Management of Cirrhotic Patients
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Case
- 50 yo male with HCV-related cirrhosis complicated by ascites (stable on low dose diuretics) and encephalopathy (stable on lactulose) presents with worsening ascites and mild encephalopathy
- Labs: Na 130, Cr 1.2, Tbil 5, Alb 3.0, INR 1.4, AST/ALT
What is Cirrhosis?

Cirrhosis

- End stage of any chronic liver disease
- Characterized histologically by regenerative nodules surrounded by fibrous tissue
- Clinically there are two types of cirrhosis:
  - Compensated
  - Decompensated
Cirrhosis

Normal

Cirrhosis

Irregular surface

Nodules surrounded by fibrous tissue
What is the Natural History of Cirrhosis?

Natural History of Chronic Liver Disease

Chronic liver disease → Compensated cirrhosis → Decompensated cirrhosis → Death

Development of complications:
- Variceal hemorrhage
- Ascites
- Encephalopathy
- Liver Cancer
Development of Complications in Compensated Cirrhosis

Probability of developing event:
- Ascites
- Jaundice
- Encephalopathy
- GI hemorrhage

Gines et al., Hepatology 1987; 7:122

Decompensation Shortens Survival

Median survival ~ 9 years
Median survival ~ 1.6 years
Gines et al., Hepatology 1987; 7:122
Cirrhosis - Diagnosis

- Cirrhosis is a histological diagnosis
- However, in patients with chronic liver disease the presence of various clinical features suggests cirrhosis
- The presence of these clinical features can be followed by non-invasive testing, prior to liver biopsy
In Whom Should We Suspect Cirrhosis?

- Any patient with chronic liver disease
  - Chronic abnormal aminotransferases and/or alkaline phosphatase
- Physical exam findings
  - Stigmata of chronic liver disease (muscle wasting, vascular spiders, palmar erythema)
  - Palpable left lobe of the liver
  - Small liver span
  - Splenomegaly
  - Signs of decompensation (jaundice, ascites, asterixis)

Laboratory

- Liver insufficiency
  - Low albumin (< 3.8 g/dL)
  - Prolonged prothrombin time (INR > 1.3)
  - High bilirubin (> 1.5 mg/dL)
- Portal hypertension
  - Low platelet count (< 175 x1000/µL)
- AST / ALT ratio > 1
In Whom Should We Suspect Cirrhosis?

**Imaging studies**
- Liver-spleen scan
  - Small liver, irregular uptake
  - Splenomegaly
  - Colloid shift to bone marrow
- CAT scan / Ultrasound
  - Nodular liver
  - Splenomegaly
  - Venous collaterals

CAT Scan in Cirrhosis

Liver with an irregular surface  
Collaterals  
Splenomegaly
Confirmatory Liver Biopsy Is Not Always Necessary in Cirrhosis

- Liver biopsy is not necessary in the presence of any of the following:
  - Decompensated cirrhosis (variceal hemorrhage, ascites, encephalopathy)
  - Liver-spleen and/or CAT scan diagnostic of cirrhosis
- Liver biopsy is not necessary for pre-transplant evaluation

Diagnostic Algorithm

Patient with chronic liver disease and any of the following:
- Variceal hemorrhage
- Ascites
- Hepatic encephalopathy

Yes No

Physical findings:
- Enlarged left hepatic lobe
- Splenomegaly
- Stigmata of chronic liver disease

Laboratory findings:
- Thrombocytopenia
- Impaired hepatic synthetic function

Yes No

Radiological findings:
- Small nodular liver
- Intra-abdominal collaterals
- Ascites
- Splenomegaly
- Colloid shift to spleen and/or bone marrow

Yes No

Liver biopsy not necessary for the diagnosis of cirrhosis

Liver biopsy
Pathophysiology of Hepatic Encephalopathy

↑ Ammonia

- Upregulation of astrocytic peripheral benzodiazepine receptors (PBR)
- Neurosteroid production
- Modulation of GABA<sub>A</sub> receptor

Hepatic encephalopathy

Hepatic Encephalopathy Is A Clinical Diagnosis

- Clinical findings and history important
- Ammonia levels are unreliable
- Measurement of ammonia not necessary
- Measurement of ammonia levels may be supportive when diagnosis is in doubt
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**Stages of Hepatic Encephalopathy**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Mental state</th>
<th>Neurologic signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Mild confusion: limited attention span, irritability, inverted sleep pattern</td>
<td>Incoordination, tremor, impaired handwriting</td>
</tr>
<tr>
<td>2</td>
<td>Drowsiness, personality changes, intermittent disorientation</td>
<td>Asterixis, ataxia, dysarthria</td>
</tr>
<tr>
<td>3</td>
<td>Somnolent, gross disorientation, marked confusion, slurred speech</td>
<td>Hyperreflexia, muscle rigidity, Babinski sign</td>
</tr>
<tr>
<td>4</td>
<td>Coma</td>
<td>No response to pain, decerebrate posture</td>
</tr>
</tbody>
</table>

