Acute Cholecystitis Presenting as a Subcutaneous Abscess and Sepsis

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Introduction

Gallstones present a significant burden on the American healthcare system, with gallstone-related disease affecting an estimated 20 million Americans annually. It is also estimated that fewer than one in five people with clinically silent gallstone disease will present with gallstone-related complaints over a 15-year period. Most commonly, patients will have biliary colic or, in certain cases, acute or chronic cholecystitis. Migratory stones may present with choledocholithiasis, gallstone pancreatitis, or ascending cholangitis. In rare cases, the stone may fistulize into either the biliary tree or the bowel. Mirizzi syndrome, marked by a large gallstone in the neck of the gallbladder which obstructs the outflow of bile from the common bile duct, is another possible complication of gallbladder disease. Finally, gallstone ileus may occur when a chronically inflamed gallbladder fistulizes into the small bowel, allowing a large stone to pass from the gallbladder into the small bowel. The large stone becomes lodged in the terminal ileum where the lumen is narrow, causing an obstruction. In severe cases, mucocele, empyema, gallbladder gangrene, or even rupture with biliary peritonitis may develop. We examine a case of acute-on-chronic cholecystitis presenting as a subcutaneous abscess and sepsis.

Case presentation

A 67-year-old female presented with complaints of abdominal pain, which seemed to worsen with a change in position. The patient denied fever, chills, nausea, chest pain, or vomiting. There were also complaints of dyspnea and difficulty taking a deep breath. The patient’s physical exam revealed a morbidly obese female with clear lung fields and normal heart sounds. Her abdomen was soft and undistended, with some mild right upper quadrant tenderness. The patient had no rebound tenderness and had normal, active bowel sounds. Bloodwork was significant for sodium of 144 mEq/L, potassium of 5.9 mEq/L, chloride of 104 mEq/L, bicarbonate of 21 mEq/L, and creatinine of 6.6 mg/dL with a BUN of 93 mg/dL. The patient’s lactic acid was normal. Total bilirubin was 1.2 mg/dL and white blood cell count was 17.6K/mL with hemoglobin of 10.2 g/dL. The patient underwent a non-contrasted CT scan of the abdomen and pelvis which showed some subcutaneous fluid and a large calcified gallstone (Figure 1). There were no skin changes or physical signs of an abscess. She was admitted to the medicine service and started on broad-spectrum antibiotics. Surgery was consulted.

Figure 1 - CT of the abdomen demonstrating a large calcified gallstone and fluid in the subcutaneous tissues.
for possible acute cholecystitis. A HIDA (hepatobiliary iminodiacetic acid) scan was obtained, which showed non-visualization of the gallbladder. The patient’s renal function did not improve with hydration. Nephrology was consulted and hemodialysis was started.

On the patient’s third hospital day, she was taken to the operating room for a possible open cholecystectomy. An incision was made in the right upper quadrant, and approximately two liters of pus was drained from a subcutaneous abscess. At the base of the wound, a small punctate hole drained bile, with the incision carried into the abdomen. There was extensive inflammation around the gallbladder, making it difficult or impossible to remove. A large malecot catheter was placed in the gallbladder as a cholecystostomy tube. The abdominal wall was closed. The cholecystostomy tube was brought out of the abdomen through a separate incision. Subcutaneous tissues were left open and a wound VAC was placed.

The patient required intermittent hemodialysis to support her renal function. She was taken back to the operating room two days later for a washout of her subcutaneous tissues. Some devitalized subcutaneous tissues were debrided. After another two days, the patient was taken back to the operating room, where she underwent a split-thickness skin graft from her flank to her large abdominal wound. Postoperatively, the patient recovered. Her renal function improved, and she was able to be weaned off hemodialysis. Her cholecystostomy tube came out approximately three months later. The patient underwent an open cholecystectomy four months after her original surgery without incident.

**Discussion**

We present the case of a ruptured gallbladder into the subcutaneous tissues. The patient presented septic with an acute kidney injury. She was admitted and placed on broad-spectrum antibiotics. After consulting nephrology and surgery, the patient underwent intermittent hemodialysis. The patient’s renal function recovered after she underwent a delayed open cholecystectomy.

The pathophysiology of acute cholecystitis is well known: A gallstone obstructs the cystic duct, causing distention of the gallbladder. The gallbladder can spasm, a symptom of biliary colic. If the obstruction is not relieved, further distension of the gallbladder can lead to ischemia and necrosis, which can easily lead to perforation. This complication is rarely seen in the United States, likely because most patients seek early medical attention. McDonald’s 1966 review of acute calculus cholecystitis showed a 10.6% incidence of gallbladder perforation. In a 1954 paper, Pines and Rabinovitch, using the Stout and Hibbard classification of gallbladder perforation (Table I), found between 2.5 and 25% incidence of gallbladder perforation. Though ruptured gallbladder is not unheard of in acute cholecystitis, Strasburg’s comprehensive 2008 review only briefly mention the complication of a ruptured gallbladder.

Since the late 1980s, laparoscopic cholecystectomy has been the preferred method for removing the gallbladder in acute cholecystitis. However, in patients with difficult gallbladders (chronic inflammation, confusing anatomy), the open method is preferred and may be required. Therefore, it is important for all general surgeons to be comfortable with the open method.

Additionally, the medical literature has stressed the proper treatment of sepsis for more than a decade, including guidelines from the Society of Critical Care Medicine, the European Society of Intensive Care Medicine, and the International Sepsis Forum. The most recent guidelines, published in 2016, stress rapid identification of sepsis through clinical examination and laboratory analysis. Once a clinician believes the patient might be septic, they should quickly obtain cultures (blood, sputum, and urine, and any obvious wounds). This must be immediately followed by appropriate antibiotics as well as fluid resuscitation. Clinicians must locate the source of sepsis, which then must be controlled. The guidelines also stress the continued serial laboratory monitoring of resuscitation, adjusting the resuscitation to ensure that the patient is not over or under resuscitated. The target blood pressure should be a mean arterial pressure of 65 mmHg. A combination of pressors and fluid should be used if fluid alone does not achieve the desired blood pressure.

When our patient presented with vague abdominal complaints, she was found to be septic and rapidly

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**Table 1. Gallbladder Perforation Classification**

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<tbody>
<tr>
<td>1</td>
<td>Perforation into a viscus</td>
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<tr>
<td>2</td>
<td>Pericholecystic perforation with localized abscess formation</td>
</tr>
<tr>
<td>3</td>
<td>Perforation into the liver</td>
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<tr>
<td>4</td>
<td>Perforation into the free peritoneal cavity</td>
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<tr>
<td>5</td>
<td>Perforation through the abdominal wall</td>
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treated according to the latest guidelines. We identified the source of her sepsis as the gallbladder. Once the abscess was drained and the source of sepsis controlled, the patient rapidly improved.

Conclusion

Perforation of the gallbladder is a rare complication of acute cholecystitis. As with any septic patient, fluid resuscitation, adequate antibiotics, and source control are the mainstays of therapy. In this patient, we obtained source control by draining the abscess and placing a cholecystostomy tube. The patient underwent a delayed open cholecystectomy without incident.

References