

## Spontaneous Bacterial Peritonitis in Cirrhosis

### The bottom line:
- SBP is common, and portends a poor prognosis
- The clinical exam is unpredictable, so consider SBP in any patient with cirrhosis
- Treatment modalities are controversial
- Consider retapping effusions when patient does not improve or has a high initial peritoneal WBC count

### The basics:
- SBP that occurs in cirrhotic patients carries a very adverse prognosis, with 30-50% survival at 1 year and 20-30% at 2 years.
- Risk factors for SBP are: worse degrees of cirrhosis (Childs C), Fluid protein < 1 g/dL (correlates with decreased opsonization activity), GI Bleeding (20% are infected at the time of the bleed, and 30-40% develop infection during the hospitalization), Previous SBP, UTI, and Lines/catheters.
- Symptoms/signs of SBP: fever (69%), abd pain (59%), encephalopathy (54%), abd tenderness (49%), no sx in 10%.

<table>
<thead>
<tr>
<th>Type</th>
<th>PMN</th>
<th>Culture</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous Bacterial Peritonitis (SBP)</td>
<td>&gt;250</td>
<td>Usually monomicrobial (60% Gram negative, 25% Gram positive)</td>
<td>See above</td>
</tr>
<tr>
<td>Culture-negative neutrocytic ascites</td>
<td>&gt;250</td>
<td>Negative</td>
<td>Clinically and prognostically like SBP; need to look for TB, carcinomatosis, pancreatitis</td>
</tr>
<tr>
<td>(CNNA)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Monomicrobial non-neutrocytic bacterascites (MNB)</td>
<td>&lt;250</td>
<td>Monomicrobial</td>
<td>With sx acts exactly like SBP (may be early SBP); without sx has better prognosis, but need to retap</td>
</tr>
<tr>
<td>Secondary Peritonitis</td>
<td>&gt;250</td>
<td>Polymicrobial</td>
<td>Needs surgical rx + antibiotics</td>
</tr>
<tr>
<td>Polymicrobial Bacterascites</td>
<td>&lt;250</td>
<td>Polymicrobial</td>
<td>Usually secondary to “tapping the bowel” (1 in 1000)</td>
</tr>
</tbody>
</table>

### Tapping Basics:
- 40% of patients with clinical manifestations suggestive of SBP and elevated PMN count have negative cultures; the yield of cultures can be increased by sending the fluid in blood culture bottles (67% vs 31%). Most common organisms are *E. coli* and *E. faecalis*.
- Tapping is safe even in coagulopathic patients (1% hematoma, 0.01% hemoperitoneum, 0.01% infection rate)
- In traumatic taps, subtract 1 PMN for every 250 RBC’s

### When to tap/retap:
- New ascites
- Admission of all patients with cirrhotic ascites
- Deterioration in clinical status
- Complication of cirrhosis (GI bleed, confusion).
- Polymicrobial culture OR positive culture with negative PMN’s (MNB that isn’t being treated)
- In general should retap 24-48 hrs after treatment has started for pts with WBC >1000 or lack of improvement. In one study PMN>1000 was associated with 88% mortality.

### Treatment:
- Various regimens have been studied; iv cephalosporins (cefotaxime, ceftriaxone), augmentin, and oral norfloxacin (for patients with uncomplicated SBP) can be used. Short course of IV (2 days), followed by oral therapy as effective as long course of IV.
• Treatment for 5 days suggested, as no better outcomes with prolonged treatment in cases where pts were improving clinically and PMN count less than 250 on day 5. If pt not improving or WBC not decreased after retapping, consider prolonged treatment or atypical organisms (tuberculosis, fungal, etc).
• IV albumin in addition to antibiotics showed reduced rate of renal impairment and death when compared with antibiotics alone (absolute risk reduction 23%).

**Prophylaxis:**
• GI Bleeds: meta-analysis demonstrated a mortality benefit; no difference if oral or iv; recommended regimen is norfloxacin 400 po q12
• Previous SBP: agents (qd norfloxacin, qwk cipro, septra) have shown a decrease in 1 year recurrence of SBP from 68% to 20% as well as a survival benefit (meta-analysis). The problem is that recurrent episodes of SBP tend to be with gram-positive organisms (including MRSA)
• Controversial: T. Protein < 1 – higher risk category, but is it worth treating? The jury is still out; some recommend treating in house only

**Hepatic Hydrothorax and Spontaneous Bacterial Empyema** (Xiol et al, Am J Medicine, 2001)
• Patients with cirrhosis can have associated pleural effusions (R>L) that are termed “hepatic hydrothorax” (10% of patients with cirrhosis). Typically, the fluid from hepatic hydrothorax is a bland transudate that is similar to ascitic fluid; the pathogenesis of hepatic hydrothorax is thought to be related to microfenestrations in the diaphragm that allow communication of fluid between the peritoneum and pleural space.
• However, in some cases, hepatic hydrothorax may exist in the absence of ascites.
• In addition, just as ascitic fluid can become infected (SBP), the hydrothorax can become infected as well, leading to spontaneous bacterial empyema (SBE).
• The dx of SBE is made by tapping the pleural fluid and obtaining a PMN count of > 250 with a positive culture or PMN count > 500 (in the absence of parapneumonic effusion). Typically, the fluid will have a low protein and high albumin gradient, but this will vary given the degree of infection.
• Studies from the literature have shown that about 15% of patients with cirrhosis on first tap of pleural fluid will have SBE (70% will have hepatic hydrothorax). In addition, the correlation between SBP and SBE is weak, with discrepancy in fluid findings found in up to 40% of cases (but small total number of patients).
• In general, SBE can be treated without chest tube drainage, but look at the fluid characteristics first.

References:
Such and Runyon, CID, 1998; Rimola et al, J. Hepatology, 2000; Sort et al, NEJM 1999; Thanopoulou et al, European Journal of Internal Medicine, 2002; Franca et al, J Gastroenterology 2002; several others....