Bilateral Pleural Effusions with Ascites

By Nancy Liao
81 year old male with history of type II diabetes, hyperlipidemia, and congestive heart failure presents as transfer from extended care facility for evaluation of acute, progressive shortness of breath and increasing abdominal girth.
Pulmonary exam significant for tachypnea. Abdomen was distended on physical exam. 2+ LE edema bilaterally.

CXR obtained for dyspnea showed bilateral pleural effusions.

U/S of abdomen was performed, which also demonstrated large bilateral pleural effusions as well as ascites.
Left-sided Pleural Effusion with Ascites
Right-sided Pleural Effusion with Ascites

Diaphragm

Ascites

PLEURAL EFF/ASCITES

Pleural effusion

Liver

Kidney
Right-sided Pleural Effusion with Lung Tip
Right Lung with Pleural Effusion
Overview of Pleural Effusions

• Abnormal collection of fluid in the pleural space

• Transudate vs. exudate
  • Normal pleural fluid: pH 7.60-7.64, protein < 1-2 g/dL, < 1000 WBCs/mm³, glucose ~ plasma glucose, LDH < 50% plasma LDH
  • Light’s criteria for exudates:
    – Pleural fluid protein/serum protein > 0.5
    – Pleural fluid LDH/serum LDH > 0.6
    – Pleural fluid LDH > 200 IU/L or 2/3 normal upper limit for serum
  • Exudate*
    – Pleural fluid total protein > 2.9 g/dL
    – Pleural fluid protein/serum protein > 0.5
    – Pleural fluid cholesterol > 45 mg/dL
    – Pleural fluid LDH/serum LDH > 0.6

Overview of Pleural Effusions

• Transudates
  – CHF (as in this patient)
  – Cirrhosis
  – Nephrotic syndrome
  – Others-urinothorax, myxedema, peritoneal dialysis

• Exudates
  – Infection
  – Malignancy
  – PE
  – Chylothorax
  – Hemothorax
  – Others-drug induced, SLE, RA, vasculitis (Wegener’s, Churg-Strauss), GI (pancreatitis, esophageal rupture)
Diagnosis and Treatment of Pleural Effusions

• Diagnostic thoracentesis
• Therapeutic thoracentesis for symptomatic effusions
• Tube thoracostomy for hemothorax
• Tube thoracostomy empyemas ± tPA
  – pus on thoracentesis, a positive Gram stain, glucose < 60 mg/dL, pH < 7.20, or elevated LDH
• Treat underlying disorder
Overview of Ascites

• Theories on pathophysiology
  – Underfilling-insufficient sequestration of fluid secondary to portal hypertension leading to activation of renin-angiotensin-aldosterone system
  – Overflow-inappropriate retention of Na and H\textsubscript{2}O by renal system, hepatorenal reflex
  – Peripheral arterial vasodilation hypothesis-vasodilation secondary to portal hypertension leading to decreased effective arterial volume and renal Na retention
  – Decreased oncotic pressure secondary to hypoalbuminemia
Etiology of Ascites

Serum ascites albumin gradient

\[ \text{SAAG (g/dL)} = \text{Serum Albumin} - \text{Ascites Albumin} \]

<table>
<thead>
<tr>
<th>( \geq 1.1 \text{g/dL} )</th>
<th>(&lt; 1.1 \text{g/dL} )</th>
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</thead>
<tbody>
<tr>
<td>Cirrhosis</td>
<td>Peritoneal carcinomatosis</td>
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<tr>
<td>Alcoholic hepatitis CHF</td>
<td>Peritoneal TB</td>
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<tr>
<td>Massive hepatic metastases</td>
<td>Pancreatitis</td>
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<tr>
<td>Vascular occlusion</td>
<td>Serositis</td>
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<tr>
<td>Fatty liver disease of pregnancy</td>
<td>Nephrotic syndrome</td>
</tr>
<tr>
<td>Myxedema</td>
<td>Bowel obstruction / infarction / perforation</td>
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Runyon et al (1992)
Treatment

• Sodium restriction

• Diuretics
  – Spironolactone (25-200 mg/d PO qd or divided bid) ± furosemide (40-120 mg/day PO divided qd-bid; start 20-80 mg POx1, increase 20-40 mg q6-8hrs or 20-40 mg mg IV/IM; titrate up 20mg q2hrs; Max: 600mg/day)

• Therapeutic paracentesis
  – Large volume > 4-6L with albumin replacement
    • 5 g of albumin/ L of ascitic fluid (over 5L) to decrease rate of complications

• TIPS
  – For diuretic-refractory ascites
  – Stent is percutaneously placed from the right jugular vein into the hepatic vein. Connects portal and systemic circulations.
Patient’s hospital course

CHF exacerbation-patient was initially diuresed with IV Lasix for severe volume overload and then transitioned to PO Demadex. He was on dobutamine for increased creatinine, which was briefly switched to milrinone for his elevated wedge and PA pressures by right heart cath. Left heart cath showed new non-obstructive coronary artery disease. Statin ACE inhibitor and aspirin were added to his medication regiment. Cardiac echo finding: severe tricuspid valve regurgitation, moderate to severe mitral valve regurgitation, moderate aortic regurgitation, and an EF of 25%. Patient was in atrial fib/flutter during hospitalization. Coumadin was held due to risks.

At time of discharge patient remained significantly volume overloaded despite diuresis of a significant volume, however the patient had improved shortness of breath and was able to ambulate with assistance. BUN and creatinine had increased from admission and Demadex dose was decrease.