# Revision of Tropical Medicine Practical

**General examination**

<table>
<thead>
<tr>
<th>Overview</th>
<th>Vital signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Mentality</td>
<td>• Pulse</td>
</tr>
<tr>
<td>• Decubitus</td>
<td>• Temperature</td>
</tr>
<tr>
<td>• Look (appearance)</td>
<td>• Blood pressure</td>
</tr>
<tr>
<td>• Built</td>
<td>• Respiratory rate</td>
</tr>
</tbody>
</table>

### 4X4 Complexions (colors)

<table>
<thead>
<tr>
<th>Regional examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Pallor</td>
</tr>
<tr>
<td>• Cyanosis</td>
</tr>
<tr>
<td>Jaundice</td>
</tr>
<tr>
<td>Pigmentation &amp; other complexions</td>
</tr>
</tbody>
</table>

### LN Importance of LN examination in abdominal cases

1. Malignant LN eg left supraclav. (virchow’s gland) in advanced cancer stomach (Troisier sign).
2. Very important in all cases of splenomegaly → (lymphoma & leukaemia).
3. T.B peritonitis → tuberculous LN

### Hand (of special importance)

<table>
<thead>
<tr>
<th>Upper limb</th>
<th>Hand</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Shape</td>
<td>• Blue in cyanosis</td>
</tr>
<tr>
<td>2. Tremors (Flapping or fine)</td>
<td>• Hyperkeratosis in vit. A def</td>
</tr>
<tr>
<td>3. Nails</td>
<td>• Xanthomatosis in hypercholesterolaemia</td>
</tr>
<tr>
<td></td>
<td>• Warm in hyperdynamic circulatoin.</td>
</tr>
</tbody>
</table>

### Upper limb

1. Koilonychia In iron deficiency anaemia.
2. Leuconychia Whitening of the nail plates. In hypoalbuminemia (L.C.F)
3. Terry’s nails Whitening of proximal 80% of the nail, having a small rim of peripheral reddening.
   - Older people.
   - Liver cirrhosis
4. Azure lunule Blue lunule occur in Wilson disease

### Nails

<table>
<thead>
<tr>
<th>Definition</th>
<th>Clubbing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not defined by ↑nail convexity but by:</td>
<td>Not defined by ↑nail convexity but by:</td>
</tr>
<tr>
<td>• Loss of subungal angle.</td>
<td>• Loss of subungal angle.</td>
</tr>
<tr>
<td>• Ballotability of the nail.</td>
<td>• Ballotability of the nail.</td>
</tr>
<tr>
<td>• Abnormal phalangeal depth ratio.</td>
<td>• Abnormal phalangeal depth ratio.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Types</th>
<th>GIT causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cyanotic (blue clubbing)</td>
<td>• Iry biliary cirrhosis.</td>
</tr>
<tr>
<td>Toxic (pale clubbing)</td>
<td>• Bilharzial intestinal polyposis.</td>
</tr>
<tr>
<td></td>
<td>• U.C &amp; Crohn’s dis.</td>
</tr>
<tr>
<td></td>
<td>• Chronic pyelonephritis.</td>
</tr>
<tr>
<td></td>
<td>• Abdominal lymphoma</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>nb</th>
<th>GIT causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Causes of unilateral clubbing = causes of unequal pulse volume.</td>
<td>• Malabsorption syndrome.</td>
</tr>
<tr>
<td>2. Causes of clubbing in LL only = causes of differential cyanosis.</td>
<td></td>
</tr>
</tbody>
</table>

