GALL BLADDER GANGRENE AND PERFORATION: REVIEW OF LITERATURE AND A CASE REPORT

Ghosh Yatin¹, Nagi Gurinderjit Singh²*

¹MS, Associate Professor, Surgery, PIMS (Punjab Institute of Medical Sciences) Medical College, Garha Road, Jalandhar, Punjab, India
²MS, Assistant Professor Surgery, PIMS (Punjab Institute of Medical Sciences) Medical College, Garah Road, Jalandhar, Punjab, India

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*Corresponding Author: Dr Gurinderjit Singh Nagi, Assistant Professor Surgery, PIMS Medical College, Garha Road, Jalandhar, 144006, Punjab, Mobile: 09814657900

ABSTRACT

Enteric fever is common among the lower socioeconomic class of society. Enteric fever has a wide range of complications which in this modern era has decreased due to better health facilities. Though enteric fever is associated with various pathologies of the gut including acute cholecystitis but perforation of the gall bladder with gangrene is a very rare event. We report a case with enteric fever with gall bladder gangrene and perforation in a 11 years old girl.

Keywords: Enteric Fever, Gall Bladder, Gangrene, Perforation.

INTRODUCTION

Typhoid fever is a common infection in the tropics and one of the common causes of intestinal perforation resulting in high morbidity and mortality¹. However it is rare in surgical practice². Gall bladder perforation is extremely rare in children³. There may be no specific presentation⁴ and diagnosis may not be established preoperatively⁵. Among various complications of typhoid fever, acute cholecystitis is uncommon while perforation of gall bladder is extremely rare with dreadful consequences. Spontaneous perforation in calculus cholecystitis is infrequent and even rarer in the absence of gallstones, but occasionally occurs following typhoid fever. If such conditions are not treated in time, mortality rates are very high⁶. At present the mortality is decreased to nil⁷-¹⁰. Gall bladder gangrene is noted to have high frequency among immune compromised patients or patients with intense inflammation of the gallbladder from enteric fever because Salmonella Typhi multiply in the bile in very high titers and are further concentrated in the gallbladder. Hollow viscous perforations are common in patients with enteric fever and are commonly seen in our hospital.

CASE REPORT

An 11 years old female presented with history of fever and abdominal pain 4 days prior to presentation to casualty in PIMS (Punjab institute of medical Sciences Jalandhar). She was apparently well when she started to have high grade fever (101-102F) not associated with chills and rigors. She was taken to local practitioner for her fever but the fever continued and on the second day she developed severe abdominal pain (epigastric) with radiation of the pain to the flanks and umbilicus. She was admitted under pediatrics and was managed conservatively. She also had history of melena two episodes and her platelet counts were only 46000 at the time of admission, for which she received 2 units of packed red blood cells. Her repeat platelet was 96,000. On examination she had pallor, no icterus, no cyanosis, no lymphadenopathy. Pulse rate of 90/min and Blood pressure of 90/50 mm Hg, respiratory rate of 15 breath/ min. Her abdomen was soft with minimal distension and no guarding, rigidity or signs of peritoneal irritation, though her bowel sounds were absent and shifting dullness was present. Rest of the examination was unremarkable. On investigation her chest x-ray was unremarkable, her Hb was 9 gms% and a total leukocyte count of 9,000 /cmm only. Widal test was positive upto 1:320 and working diagnosis of enteric fever was made. A previous ultrasound done had shown a collection in the RT iliac fossa with internal echoes and a distended gall bladder. The CECT abdomen had shown no free air in the peritoneum and a distended gall bladder with thickened wall and a periholecystic collection and collection in the Morrison pouch. Perforation of the gall bladder was suspected. Initially a decision to defer the surgery was made due to absence of features of peritoneal irritation/peritonitis and hollow viscous perforation. A decision to tap the abdomen in right flank proved worthwhile as it yielded frank bile from the aspirate which was send for culture. The patient was immediately
taken up for laparotomy and the operative findings were Biliary peritonitis, gangrenous distended gall bladder with perforation in the body. After Cholecystectomy, thorough peritoneal cavity lavage was done with saline and abdomen was closed in layers after putting in drain in the Morrison’s pouch. The patient was shifted to ICU after extubation. The intestinal peristalsis was present and the patient passed flatus on the third day. The patient remained afebrile after day 2 and vitals were within normal range. The oral feed was started on fourth day which she tolerated well.

Figure: (A)

Figure (A) and (B) are Photographs taken during surgery

DISCUSSION

Enteric (typhoid) fever is a systemic disease characterized by fever and abdominal pain and caused by dissemination of S. typhi or S. paratyphi. Typhoid fever is a common infection in the tropics and one of the common causes of intestinal perforation resulting in high morbidity and mortality. Surgical complications of typhoid usually involve the gut rather than the gallbladder and salmonella typhi is more common than parathiyphi. Typhoid complicated by cholecystitis has a reported incidence of 2.8% with 1.7% being acalculous. Acute acalculous cholecystitis was first described in 1844 by Duncan and Lothrop reported the first case of acute acalculous cholecystitis as a complication of typhoid fever. Gallbladder perforation usually occur in acute obstructive (calculus) cholecystitis, and its incidence is high in patients with diabetes mellitus, recent severe trauma, critical illness, severe burn. Spontaneous gallbladder perforation (GBP) may be caused by an inflammatory reaction, weakness and necrosis of its wall. The most common site of gallbladder perforation is the fundus, because this is the most distal part with regard to blood supply. For prompt diagnosis early diagnosis and early intervention is needed. Typhoid GBP is a rare surgical sequel of typhoid infection and rarely suspected preoperatively where typhoid ileal perforation is suspected. Immunosuppression, virulent organisms and presence of intense inflammation has been attributed in cases of acalculous typhoid gall bladder perforation. On rare occasions; typhoid bacilli may directly infect the gallbladder. Routes of gallbladder infection by Typhoid bacilli: may be through blood stream, biliary system, contiguous infected organs or lymphatics from gastrointestinal tract and rarely typhoid bacilli may infect Gall bladder directly. Delay in diagnosis and intervention for acute or gangrenous cholecystitis appears to increase the risk of perforation. Ultrasound has low sensitivity in detecting perforation and Computerized tomography scan has shown to improve the diagnostic accuracy but still fail to detect all cases. Ultrasound finding of gallbladder wall thickening (>3.5 mm), gallbladder distention, pericholecystic fluid, and positive sonographic Murphy's sign are significant and some time present in GBP.

CONCLUSION

Although we manage uncomplicated enteric cholecystitis but complicated cases like gangrenous or perforated cases of gall bladder are managed surgically requiring cholecystectomy. Gall bladder should also be considered in differential diagnosis of peritonitis in cases of enteric fever. Though abdominal USG and CECT have it limitation in making the diagnosis of GB perforation, yet GB wall thickening, GB distention, pericholecystic fluid, positive sonographic Murphy's sign are significant and tapping the abdomen and aspiration of free bile invariably will clinch the diagnosis.

REFERENCES


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