.,<sup>[15]</sup> studied 142 cases of snake bite, of which 52 were viper bites having hemostatic abnormalities. Of these 27 developed AKI. Various procoagulant enzymes are found in viper venoms which activate different steps of clothing cascade resulting in a state of DIC. Bleeding tendencies can also cause severe blood loss resulting in hypotension, further adding to renal insult or causing one when none

# ORIGINAL ARTICLE

existed. Fibrin thrombi in renal microvasculature, glomerular capillaries, microangiopathic hemolytic anemia and thrombocytopenia in patients with cortical necrosis suggests that DIC plays a major pathogenic role in snake bite induced cortical necrosis. Thus, bleeding tendencies secondary to DIC are a major factor in development of AKI in patients of snake bite, especially those involving vipers. The above studies strongly support the present study.

**CONCLUSION:** This study concludes that acute kidney injury occurs in 23.3% of victims of hemotoxic snake bite. The etiology of AKI in snake bite is multifactorial. DIC was the most common etiologic factor. Other etiologic factors are direct nephrotoxicity, circulatory failure, DIC with capillary leak syndrome and DIC with sepsis.

Total No. of cases	Renal Failure	Percentage
14	50	23.3

Table I: Incidence of renal failure in hemotoxic snake bite

Sex	Total no. of Patients	Renal Failure	
		No. of patients	Percentages
Male	150	34	22.66
Female	64	16	25
<b>Total</b>	<b>214</b>	<b>50</b>	<b>23.4</b>

Table II: Sex distribution of AKI in hemotoxic snake bite

Sl. No.	Probable Etiology	No. of cases	%
1.	DIC	24	48
2.	DIC with hypotension	8	16
3.	DIC with shock and capillary leak syndrome	4	8
4.	DIC with shock and sepsis	2	4
5.	Direct nephrotoxicity	12	24
	<b>Total</b>	<b>50</b>	<b>100</b>

Table III: Distribution of probable etiology of AKI in snake bite

## REFERENCES:

1. AI – Homrany M. Acute renal Failure following snake bite. A case report and review of the literature. Saudi J Kidney DIS transpl. 1996; 7: 309-12.
2. Basu J, Majumdar G, Dutta A, et al. Acute renal failure following snake bites (viper). J. Assoc. Physicians India. 1977. 25: 883-90.
3. Bhat RN. Viperine Snake bite poisoning in Jammu. J Indian Med Assoc. 1974. 63; 383-92.
4. Chippaux JP. Snake bites: appraisal of the global situation. Bull World Health Org. 1998; 76(5): 515-24.

## ORIGINAL ARTICLE

5. Chugh HS, Pal Y, Chakravarthy RN, et al., Acute renal failure following poisonous snake bite. AMJ Kidney Dis. 1984; 4 (1): 30-38.
6. Albright RL Jr. Acute renal failure: A practical update. Mayochn Proc. 2001; 76(1): 67-74.
7. Norris RL, Averbach. PS. Disorders caused by reptile bites and marine animal exposure, Harrison's Principles of Internal medicine 15<sup>th</sup> ed. Mc. Graw-Hill: 2005.
8. Mittal BV. Acute renal failure following poisonous snake bite. J Post grad. Med. 1994: 40(3): 123-26.
9. Norris RL, Helling JA, Shah SV, Molitoris BA, Ronco C, Warnock DG, Levin A. Acute Kidney Injury Network: report of an initiative to improve out come in acute kidney injury. Crit care. 2007: 11(2): R31.
10. Myint – Lwin, Warrell DA, Phillips PS RE, et al. The bites of Russell's viper in Burma: Haemostatic, vascular and renal disturbances and response to the treatment. Lancet. 1985; 2: 1259 – 64.
11. Punde DP. Management of snake bite in rural Maharashtra: a 10- year experience. Natl Med. J India 2005: 18: 71-75.
12. Ali G, Kak M, Kumar M, Bali SK, Tak SI et al., Acute renal failure following echis carinatus (Saw-scaled viper) envenomation. Indian J Nephrology 2004: 14: 177-81.
13. Reid HA. Animal Poisons, in Manson S Tropical Diseases. 18<sup>th</sup> ed. In: Manson – Bahr PE, Apted FI, (eds.) London, Balliere Tindall. 1982; 544-66.
14. Athappan G, Balaji MV, Navaneethan V, Thiru Malikolundu Subramanian P. Acute renal failure in snake envenomation: A large prospective study. Saudi J Kidney Dis. Transpl. 2008: 19(3): 404-10.
15. Sharma SK, Koirala S, Dahal G, Sahl. The Clinico- epidemiological features of snake bite: A study from eastern Nepal. Trop. Doct. 2004 Jan: 34(1): 20-22.

### **AUTHORS:**

1. B. Purushottam Rao
2. P. Siva Teja
3. V. V. Satyanarayana

### **PARTICULARS OF CONTRIBUTORS:**

1. Associate Professor, Department of Medicine, Andhra Medical College.
2. Post Graduate, Department of Medicine, Andhra Medical College.
3. Professor, Department of Medicine, Andhra Medical College.

### **NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:**

Dr. B. Purushottam Rao,  
# 44, Dasapalla Hills,  
Visakhapatnam-530003,  
Andhra Pradesh.  
E-mail: purushottamr@ymail.com  
rlgujju832@gmail.com

Date of Submission: 07/02/2015.  
Date of Peer Review: 08/02/2015.  
Date of Acceptance: 20/02/2015.  
Date of Publishing: 27/02/2015.