### Lower limb

<table>
<thead>
<tr>
<th>Lower limb</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Lymph nodes</td>
<td>Inguinal LN examination</td>
</tr>
<tr>
<td>2. Pulsation</td>
<td>Femoral artery, popliteal, post tibial, dorsalis pedis</td>
</tr>
<tr>
<td>3. Blood pressure</td>
<td>In AR &amp; coarctation of the aorta</td>
</tr>
<tr>
<td>4. Cyanosis &amp; clubbing of toes</td>
<td></td>
</tr>
<tr>
<td>5. Pellargric rash (over greater trochanter) and signs of vitamin A deficiency</td>
<td></td>
</tr>
<tr>
<td>6. Ulcers</td>
<td>DM, Leprosy, trauma, VV, ischemia, hemolytic anemia</td>
</tr>
<tr>
<td>7. Muscle wasting</td>
<td></td>
</tr>
<tr>
<td>8. Edema</td>
<td></td>
</tr>
</tbody>
</table>
### Abdominal examination

<table>
<thead>
<tr>
<th>Inspection</th>
<th>Mild inspection</th>
<th>Inspection of sides</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Inspection</strong></td>
<td>1. Subcostal angle</td>
<td>4. Umbilicus</td>
</tr>
<tr>
<td></td>
<td>2. Epigastric pulsation</td>
<td>5. Suprapubic hair distribution</td>
</tr>
<tr>
<td></td>
<td>3. Divercation of recti</td>
<td>6. Hernial orifices</td>
</tr>
<tr>
<td></td>
<td>4. Umpilicus</td>
<td>1. Contour of abdomen</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Dilated veins</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Skin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4. Scars</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5. Movement with resp</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6. Visible peristaltis</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Causes of abdominal enlargement</th>
<th>Generalized</th>
<th>Localized</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fluid (ascites)</td>
<td>1. Hernias → size ↑ with cough</td>
<td></td>
</tr>
<tr>
<td>2. Fat (obesity)</td>
<td>2. Masses in abdominal wall (abcess &amp; tumors)</td>
<td></td>
</tr>
<tr>
<td>3. Flatus and faeces</td>
<td>3. Enlargement of intra-abdominal organs</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Causes of hepatomegaly</th>
<th>1. Infection</th>
<th>2. Congestion</th>
<th>3. Infiltration</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Viral: Viral hepatitis, IMN, CMV</td>
<td>• Rt side ht failure</td>
<td>• Amyloidosis</td>
<td></td>
</tr>
<tr>
<td>• Bacterial: Brucellosis, T.B</td>
<td>• Tricusbed valve disease</td>
<td>• Leukemia</td>
<td></td>
</tr>
<tr>
<td>• Parasitic: Bilharziasis, Malaria, Fasciola</td>
<td>• Constrictive pericarditis</td>
<td>• Lymphoma</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Causes of splenomegaly</th>
<th>4. Neoplastic</th>
<th>5. Miscellaneous</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Anemia</td>
<td>4. Blood disease:</td>
<td>5. Neoplastic</td>
</tr>
<tr>
<td>• Polycythemia</td>
<td>• Viral: IMN, CMV</td>
<td>6. Miscellaneous</td>
</tr>
<tr>
<td>• Myeloproliferative disease</td>
<td>• Bacterial: Septecemia, Typhoid fever, Brucellosis T.B, Syphilis</td>
<td>• Collagen disease</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Grades of splenomegaly</th>
<th>Mild</th>
<th>Moderate</th>
<th>Huge</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Spleen just palpable under costal margin</td>
<td>Spleen is palpable between costal margin and umbilicus</td>
<td>Spleen is palpable below the umbilicus</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Causes of huge splenomegaly</th>
<th>1. Infection</th>
<th>2. Congestion</th>
<th>3. Infiltration</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Bilharziasis</td>
<td>• Leukemia</td>
<td>• Amyloidosis</td>
<td></td>
</tr>
<tr>
<td>2. Chronic malaria</td>
<td>• Infective endocarditis</td>
<td>• Sarcoiosis</td>
<td></td>
</tr>
<tr>
<td>3. Kala azar</td>
<td>• Typhoid fever</td>
<td>• Lipid storage disease</td>
<td></td>
</tr>
<tr>
<td>4. Chronic myloid leukemia</td>
<td>• Causes of portal hypertension</td>
<td>• Gucher disease</td>
<td></td>
</tr>
<tr>
<td>5. Hairy cell leukemia</td>
<td>• Amyloidosis</td>
<td>• Leukemia</td>
<td></td>
</tr>
<tr>
<td>6. Myelofibrosis, myelosclerosis</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Causes of tender spleen</th>
<th>4. Causes of huge splenomealy</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Septicemia</td>
<td>1. Infection</td>
</tr>
<tr>
<td>• Infective endocarditis</td>
<td>2. Infarction</td>
</tr>
<tr>
<td>• Typhoid fever</td>
<td>*refer above</td>
</tr>
<tr>
<td></td>
<td>Perisplenitis, splenic rub</td>
</tr>
<tr>
<td></td>
<td>3. Sickle cell anaemia</td>
</tr>
<tr>
<td></td>
<td>4. Causes of huge splenomealy</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Differences between splenic swelling and kidney swelling</th>
<th>Spleenic swelling</th>
<th>Kidney swelling</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Notch on anterior border (pathognomonial)</td>
<td>No notch; reniform in shape</td>
</tr>
<tr>
<td></td>
<td>Hand cannot be insinuated ( ) swelling &amp;costal margin</td>
<td>Can be insinuated ( ) swelling &amp; costal margin</td>
</tr>
<tr>
<td></td>
<td>Does not fill renal angle</td>
<td>Fills the renal angle</td>
</tr>
<tr>
<td></td>
<td>Dull on percussion &amp; continuous with splenic dullness</td>
<td>Percussion above swelling → band of colonic resonance anteriorly</td>
</tr>
<tr>
<td></td>
<td>Moves with respiration</td>
<td></td>
</tr>
<tr>
<td></td>
<td>No posterior ballotment</td>
<td>Posterior ballotment</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Traub’s area</th>
<th>Area of tympanetic resonance over fundus of the stomach</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>From above</td>
</tr>
<tr>
<td></td>
<td>From left</td>
</tr>
<tr>
<td></td>
<td>From right</td>
</tr>
<tr>
<td></td>
<td>From below</td>
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### Auscultation

