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Case Report

Acute acalculous cholecystitis as a presenting feature of neurotoxic snake bite: a case report and review of literature

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ABSTRACT

Snakebite is classified by the WHO as a neglected tropical disease. Neurotoxic snake bites pose a great diagnostic challenge to the physicians in tropical and subtropical regions of the world. There is considerable variation between individual patients in the clinical manifestations following envenoming by neurotoxic snake bite. Here authors describe a rare case who presented to emergency department as acute acalculous cholecystitis following unknown neurotoxic snake bite. This acalculous cholecystitis resolved post administration of anti-snake venom. The possibility of this rare but potentially fatal complication needs to be considered in patients with snake bite and abdominal symptoms so that prompt management can prevent mortality in such patients.

Keywords: Acalculous cholecystitis, Antisnake venom, Neurotoxic snake bite, Phospholipase A2

INTRODUCTION

Acute acalculous cholecystitis constitutes 10% of all cases of cholecystitis and is a potentially fatal complication if prompt management is not instituted.[1] It is an acute inflammation of the gall bladder without evidence of calculi or sludge. It has been documented in patients with burns, infections, diabetes, major non-biliary surgery, critically ill patients, trauma and prolonged total parenteral nutrition.[1-3] The exact mechanism of acalculous cholecystitis is not known. The possible mechanisms postulated are ischemia and reperfusion, infection, endotoxins, stasis and changes in bile, microangiopathy, and disruption of neurotransmitter release leading to gall bladder dysmotility.[4] The diagnosis of this type of cholecystitis requires a high degree of suspicion, as clinical and imaging evaluations can be misleading because of comorbidities. Here authors report a rare case of 30-year-old labourer presenting as acute acalculous cholecystitis to emergency department following envenomation by neurotoxic snake bite without neuromuscular paralysis in the initial stage.

CASE REPORT

A 30 year old labourer came to emergency department at 9 a.m. with history of acute colicky pain in the right hypochondrium associated with vomiting since 5 a.m. in the morning. There was no history of fever, jaundice, and loose motions. On examination, his vitals were stable and there was minimal guarding associated with marked tenderness in the right hypochondrium. Bowel sounds were normal. Ultrasound revealed gall bladder wall edema with minimal pericholecystic fluid but absence of biliary sludge or gall stones. Patient was admitted in the department of surgery as acute acalculous cholecystitis and was started on antibiotics and intravenous fluids. Two hours later, he developed acute bilateral ptosis and ophthalmoplegia progressing to respiratory muscle weakness. On enquiry, he did not reveal any history of snake bite the night prior although he had slept on the

floor. On close inspection, small probably fang marks were noticed in the upper aspect of the back. Patient was shifted to ICU, intubated and connected to ventilator support. He received 20 vials of anti-snake venom and neostigmine. Hematological investigations revealed normal clotting time. His Hemoglobin was 13 gm%, Total leucocyte count 15500 cell/mm^3 with neutrophilic predominance of 80%. Platelet count was 1.5 lakh/mm^3 and INR was 1. His renal parameters were normal. Liver function tests showed mild rise in bilirubin of 2.5 mg/dl with SGOT (AST) 124 IU/L, SGPT (ALT) 234 IU/L and alkaline phosphatase 245IU/L. Patient's ptosis and ophthalmoplegia subsequently improved post anti snake venom administration after 24 hours and he was weaned from ventilator support. His abdominal pain also settled after ASV administration and repeat ultrasound showed resolution of gall bladder edema and pericholecystic fluid. Repeat liver function tests done 48 hours later showed resolution and patient was discharged after 72 hours of admission without any neurological deficits.

DISCUSSION

Neurotoxicity is a known characteristic of envenoming due to elapids (family Elapidae) such as kraits (Bungarus spp), cobras (Naja spp.), taipans (Oxyuranus spp.), coral snakes (Micrurus spp.) death adders (Acanthophis spp.), and tiger snakes (Notechis spp.).[5] Acute neuromuscular paralysis is the main type of neurotoxicity and is an important cause of morbidity and mortality related to snakebite. Mechanical ventilation, intensive care, antivenom treatment, antibiotics form an important role in the management of the patients with neurotoxicity. There is considerable variation between individual patients in the clinical manifestations following envenoming by any particular species. Snake venom is a complex cocktail of enzymes, polypeptides, non-enzymatic proteins, nucleotides, and other substances, many of which may have different neurotoxic properties.[6]

Many atypical delayed manifestations have been reported in neurotoxic snake bite including cardiotoxicity, critical illness neuropathy, myopathy and GB syndrome.[7] But acute acalculous cholecystitis following neurotoxic snake bite is a rare manifestation. This is one of the rare cases where patient presented initially with early morning acute acalculous cholecystitis in the absence of bite history or neurological features.[8,9] Such manifestation can pose a great diagnostic challenge to the physician. Therefore, detailed examination and close monitoring of such patients is required for clinical recovery of such patients. Snake bite has to be also kept in mind when patients with early morning neuromuscular paralysis are encountered in emergency department as they may not give relevant bite history.

The exact mechanism of acute acalculous cholecystitis in studied patient is not known. Snake venom probably undergoes hepatobiliary mode of excretion as demonstrated by Rocha et al in mice and this theory could explain the gall bladder inflammation in our case.[2] Snake venom also contains phospholipase A2, which produces local inflammation and tissue necrosis and hemorrhagin, which causes endothelial damage.[10] Also, neurotoxins and coagulants can release inflammatory mediators causing local inflammation and stasis of bile. Another possible theory postulated is change in the chemical composition of bile probably caused by phospholipase A2, which converts phospholipids in bile into toxic fatty acids and lysolecithin.[4] Formation of microcirculatory thrombi secondary to disseminated intravascular coagulation is another possible explanation for the occurrence of cholecystitis.

Emergency cholecystectomy is often considered because perforation or gangrene may occur in more than 50% of patients. However, in our case abdominal symptoms improved with antisnake venom and conservative treatment, and a follow-up ultrasound showed resolution of gall bladder oedema.

CONCLUSION

Acute acalculous cholecystitis occurs in critically ill patients due to stasis of bile and ischemia of the gallbladder wall from hypotension and microcirculatory thrombosis. Phospholipase A2, which causes local inflammation and necrosis, may play an important role in the pathogenesis of acute acalculous cholecystitis. The possibility of such complication as a presenting symptom following neurotoxic envenomation can pose a diagnostic challenge to the physician. Therefore, a detailed clinical examination and close monitoring of such patients is warranted for recovery of such patients.

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