CASE REPORT

A rare case of acute pancreatitis following snake envenomation with fatal outcome

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Abstract

The case study herein reported depicts a case of neurotoxic envenomation with common krait (*Bangarus caeruleus*) in a young farmer from a small village in India, which was unexpectedly complicated with acute pancreatitis. Patient developed features of acute abdomen on the third day of envenomation, and based on the laboratory and radiological findings, acute pancreatitis was diagnosed. Subsequently, consumptive coagulopathy, renal failure, and respiratory insufficiency set in. Despite timely administration of anti-snake venom and supportive treatment including hemodialysis, fresh frozen plasma and thrombocyte transfusion, and mechanical ventilation, the patient succumbed to severe toxemia of pancreatitis on the seventh day of envenomation. Pancreatitis associated with snake bite is an exceptional complication, and hence, we report this case on account of its rarity.

Keywords: Acute pancreatitis, *Bangarus caeruleus*, coagulopathy, envenomation, krait, renal failure, snake bite

Introduction

Snake envenomation is a well-known hazard across the tropics. Though local and systemic effects such as neurotoxicity, consumption coagulopathy, and renal failure are well-appreciated manifestations of snake bite, acute pancreatitis after snake bite is a very rare occurrence. The case reported here represents a more exceptional serious course of envenomation with profound multiorgan dysfunction as a result of toxaemia of pancreatitis. Moreover, this case study is the first report of acute pancreatitis after krait bite.

Case Report

A 30-year-old farmer presented to the emergency department of RL Jallappa hospital with complaints of snake bite over the dorsum of left hand 3 h before arrival to the hospital. The snake was identified to be a common krait [Figure 1]. He had no notable past medical history. General physical examination revealed fang marks along with minimal local reaction over the left hand. The 20-min whole blood clotting time was within normal limits. As expected with krait bite, patient had features of neurotoxic envenomation in the form of dysphagia and ptosis. In view of systemic envenomation, patient was treated with anti-snake venom (ASV). Clinical improvement was noted over the next 24 h, with features of neurotoxicity resolving. All laboratory parameters such as coagulation profile, thrombocyte count, renal function tests, and liver function tests were within normal limits on admission. On the third day of envenomation, patient complained of acute-onset colicky abdominal pain. Examination revealed marked guarding and tenderness over the epigastrium. Subsequently, the patient developed hematuria and a progressive decrease in the urine output, progressing on to anuria. Laboratory tests revealed evidence of renal insufficiency, severe metabolic acidosis, consumptive coagulopathy (thrombocytopenia, prolonged activated partial thromboplastin time, and high levels of fibrinogen degradation products), and deranged liver function tests [Table 1]. Serum amylase and urinary amylase levels were 556 U/L and 1453 U/L, respectively (normal range: 30-110 U/L and 24-400 U/L, respectively). Serum lipase levels were increased to 3485 U/L (normal range: 5-208 U/L). Abdominal ultrasonography revealed a bulky pancreas and free fluid in the intraperitoneal and retroperitoneal spaces,

Access this article online	
Quick Response Code:	Website: www.tjmrjournal.org
	DOI: 10.4103/1119-0388.140446

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Figure 1: The snake responsible for the envenomation identified as the common krait (*Bangarus caeruleus*)

Table 1: Profile of laboratory investigations

Parameters	Normal adult range (units)	Day of envenomation		
		3	5	7
Activated partial thromboplastin time	18-45 s	58	65	52
INR	0.9-1.2	4.5	3.5	2.2
Platelet count	1.5-4.5×10^9/L	18	26	80
D dimer	<500 ng/ml	4534	5564	4345
White blood cell count	4-11×10^3/mm^3	18	24	23
Blood urea	7-21 mg/dl	213	184	160
Serum creatinine	0.7-1.0 mg/dl	10	12	9.8
Serum potassium	3.5-5.5 mEq/L	6.0	5.6	4.8
Total bilirubin	0.2-1.2 mg/dl	3.8	4.3	5.9
Aspartate transaminase	5-40 IU/L	230	198	178
Alanine transaminase	7-56 IU/L	245	200	186
Alkaline phosphatase	30-120 IU/L	333	357	423
Serum amylase	30-110 U/L	556	674	526
Serum lipase	5-208 U/L	3485	5568	6943
Urinary amylase	24-400 U/L	1453	2531	2943
Arterial blood gas pH	7.35-7.45	7.12	7.26	7.36
HCO3	21-26 mEq/L	10	13	18
PaO_2	80-100 mm Hg	88	75	70

INR=International Normalised Ratio

suggestive of exudative pancreatitis. The consumptive coagulopathy was corrected by administration of units of thrombocytes and fresh frozen plasma. Antibiotic therapy with Imipenem was initiated. The progressive renal insufficiency necessitated the initiation of hemodialysis. Besides this, the patient received other supportive treatment in the form of supplemental oxygen therapy, adequate hydration and pain relief, and correction of metabolic acidosis. Over the next 48 h, i.e. on the fifth day of envenomation, patient developed progressive dyspnoea and in view of persisting respiratory insufficiency, mechanical ventilation was started. The patient satisfied all the criteria for acute respiratory distress syndrome. With adequate replacement therapy, thrombocytopenia and coagulopathy was corrected, but the renal failure persisted for which hemodialysis was continued. However, despite all the supportive treatment and the timely administration of ASV, the patient ultimately succumbed to the severe toxaemia of pancreatitis on the seventh day of envenomation.

