Case Report

Gall Bladder complications of Typhoid

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Abstract

Surgical complications of typhoid fever usually involve gut but infrequently typhoid fever also involves gallbladder. Complications range from acalculous cholecystitis to perforation. We present a series of three cases which included acalculous cholecystitis, gangrenous cholecystitis and perforation of gallbladder. Investigations revealed them to be a complication of Enteric fever. Acalculous cholecystitis was managed conservatively; gangrenous cholecystitis and perforated gallbladder underwent emergency cholecystectomy. All three cases were discharged after treatment and are doing well on follow up.

Key words: Gallbladder, perforation, typhoid

Introduction

Typhoid fever is a common infection in the tropics and one of the common causes of intestinal perforation resulting in high morbidity and mortality.[1] Among various complication of typhoid fever, acute cholecystitis is uncommon while perforation of gall bladder is extremely rare with dreadful consequences.[2] Surgical complications of typhoid usually involves the gut rather than the gallbladder and salmonella typhi is more common than parathyphi.[3] We present a case series of typhoid related complications of gall bladder ranging from inflammation to perforation.

Case 1

A 19 year old male presented with fever for 10 days along with right upper quadrant pain and vomiting for five days. Fever was of high grade without chills and rigor. Patient had undergone mitral valve replacement for mitral stenosis due to rheumatic heart disease. Symptomatology was not suggestive of infective endocarditis. Patient was febrile with tenderness, guarding and rigidity on right upper abdomen. Investigations revealed leucocytosis. Liver function test (LFT), Renal function test (RFT) and serum amylase was normal. Abdominal ultrasonography showed thickened gallbladder wall with positive sonographic Murphy's sign. No calculi were seen in the gallbladder and common bile duct. With clinical suspicion of acalculous cholecystitis due to enteric fever, patient was managed conservatively. Later diagnosis was supported by positive Widal test for Salmonella paratyphi. After seven days of conservative management, patient was discharged on oral antibiotics. On follow up patient had been doing well.

Case 2

A 40 year old male presented with history of fever for 8 days and sudden severe pain abdomen for a day. Pain was continuous and more on upper abdomen. There was no significant past medical, surgical or any drug history except that he had been taking NSAIDs for fever. He was ill looking, febrile with tachycardia. Abdominal examination was suggestive of peritonitis with tenderness more on upper abdomen. Leukocyte count was markedly raised (21,800/cu mm) with normal renal and liver function tests. Ultrasound abdomen showed free fluid in the peritoneal cavity without any obvious abnormality. With the suspicion of duodenal ulcer perforation peritonitis, exploratory laparotomy was done with upper abdominal incision. Gall bladder wall was significantly thickened with part of the body showing

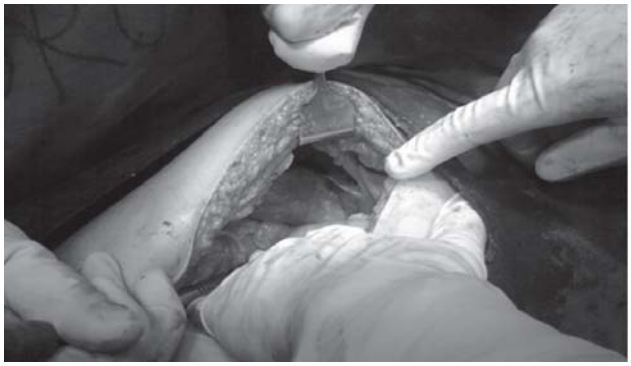

Fig. 1. Showing gangrenous patch on the wall of Gall Bladder

gangrenous changes. Thorough evaluation of the abdominal organs was done and no other abnormalities were observed. Cholecystectomy was done. Postoperatively antibiotics were continued and postoperative period was uneventful. Bile culture was positive for Salmonella typhi and sensitive to Ceftriaxone/ amikacin/ Gentamycin. This confirmed that the cause of the gangrenous gallbladder was salmonella typhi. Patient was discharged on 5th post operative day and was doing well on follow up.

Case 3

A female of 48 years presented with history of fever of 7 days. Fever was of high grade, continuous, associated with mild abdominal pain till a day before admission after which it became severe and generalized suggestive of peritonitis. There was no history of drug abuse, alcoholism, jaundice, trauma, recurrent biliary colic or chronic cholecystitis. Patient was ill looking and had tachycardia, tachypnea with raised temperature. Blood pressure was maintained. Abdomen was distended, rigid and tender with generalized rebound tenderness. There was leucocytosis but chest X ray did not reveal gas under the dome of diaphragm. USG abdomen revealed gross fluid collection in the peritoneal cavity with mild splenomegaly and thickened gallbladder wall. On this background, provisional diagnosis of typhoid fever induced small bowel perforation was made. After resuscitation, patient underwent laparotomy. On exploration, around two liters of bilious fluid was drained. After complete evaluation of the bowel loops and other parts of the gastrointestinal tract, grossly inflamed gallbladder with a small perforation of around 0.5cmX 0.5cm near the body was found (Fig 2). After thorough evaluation, it was confirmed that peritonitis was due to gallbladder perforation. Patient underwent cholecystectomy. Postoperative period remained uneventful and she was discharged after completing the course of antibiotics. Widal test was strongly positive for type O and H for S typhi but blood and bile culture did not grow any organisms.

