Case Report

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Gallbladder perforation in acute acalculous cholecystitis: an atypical manifestation of dengue fever

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ABSTRACT

Among the atypical manifestations of dengue fever, comprising 15.8% of all cases, acalculous cholecystitis forms a small subset. Acalculous cholecystitis is managed conservatively in majority of cases and a chance of gall bladder perforation is low (2-18%). Management of gall bladder perforation in the scenario of Dengue is sometimes complicated by presence of thrombocytopenia and its complications thereof. Mortality associated with gall bladder perforation is relatively high. Gallbladder usually perforates at the fundus and is to be dealt with surgically if it doesn't localize. Laparoscopic management of gall bladder perforation is feasible as calot's is relatively virgin (as there is no calculous disease). Thorough peritoneal toileting is possible and a chance of intra-abdominal abscess in post-operative period is a mere speculation. Laparoscopic management results in early recovery and fewer wound complications and lesser hospital stay.

Keywords: Dengue fever, Gall bladder perforation, Laparoscopic management

INTRODUCTION

Acalculous cholecystitis and gall bladder perforation are lesser known complications of Dengue. Atypical manifestations of Dengue fever are present in 15.8% of patients and include fulminant hepatitis, encephalopathy, cardiomyopathy, acute pancreatitis and acalculous cholecystitis. Incidence of acute acalculous cholecystitis in dengue is 27.5 %. Gall bladder perforation in acalculous cholecystitis has been reported in the range of 2-18%. We wish to report a rare case of Gall bladder perforation, complicating acute acalculous cholecystitis in dengue fever.

CASE REPORT

A 47-year-old male, hypertensive, presented with complaints of pain in upper abdomen (more in RUQ) for 1 week. It was associated with a high-grade fever. On

evaluation, he was found to have thrombocytopenia and Ig M and Ig G positive for dengue virus. USG of abdomen showed a mildly enlarged liver with altered echotexture, a distended gall bladder with wall edema and minimal ascitis. His Liver function test (LFT) did not show any derangement.

He was treated with antipyretics, intravenous fluids and monitored for any drop in platelet count. Pain in upper abdomen increased in intensity over the next few days, gradually spreading to the whole abdomen and was associated with high grade fever with chills and rigor. On examination he had high fever and sinus tachycardia (>120 per min).

Abdomen was soft except for severe tenderness in right hypochondrium with guarding & rigidity and sluggish bowel sounds. CECT of abdomen showed features of acalculous cholecystitis with focal contained perforation in fundal region with adjacent loculated collection in right subhepatic region and mild to moderate pleural effusion on the right side (Figure 1). He was started on antibiotics empirically (Piperacillin, Tazobactam and Doxycycline) and was planned for percutaneous intervention (drainage), but the procedure was deferred in view of thrombocytopenia. Random Donor platelets were transfused and patient was taken for a percutaneous drainage of collection. Percutaneous drainage was done and a 10 F malecot's catheter was placed in the right subhepatic space with the plan for a definitive surgery at a later date. Approximately 800 ml of bile drained over 24 hours. An ERCP was done which showed a contrast leak from gallbladder in gallbladder fossa (Figure 1). A stent (8Fr X 8 cm) was placed in the CBD. USG abdomen showed approximately 650 ml collection with multiple septations in right iliac fossae, prevesical space and left lumbar region. Another collection of approximately 120 ml was noted in right subhepatic space with catheter tip in situ and mild free fluid in perisplenic, left paracolic gutter and right hepatorenal pouch.

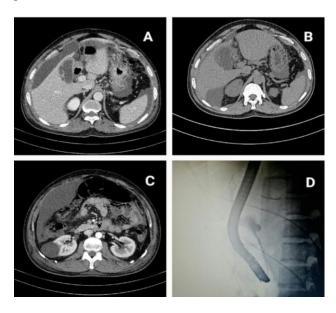


Figure 1: A- GB perforation with pericholecystic, perihepatic, perisplenic collections, B-Sub hepatic collection, C-RIF collection, D-ERCP showing contrast leak.