| 1. Intestinal sounds |
| 2. Vascular sound: Arterial bruit, Venous hum |
| 3. Friction rub : Splenic rub, Hepatic rub |
| 4. Succussion splash |
| 5. Minimal ascites (puddle sign) |
| 6. Lower border of liver (scratching method) |
### Approach to ascites

**Definition**

Ascites is defined as pathological accumulation of fluid within the peritoneal cavity.

**Liver diseases:**
- Portal hypertension is a prerequisite for ascites formation
- Cirrhosis
- Alcoholic hepatitis
- Fulminant hepatitis
- Veno-occlusive disease
- Budd-Chiari syndrome
- Massive liver metastasis

**Cardiac disease:**
- High output and low output heart failure → decrease effective arterial blood volume → renal sodium retention
- Pericardial disease: rare cause of cardiac ascites

**Differential diagnosis**

**Liver diseases:**
- Portal hypertension is a prerequisite for ascites formation
- Cirrhosis
- Alcoholic hepatitis
- Fulminant hepatitis
- Veno-occlusive disease
- Budd-Chiari syndrome
- Massive liver metastasis

**Pancreatic ascites:**
- Occur as a complication of:
  1. Severe acute pancreatitis
  2. Pancreatic duct rupture
  3. Leakage form pancreatic pseudocyst
  4. Many patients with pancreatic ascites have underlying cirrhosis

**Cardiac disease:**
- High output and low output heart failure → decrease effective arterial blood volume → renal sodium retention
- Pericardial disease: rare cause of cardiac ascites

