CASE REPORT

Acute Acalculous Cholecystitis: A Rare Complication of Snake Bite

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Snake bite is an environmental and occupational hazard in many tropical and subtropical countries. It demands a high level of knowledge and skill in managing the envenomation syndrome. Herein, we describe a rare case of acute acalculous cholecystitis (AAC) that developed in a 36-year-old man after an Indian cobra (*Naja naja*) bite in the absence of any other predisposing factors for AAC. The probable mechanisms for the occurrence of AAC have been highlighted. Recognizing the possibility of such a complication after envenomation will definitely aid in early diagnosis and, subsequently, a better outcome.

Key words: cobra envenomation, acute acalculous cholecystitis, *Naja naja*

Introduction

Snake bite is an environmental and occupational hazard in many tropical and subtropical countries. The principal effect of envenomation is noticed on the nervous system, blood coagulability, and the site of the bite. We describe a rare case of acute acalculous cholecystitis (AAC) that developed in a 36-year-old man after a cobra bite in the absence of any other predisposing factors for AAC.

Case Report

A 36-year-old lean, male farmer without any comorbidity was bitten by an Indian cobra (*Naja naja*) on his right hand while he was cutting grass in the field, and exhibited features of envenomation. He was taken to a local hospital in South India 6 hours after the snake bite, where he was provided with 150 mL of polyvalent snake antivenom and supportive care. He did not require ventilator support. Although he was stable on the third day of the

bite, he had vomiting and right upper abdominal pain for which he was managed conservatively for approximately 12 hours. As the pain started increasing in severity, he was transferred to the emergency department (ED) of a tertiary care center. The pain was abrupt in onset, with associated right-shoulder discomfort. In the recent past he was not treated for burns, trauma, vasculitis, surgery, or sepsis. Also, he was not on any illicit drugs or herbal medications or immunocompromised.

In the ED, he was conscious, well oriented, febrile, and icteric. His vital signs were stable, and systemic examination was unremarkable. Abdominal examination revealed mild abdominal distention, guarding, and rigidity with tenderness in the right upper quadrant. Bowel sounds were hyperactive.

Laboratory results were unremarkable except for elevated serum total bilirubin (4.8 mg/dL) and liver enzymes. Aspartate transaminase (AST), alanine transaminase (ALT), and alkaline phosphatase (ALP) were 387, 284, and 289 IU/L, respectively. Infectious hepatitis screening panel was negative. Blood and urine cultures were negative. Admission electrocardiogram showed tachycardia with nonspecific T-wave changes. Chest radiography was not contributory. An urgent ultrasound (USG) of the abdomen revealed a thickened gallbladder

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Figure. Ultrasonogram of the abdomen showing distension of the gall bladder, thickening of the gallbladder wall (4 mm), a clear rim of pericholecystic fluid collection, and absence of gallstones.

wall (7 mm) and pericholecystic fluid collection without evidences of biliary dilatation, gallstones, or sludge (Figure). There was a positive sonographic Murphy's sign.

Based on the clinical and USG findings, a diagnosis of snake bite with envenomation complicated with AAC was made. On retrospective interrogation, there was no evidence of any hepatobiliary comorbid condition including alcohol consumption, cancer, hepatitis, and environmental toxin exposure. He was started on intravenous fluids, antibiotics, antipyretics, and analgesics. He became afebrile on the second day. Within a week's time abdominal pain subsided, and he was discharged on day 10. Repeat USG done 2 weeks later showed complete resolution of the previous ultrasonography findings.

Discussion

Acute acalculous cholecystitis is an uncommon but a potentially fatal abdominal emergency.^{1,2} The term AAC represents an acute necroinflammatory process of the gallbladder without evidence of calculi or sludge, and is usually seen in critically ill patients. Acute acalculous cholecystitis comprises only 10% of all cholecystitis,³ but has a more fulminant course compared with calculous cholecystitis. Mortality can be as high as 50% in such cases, and it depends mainly on coexisting medical or surgical conditions.⁴ Although the pathogenesis of AAC is not well defined, commonly postulated mechanisms are ischemia and reperfusion, infection, endotoxins, stasis and changes in bile, microangiopathy, and disruption of neurotransmitter release leading to gallbladder dysmotility.⁵ Multiple risk factors such as trauma, burns, infection, long-term total parenteral nutrition, major nonbiliary surgery, cholesterol emboli, multiple transfusions, vasculitis, mechanical ventilation,

and cardiopulmonary bypass have been associated^{6,7} with AAC, but none of them were present in our patient.

The exact mechanism of AAC in our patient is not clear. The implicated potential pathogenic mechanism in our patient is likely attributable to snake venom, which contains a mixture of proteins, enzymes, and other chemical agents. Any one of them or a combination of them might have caused a cytotoxic effect in the gallbladder and triggered the process. Also, the neurotoxins and coagulants might have contributed to the release of mediators resulting in local inflammation and stasis of bile, with alterations in the chemical composition of bile probably caused by phospholipase A₂, which converts phospholipids in bile into toxic fatty acids and lysolecithin.⁴ Rocha et al⁵ demonstrated excretion of a large portion of radiolabeled venom via the hepatobiliary route into the gallbladder in mice when it was administered by needle. Formation of microcirculatory thrombi secondary to disseminated intravascular coagulation is another plausible explanation for the occurrence of AAC.

The diagnosis of AAC requires a high degree of suspicion, as the sensitivity and specificity of both clinical and imaging evaluations are often misleading because of comorbidities. Ultrasound is the investigation of first choice and should be done whenever AAC is suspected. It is relatively inexpensive and can be performed at the bedside. Ultrasonographic features of AAC are thickening of the gallbladder wall (>5 mm) with pericholecystic fluid in the absence of gallstones or biliary sludge, positive Murphy's sign induced by the ultrasound probe, and lack of visualization of gallbladder and emphysematous cholecystitis with gas bubbles in the fundus of the gallbladder ("champagne sign"). Hepatobiliary scintigraphy, which is used when the diagnosis is difficult by ultrasonography, was not deemed necessary.⁷

Management should be instituted promptly with intravenous antibiotics after drawing blood for culture. Emergency cholecystectomy is often contemplated because perforation or gangrene may occur in more than 50% of patients. However, the surgery was not warranted in our case, as his symptoms subsided with conservative treatment, and a follow-up ultrasound demonstrated a nondilated, thin-wall gallbladder without lithiasis or sludge. Most cases reported in the literature had a poorer outcome than for the calculus variety of the disease owing to variable pathophysiology, associated comorbid conditions, and delay in diagnosis and treatment.

Acute acalculous cholecystitis was recognized by Nalawade et al⁸ in a patient with viper envenomation, who also had multiple-organ dysfunction and eventually succumbed to death. To the best of our knowledge, this is the first reported case of AAC after cobra snake envenomation. Unlike the fulminant course described by

Nalawade et al,⁸ our patient had a rather benign course with complete recovery. It is difficult to speculate as to why these patients had entirely different clinical courses. They had similar physical profiles, but had different types of envenomation, which could be a probable contributory factor. The possibility of this rare but potentially fatal complication needs to be considered in patients with snake bite and abdominal symptoms. Recognizing the possibility of such complication after envenomation will definitely aid in early diagnosis and, subsequently, a better outcome.

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