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Gall bladder perforation: report of 3 cases

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Summary
We report 3 cases of gall bladder perforation treated in this centre between January 1996 and June 2001. These were compared with Neimier's original classification of types of gall bladder perforation and areas of difficulty in management emphasised. The literature was also reviewed.

Keywords: *Gall bladder perforation, subtotal cholecystectomy.*

Résumé
Nous rapportons 3 cas de perforation de la vésicule biliaire traités dans ce centre entre janvier 1996 et juin 2001. Ceux-ci ont été comparés avec la classification de l'originale de Neimier de types de perforation de la vésicule biliaire et des zones de difficulté dans la gestion ont ete accentuée. L'ouvrage a aussi été revue.

Introduction

Gall bladder perforation is one of the complications of acute cholecystitis. This complication though more common with calculus cholecystitis, a significant number of perforations occur in patients with acalculus cholecystitis [1,2].

Cholelithiasis is less common in the West African sub-region with prevalence ranging from 0.007% in the general population to about 2% in pregnant women [3]. The incidence in other parts of the world vary. It is about 10% in America. In England, it occurred in 6.2% of male adults and 12.2% of female adults in South Wales [4,5].

Gall bladder perforation is seen in 4.6% to 10.6% of patients with acute cholecystitis [1,2]. The rate of this complication in Africans is not well documented. Thirty-four (34) cholecystectomies were done in this centre between January 1996 and June 2001. Three (3) of these patients had gall bladder perforation (8.8%). Each case typically exemplifies one type of gall bladder perforation described by Neimier.

Case 1

Mrs. I. A., a 60 year old woman presented at the emergency unit with one day history of periumbilical colicky abdominal pain. There was associated nausea and she had vomited about ten times. There was associated fever and rigors. She had more than twenty (20) years history of recurrent epigastric pain. The last episode occurred about 3 years prior to presentation and was managed in a private hospital.

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On examination, she was ill looking, dehydrated and mildly pale. She had a pulse rate of 104/min and a blood pressure of 100/70mmHg. Her abdomen was distended. She had an everted tender umbilicus and a reducible paraumbilical hernia with a defect about 4cm in diameter. There was generalised tenderness with guarding and rebound. Bowel sounds were hypoactive.

A diagnosis of peritonitis due to perforated peptic ulcer disease was made. PCV was 34% and total WBC 8 x 10^9/L. An initial urea level of 8.0mmol/L was corrected after resuscitation with intravenous fluids and antibiotics.

She had an exploratory laparotomy 23 hours after presentation. A huge gall bladder filled with purulent material and many dark stones was found (Fig1). A big stone with a diameter of about 4.5cm was found in the Hartmann's pouch. There was a perforation on the inferior side of the Hartmann's pouch adjacent to the stone. The Calot's triangle could not be assessed.

Fig. 1:

She had a subtotal cholecystectomy and a corrugated rubber drain was inserted in the subhepatic space. She had an uneventful postoperative period and was discharged nine (9) days post operation. She was followed up for a year with no complaints.

Case 2

Mr. A. J., a 75 year old man presented at the emergency unit with three weeks history of fever and abdominal pain. The fever was associated with rigors and vomiting. He was ill-looking, febrile and had a distended abdomen that was tender. There was guarding and rebound tenderness. His preoperative diagnosis was peritonitis due to ileal perforation from typhoid fever. He had a PCV of 31% and a total WBC of 3.0 x 10^9/L. The plain abdominal radiograph showed abdominal distension with a generalised ground glass appearance. There were multiple air fluid levels. There was no visible free air in the peritoneal cavity. He was

operated upon on the day of admission. He had subhepatic and right subphrenic collection of frank pus walled off by the omentum, stomach and transverse colon from the general peritoneal cavity. The fundus of the gall bladder was gangrenous and the Calot's triangle was difficult to define. A gallstone was found free in the peritoneal cavity

A subtotal cholecystectomy was done piece meal and the subhepatic and right subphrenic spaces drained. Post operatively, he had acute renal failure which was managed with fluid restriction and drugs. He was discharged two weeks after operation. He was readmitted two weeks after discharge with periumbilical pain and features of septiceamia. WBC was 14.7 x 10^9 and PVC 27%. He had hypoproteineamia and ascites. He improved on antibiotics and blood transfusion and was discharged 17 days after readmission. He was last seen in clinic 5 months after discharge.

Case 3

Mrs. M. N., a 35 year old woman presented at the emergency unit with epigastric and right hypochondrial pain of 6 days duration. There was associated vomiting and fever. She had previous episodes that were managed in private hospitals. On one of such episodes about 2 years prior to presentation, an abdominal ultrasound scan suggested acute calculus cholecystitis. She was ill-looking and in pain. She was not pale, not jaundiced and afebrile. Pulse rate was 72/min and BP 100/80mmHg. She had right hypochondrial tenderness and a positive Murphy's sign. Her PCV was 34% and total WBC of 4.0 x 10^9/L. Abdominal ultrasonography did not visualize the gall bladder but showed three gall stones in the area of gall bladder and suggested stones within the cystic duct. She improved on antibiotics and analgesics and was discharged home after one week.