**Infectious diseases**
- E.g. TB peritonitis
- Persistent abdominal pain and fever
- Often extraperitoneal tuberculosis

**Malignant ascites:**
- Malignancy-related ascites signify advanced disease in most cases
- Mechanism of ascites formation:
  - Depend on location of tumour
  - Peritoneal carcinomatosis → exudation of proteinaceous fluid into the peritoneal cavity
  - Massive liver metastasis → PHT
  - HCC → PHT, tumor-induced portal vein thrombosis

**Pathogenic ascites**

**1. Vasodilatation theory**
- Portal hypertension → vasodilatation and ↓ effective arterial bl. Volume → ↑ renal sodium retention → fluid accumulation

**2. Lymph imbalance theory:**
- The lymph production or the lymph quantity can no longer be drained via the lymph vessels. (Weeping liver)

**Biliary ascites:**
- Gall bladder rupture due to gangrene of GB
- After biliary surgery or intestinal perforation → bile accumulate

**Nephrogenic ascites:**
- E.g. Nephrotic syndrome
- Protein loss in urine → decrease intravascular volume → increase renal sodium retention

**Miscellaneous disorders**

1. Myxedema
2. Ovarian disease
3. SLE
4. Megs syndrome

**Chylous ascites:**
- Obstruction or damage of chyle-containing lymphatic channels (lymphoma, other malignancy)
- Ascetic fluid is milky with triglyceride content >200 mg/dL

**Pathogenesis of cirrhotic ascites**

**Investigations**

**1. Liver function test:**
- ALT, AST, ALK P
- Prolonged prothrombin time in liver disease
- Hypoalbuminemia

**2. Thrombocytopenia:**
- Suggest liver disease

**3. Serum creatinine**

**4. Urine analysis**
- Protein loss in nephrotic syndrome or bilirubinuria with jaundice

**5. α fetoprotein in HCC**

**6. Ascetic fluid examination**

**7. Chest x-ray: TB**

**8. Abdominal U/S:**
- Detect as little as 100 mL of ascetic fluid.
- Confirm presence of portal hypertension
- Differentiate obesity from ascites
- Detect mass lesion in liver, ovaries, pancreases
### Ascetic fluid analysis

| Colour | 1. **Serous ascites**: can be clear or turbid, green, straw-colored, bile stained e.g hepatogenic, pancreatic, malignant, inflammatory  
2. **Hemorrhagic ascites**: in malignant disease  
3. **Turbed ascites**: e.g bacterialy infected, malignant, pancreatogeic, in budd-chiari syndrome  
4. **Chylous ascites**: in liver cirrhosis due to impaired lymphatic drainage, malignant disease, TB, intestinal lymphangictasia, Whipple disease |
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Cell count</td>
<td>Fluid with &gt; 250 PMNs/mm3 is presumed infected.</td>
</tr>
</tbody>
</table>

### Total protein

- **SAAG <1.1 g/dL**
  - Peritoneal
  - Carcinomatosis
  - TB peritonitis
  - Pancreatic ascites
  - Biliary ascites
  - Nephrotic syndrome
- **SAAG ≥ 1.1 g/dL**
  - Cirrhosis
  - Alcoholic hepatitis
  - Cardiac ascites
  - Budd-Chiari syndrome
  - Acute fatty liver of pregnancy
  - Myxedema

### Bacteriology

- Gram –ve bacteria can be detected in 70%
- Gram +ve bacteria in 25%
- Anaerobe in about 5%

### Cytology

- Has specificity of 97-100% for detection of malignant ascites

### Refractory ascites

**Definition:** Ascites that cannot be mobilized or the early recurrence of ascites (i.e., after, therapeutic parancentesis) cannot be satisfactorily prevented by medical therapy

1. **Diuretic resistant:** Lack of response to dietary sodium restriction and intensive diuretic treatment
2. **Diuretic intractable:** Development of diuretic-induced complication precludes the use of effective diuretic doses

