

Case Report

Two cases of viper bite: still an important health problem

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ABSTRACT

Viper venoms act mainly as hemotoxic. Manifestations of snakebites depend on specific toxins that constitute the venom. The local and systemic snake bite related symptoms are directly linked to the toxicity of the venom. Edema, ecchymoses, hematoma, and gangrenous lesions are reported to occur as local symptoms. Systemic symptoms may include fever, nausea, vomiting, delirium, jaundice, circulatory collapse, convulsions, and coma. Death from secondary infections, neurotoxicity, disseminated intravascular coagulation (DIC), intracranial hemorrhage, and acute renal failure are the well-known facts. For reduction of morbidity and mortality, it is important that antiserum is administered at the appropriate dose as early as possible after snake bite. There are several case reports about various complications of viperid bite. Here we are discussing two cases of viper bite. These cases are unique because of the extensive tissue necrosis. One of them succumbed to septicemia after acute pancreatitis.

Keywords: Snake bite, Viper, Pancreatitis, Acute renal failure, Necrosis

INTRODUCTION

Russel viper bite is a common incidence in India. Most of the people live in rural area, and they are exposed to increased risk of snake bite. Some are fortunate enough to encounter nonpoisonous snake bite, but some are victims of poisonous snake bite. Vasculotoxicity, neurotoxicity, or myotoxicity are commonly discussed complications of Russel viper bite. Approximately 45,900 are reported to be fatal in a year.¹ A previous community-based survey in the state revealed that only 22% snake bite victims attended hospitals.² Apart from systemic effect snake envenomation has some local tissue destroying action also. Pain, edema, ecchymoses blisters and cellulitis can occur due to the local action of venom. We are here discussing two cases who present with extensive cellulitis. In both of the cases, there were the acute renal failure (ARF). But in one case patient developed pancreatitis and succumbed to disseminated intravascular coagulation (DIC) and septicemia.

CASE REPORT

A 30-year-old male farmer was a victim of a viper bite during spraying insecticides in the paddy field. He got admitted to the local hospital, and there anti-snake venom (AVS) was given. But due to some inevitable situation, there was 5 hours delay in starting AVS treatment. After 48 hour's patient developed severe epigastric pain and vomiting. Ultrasonography whole abdomen showed bulky pancreas and free fluid in intraperitoneal space. An emergency contrast-enhanced CT abdomen showed bulky pancreas with peripancreatic collections with distended bowel loops (Figure 1 and 2). At that time his serum amylase was 800U/L (average value-25-85U/L) and lipase level was 1400U/L (normal value- 0-160 U/L). With a diagnosis of acute pancreatitis after a viperid bite patient was referred to our institution. At the time of admission patient's hemoglobin (Hb) was 7gm/dl, WBC count was 24000/cumm, and platelet count was 1.2L /cumm. His serum creatinine level was 1.8mg/dl. He has

been started IV antibiotics, and IV fluid was also given. In the meantime, he has developed local tissue destruction at the site of snake bite that is left foot. Immediately debridement was also done, and pus was sent for culture sensitivity (Figure 3 and 4). Within 24 hour of admission patient developed subconjunctival hemorrhage (Figure-5 and 6). Patient was still conscious. His repeat blood report showed Hb-7gm/dl, WBC count-23700/cumm and platelet count was 90000/cumm. His creatinine level was raised to 2.8mg/dl. Till evening of the second day of admission patient passed only 150 ml

of urine. His ultrasonography showed increased cortical echogenicity in bilateral kidneys. He was immediately sent for hemodialysis. The patient was shifted to intensive care unit and was kept under close observation. Next day his creatinine level was detected to be 4.8mg/L and platelet count was 56000/cumm. Hemodialysis was continued. Patient succumbed on the fourth day after receiving the third hemodialysis. His blood reports have been expressed in the table below (Table 1).

Table 1: Serial blood reports of the first patient.

Parameters	Normal value	Day 1	Day 2	Day 3	Day 4
Hemoglobin	13.5-17.5gm/dl	7gm/dl	7gm/dl	6.8gm/dl	6.5gm/dl
WBC count	4500-10000/cumm	24000/cumm	23700cu/mm	25000cu/mm	24600/cumm
Platelet count	1.5-4.5×10 ⁹ /L	1.2/L	90000/L	56000/L	40000/L
D dimer	<500 ng/ml	1600ng/ml	2000ng/ml	1800ng/ml	1500ng/ml
Blood urea	7-21 mg/dl	60mg/dl	110mg/dl	120mg/dl	150mg/dl
Blood creatinine	0.7-1.0 mg/dl	1.8 mg/dl	2.8mg/dl	4.8mg/dl	6.0mg/dl
Activated partial thromboplastin time	18-45 s	42s	54s	60s	52s
International normalised ratio	0.9-1.2	1.8	2.2	3.8	4.0
Arterial blood gas pH	7.35-7.45	7.20	7.10	7.32	7.34

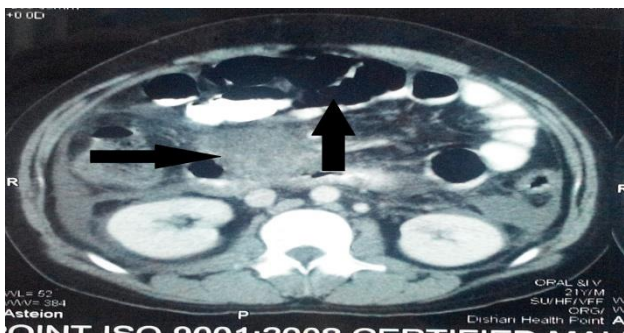


Figure 1: Shows bulky pancreas and distended bowel loops (black arrows).