Discussion

Venomous animal bites are a significant health problem for rural populations in many parts of the world. Snake venom is one of the most potent animal venoms. Snake venom has a very complex heterogeneous composition, containing enzymes, lethal peptides, nonenzymatic proteins, metals, carbohydrates, lipids, biogenic amines, free amino acids, and direct hemolytic factors. This composition makes it possible to see many different symptoms after envenomation. Though features such as local tissue necrosis, renal toxicity, neurotoxicity, and consumptive coagulopathy are well known with snake bite, acute pancreatitis is a very rare occurrence.

The exact pathogenesis of pancreatitis occurring as a complication following snake envenomation is not well established. Various mechanisms have been suggested for this exceptional complication. Phospholipase A2, a lipolytic enzyme, is present in almost all snake venoms as a basic protein called "direct lytic factor," which has been implicated in diseases like acute pancreatitis.[1,2] It has also been suggested earlier that adder venom contains enzymes which can, at least theoretically cause acinar cell damage and thus trigger acute pancreatitis.[3] Another mechanism implicated is the imbalance of the collagen metabolism resulting in fibrosis of pancreatitis.[4] A possible role of serum complement system in the formal pathogenesis of acute pancreatitis has also been described.[5] Krait venom constitutes primarily of pre-synaptic and post-synaptic neurotoxins (b-bungarotoxin and a-; and k-bungarotoxins, respectively). Phospholipase A2–induced pancreatitis (an enzyme thought to be present in all snake venoms, as mentioned earlier) seems to be the most likely possibility in our patient.

The case herein described is noteworthy for various reasons. Till date, very few cases of acute pancreatitis following snake envenomation have been reported.[3,6,7] The common krait is well recognized for its neurotoxic envenomation, and to the best of our knowledge, this is the first case of acute pancreatitis to be reported following krait envenomation. While most of the other case studies of snake bite associated pancreatitis reported a favourable course and outcome, the case herein reported

represents that of an exceptionally serious envenomation which progressed on to develop multiorgan dysfunction. The patient reported to us immediately following envenomation and immediate treatment with ASV was initiated. An unexpected fatal course was observed in our patient, and despite appropriate treatment and supportive measures, our patient eventually succumbed to severe toxaemia of pancreatitis.

Acknowledgment

The authors express their gratitude toward Sri Devaraj Urs Medical College and the Department of Medicine for their invaluable support.

References

1. Kihara Y, Yoshikawa H, Honda H, Fukumitsu K, Yamaguchi T, Otsuki M. Natural disruption of group 2 phospholipase A2 gene protects against choline-deficient ethionine-supplemented diet-induced acute pancreatitis and lung injury. Pancreas 2005;31:48-53.
2. Rönkkö S, Rekonen P, Kaarniranta K, Puustjarvi T, Teräsvirta M, Uusitalo H. Phospholipase A2 in chamber angle of normal eyes and patients with primary open angle glaucoma and exfoliation glaucoma. Mol Vis 2007;13:408-17.
3. Kjellström BT. Acute pancreatitis after snake bite. Case report. Acta Chir Scand 1989;155:291-2.
4. Ogawa M, Nakano I, Funatomi H, HattaY. Collagen metabolism in ethionine induced rat chronic pancreatic injury. Nihon Shokakibyo Gakkai Zasshi 1991;88:65-70.
5. Seelig R, Seelig HP. The possible role of serum complement system in the formal pathogenesis of acute pancreatitis II. Cobra venom factor pancreatitis: Sodiumtaurocholate and deoxycholate pancreatitis. Acta Hepatogastroenterol (Stuttg) 1975;22:335-46.
6. Sagheb MM, Sharifian M, Moini M, Salehi O. Acute renal failure and acute necrotizing pancreatitis after *Echis carinatus sochureki* bite, report of a rare complication from southern Iran. Prague Med Rep 2011;112:67-71.
7. Valenta J, Stach Z, Svítek M. Acute pancreatitis after viperid snake *Cerastes cerastes* envenoming: A case report. Prague Med Rep 2010;111:69-75.

How to cite this article: Malkarnekar S, Naveen L. A rare case of acute pancreatitis following snake envenomation with fatal outcome. Trop J Med Res 2014;17:137-9.

Source of Support: Nil, **Conflict of Interest:** None declared.