

5-2019

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Recommended Citation

Medina, Miles and Cronovich, Heather, "Sarcoidosis and Acute Cholecystitis" (2019). *Case Reports*. 73.
<https://scholarlycommons.henryford.com/merf2019caserpt/73>

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Sarcoidosis and Acute Cholecystitis

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Introduction

50 year old male with a history of sarcoidosis presented with a chief complaint of pleuritic chest pain. The patient experienced relief after treatment with a “GI cocktail.” Patient was given return instructions as well as gastrointestinal follow up. Patient returned 3 days later after undergoing esophagogastroduodenoscopy with findings of erosive gastritis. Patient returned as he was experiencing nausea, vomiting, and abdominal pain. Imaging displayed pericholecystic inflammatory changes concerning for acute cholecystitis.

Intravenous antibiotics were started and patient was taken for operative intervention the following morning. A laparoscopic cholecystectomy was performed and 4 days later, the patient was discharged with no complications. This case explores the atypical presentations of acute cholecystitis as well as speculates the relationship of sarcoidosis with cholecystitis etiologies.

Case Presentation

50 year old male presented to the emergency department with chest pain. The patient was working his night shift when this pain started gradually. Patient described his pain as pressure-like, constant, non radiating, worsened with inspiration, and worsening over time. Patient had a medical history remarkable for sarcoidosis, hiatal hernia, and esophagogastroduodenoscopy 6 months prior remarkable for a Schatzki’s ring. 7 months prior, the patient had been seen by a cardiologist with 2 catherizations displaying no evidence of coronary artery disease over the last 3 years including a negative stress test. Patient had a blood pressure of 118/85 mmHg, pulse of 87 bpm, temperature of 98.2 F, respirations at 18 bpm, and SpO2 at 100%. There were no abnormalities found on physical examination. Patient denied diarrhea, vomiting, diaphoresis and admitted to nausea.

Patient was given aspirin and morphine which resulted in mild relief of his pain. He had a heart score of 2 given his age, obesity, and low suspicion of clinical history. His EKG was normal sinus rhythm at 83 beats per minute, no ST changes, and PR intervals within normal limits. He was given pepcid, carafate, and viscous lidocaine. He experienced a great deal of relief afterwards. Labs were unremarkable with a chest x-ray revealing no acute intrathoracic process. ALT, AST, bilirubin and two troponins within 3 hour windows were included which were within normal limits. Patient was then discharged with gastrointestinal follow up and carafate.

3 days later, the patient returned with a chief complaint of abdominal pain. The pain was epigastric in nature, did not radiate, unchanged with eating, and had associated nausea with decreased appetite. Of note, the patient received a EGD 1 day prior which displayed fundal erosive gastritis with a small hiatal hernia. Patient admitted to having to both nausea and vomiting with this visit as well as abdominal bloating with a sensation of feeling “gassy.”

Vital signs for the second encounter were a blood pressure of 128/79 mmHg, temperature of 99.7 F, respirations at 16 bpm, pulse at 90 bpm, and SpO2 of 97%. Physical exam revealed diffuse abdominal tenderness. A CT scan of the abdomen and pelvis with IV contrast demonstrated gallbladder wall thickening and pericholecystic inflammation highly concerning for changes of acute cholecystitis. No focal intrahepatic lesions were present nor was there intrahepatic or extrahepatic biliary ductal dilatation. Laboratory values did not demonstrate elevations with ALT, AST, bilirubin, or lipase.

Notable values was a WBC of 5.1 K/uL and a lactic acid of 1.9 mmol/L. General Surgery was consulted for further management and an ultrasound of the right upper quadrant was ordered. The ultrasound demonstrated gallbladder stones, sludge, and wall thickening consistent with acute cholecystitis. Intravenous antibiotics were started and patient was taken to the operating room shortly. During the laparoscopic cholecystectomy, it was found that the

Case Presentation Continued patient had an inflamed, friable gallbladder with omental adhesions, and frank purulence upon grabbing and retraction of the gallbladder

The patient tolerated the procedure well and transitioned onto the general medical floor. He progressed well post-operatively and was discharged on post operative day 3. Patient had no complications and was discharged with PO antibiotics, pain medications, and laxatives. Pathology did not display any abnormalities in surgical specimen.

