TITLE OF CASE

Perforated Gallbladder: A Complication of Acute Cholecystitis

AUTHORS OF CASE

Stephanie LaGrow

SUMMARY

Patient was a 71 year old male with a known cardiac history admitted with chest pain and pressure. On the second day of admission, patient complained of right lower then right upper quadrant (RUQ) pain with nausea and vomiting. He also developed a low-grade fever, leukocytosis and elevated liver enzymes. Abdominal ultrasound suggested acalculous cholecystitis. Abdominal/pelvic CT suggested acute cholecystitis with hyperdensity, possible cystic duct obstruction.

Patient was taken for a laparoscopic cholecystectomy with intra-operative cholangiogram and found to have an inflammatory phlegmon of omentum and a gangrenous, perforated gallbladder. The gallbladder was removed, the RUQ was irrigated and a Jackson-Pratt drain was placed. Patient was started on Cipro and Flagyl.

Patient’s nausea and vomiting ceased, he became afebrile, his leukocytosis resolved, and the JP drain was removed. Patient was discharged home on post-op day (POD) four with oral antibiotics, pain medications and instructions to follow up with the surgeon in two weeks.

BACKGROUND

Acute cholecystitis is a very common surgical diagnosis that almost always requires surgical intervention. Gallbladder perforation is a potentially fatal complication that is often hard to diagnose preoperatively and therefore must always be considered in cases of acute cholecystitis. Proper and prompt initiation of treatment and surgery may help to avoid severe illness and prolonged hospital admission.

CASE PRESENTATION

Chief Complaint: Chest pain and pressure
History of present illness: 71 year old Caucasian male who presented to the emergency department on 12/14/08 with chest pain and pressure after eating steak. Initial cardiac studies were negative, and he was admitted under the cardiology service for further cardiac work-up. He then began having pain in the periumbilical region on 12/15/08, which then moved to the right lower quadrant (RLQ). He also developed severe nausea and vomiting with a low-grade fever. Patient did not have fevers or chills previously at home, but did have nausea without vomiting earlier in the week. On 12/16/08, patient continued to complain of RLQ pain, but now had greater pain right upper quadrant (RUQ) with continued nausea, vomiting, fever and leukocytosis.

Past medical History: CAD, MI, home oxygen (2-3L)

Past Surgical History: CABG x3, cardiac stent x3, right foot surgery, bowel surgery for volvulus

Meds: Cardizem, aspirin, Lopid, Nitrostat, Ambien, Darvocet, vitamin B12

Allergies: Isordil, Penicillin, Lipitor, Sulfa, and Prozac.

Family History: Unknown
Social History: Lives with wife, retired. Quit smoking 40 years ago, social ETOH use, and no illicit drug use.

Review of Systems 12/16/08 (Date of surgical consult):
General: Decreased appetite, recent 5# weight loss
HEENT: Denies URI symptoms, dysphagia
Cardiovascular: Mild substernal pain
Respiratory: No cough, sputum production or SOB.
GI: RUQ and RLQ pain, nausea and vomiting
GU: Denies frequency, burning or decreased flow with urination.
Musculoskeletal: Denies joint or muscle pain in extremities and neck.
Skin: Denies rashes and lesions
Neurologic: Denies HA, dizziness, paresthesia.

Physical Examination 12/16/08:
General: Pt. ill appearing with difficulty finding comfortable position in bed. A&Ox3.
HEENT: Normocephalic, atraumatic. No sclera icterus.
Neck: Trachea midline, supple
Cardiovascular: S1, S2. RRR. No murmurs, rubs or gallops.
Respiratory: Equal breath sounds, clear to auscultation
GI: (+) bowel sounds, soft, tenderness in RUQ and LUQ, guarding with RUQ palpation. No rebound. No Rovsing sign. Positive obturator and Murphy’s signs.

## INVESTIGATIONS

### Complete Blood Count

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<th>Date</th>
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<tr>
<td>12/16/08</td>
<td>14.1 H</td>
<td>12.5 H</td>
</tr>
<tr>
<td>12/17/08</td>
<td>17.8 H</td>
<td>15.9 H</td>
</tr>
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<td>13.4 H</td>
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</tr>
<tr>
<td>12/19/08</td>
<td>10.1</td>
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<td>12/21/08</td>
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<td>7.5 H</td>
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### Hepatic Function Panel

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<th>(40-130 U/L)</th>
<th>ALB, ALT, Bili, Dbili WNR</th>
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<td>(40-130 U/L)</td>
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**Note:** Dates for laboratory results are placeholders; actual dates should be provided.
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<tr>
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<tr>
<td>Dbili</td>
<td>2.1</td>
<td>(0-.5 mg/dl)</td>
</tr>
<tr>
<td>AST</td>
<td>409</td>
<td>(0-50 U/L)</td>
</tr>
</tbody>
</table>

12/19/08 -
| ALKP  | 703       | (40-130 U/L)      |
| ALB   | 2.2       | (2.8-5.5 g/dl)    |
| ALT   | 167       | (0-69 U/L)        |
| Bili  | 1.7       | (.1-1.6 mg/dl)    |
| Dbili | 1.5       | (0-.5 mg/dl)      |
| AST   | 366       | (0-50 U/L)        |