### Causes

| 1. Excessive sodium levels in the body  
2. Hypovolaemia  
3. Absence of peripheral edema  
4. Excessive volume of ascetic fluid in abdomen  
5. Spontaneous bacterial peritonitis  
6. Deterioration of liver function (GIT bleeding)  
7. Deterioration of renal function  
8. Unfavourable diuretic effects  |  |
|---|---|
| - Inadequate diuretic absorption  
- Unsuitable diuretic agent  
- Incorrect dosage  
- Drug interactions e.g. nonsteroidal |

### Mechanical complications

| 1. Increased physical immobility  
2. Dyspnea (hepatic hydrothorax, elevate diaphragm, atelectasis)  
3. Elevated portal venous pressure  
4. Compression of vessels (IVC-renal vein)  
5. Formation and ruptured of hernias  
6. Dislocation of organs  
7. ↑ gastro-esophageal reflux  |  |

### Bacterial complications

- Spontaneous bacterial peritonitis

### Metabolic complications

| 1. Disturbances of electrolyte metabolism  
2. Disturbances of protein metabolism  
3. Hepatic encephalopathy  |

### Hepatorenal syndrome
<table>
<thead>
<tr>
<th>Treatments</th>
<th>Non – cirrhotic ascites</th>
<th>Cirrhotic ascites</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Peritoneal carcinomatosis</strong></td>
<td></td>
<td>General principles:</td>
</tr>
<tr>
<td>▪ Diuretics for peripheral edema</td>
<td>▪ Establish the diagnosis</td>
<td></td>
</tr>
<tr>
<td>▪ Therapeutic paracentesis</td>
<td>▪ Identify precipitating factors of ascites</td>
<td></td>
</tr>
<tr>
<td>▪ Chemotherapy</td>
<td>▪ Sodium balance is the key to successful ttt</td>
<td></td>
</tr>
<tr>
<td><strong>Tuberculous ascites</strong></td>
<td>▪ Sodium restriction is mainstay of ttt</td>
<td></td>
</tr>
<tr>
<td>▪ Anti-tuberculous therapy</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Lymphatic leak</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>▪ Resolve spontaneously</td>
<td></td>
<td></td>
</tr>
<tr>
<td>▪ Peritoneovenous shunting</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Diuretic therapy:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. <strong>Spironolactone</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>▪ The diuretic of choice for single-agent therapy</td>
<td></td>
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</tr>
<tr>
<td>2. <strong>Combination of spironolactone &amp; furosemide</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>▪ Spironolactone 100mg , furosemide 40mg</td>
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<tr>
<td>▪ Increased every 3-5 days if weight loss and urinary sodium excretion is inadequate</td>
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<tr>
<td>▪ Max dose: spironolactone 400 mg+ furosemide 160 mg</td>
<td></td>
<td></td>
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<tr>
<td>3. <strong>Amiloride 10- 40 mg</strong> can be substituted for spiranolate (painful gynaecomastia)</td>
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<tr>
<td><strong>Monitoring:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>▪ Daily weight</td>
<td></td>
<td></td>
</tr>
<tr>
<td>▪ Daily monitoring of Electrolytes/Urea/Creatinine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>▪ Patients with massive edema can be allowed to loose weight rapidly</td>
<td></td>
<td></td>
</tr>
<tr>
<td>▪ Once edema has subsided 0.5 kg daily weight loss is considered appropriate</td>
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</tbody>
</table>

1. **Large volume paracentesis (LVP)** |
| ▪ Removal of ≥ 5 L of fluid by therapeutic paracenteses |
| ▪ Safe and effective treatment. |
| ▪ Not performed more often than every 2 weeks |
| ▪ Rapid resolution of tense ascites than diuretic |
| ▪ IV volume expenders are unnecessary for paracenteses of < 5 L of fluid |