His condition showed no improvement and in view of rising TLC, persistent tachycardia and fever he was planned for surgery (diagnostic laparoscopy and proceed). A traditional four port position was used-10mm umbilical (camera), 10 mm epigastric (first working port), 5mm right midclavicular line beneath the coastal margin (second working port) and 5 mm right anterior axillary line 2 inches above iliac crest (assistant). Gall bladder was covered by omentum, transverse colon and stomach with bile in right subhepatic space. There was bile in pelvis and adhesions of small bowel with the parieties in right iliac and left lumbar region. Gentle adhesiolysis using blunt dissection with a suction irrigation instrument was performed which showed a

perforation at the fundus/body junction with the lateral edge of gallbladder. Calot's was relatively preserved (Figure 2) and a total cholecystectomy was performed, and specimen retrieved in bag through the 10 mm epigastric port. OT set up was changed and monitor was brought to the foot end of the table. Umbilical port was used as the first working port; camera was shifted to the epigastric port. An extra 5mm port was placed at the left lumbar region. Right anterior axillary and left lumbar port were used as the first and second working port respectively.

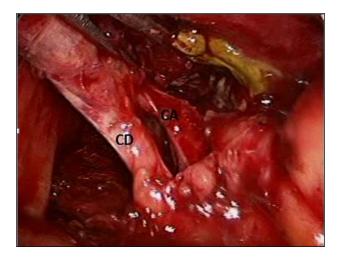


Figure 2: CA-Cystic artery, CD-Cystic duct.

Adhesions between small bowel and parieties were taken down and loculated collections were broken and emptied. Wide bore drains put through the right anterior axillary line in the Hepatorenal pouch of Morrison and through the left lumbar port in the pelvis. Surgery was done under cover of fresh frozen plasma (FFP) and platelets (RDP's). Biopsy of gall bladder revealed myonecrosis with features of inflammation (Figure 3).

Patient recovered over next few days and was discharged on fourth postoperative day.

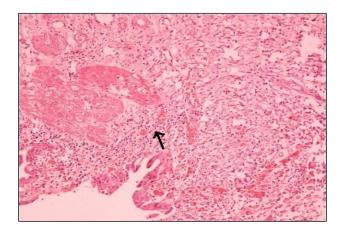


Figure 3: Histopathology directing towards myonecrosis.

DISCUSSION

Acalculous cholecystitis in the course of dengue is usually a self-limiting disease. Cholecystectomy is usually not indicated in these patients due to a high risk of bleeding.⁴ However, it may be necessary if gangrene, gallbladder wall perforation or diffuse peritonitis is suspected. Gallbladder perforation occurs most commonly at the fundus, which has the least blood supply. This underlines the ischemic mechanism of gallbladder perforation.^{3,5,6}

In 1934, Neimeier proposed a classification for GB perforation:⁷

- Type I Acute free perforation into peritoneal cavity.
- Type II- Subacute perforation with pericholecystic collection/abscess.
- Type III- Chronic perforation with cholecystoenteric fistula formation.

A number of modifications have been proposed but basic classification still stands good. Type I perforation are usually seen commonly in patients without any predisposing disease in the gall bladder and are seen as a result of trauma, iatrogenic or idiopathic spontaneous perforations. Type II perforations are common in calculus gallbladder disease. Type III perforations are seen in complicated calculus gall bladder disease. Management of patients with GB perforation mainly depends on general condition of patient. As most of them are critically ill, minimally invasive procedures like percutaneous drainage of collection with pigtail catheterization/ malecot's catheterization can be tried along with good antibiotic coverage, supportive treatment and plan for definitive surgery; once patient is stable. In patients who can sustain a laparotomy, the threshold for an early surgery should be low.8,9 The mortality associated with GB perforation is quite high.

In a study by Roslyn and Busutte, a mortality of 12-16 % was seen following GB perforation. Another study by Lennon F et al reports mortality in the range of 11-16 %. ¹⁰ In type I&II perforations, Calot's area is reasonably virgin and therefore laparoscopy should always be attempted first. In our patient, owing to thrombocytopenia and risks of bleeding thereof an initial decision for conservative management was taken.

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Institutional Ethics Committee

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