12/21/08 -
| ALKP  | 813       | (40-130 U/L)      |
| ALB   | 2.3       | (2.8-5.5 g/dl)    |
| ALT   | 103       | (0-69 U/L)        |
| Bili  | 0.4       | (.1-1.6 mg/dl)    |
| Dbili | 0.1       | (0-.5 mg/dl)      |
| AST   | 66        | (0-50 U/L)        |

Serial Cardiac Enzymes - all negative
Urinary Analysis (UA) - negative

Chest X-Ray (CXR) 12/15/08 - Bibasilar scarring/atelectasis
Ultrasound (US) Abdomen 12/16/08 - Findings suggestive of acalculous cholecystitis
CT Abdomen/pelvis with IV contrast 12/16/08 -
1. Findings consistent with acute cholecystitis with approximately 1cm hyperdensity in cystic duct.
2. Uncomplicated diverticulosis of recto sigmoid colon
3. Cardiomegaly.

**DIFFERENTIAL DIAGNOSIS**
Biliary: acute cholecystitis, cholelithiasis, cholangitis
Appendicitis
Colonic: colitis, diverticulosis, diverticulitis, volvolus (pt. Had previously), SBO
Hepatic: mass, hepatitis, abscess
Gastric: esophagitis, gastritis, PUD
Renal: nephrolithiasis, pyelonephritis
Other: viral gastroenteritis (3)

**TREATMENT**
After the diagnosis of acute cholecystitis, the patient was started on Cipro and Flagyl, kept NPO and was continued on IV fluids and anti-emetics. He then underwent a laparoscopic cholecystectomy with intra-operative cholangiogram and was found to have a gangrenous, perforated gallbladder. The surrounding omentum, which was containing the spillage, was removed and a RUQ intra-abdominal irrigation was completed. A Jackson-Pratt (JP) drain was placed in the RUQ and externalized.

**OUTCOME AND FOLLOW-UP**
The patient was returned to the general medical floor after surgery. The nausea and vomiting ceased and he began to tolerate a full liquid to soft, low-fat diet. The JP drain was removed on POD #2 with a total of 30cc of output. The patient gradually increased activity, bowel function returned on POD #3 and he was discharged home on POD #4 with po Cipro and Flagyl and pain medications. Patient was to follow up with surgeon in 2 weeks.
1. What are the incidence, mechanism and presentation of a gallbladder perforation in acute cholecystitis?
2. What is the best initial method of diagnosing a gallbladder perforation?
3. What is the best management and potential complications after a gallbladder perforation?
4. What is the role of the omentum in gallbladder perforation?
5. How does this case presentation compare to other cases of gallbladder perforation?

1. Gallbladder perforation occurs in 3-10% of patients with acute cholecystitis, with a mortality rate of 12-16%. A perforation may occur two days to several weeks after the onset of acute cholecystitis. Most cases are associated with gallstones, which causes outflow obstruction and an increase in bile concentration which then acts as an irritant that leads to inflammation (1). Inflammation of the gallbladder then results in increased intra-luminal pressure, which impedes venous and lymphatic drainage and leads to necrosis and perforation of the gallbladder wall. In addition to cholelithiasis, other factors that predispose patients to perforation are infection, malignancy, trauma and drugs, such as corticosteroids. Most patients with gallbladder perforation are elderly and present with RUQ pain usually less than three days (4). These patients also usually present with guarding, leukocytosis and fever. (1)

Most perforations occur at the gallbladder funds because this is the most likely site of vascular compromise. Perforations are more likely to occur in patients who delayed medical treatment or who did not respond to conservative management. Once the gallbladder has perforated, most patients experience a relief of symptoms because the gallbladder decompresses. However, peritonitis may soon develop. Most perforations are localized, in which the surrounding omentum and viscera contain the contents spilled from the gallbladder (4)

A study was conducted from 1982 to 2002 in which 11,360 patients who underwent cholecystectomy, all within the same facility, were followed. Of those patients, 30 were diagnosed with a gallbladder perforation, and in 9 out of 21 patients, the perforation was contained. The incidence of perforation occurred more in men than women, with the average age of 60 years. In addition, 29% of those patients with gallbladder perforation had a cardiac co-morbidity, 37% had post-operative complications, and 33% required an ICU admission (12)

Gallbladder perforation may not always be caused by the disease process itself, and may also occur intraoperatively during a cholecystectomy. One study looked at 110 patients undergoing laparoscopic cholecystectomy, and found that 26.3% sustained an intra-operative gallbladder perforation. The injuries were caused during the gallbladder dissection, by retraction with grasping laparoscopic instruments, during extraction from the abdomen or because of cystic duct clip spillage, which then leaked bile and stones (6).

