Typhoid Perforation of Gallbladder- A Rare Pathology
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Abstract:
Surgical complication of Typhoid fever when occurs, it commonly involves gut rather than the gallbladder. This is a case report of 24 years young male who came to emergency with the complaint of fever of 12 days duration and sudden severe pain in the abdomen for 2 days. Patient was evaluated and investigated. He underwent laparotomy; on laprotomy, gallbladder was found to have perforation and therefore, cholecystectomy was done. The investigations supported the diagnosis of Typhoid fever. Patient recovered uneventfully and was discharged after 10-days.

Key Words: Perforation peritonitis, Gallbladder, Typhoid.

Introduction:
Typhoid infection is common in this region of our country. It is a common cause of intestinal perforation resulting in high morbidity and mortality (Archampong, 1976 ; Ugwu et al , 2005) . Among the various complications of this condition, gallbladder perforation is uncommon and has high mortality if not managed promptly. Acute abdominal pain is one of the common presentations in emergency but the diagnosis of gallbladder perforation is infrequently considered in the absence of pre-existing gallbladder disease. The clinical presentation of gallbladder perforation may range from acute generalized peritonitis to non-specific abdominal presentations. Often the radiological investigations may not detect this pathology so as to make an early diagnosis.

Case Report:
A 24 year male presented to the Emergency of People’s Hospital with fever of twelve days duration and moderate abdominal pain. Two days prior to his admission he had severe abdominal pain with two episodes of vomiting. He was hemodynamically stable. The abdominal examination showed distension, diffuse tenderness and guarding. Abdominal X-ray showed mildly distended bowel- loops without any gas-fluid levels or free peritoneal gas; chest radiograph was within normal limits and there was no free gas under the diaphragm. Abdominal sonography revealed moderate ascites with internal septation. Wall of the gall bladder was found to be thickened and thick echogenic sludge was seen within it. Laboratory reports documented a white blood cell count of 9,500 cu/mm with 85% neutrophils and liver function tests were within normal limits. The widal test was positive for ‘O’ & ‘H’ antigen in high titres.

Fig. I: Photograph showing collapsed gallbladder and fluid in peritoneal spaces

A contrast enhanced computerized tomography scan of the abdomen suggested fluid accumulation in the perihepatic, subphrenic, and both paracolic gutters; collapsed gallbladder with poor definition of its thickened wall, two air pockets within the pericholecystic fluid; enlarged mesenteric lymph nodes and mild hepatosplenomegaly.

In view of signs of generalized peritonitis, laparotomy was planned. During surgery, 1.5-2 litre of bile stained peritoneal fluid was drained. While evaluating the duodenum, a moderately inflammed gall bladder was seen with a perforation of
approximately 8mm near the fundus. The gall bladder was filled with biliary sludge and it was non-lithiatic. The peritoneal cavity was thoroughly explored and it was concluded that primary pathology causing peritonitis was gall bladder perforation. Cholecystectomy was performed. The patient had an uneventful recovery and was discharged after 10 days.

**Discussion:**

The chances of gall bladder perforation is more in cases of obstruction of the Gallbladder, resulting in steep rise of intraluminal pressure as it happens in calculus disease of gallbladder. Non obstructive cholecystitis is less likely to cause perforation of the viscus (Essenhigh, 1968).

The intense inflammation coupled with infection with more virulent organisms and existence of a immuno compromised state leads to thrombosis of the blood vessels which predisposes to the setting of transmural necrosis which eventually leads to perforation. Roslyn & Bustte suggested that spontaneous gall bladder perforation is caused by hypoperfusion of the viscera, secondary to systemic disease and also reported that fundus of the gall bladder is the most common site of the perforation (Archampong, 1976). Derici et al (2006) suggested that when GB is perforated at the fundus, there is a possibility that it is not covered by omentum and thus bile drains into the peritoneal spaces leading to peritonitis as it occurs in the other common causes like the peptic ulcer perforation. The occurance of spontaneous gallbladder perforation (GBP) or gangrene in acalculous cholecystitis is rare following typhoid fever (Niemeier, 1934). Perforations of the gallbladder usually occurs in the presence of gall-stone disease, gallbladder malignancies, presence of risk factors e.g. atherosclerosis, diabetes mellitus, congenital anomalies of the biliary system, Gallbladder infections, and pancreatitis. Perforation of the gallbladder was first reported by Duncan of the Royal Infirmary,Edinburg about 135 years ago (Gonsalves, 1979). Gall bladder perforations due to typhoid fever, though known, have been rarely reported over the past two decades (Saxena et al, 2007). Ergul & Gozetlik (2008) in their retrospective study of 1042 cases of Acute Cholecystitis found the incidence of Gallbladder perforation to be 3.55% (37 cases); of which 4 cases were of acalculous type (10.8%). 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 rate ranging from 6% -67 %. Typhoid complicated by cholecystitis has a reported incidence of 2.8% with 1.7% being acalculous (Bhandari et al, 2009). Further, with limited successes of the ultrasound and CT scan for detecting gallbladder perforation and in the absence of specific clinical features/laboratory inputs, an early surgery (laparotomy) or a diagnostic laparoscopy are the final means to reach the diagnosis (Ergul & Gozetlik, 2008). Kim et al (1994) in their comparative study of the CT and Ultrasonography with gallbladder...
perforation, detected the site of perforation in 50% of the patients on CT but in none of the patient on ultrasonography.

Peritonitis due to gall bladder perforation is associated with high mortality rate of 39.1% (Essenhigh, 1968). Due to high mortality, timely diagnosis and management of perforated gall bladder is highly crucial. Perforation of the gall bladder must be treated surgically when the diagnosis is made or suspected. Niemeier (1934) classified gallbladder perforations: generalized peritonitis as acute or type I; pericholecystic abscess and localized peritonitis as subacute or type II and Cholecystoenteric fistula as chronic or type III.

The stage & type of presentation may vary; in the present case presentation was delayed. Every case of gallbladder perforation may not have necrosis or gangrene, but may have small perforation as seen by Gunturi et al (2012).

The diagnosis of gallbladder perforation requires high index of suspicion, often it is a per-operative diagnosis. The situation demands prompt and challenging management (Budensab & Annigeri, 2012). The outcome is invariably good with early intervention and cholecystectomy. The preferred procedure is emergency cholecystectomy. Cholecystostomy is an acceptable alternative especially in poor risk patients (Singh et al, 2010).

Conclusion:

Spontaneous perforation of the gallbladder is rarely seen in the absence of gallstone but gall bladder perforation is an occasional complication of typhoid. In patients of typhoid, signs of peritonitis are commonly due to intestinal perforation. Gallbladder perforation secondary to typhoid fever should be considered as a differential diagnosis in patients with suspected typhoid enteric perforation. Abdominal Ultrasound and Computer tomogram scan has limitations and may not be of help in diagnosing this pathology. However, an early surgical intervention should lead to the intra-operative diagnosis and definitive management.

References: