

Congestive Hepatopathy: A Case Report and Literature Review

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INTRODUCTION

- Congestive hepatopathy is diffuse venous congestion within the liver that results secondary to heart failure.
- Awareness of this pathology can help to avoid the use of unnecessary treatments.
- We are presenting a case of a patient with multiple comorbidities including heart failure initially treated for acute cholecystitis who was found to have congestive hepatopathy. Patient's symptoms improved after heart failure treatment optimization.

CASE PRESENTATION

A 39-year-old African American woman presented to the emergency room with five-day history of right-sided colicky abdominal pain associated with nausea and decreased appetite. Her past medical history was remarkable for hypertension, congestive heart failure NYHA class III, COPD, active cigarette smoker with 5.7 pack-year history. She denied alcohol or drug abuse. Ten days before this visit, she was admitted in a different hospital for a similar episode where she completed a course of intravenous antibiotics for possible acute cholecystitis.

On physical examination, patient was in non-acute distress, afebrile, blood pressure of 129/83 mmHg, heart rate 76 bpm, respiratory rate of 16 rpm, oxygen saturation of 92% on room air. Abdominal exam was remarkable for tenderness to palpation of the right upper quadrant and hepatomegaly of 4-6 cm below the right costal border. No guarding or rebound. Lower extremities with pitting edema up to the knee level. No additional physical exam findings.

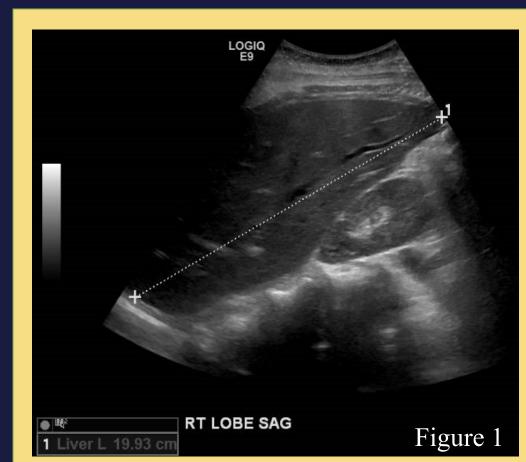
Initial laboratory workup revealed white blood cell count of 4,100/uL, alanine aminotransferase (ALT) of 20 IU/L, aspartate aminotransferase (AST) of 50 IU/L, total bilirubin 3.4 mg/dL, direct bilirubin 0.9 mg/dL, alkaline phosphatase 77 UI/L, albumin 3.2 mg/dL, lipase 20 U/L and creatinine 1.1 mg/dl. Brain natriuretic peptide was 3496 pg/ml. Pregnancy test negative.

Hepatitis C antibody was non-reactive. Hepatitis B panel was compatible with acquired immunity from prior infection. Ultrasound of the abdomen showed hepatomegaly of 19.9 cm (Figure 1), diffuse thickened gallbladder (8 mm) without pericholecystic fluid or gallstones, no biliary ductal dilatation, and positive sonographic Murphy's sign.

Patient was evaluated by Surgery that recommended no acute surgical intervention needed. Further workup was directed towards detecting gallbladder pathology. Patient was started on treatment for acute cholecystitis with antibiotics, analgesics and intravenous hydration.

Patient underwent hepatobiliary iminodiacetic acid (HIDA) scan which showed no scintigraphy evidence of acute cholecystitis. Antibiotics were discontinued. Computed tomography (CT) of the abdomen with contrast to rule out possible Budd-Chiari syndrome revealed fatty liver infiltration, marked hepatomegaly with a liver spanning the entire transverse diameter of the abdomen (Figure 2) and measuring 20 cm in craniocaudal extent. No focal liver lesions, normal gallbladder in size, small amount of pericholecystic fluid, no gallstones. Subcutaneous fat infiltration of soft tissues suggestive of anasarca, ground glass opacities in the lower lung fields consistent with edema and a small right pleural effusion.

Patient was considered to have congestive hepatopathy. Her poor appetite was most likely multifactorial from cardiac cachexia or organomegaly compressing on the stomach. Patient was aggressively diuresed and had her heart failure medications optimized. Her symptoms improved and she was discharged home with outpatient Cardiology follow up.



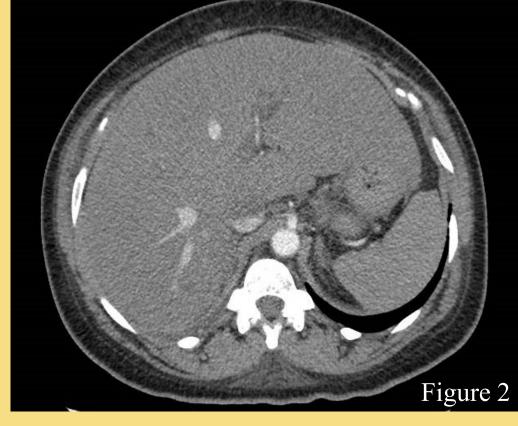


Figure 1. Ultrasound of the abdomen showing hepatomegaly of 19.9 cm in transverse diameter. Figure 2. Contrast CT of the abdomen and pelvis showing marked hepatomegaly

DISCUSSION

Acute cholecystitis, the acute inflammation of the gallbladder, is most commonly associated with gallstone disease. Signs and symptoms of cholecystitis may include severe right upper quadrant or epigastric pain that may radiate to shoulder or back, fever, nausea, vomiting, leukocytosis, abdominal guarding and a positive Murphy's sign. Usually, these symptoms occur after a meal, particularly a large or fatty meal. Confirmation of the diagnosis of acute cholecystitis requires demonstration of wall thickening or edema, a sonographic Murphy sign or failure of the gallbladder to fill during cholescintigraphy.

The 2013 Tokyo guidelines for the diagnosis of acute cholecystitis are as follows (1):

- A. Local signs of inflammation, etc.:
- (1) Murphy's sign, (2) RUQ mass/pain/tenderness
- B. Systemic signs of inflammation, etc.:
- (1) Fever, (2) elevated CRP, (3) abnormal WBC count
- C. Imaging findings: imaging findings characteristic of acute cholecystitis

Definite diagnosis

- (1) One item in A + one item in B are positive
- (2) C confirms the diagnosis when acute cholecystitis is suspected clinically

Imaging findings characteristic of acute cholecystitis are the presence of stones, gallbladder wall thickening (greater than 4 to 5 mm) or edema (double wall sign), and a sonographic Murphy sign which is more accurate than hand palpation (2). When the diagnosis remain uncertain following ultrasonography, as in our patient, HIDA scan is indicated. Computed tomography of the abdomen is useful only when complications of acute cholecystitis are suspected or additional diagnosis are being considered.

Diagnosis of conditions other than acute cholecystitis remain a challenge for the clinician. In patients with passive congestion due to decompensated congestive heart failure, congestive hepatopathy may develop. These type of patients are usually asymptomatic.

Congestive hepatopathy should be suspected in any patient with congestive heart failure or other cardiac condition associated with elevated central venous pressure (3). In these patients, the increase congestion or edema seen during acute exacerbation of congestive heart failure usually causes stretching of the liver capsule provoking right upper quadrant pain (4). The diffuse gallbladder wall thickening related to elevated portal and systemic venous pressures is typically not thought to cause pain or a positive Murphy sign (5). A case report suggested that this diffuse gallbladder thickening may cause obstruction and mimic biliary pain (4).

The most common signs and symptoms of congestive hepatopathy and its frequency are illustrated below (4):

Marked hepatomegaly	95-99%
Palpable hepatomegaly (> 5cm below right costal margin)	49-57%
Peripheral edema	71-77%
Pleural effusion	17-25%
Ascites	7-20%
Splenomegaly	20-22%
Jaundice	10-2%

The most common laboratory abnormalities attributed to congestive hepatopathy are decreased albumin, mild elevation of serum bilirubin and alkaline phosphatase, and usually normal aminotransferases (5,6). Ultrasonography findings in congestive hepatopathy are non-specific.

Treatment of congestive hepatopathy is aimed to the treatment of the underlying heart disease. Symptoms usually improve after diuretic therapy. Excessive diuresis should be avoided as it can impair hepatic perfusion.

CONCLUSIONS

Congestive hepatopathy is a challenging diagnosis as it could potentially be mislead with gallbladder pathology. Taking this into consideration, clinicians can avoid unnecessary treatment including surgical intervention.

Contact Information