2. The diagnosis of gallbladder perforation is very difficult. One study found that only 3% of patients were diagnosed with a gallbladder perforation preoperatively (12). Another study found that only 11.1% of patients were diagnosed correctly preoperatively (1). Ultrasonography (US) is the initial recommendation for investigation in a suspected gallbladder perforation. According to the literature, there are no imminent signs that gallbladder perforation is about to occur. However, once a perforation does occur, the following is often seen on US: The usual signs of acute cholecystitis, a defect in the wall of the gallbladder at the site of perforation, serpiginous intra-luminal membranes due to sloughed mucosal
lining, and one or more hypo echoic masses adjacent to the gallbladder, which represents an abscess or peri-cholecystic fluid collection (9).

Another method is CT, which is 88% sensitive for gallbladder perforation (8). One study looked 23 patients with gallbladder perforation, and found that US detected gallbladder perforation in 70% of cases, whereas a CT detected a perforation in 78% of cases. (11) Another study compared the use of US versus CT in the diagnoses of gallbladder perforation. They found that US alone showed the most common findings of peri-cholecystic fluid collection and lying of the gallbladder wall. The CT group showed peri-cholecystic fluid and layering of the GB wall, in addition to streaking of the omentum or mesentery, and the gallbladder wall defect. They concluded that gallbladder perforations where detected on US in 38.5% of patients and in 69.2% of patients by CT (5). In a study by Vogt (2002), they concluded that CT scanning was not as accurate at detecting gallstones as US, but that CT scanning was far more superior in displaying thickening of the gallbladder wall and peri-cholecystitis fluid, which is found with perforation (14).

There appears to be some controversy over the best diagnostic tool for gallbladder perforation. Overall, the majority of the literature suggested that the best diagnostic tool appears to be US initially, and if determined inconclusive, then a CT should be completed. (1,9,11)

3. Patients with a suspected or known perforated gallbladder should be kept NPO, given IV fluids and pain medications and started on antibiotics to cover the most common bacteria, which include Escherichia coli and Streptococcus faecalis (1). Surgical management includes laparoscopic cholecystectomy, irrigation of the abdominal cavity in the area of the perforation and drainage of the abdominal cavity. As with all laparoscopic procedures, conversion to an open procedure may be necessary. (13)

Complications of gallbladder perforation include contamination of the abdominal cavity with bacteria from inside the gallbladder and spillage of calculi. Attempts should be made to irrigate the abdominal area and retrieve as many calculi as possible (6).

4. The omentum is often referred to the “policeman of the abdomen” because of its unique ability to contain bowel wall injuries and abdominal organ perforations. The omentum is composed of not only fat, but also lymphatics, blood vessels and cellular tissues of the immune system, and has a large protective role in phagocytosis and inflammatory processes (10). In 1996, a group of researchers found that the tissue factor concentration of the omentum is greater than twice the amount per gram that tissue factor concentration found in muscle. The large tissue factor concentration facilitates activation of coagulation at sites of infection, trauma and inflammation within the peritoneal cavity. The local production of fibrin, allows the omentum to adhere to the areas of inflammation or injury and perform a protective barrier to contain the area. (2). Studies have also shown that the absorption, adhesion, neovascularisation and infection defence of the omentum protects against irradiation damage, increases the rate of healing in dead space, and decreases the complication rate after application to a wound bed (7).

Regarding gallbladder perforation, one study found that only 0.5% of patients undergoing conservative treatment for acute cholecystitis with perforation have gross contamination into the peritoneal cavity. The vast majority do not have gross contamination of the peritoneal cavity because of the unique ability of the omentum and parietal peritoneum to form the protective barrier that prevents further contamination (13).
5. According to the literature review that I completed, the patient in this case study appears to be a typical presentation of a gallbladder perforation. The patient was male, older than 60 years, had a cardiac co-morbidity and signs and symptoms consistent with acute cholecystitis. The only significant difference was in addition to the classic RUQ pain, he also presented with substernal chest pain and pressure and RLQ pain. The patient underwent both an US and then a CT due to right side abdominal pain and the history of volvolus. Neither the US or CT visualized an abscess or peri-cholecystic fluid, suggestive of a gallbladder perforation. However, the CT of the abdomen and pelvis did show a hyperdensity in the cystic duct which may have actually been the perforation and was just assumed to be a gallstone. There is also a possibility that the patient did not perforate until after the studies where completed. The patient was appropriately placed on antibiotics, taken for cholecystectomy with intra-abdominal irrigation and had a JP drain placed. The case was handled promptly, thus possibly avoiding further complications and the need for ICU placement. The patient showed a marked improvement in pain, mobility, and appetite after surgery as compared to before surgical intervention. The medical and surgical management of this patient in the case study compared to a majority of the cases that were reviewed in the literature (1,4,6,12,14)

**LEARNING POINTS/TAKE HOME MESSAGES**

1. Acute cholecystitis has the potential to cause several complications, such as gallbladder perforation, that can be life threatening.

2. Although imaging may not always show a gallbladder perforation, clinicians should be aware that this can be a complication of acute cholecystitis. Antibiotics, fluids and cholecystectomy should be completed as soon as possible in a patient with symptomatic acute cholecystitis with suspected perforation. Don’t depend on a test to tell you it is there because you may not be able to see it with diagnostic testing!

3. Diagnosis of acute abdominal pain can often be complicated and may present atypically.

**REFERENCES**


