Delayed Psoas Hematoma after *Echis Carinatus* Bite: An Unusual Manifestation

Manoj Lakhotia¹, Hans Raj Pahadiya^{2,*}, Harish Kumar², Jagdish Singh², Gopal Raj Prajapati³, Ravi Sangappa Jainapur⁴

¹Senior Professor, Department of medicine, Dr. S.N. Medical College, Jodhpur, Rajasthan, India
²Senior Resident, Department of medicine, Dr. S.N. Medical College, Jodhpur, Rajasthan, India
³Assistant Professor, Department of medicine, Dr. S.N. Medical College, Jodhpur, Rajasthan, India
⁴Resident, Department of medicine, Dr. S.N. Medical College, Jodhpur, Rajasthan, India
*Corresponding author: drhans05sms@gmail.com

Received July 28, 2014; Revised August 15, 2014; Accepted August 19, 2014

Abstract Bleeding manifestations are common with viperine snake bite. Common bleeding manifestations include bleeding from site of bite, bleeding gums, epistaxis, hemoptysis, hematuria, hematemesis and intracranial bleed. Bleeding in retroperitoneal, plural and pericardial spaces have also been described. Delayed bleeding in the muscle is a rare complication. We report a patient of viperine bite who developed Psoas hematoma after eight days of bite. The patient had low platelet count and normal coagulation profile. He responded to intravenous dexamethasone. In appropriate setting, the possibility of soft tissue bleeding should be considered in a patient of snake bite inspite of coagulation profile being normal. To the best of our knowledge this is the first case report of delayed Psoas hematoma after *Echis carinatus* bite.

Keywords: Viperine bite, Echis carinatus, Psoas hematoma

Cite This Article: Manoj Lakhotia, Hans Raj Pahadiya, Harish Kumar, Jagdish Singh, Gopal Raj Prajapati, and Ravi Sangappa Jainapur, "Delayed Psoas Hematoma after Echis Carinatus Bite: An Unusual Manifestation." *American Journal of Medical Case Reports*, vol. 2, no. 8 (2014): 152-154. doi: 10.12691/ajmcr-2-8-2.

1. Introduction

Saw scale vipers or *Echis carinatus* are commonly found in the semi-arid deserts of Western Rajasthan with predominant nocturnal habitus. The occurrence of local and systemic snake bite related symptoms is directly related to the toxicity of the venom. Bleeding is a major manifestation of viper bite and may occur from multiple sites including gums, nose, gastrointestinal tract, urinary tract, injection sites and even as multiple petechiae and purpurae over skin. [1] Edema, ecchymosis, hematoma, and gangrenous lesions may occur as local symptoms, whereas systemic symptoms may include fever, nausea, vomiting, delirium, jaundice, circulatory collapse, convulsions, and coma. Disseminated intravascular coagulation (DIC), intracranial haemorrhage, neurotoxicity, infections, and acute renal failure are the major cause of mortality after viper bite. [2,3] To the best of our knowledge this is the first case report of delayed Psoas hematoma after Echis carinatus bite. We report a case of viperine bite who developed Psoas hematoma after eight days of bite.

2. Case Report

A 48 years old male, was admitted in a local hospital three hours after he being bitten on his right ankle by a viper snake while working on the farm. The killed snake was identified as Echis carinatus by the initial attending medical officer. At the local hospital, management consisted of antibiotics, anti-tetanus prophylaxis, analgesics and 10 vials of polyvalent anti snake venom (ASV). ASV was repeated on the next two days. The patient developed hematuria and persistent vomiting on the 3rd day and hence was referred to the medical college. There was no other site of bleeding. Patient was not suffering from any chronic illness and had no addiction. At the time of admission patient had swelling of the right ankle extending up to the middle of leg. His blood pressure was 110/70 mmHg, pulse rate was 78 beats/min with normally palpable peripheral pulses, respiratory rate was 18 breaths/min. Lungs were bilaterally clear and cardiac auscultation was normal. Laboratory investigations revealed a haemoglobin (Hb) of 10.7 g/dL, total leukocyte count of 10,280/mm³, with normal differential count. Platelets were 54,000/mm³. Renal, liver functions and serum electrolytes were normal. Urine was dark red in colour and was full of red blood cells. Coagulation profile showed bleeding time (BT) 9.5 min (normal up to 9 min), clotting time (CT) 3.25 min (normal up to 8 min), prothrombin time (PT) 16 sec (Control 14 sec) with INR of 1.05, thromboplastin time with kaolin (PTTK) 38 sec (control 35 sec) and fibrinogen degradation product (FDP) was 0.50 µg/mL (0-1). Oxygen saturation was 99% on room air. Two units of whole blood were transfused. On day 5th of admission the patient started

complaining of pain in both the inguinal area more on the left. He assumed a position of flexion at hip joint. Extension of the joint was painful. There was no neurological deficit. He had no fever and lymph nodes were not palpable. Traction was applied on both lower limbs. MRI of pelvis including both hip joint regions was ordered which showed lobulated altered signal intensity in the right Psoas muscle and left Iliopsoas extending up to upper thigh suggestive of hematoma.