Figure 3: Local tissue injury after debridement.

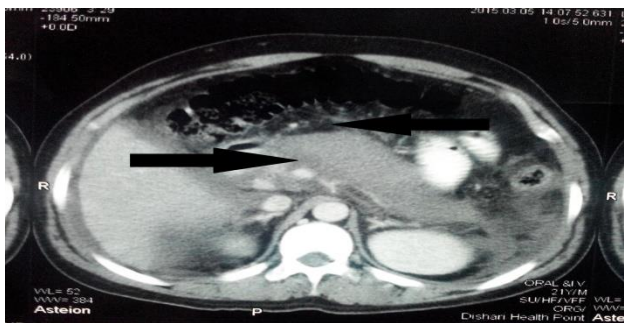


Figure 2: Shows bulky pancreas and peri pancreatic fluid collection (black arrows).



Figure 4: Local tissue injury after debridement (closer view).



Figure 5: Subconjunctival hemorrhage front view.



Figure 6: Subconjunctival hemorrhage side view.



Figure 7: Local tissue necrosis of second patient side view.



Figure 8: Local tissue necrosis of second patient.

Another case was also admitted directly to our institution after Russel viper bite. AVS was given within 3 hours of snake bite. Here also the patient developed acute renal failure (ARF), but he was successfully managed after seven episodes of hemodialysis. Here also, patient

developed extensive local tissue necrosis (Figure 7 and 8). After IV antibiotics the systemic infection was controlled, and the patient was discharged in a stable condition.

DISCUSSION

The Viper venom has thrombin-like actions. They may cause local vasculopathy causing swelling, blisters and necrosis. There is 2-3% chance of tissue necrosis with viper venom. This local injury is a site for bacterial growth, and subsequently, it can cause septicemia also. There are several studies supporting the high chance of necrosis at the site of viperid bite.³ There are mixtures of multiple enzymes and low molecular weight peptides in viper venom. Some of which are responsible for the bleeding manifestations. Several mechanisms have been suggested for the bleeding after envenomation. Disseminated intravascular coagulation (DIC) may be due to spontaneous activation of factor V and factor X by pro-coagulants present in the venom. This results in the production of a fragile fibrin leading to bleeding manifestations. Direct vascular endothelial damage caused by the direct effect of the venom also contributes to bleeding manifestations.⁴ Exact pathogenesis of pancreatitis occurring as a complication of snake envenomation is a matter of research till now. Phospholipase A2, a lipolytic enzyme, is found in almost all snake venoms as a basic protein called "direct lytic factor." It may result in acute pancreatitis. The impaired collagen metabolism resulting in fibrosis of pancreatitis has also been postulated to cause acute pancreatitis. Serum complement system has also been proposed in the pathogenesis of acute pancreatitis.⁵ ARF can develop as early as within 24 hours and as late as after one week after hemotoxic snake bite. Renal vascular blockage by microthrombi, ischemia, and shock cause decreased renal perfusion. Hemoglobinuria and myoglobinuria may cause direct nephrotoxicity. Late involvement of kidney can be explained by shock due to hypovolemia. This is caused by loss of plasma and blood in the extravascular compartment and pulmonary intravascular clotting. The viper venom acting directly on the heart may also result in hypotension. Increased vascular permeability causes peripheral pooling of blood resulting in decreased perfusion of renal circulation.¹

The purpose of describing these two cases is to depict that delay in starting of AVS therapy in our first case results in a devastating outcome. We have got the patient after 72 hours of the bite. In the second case, we started AVS as early as possible, but this patient also developed severe local tissue injury and ARF also. Timely hemodialysis and antibiotic therapy saved our case.

World Health Organization in the year 2009 includes snake bite in the list of neglected tropical diseases. Very few epidemiological surveys are there on the snake bite problem in India. More data regarding clinical course of snake bite is necessary.² We need to study further about

the relationship between the time interval of commencement of AVS and outcome of the patients.

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REFERENCES

1. Vikhe VB, Gupta A, Shende P, Jain J. Vasculotoxic snake bite presenting with sepsis, acute renal failure, disseminated intravascular coagulation, and acute respiratory distress syndrome. Medical Journal of Dr. D.Y. Patil University. 2013;6(2):197-9.
2. Majumder A, Sinha A, Bhattacharya SK, Ram R, Dasgupta U, Ram A. Epidemiological Profile of Snake Bite in South 24 Parganas District of West Bengal with Focus on Underreporting of Snake Bite Deaths. Indian Journal of Public Health. 2014;58(1):17-21.
3. Balaji G, Kumar A, Menon J. Snake bite induced cellulitis leading to infected open dislocation of the first metacarpophalangeal joint-A rare complication. J Clin Orthop Trauma. 2015;6(3):195-8.
4. Lakhota M, Kothari D, Choudhary DR, Sharma S, Jain P. A Case of Saw Scale Viper Snakebite Presenting as Pleuro-pericardial Haemorrhage. JIACM. 2002;3(4):392-4.
5. Malkarnekar S, Naveen L. A rare case of acute pancreatitis following snake envenomation with fatal outcome. Trop J Med Res. 2014;17(2):137-9.

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