Variable Clinical Presentations of Acute Cholecystitis and Sarcoidosis

Chest pain is an atypical complaint for cholecystitis. There have been a few cases that have acute cholecystitis coexisting or being misdiagnosed as a cardiovascular etiology. Most of the missed cases have had existing cardiac problems such as previous acute coronary syndromes, positive biomarkers during initial evaluation, and electrocardiography changes suspicious for ischemia. Most of these patients were hospitalized for this and were later found to have abdominal complaints resulting in gastrointestinal consultation and eventual diagnosis of cholecystitis. A study demonstrated 16 out of 5552 patients admitted for cardiac work ups with later diagnoses of acute cholecystitis. Of these 16, 5 had been hospitalized in the ICU. Of note, this study was limited as there was little history provided on these patients background of both chronic disease or hepatobiliary disorders³.

Two cases with known sarcoidosis are as follows:

- Patient with abdominal pain who is post sarcoidosis treatment with immunosuppressants and high dose steroids found to have non caseating granulomas in gallbladder biopsy post cholecystectomy, similar to cutaneous sarcoidosis – see Figure 1
- Patient currently receiving treatment for sarcoidosis with concern for GI infiltration due to epigastric pain and weight loss found to have abnormal endoscopy with non caseating granuloma on endoscopic biopsy – see Figure 2

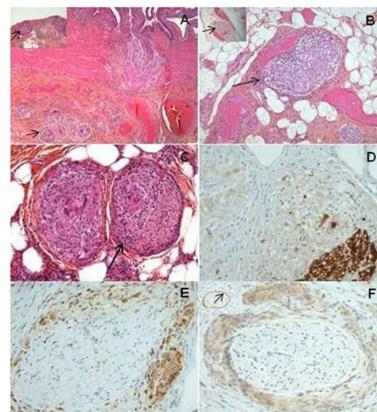


Figure 1²

Figure 1. Gallbladder wall demonstrating epithelioid and giant cell granulomas. Picture A demonstrates the level of the serosa, picture B in the perivascular, picture C-F in the perineural spaces. C-F have different markers that were expressed in both granulomas epithelioid and multinucleated giant cells.

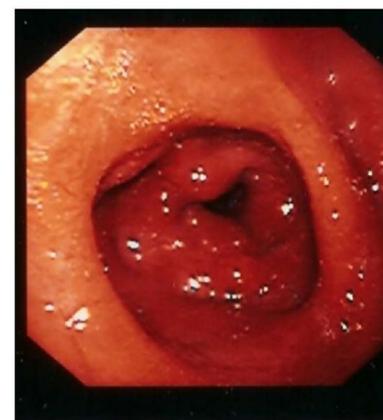


Figure 2³

Figure 2. Upper gastrointestinal endoscopy showing linitis plastica appearance and diffuse erythema.

Discussion

The patient initially presented with chest pain with negative biomarkers from a cardiac standpoint. He was discharged and subsequently had an EGD with findings of fundal erosive gastritis similar to the endoscopy findings of the patient in Figure 2. This could indicate sarcoidosis infiltration into the GI tract. In addition, the patient’s history of high dose steroids and immunosuppressants suggest decreased biliary motility playing a crucial role in the presentations of cholecystitis in sarcoidosis.

As noted in Figure 1, most of the granulomas were found in the perineural spaces which further contributes to decreased motility of the biliary tract. In most of the cases, there have been the primary complaint of abdominal pain with no known reported cases initially as chest pain. However, due to the perineural nature of the granuloma formation, there is evidence to suggest atypical presentations as nerve fibers cross and synapse. Table 1 compares and contrasts these three cases.

	Patient	Figure 1	Figure 2
Chief Complaint	Chest pain to abdominal pain	Abdominal pain	Abdominal Pain
Lab Changes	None	ALT, AST, Bilirubin	ALT, AST, Bilirubin
Biopsy	No abnormalities	Perineural non caseating granulomas on gallbladder biopsy	Non caseating granulomas on endoscopic biopsy

Table 1. Note: these three cases all had treatment or currently being treated with both corticosteroids and immunosuppressants

Conclusion

Sarcoidosis with GI involvement is exceedingly rare. Out of the few cases, there seems to be a diverse set of presentations. What appears to be consistent is a chief complaint of epigastric pain and previous treatment with immunosuppressants and corticosteroids. Variations of gallbladder biopsy range from granuloma formation to no abnormalities. Laboratory findings in ALT, AST, and bilirubin appear to be varied as well.

With most CT scans and ultrasounds demonstrating gallbladder stones, decreased biliary motility seems to be the leading contributor towards the diagnoses of acute cholecystitis in the setting sarcoidosis. Whether it is the corticosteroids, immunosuppressants, or granuloma formation in the perineural space are responsible is a question that requires more study.

Though this patient did not appear to be in active flare in sarcoidosis it is important to recognize sarcoidosis’s active role in the pathology and to consider corticosteroid therapy/immunosuppressants as granulomatous formation may extend beyond the biliary tree.

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