2. **Transiugular intrahepatic portosystemic stent-shunt (TIPS)** |
| ▪ Bridges a branch of the hepatic vein with a branch of portal vein. |
| ▪ Promptly reduces portacaval pressure gradient. |
| ▪ Effective in 90% of patients. |
| ▪ Ascites improves within 1-3 months after TIPS |

3. **Peritoneovenous shunting (PVS):** |
| ▪ Not change rates of survival |
| ▪ Complications : |
| ▪ Bacterial infections |
| ▪ Congestive heart failure |
| ▪ DIC |
| ▪ Shunt thrombosis |

4. **Liver transplantation (LT)**
**Spontaneous Bacterial Peritonitis (SBP)**

<table>
<thead>
<tr>
<th>Definition</th>
<th>Infection of the ascitic fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nb</td>
<td>10-27% patients with ascites admitted to hospital have SBP</td>
</tr>
</tbody>
</table>

**Pathogenesis of ascitic fluid infection**

1. Reduction in opsonic and bactericidal activity of ascitic fluid
   - Decrease C3 in ascitic fluid (C3 <20mg/dl)
   - Ascitic protein value (<1.0g/dl)
2. Impaired function of RES phagocytosis system
3. Dysfunction of polymorphonuclear neutrophils

This multifactorial weakness in defense allow bacterial penetration of ascitic fluid to be effected by:

1. Transmural migration in portal hypertension with greater permeability of intestinal wall
2. Systemic bacteremia
3. Invasion of bacteria via fallopian tube
4. Lymphatic flow into ascitic fluid

**Risk for ascitic fluid infection**

1. Prior episode of SBP
2. GIT bleeding (esp, variceal hemorrhage).
3. An ascitic fluid total protein <1.0 g/dL

**Signs**

- Abdominal tenderness
- Fever
- Hypotension
- Tachycardia

**Symptoms**

- Leukocytosis
- Azotemia
- Increasing bilirubin

- Abdominal pain
- Rigors
- Nausea
- Vomiting

- Diarrhea
- Encephalopathy
- Malaise

**Classification**

<table>
<thead>
<tr>
<th>Ascitic fluid analysis</th>
<th>Category</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous bacterial peritonitis (SBP)</td>
<td>PMN ≥250/mm³, single organism</td>
</tr>
<tr>
<td>Culture-negative neutrocytic ascites (CNNA)</td>
<td>PMN ≥250/mm³, negative culture</td>
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<tr>
<td>Monomicrobial non-neutrocytic bacterascites (MNB)</td>
<td>PMN&lt;250/mm³, single organism</td>
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<tr>
<td>Polymicrobial bacterascites</td>
<td>PMN&lt; 250/mm³, multiple organisms</td>
</tr>
<tr>
<td>Secondary bacterial peritonitis</td>
<td>PMN≥250/mm³, multiple organisms</td>
</tr>
</tbody>
</table>

**Empiric antibiotic**

1. **3rd-generation cephalosporins (Cefotaxime)**
   - The antibiotic of choice for empiric treatment.
   - Cover > 95% of the flora responsible for SBP
   - Dose is 2 g IV. every 8 h. for 5 day
2. **Alternative antibiotic regimens**
   - Amoxicillin-clavulanic acid
   - Fluoroquinolones.

**Follow-up paracentesis**

Indicated whenever:

1. Secondary (surgical) bacterial peritonitis is suspected.
2. The typical clinical response to cefotaxime does not occur (i.e., fall in serum WBCs.).

**Intravenous volume expanders (e.g., albumin)**

1. Increase central volume and maintain renal perfusion
2. Administration of IV albumin 1.5 g/kg at the time of diagnoses SBP and 1.0 g/kg on day 3 of antibiotic treatment decreased the risk of renal insufficiency and SBP-related mortality

**Prophylaxis**

Indications: Patients at high risk for development of SBP

Prophylactic antibiotic regimens:
1. Norfloxacin: (400 mg orally daily)
2. Trimethoprim-sulfamethoxazole.