

Unusual Presentation of Gall Bladder Perforation

*Egwuonwu OA¹, Chianakwana GU², Ulebe OA²
Mbaeri TU²

ABSTRACT

Background: *Gall bladder perforation (GBP) is a rare but life threatening complication of cholecystitis. Therefore, it continues to be an important problem for the surgeon.*

Aim: *To report a case of perforated gall bladder mimicking perforated peptic ulcer disease.*

Methods: *A case report of gall bladder perforation that was managed at the Nnamdi Azikiwe University Teaching Hospital.*

Result: *A 67 years old woman who had a clinical diagnosis of perforated peptic ulcer disease secondary to chronic NSAID use but with intra-operative finding of Niemeier's type II gall bladder perforation.*

Conclusion: *High index of suspicion is required to diagnose perforated gall bladder mimicking perforated peptic ulcer disease.*

Keywords: Gall bladder perforation, Peptic ulcer disease, Niemeier's Classification

INTRODUCTION

Gallbladder perforation (GBP) is a rare but life threatening complication of cholecystitis. Therefore, it continues to be an important problem for the surgeon. Despite the fact that early recognition and treatment would decrease the mortality and morbidity, the disease still remains a diagnostic dilemma. The purpose of presenting this case is that perforated gall bladder mimicking perforated PUD should be kept in mind as one of the differential diagnoses of perforated viscus.

CASE REPORT

Mrs N. T, a 68 year-old trader, presented to the accident and emergency department of Nnamdi Azikiwe University Teaching Hospital (NAUTH), Nnewi with complaint of abdominal pain of three weeks duration. She was in apparent good health until three weeks prior to presentation when she developed abdominal pain which was of sudden onset following ingestion of non-steroidal anti-inflammatory drugs (NSAIDs) for

treatment of low back pain. The abdominal pain was located in the epigastrium and right hypochondrium. It was initially dull and intermittent but it later became severe and continuous. It did not radiate, and it was not related to ingestion of any type of food. There was no known relieving or aggravating factor. Two days into the illness, the patient vomited twice. Vomiting was non-projectile and the vomitus was bilious, and measured about 150ml in each episode. There was no haematemesis, abdominal distension or abdominal mass. Although the patient was not known to be suffering from peptic ulcer disease (PUD), she had history of passage of melena stool. There was history of chronic ingestion of NSAIDs for low back pain. She did not have yellowness of the eyes or fever. The patient did not have any trauma to the abdomen. She had a similar episode of abdominal pain 2 years before presentation and was treated with antacid and proton pump inhibitor at a peripheral hospital as an out-patient. She had full recovery at that time. At the onset of this present episode of abdominal pain, the patient presented to a peripheral hospital where she was admitted for two days and was placed on parenteral antibiotics and anti-ulcer regimen, without any improvement. She was therefore referred to NAUTH for further management. She had never been on admission in a hospital prior to this present illness. The patient neither ingested alcohol nor used tobacco in any form.

Examination revealed an obese woman in painful distress, afebrile, not pale, anicteric, not dehydrated and with no pedal oedema, digital clubbing or peripheral lymphadenopathy. Her pulse rate was 100 beats/min; her blood pressure was 120/70 mmHg, and her respiratory rate was 30 cycles/min, with a temperature of 37.1°C. Her abdomen was full and moved with respiration; her umbilicus was inverted, and her hernia orifices were intact. There was marked tenderness over the epigastrium and right hypochondrium with guarding and rigidity. The liver and spleen could not be assessed due to tenderness. The kidneys were not ballotable. There was no demonstrable ascites, and abdominal percussion notes were tympanitic. Her bowel sounds were normoactive. Rectal examination was normal and other systems were essentially normal.

A clinical diagnosis of perforated peptic ulcer disease secondary to chronic NSAID use was made.

Radiograph of chest and upper abdomen did not show any evidence of air under the diaphragm. The

¹Department of Surgery, Federal Medical Center, Owerri

²Department of Surgery, Nnamdi Azikiwe University Teaching Hospital, Nnewi

*E-mail: egwuobi@yahoo.com.

abdominal ultrasound scan report noted that gall bladder was not visualized post prandial, and that there was a right sub-phrenic collection containing internal debris and strands, plus presence of free pelvic fluid. Other viscera were sonographically normal. Because of these findings, the impression of sub-phrenic abscess was made. CT-scan was advised but could not be done because of financial constraint.

The patient's haemoglobin concentration was 11.3g/dl, WBC= 10,000 cells/microliter; with a differential neutrophil count of 65.0%. Urinalysis result was normal.

Nasogastric tube was passed for drainage and urethral catheter was inserted for urine monitoring. The patient was worked up for emergency exploratory laparotomy and the intra-operative findings were as follows:

- (1) Approximately three (3) litres of bilious fluid, mostly in the sub-phrenic space.
- (2) Multiple biliary stones. Some were found inside the gall bladder and some were scattered in the bilious fluid, each < 0.5 cm in diameter. The total number of stones recovered from the sub-phrenic fossa and gall bladder was 39.
- (3) Perforated gall bladder. The perforation was at the fundus, and the wall of the gall bladder was fibrotic.
- (4) Thick, fibrous adhesions binding the omentum, the gall bladder, the left lobe of the liver, the stomach and the duodenum.

Tube cholecystostomy was done using Foley's catheter size 20-G. The right sub-hepatic fossa was drained with Foley's catheter size 22G. Immediate postop status was satisfactory. The intra-operative diagnosis was perforated gallbladder (Niemeier's Type II) secondary to calculous cholecystitis.