Figure 1. Coronal T1W MR image showing diffuse enlargement and complex hyper intensity of bilateral Iliopsoas muscle suggestive of hematoma

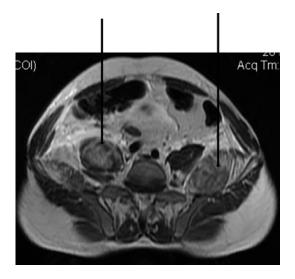


Figure 2. Axial T2W MR image showing bilateral bulky Psoas with heterogeneous signal suggestive of hematoma

After 3 days Haemoglobin dropped to 6.2 g/dL, platelets count was 60,000/mm³ and there was persistent hematuria. Urinary Hb was 60 mg/L (normal <0.5 mg/L). Three more units of whole blood and 18 bags of random donor platelets (RDP) were transfused over the next three days. However hematuria and pain in the inguinal region persisted hence dexamethasone 8mg q8h for 3 days was given. Urine cleared over the next 2 days. The pain in the inguinal region started subsiding and by the end of a week the patient could stand up and walk.

3. Discussion

The *Echis carinatus* is primarily vasculotoxic. Its venom is a highly complex mixture of a variety of biological substances including protein (enzymes & nonenzyme polypeptides) and non protein toxins. [1] The venom causes haemostatic derangement through the

combined pro-coagulant, fibrinolytic, antiplatelet, and haemorrhagic components. [4] Activation of the coagulation pathway at varied points by procoagulants toxins can lead to Venom induced consumptive coagulopathy (VICC). [5] Activation of prothrombin by these factors result in consumptive coagulopathy with variable deficiency in fibrinogen, factor V and factor VII. [5] The venom also contains factor X activator and many other compounds which increases its capacity to cause coagulopathy such as platelet aggregation inhibitors. [1,5]

Direct vasculotoxic effect is brought about by haemorrhagins which are zinc-containing metalloproteases proteolytic enzyme found in viper venom. They damage the vascular endothelium directly by selectively cleaving the proteins in the capillary wall. The local effect of haemorrhagins is seen when venom is introduced by subcutaneous or intravenous route. However if the venom reaches the systemic blood circulation it can produce systemic effect damaging the vascular endothelium at distant site. Overall effect also depends upon the other component of venom that affects platelets function and coagulation system. [6]

The bleeding may occur from multiple sites after a viperine bite. [1] Common sites are skin, gums, nose, gastrointestinal tract, and urinary tract. Bleeding from the site of bite is also frequent. Amongst the closed spaces, bleeding in retroperitoneal space and intracranial bleeding are the most frequent. Bleeding in closed space like peritoneum [4], pleuropericardium [7] and hemothorax [8] have also been reported. Of the soft tissues hematoma in broad ligament have been reported. [9]

Development of hematoma in the Psoas muscle has not been described in the past. Our patient developed haematoma in the Psoas muscle in spite of coagulation profile being normal. He continued to have hematuria during this period and responded to the use of dexamethasone. Though this patient had reduced platelets but spontaneous deep soft tissue bleeding cannot be attributed to it. It is difficult to postulate any mechanism to this delayed bleeding except for delayed effect of haemorrhagins. The response to steroid also favours this hypothesis.

Delayed bleeding in viperine bite is rare. Saini et al [10] have documented delayed bleeding following snake bite in spite of normal coagulation profile in 4 patients. They have attributed it to the release of venom which is deposited in the bleb at the site of bite. However our patient did not have any bleb at the site of bite which could be attributed to the depot effect of venom and its haemorrhagins. But the patient did not have gross swelling of limb which may have delayed absorption of venom and its haemorrhagins.

The case is being reported because of delayed bleeding after the bite and bleeding in the muscle both of them being very rare complication of viperine bite. It is presumed that delayed bleeding in the soft tissue inspite of normal coagulation profile occurring after snake bite is due to delayed release of hemorrhagins.

References

 Warrell DA, Davidson NMD, Greenwood BM, Ormerod LD, Pope HM, Watkins BJ, Prentice CR: Poisoning by bites of the

- saw-scaled or carpet viper (Echis carinatus) in Nigeria. Q J Med 1977; 46 (181): 33-62.
- [2] Kerrigan KR. Venomous snakebite in Eastern Ecuador. Am J Trop Med Hyg 1991; 44: 93-99.
- [3] Benvenuti LA, Franca FO, Barbaro KC, Nunes JR, Cardoso JL. Pulmonary haemorrhage causing rapid death after Bothrops jararacussu snakebite: a case report. *Toxicon* 2003; 42: 331-4.
- [4] Ahn JH, Yoo DG, Choi SJ, LeeJH, Park MS, Kwak JH, et al. Hemoperitoneum caused by hepatic necrosis and rupture following a snake bite. *Korean J Radiol*. 2007; 8: 556-60.
- [5] Isbister GK. Snakebite doesn't cause disseminated intravascular coagulation: coagulopathy and thrombotic microangiopathy in snake envenoming. Semin Thromb Hemost 2010; 36 (4): 444-51.
- [6] Hati R, Mitra P, Sarker S, Bhattacharyya KK. Snake venom hemorrhagins. Crit Rev Toxicol 1999; 29 (1): 1-19.
- [7] Lakhotia M, Kothari D, Choudhary DR, Sharma S, Jain P. Pleuropericardial Haemorrhage after a Saw Scale Viper snakebite- a case report. *JIACM* 2002; 3 (4): 392-94.
- [8] Singh V, Digra SK, Slathia SS, Kakkar T. Hemothorax following Snakebite. *Indian Pediatr*. 2012; 49: 242-3.
- [9] Addo V, Kokroe FA, Reindorf RL. Broad ligament haematoma. Ghana Med J. 2009; 43 (4): 181-82.
- [10] Saini RK, Sharma S, Singh S, Pathania NS. Snake bite poisoning: A preliminary report. J Assoc Phys India 1984; 32 (2): 195-97.