Fig. 2. Showing perforation on the wall of Gall Bladder

Discussion

Acute acalculous cholecystitis describes inflammation of the gallbladder without evidence of stones. It occurs in approximately 2-15% of cases of acute cholecystitis with mortality rates ranging widely from 6% -67 %.[4] Typhoid complicated by cholecystitis has a reported incidence of 2.8% with 1.7% being acalculous.[5] Acute acalculous cholecystitis was first described in 1844 by Duncan.[6] In 1915, Lothrop reported the first case of acute acalculous cholecystitis as a complication of typhoid fever.[7] Typhoid fever may either be a primary or secondary event leading to acute acalculous cholecystitis. The pathophysiology of acalculous cholecystitis complicating typhoid fever is incompletely defined. Endotoxin-mediated injury seen in gram-negative sepsis is one proposed mechanism for the development of acute acalculous cholecystitis. These mediators lead finally to biliary stasis which results in increased bile viscosity, sludge formation and increases the gallbladder mucosal damage.[8] In turn, these mechanisms often result in functional or secondary mechanical obstruction of the cystic duct from inflammation and bile viscosity. Fasting, dehydration and narcotic analgesics are additional factors that predispose patients to biliary stasis. One of the proposed mechanisms includes abnormal permeability of serous membranes and capillary leakage as a result of direct invasion of the gallbladder wall by typhoid bacilli; in turn these mechanisms result in gallbladder distension and wall thickening. In our patient typhoid fever causing dehydration is probably a primary event that lead to acute acalculous cholecystitis .On rare occasions; typhoid bacilli may directly infect the gallbladder.[9] Routes of gallbladder infection by Typhoid bacilli: may be through blood stream, biliary system, contiguous infected organs

or lymphatics from gastrointestinal tract.[10] Typhoid cholecystitis is extremely rare among adults. A MEDLINE search showed only five cases of adult typhoidal acute acalculous cholecystitis reported in the literature. [7-14] We had reported a 20 year male who presented few years back at a different institute with fever for 10 days. He had generalized peritonitis and was found to have a perforated gallbladder on exploration. Widal was positive but the bile and blood cultures did not yield any growth.[21]

Uncomplicated enteric cholecystitis is managed conservatively with intravenous antibiotics while the complicated ones (gangrenous cholecystitis, perforation, chronic carrier state or relapse) are managed with cholecystectomy.[13]

Inflammation may progress and may cause ischemia and necrosis leading to perforation in 2-11% of patients with acute cholecystitis. Persistent occlusion of the cystic duct by the presence of an impacted stone, increased GB wall tension, epithelial injury, release of phospholipases, degradation of adjacent cell membranes, and intense inflammatory reaction lead to perforation.[15] In only 2% to 30% of patients, the disease progresses to gangrenous cholecystitis with wall perforation. Fundus of the gall bladder (GB) is the most distal site of vascular supply and most common site for perforation. In our patients the site of gangrenous change and perforation was body in contrast to that observed in the literature.[16] According to Niemeirer, spontaneous gallbladder perforation is classified into three categories: type I includes patients with free perforation in the peritoneal cavity, type II includes those with localized perforation, and type III consists of cholecysto-enteric fistulas.[17] Our patient had a Niemeirer's type I GB perforation, which was managed successfully by open cholecystectomy.

Delay in diagnosis and intervention for acute or gangrenous cholecystitis appears to increase the risk of perforation.[18] Early detection and intervention might decrease morbidity and mortality.[19] Role of imaging modalities in the diagnosis of perforation is yet to be established. Ultrasound has repeatedly been shown to have low sensitivity in detecting perforation. Computerized tomography scan, cholescintigraphy or a combination has shown to improve the diagnostic accuracy but still fail to detect all cases.[20]

Conclusion

Typhoid usually involves bowel when complicated but gall bladder complications are still prevalent in the tropics. Complications vary from cholecystitis to perforation. It requires high clinical judgment, supported by investigations for establishing the diagnosis of typhoid cholecystitis, gangrenous cholecystitis and perforation. Timely diagnosis and management has good outcome.

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