The patient was placed on nil per os, intravenous fluids (Two litres of 5% dextrose water alternated with 1 litre of Normal saline in between), given in the first 48 hours. On the third day post-op, with adequate urine output, one litre of full strength Darrows solution replaced one litre of 5% Dextrose in water. She was also placed on intravenous pentazocine, 30mg 8 hourly for the first 72 hours. This was replaced with intramuscular diclofenac sodium injection 100mg daily for 48 hours. She was also placed on subcutaneous low molecular weight heparin (40mg of Clexane) once a day.

With the passage of flatus, graded oral sips were commenced on 5th post-operative day after removal of the nasogastric tube. The patient was allowed low residue diet and was later stepped up to regular diet. Skin sutures were removed on the 9th post-operative

day. Wound drain was removed on the 13th post-op day. The cholecystostomy tube was removed on the 17th post-op day. The resultant cholecystocutaneous fistula closed on the 28th day post-op, following which the patient was discharged home and was booked for Interval cholecystectomy which was to be done in the next six (6) weeks. She was seen two (2) weeks after discharge and was still in good health. A repeat of abdominal ultrasound scan done 4 weeks after discharge showed a fibrotic, chronically inflamed gall bladder containing multiple stones. The patient thereafter, defaulted from clinic appointments and lost to follow up as her caregivers were not interested in further surgical procedures despite repeated counseling.

DISCUSSION

Niemeier⁴ in 1934, classified GBP into 3 types: free gallbladder perforation and generalized biliary peritonitis as Acute or Type 1 GBP; pericholecystic abscess and localized peritonitis as Sub-acute or Type 2 GBP, and cholecystoenteric fistula as Chronic or Type 3 GBP. This classification is still in use. There are forms that are unclassified; they include intrahepatic rupture and cholecysto-choledochal fistula. Most cases can only be diagnosed during surgery^{1,2}. Morbidity and mortality are significant, with mortality being up to 42.0%³ in neglected cases. Morbidity and mortality are higher in the elderly, in immunodeficiency, in cases of malignancy and in acute perforation where the outcome is 10 times worse than in the sub-acute perforation.

Our case falls into Type II. But the incidence of chronic cholecystitis by clinical history in cases of sub-acute gall bladder perforation is about 35.0%⁵. It has been reported that type?GBP occurs more frequently^{1,6,7}.

As in our case, GB fundus, the most distal part with regard to blood supply, is the most common site of perforation⁷.

Despite improvement in radiological diagnosis with the advent of ultrasonography (US), computed tomography, and biliary scintigraphy, it is still difficult to diagnose type II and type III perforation. Kim et al⁸ and Derici et al⁹ reported that the site of defect could not be visualized on US in any patients, just like in our case. On the other hand, CT-scan can show more accurate signs of free intra-peritoneal fluid, pericholecystic fluid, and abscess^{1,10}. CT-scan can also show GB wall thickness and the defect on the wall due to perforation^{10,11}. CT-scan was not available in our centre at the time of this work. The nearest centre where it was available when the patient presented was about 200km away hence CT-scan could not be done even though it was

recommended by the radiologist after the abdominal ultrasound scan.

Although the intra-operative finding in our patient was GBP, the clinical history, especially of chronic NSAIDs use, the clinical signs and the abdominal ultrasound findings made us to initially entertain the diagnosis of perforated peptic ulcer disease which was probably sealed by omentum. To make a pre-op diagnosis of type II GBP, there must be a high index of suspicion. Efforts must be made to get a CT-scan which would improve diagnostic accuracy.

REFERENCES

1. Roslyn JJ, Thompson JE Jr, Darvin H, DenBesten L. Risk factors for gallbladder perforation. *Am J Gastroenterol* 1987; 82: 636-640.
2. Ong CL, Wong TH, Rauff A. Acute gall bladder perforation-a dilemma in early diagnosis. *Gut* 1991; 32: 956-958.
3. Glenn F, Moore SW. Gangrene and perforation of the wall of the gallbladder. A sequele of acute cholecystitis. *Arch Surg* 1942; 44: 677-686.
4. Niemeier OW. Acute free perforation of the gall bladder. *Ann Surg* 1934; 99: 922-924
5. Roslyn JJ, Thompson JE, Darvin H, et al. Risk factors for gall bladder perforation. *Am J Gastroenterol* 1987; 82 : 636-40.
6. Lennon F, Green WE. Perforation of the gallbladder. A review of 32 cases. *J R Coll Surg Edinb* 1983; 28: 169-173.
7. Isch JH, Finneran JC, Nahrwold DL. Perforation of the gallbladder. *Am J Gastroenterol* 1971; 55: 451-458.
8. Kim PN, Lee KS, Kim IY, Bae WK, Lee BH. Gallbladder perforation: comparison of US findings with CT. *Abdom Imaging* 1994; 19: 239-242.
9. Derici H, Kara C, Bozdog AD, Nazli O, Tansug T, Akca E. Diagnosis and treatment of gallbladder perforation. *World J Gastroenterol* 2006; 12(48): 7832-7836.
10. Sood BP, Kalra N, Gupta S, Sidhu R, Gulati M, Khandelwal N, Suri S. Role of sonography in the diagnosis of gallbladder perforation. *J Clin Ultrasound* 2002; 30: 270-274.
11. Forsberg L, Andersson R, Hederstrom E, Tranberg KG. Ultra-sonography and gallbladder perforation in acute cholecystitis. *Acta Radiol* 1988; 29: 203-205.