



Case Study

Acute Hearing Loss And Acute Pancreatitis – Occurrence Of Two Rare Complications Of Snake Bite In The Same Patient

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ABSTRACT

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Snake bite is a significant health issue in many countries including India. Neuroparalysis and Coagulopathy are the common complications. Sudden bilateral deafness following snake bite has been reported very sparsely earlier in literature. The development of acute pancreatitis after envenomation is a known but rare complication. The occurrence of both deafness and pancreatitis in the same patient following a viperian snake bite is presented with a discussion on etiopathogenesis of both complications.

INTRODUCTION:

Snake bite is a major public health issue in many countries. Hematological toxicity and neuroparalysis are the common complications. Several less frequent consequences occur. There have been very few reports of sudden bilateral deafness [1]. Acute pancreatitis after envenomation is an equally rare complication. [2, 3, 4] The occurrence of both in the same patient has not been reported hitherto. This report is about a young male farm worker who developed both the rare complications after a viper bite.

CASE REPORT:

A 42 year old male farm worker was brought to the hospital with history of snake bite on the dorsum of the left foot about six hours prior to arrival. The description of the snake suggested Russel's Viper bite. On examination he was alert and oriented. BP was 110/70mmHg, Pulse 104/min, Respiratory Rate was 20/min. There was no bleeding from any site. There were two fang marks on the dorsum of the left foot with surrounding cellulitis. Laboratory data revealed Haemoglobin of -8.2gm/dL. Total Leukocyte Count -9700/mm³ Differential Count – Neutrophils :96% Lymphocytes:4% ESR-5mm at end of first hour, PCV -26.2% RBC-5.1million/mm³ Platelet count-2.33 lakh/mm³. Urea-33mg/dl, Creatinine-1.6mg/dl Na-137mEq/L K-3.5mEq/L Cl-106mEq/L. Prothrombin Time- INR-1.33 and aPTT-1min 36 secs. The 20 minute whole blood clot lysis test was positive. Treatment with Anti Snake Venom(ASV) was started. Antibiotics, analgesics, IV fluids and Fresh Frozen Plasma transfusion were also given. On the second day the cellulitis extended to mid leg. Investigation revealed worsening of parameters with Hb%-5.6gm/dL PCV-20.2, Platelet Count-50,000/mm³ Urea-80mg/dL, Serum creatinine -2.0mg% Na-128mEq/L K-4.4mEq/L Cl-102mEq/L. Prothrombin Time- Control 13 sec Patient 28.6sec INR-2.3 APTT- Control 30 secs Patient 64.12secs. D-Dimer was positive (>200 mg/dl -positive.)

The patient was treated with two transfusion units of fresh frozen plasma. Hemodialysis was initiated. On the fifth day the patient was noted to be not replying even to loudly spoken oral commands. Questioning by gestures elicited the reply that he had difficulty in hearing. Otological examination revealed intact tympanic membranes. In view of the patient being on treatment in ICU detailed ENT investigations could not be performed.

On the same evening the patient complained of pain abdomen. Abdominal examination revealed moderate ascites. Serum amylase of 259 u/l and Serum lipase of 259.22 u/l were indicative of pancreatitis. CT scan abdomen confirmed acute pancreatitis. Ascitic fluid analysis showed glucose-124mg/dl protein - 2.8mg/dl cell count- 20 cells/cu mm predominant lymphocytes.

The patient was managed as per standard guidelines for pancreatitis. He steadily improved as far as pancreatitis was concerned. Renal parameters improved after dialysis over the next five days. But he continued to have impaired hearing. Pure tone audiometry (Done after recovery from pancreatitis) showed profound bilateral hearing loss. The patient was discharged in good general condition on the fifteenth day after admission. Hearing loss persisted and he was advised hearing aid.

DISCUSSION:

Snakes have evolved the most complex of all venoms. Most venoms contain more than 100 different components [5]. 90 -95% of viper venom consists of enzymes including digestive hydrolases, endopeptidases, phospholipases and hyaluronidase. They damage mitochondria, red blood cells and white blood cells. Hyaluronidase promotes spread and metalloproteinases lead to myonecrosis.

Venom contains thrombin-like enzymes and other components activating factor X. This leads to activation of coagulation cascade with consumption coagulopathy. Components of the venom cause degradation of fibrinogen

The common syndromes of envenomation are neurotoxicity, coagulopathy, shock, rhabdomyolysis and renal failure. Rarer complications are also recognised. Many reports of acute pancreatitis [2] following snake bite are available. Kjellström [3] reported a case of acute pancreatitis which was diagnosed after exploratory laparotomy in a young, previously healthy man who had severe abdominal symptoms after an adder bite. In the case report by Valenta [4], acute exudative pancreatitis was diagnosed on the third day after envenomation following viper bite. Consumption coagulopathy with thrombocytopenia and haemolysis, colicky pain in the epigastrium and acute renal failure were the other features present in their patient. Similar features were observed in the present case.

Damage to acinar cells by the venom is one main causative phenomenon of pancreatitis after snake bite. [3] It has been postulated that Adder venom contains enzymes which cause acinar cell damage triggering acute pancreatitis. A similar mechanism is hypothesized in pancreatitis caused by Viper bite.

The term Sudden Sensory Neural Deafness is used to indicate acute cochlear-retrocochlear hearing loss of any cause [6] and it constitutes an otological emergency. In most of the instances no apparent cause is found. Specific causes such as trauma, infectious diseases, drugs and neoplasia are well described in earlier literature. Sudden sensorineural deafness following a snake bite has been reported very infrequently with only one report from India [1] whereas the occurrence of vertigo, mild impairment of auditory perception and hyperacusis after snake bite have been described earlier. [7]

As regards the mechanism of deafness after snake bite the various possibilities might be as follows: a) A direct neurotoxic effect of snake venom or toxic neuritis. A similar mechanism has been implicated in the rare occurrence of optic neuritis and optic atrophy following snake bite [8,9]. b) Vasculotoxicity of snake venom producing ischemic neuropathy c) thrombosis

in the labyrinthine artery d) spasm of labyrinthine artery due to autonomic imbalance e) Ischemia secondary to Hypotension. f) Hypersensitivity reaction to snake venom can be another mechanism and g) Hypersensitivity reaction to Anti Snake Venom. Mair and Everland [10] reported a case of bilateral deafness following revaccination against tetanus and diphtheria. They reviewed nine cases of deafness following vaccination and postulated a local hypersensitivity reaction affecting the eighth cranial nerve in the internal auditory meatus.

In the present case there was no hypotension, no hypersensitivity reaction either to snake venom or antivenom. Hence it is surmised that the most probable cause of deafness was toxic neuritis. The unique feature of this case is that both the rare complications, pancreatitis and deafness, have occurred in the same individual at around the same time.

CONCLUSION

This case report highlights two of the rarer complications of venomous snake bite reminding us of the protean ways snake bite can manifest. The clinician must be aware of not only the typical presentations but also the rarer manifestations.

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