She had cholecystectomy two weeks after. At operation, a gall bladder with severe fibrosis and a fistula between the Hartmann's pouch and the duodenum was found. No stone was found in the gall bladder. Cholecystectomy and closure of the fistulous opening into the duodenum was done. A drain was inserted in the subhepatic space.

Post operatively, she had chest pain that improved after chest physiotherapy. She was discharged twelve (12) days after operation. She defaulted after a follow up of about 6 weeks.

Discussion

The pathogenesis of gall bladder perforation is not well documented [6]. It was believed that gall bladder ischeamia was the principal aetiology because it occurred more in elderly patients who tended to have systemic illness such as coronary artery disease. Several studies also showed that the gall bladder fundus (which has the least vascularity) was the most common site of perforation [6].

More recently, it has been hypothesized that the pathogenesis differ in the different types of perforation [7]. Neimier classified gall bladder perforation into three types. Types I and II perforations usually follow acute cholecystitis with gangrene of the gall bladder. Gangrenous cholecystitis occurs after acute cholecystitis and it is more common in the elderly [8.9.10].

Type I perforation is an acute perforation of the gall bladder into the free peritoneal cavity without protective adhesions (Case I). It has been suggested that this type tends to occur in immunocompromised patients where a mild inflammatory process leads to severe oedema and gangrene. This type of perforation has the highest mortality rate and could be as high as 46% [8].

Type II perforation is a subacute perforation where the perforated gall bladder is surrounded by an abscess walled off by adhesions from the peritoneal cavity (Case 2). This is the commonest type in most series [6].

Type III perforation is a chronic perforation with the presence of a fistulous communication between the gall bladder and another viscus or the skin (Case 3). Viscera commonly involved are the duodenum, the stomach, the colon and the common bile duct [9]. Patients with this type tend to be older and have a history of biliary tract disease [7]. Type III perforation results from pressure necrosis and inflammation around impacted gall stones.

The presentation of gall bladder perforation is not specific and often difficult to differentiate from acute cholecysitis though fever and abdominal mass occur more commonly after perforation [7]. This difficulty usually leads to delay in diagnosis and mismanagement. Advocates of early surgery for acute cholecystitis hoped to reduce this complication.

The only preoperative laboratory value constantly associated with perforation after gall bladder gangrene is a leucocytosis above 15,000 WBC/ml [8]. Imaging studies are neither sensitive nor specific [6]. Plain radiographs of the abdomen are only useful if they show gall stones in the peritoneal cavity or air in the biliary tract [6,13]. Though a gall stone was found free in the peritoneal cavity in Case 2, it did not show on preoperative plain abdominal X-ray.

Cholescintigraphy is more sensitive in Type I perforation if extravasation of the radionuclide is seen. This requires patency of the cystic duct which is usually not present in acute cholecystitis [6,13]. In Type II perforation, subtle cholescintigraphy findings include pericholecystic hepatic activity along the inferior hepatic border, an enlarged well demarcated photogenic region in the gall bladder fossa, a mass effect on the right inferior hepatic lobe and a medial displacement of the common bile duct. These findings however have a low sensitivity [6,3]. CT scanning and ultrasonography effectively demonstrates fluid collections in Types I and II perforations but these findings are nonspecific.

Difficulty in preoperative diagnosis in these patients cannot be over emphasised. None of the three patients presented was diagnosed preoperatively. Ultrasonography which might have been useful in diagnosis in Cases I and 2 was not done. This is probably because gall bladder perforation was not suspected as the cause of generalised peritonitis in these patients. In some series, accurate preoperative diagnosis was made in only 10% of patients. Most diagnosis are made at surgery or at autopsy [2,11].

The treatment of gall bladder perforation should be surgical as patients treated medically may die. Cholecystectomy or cholecystostomy with drainage of collections is the treatment of choice. Two of the patients presented had subtotal cholecystectomy described as the removal of the gall bladder (sometimes in piece meal) leaving behind the cystic duct. The duct is secured at its origin with a purse string suture [14,15,16]. This procedure is recommended in difficult gall bladder surgery in patients with severe inflammation or fibrosis in the Calot's triangle and in patients with portal hypertension. The rate of post cholecystectomy syndrome in patients treated with subtotal cholecystectomy is low [15].

Significant morbidity occurred in Case 2 and can be attributed to septicaemia. He had renal failure and was later readmitted with symptoms of sepsis and hypoprotemeamia. Morbidity and mortality rates after gall bladder perforation can be extremely high. Perioperative mortality rates vary between 10%-25% and morbidity rates may be greater than 50% [6]. These high rates seem to be attributable to delay in diagnosis of perforation with subsequent delay in treatment. Heightened vigilance with resulting decrease in preoperative delay can reduce this appreciably [1].

In conclusion, gall bladder perforation is a relatively infrequent complication of acute cholecystitis. It carries a high morbidity and mortality that can be reduced appreciably by increased awareness of the condition. Emergency cholecystectomy should be considered in the elderly with acute cholecystitis who present with leucocytosis or fail to improve appreciably on antibiotics.

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