**Treatment of Hepatic Encephalopathy**

- Identify and treat precipitating factor
  - Infection
  - GI hemorrhage
  - Prerenal azotemia
  - Sedatives
  - Constipation
- Protein restriction should be avoided
### Lactulose

- **Currently the mainstay of therapy of HE; ~70% to 80% of patients with acute and chronic HE improve with lactulose treatment**
- **Administered orally, by mouth or through a nasogastric tube or via retention enemas**
- **Adjust to 2-3 bowel movements/day**
- **Principal side effects include abdominal distension, cramping, diarrhea, electrolyte changes, and flatulence**

Bajaj JS. *Aliment Pharmacol Ther* 2010;31:537-547

### Current HE Treatment Options

<table>
<thead>
<tr>
<th>Drug Name</th>
<th>Drug Class</th>
<th>Indication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactulose</td>
<td>Poorly absorbed disaccharide</td>
<td>Decrease blood ammonia concentration. Prevention and treatment of portal-systemic encephalopathy</td>
</tr>
<tr>
<td>Rifaximin</td>
<td>Non-aminoglycoside semi-synthetic, nonsystemic antibiotic</td>
<td>Reduction in risk of overt hepatic encephalopathy (HE) recurrence in patients ≥ 18 years of age.</td>
</tr>
<tr>
<td>Neomycin</td>
<td>Aminoglycoside antibiotic</td>
<td>Adjuvant therapy in hepatic coma Ototoxicity and nephrotoxicity</td>
</tr>
<tr>
<td>Metronidazole</td>
<td>Synthetic antiprotozoal and antibacterial agent</td>
<td>Not approved for HE Peripheral neurotoxicity</td>
</tr>
<tr>
<td>Vancomycin</td>
<td>Aminoglycoside antibiotic</td>
<td>Not approved for HE Bacterial resistance and renal toxicity</td>
</tr>
</tbody>
</table>

**Rifaximin**

- Minimally absorbed (<0.4%) oral antibiotic
- No clinical drug interactions reported
- No dosing adjustment required in patients with liver disease or renal insufficiency
- In registration trials, 91% of patients were given lactulose concomitantly
  - 58% reduction in risk of recurrent hepatic encephalopathy (NNT=4)
  - 50% reduction in risk of hospitalizations (NNT=9)


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**Cirrhosis**

- **Hepatic venous outflow block**
- **Arteriolar resistance (vasodilation)**
- **Effective arterial blood volume**
- **Activation of neurohumoral systems (renin, angiotensin, aldosterone)**
- **Sinusoidal pressure (HVPG \( \geq 10-12 \text{ mmHg} \))**
- **Ascites**
- **Sodium and water retention**
Natural History of Ascites

- **Portal Hypertension No Ascites**
  - HVPG <10 mmHg
  - Mild Vasodilation

- **Uncomplicated Ascites**
  - HVPG >10 mmHg
  - Moderate Vasodilation

- **Refractory Ascites**
  - HVPG >10 mmHg
  - Severe Vasodilation

- **Hepatorenal Syndrome**
  - HVPG >10 mmHg
  - Extreme Vasodilation

Ascitic Fluid Analysis

<table>
<thead>
<tr>
<th>Ascitic Fluid Laboratory Data</th>
<th>Routine</th>
<th>Unusual</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cell count and differentials</strong></td>
<td>Acid fast bacillus smear and culture</td>
<td>Cytology</td>
</tr>
<tr>
<td>Albumin</td>
<td>Triglyceride</td>
<td>Bilirubin</td>
</tr>
<tr>
<td>Total protein</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Serum-ascites albumin gradient (calculated)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Optional</td>
<td>Unhelpful</td>
<td></td>
</tr>
<tr>
<td>Culture in blood culture bottles</td>
<td>pH</td>
<td></td>
</tr>
<tr>
<td>Glucose</td>
<td>Lactate</td>
<td></td>
</tr>
<tr>
<td>Lactate dehydrogenase</td>
<td>Cholesterol</td>
<td></td>
</tr>
<tr>
<td>Amylase</td>
<td>Fibronectin</td>
<td></td>
</tr>
<tr>
<td>Gram’s stain</td>
<td>Glycosaminoglycans</td>
<td></td>
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</tbody>
</table>

Serum-Ascites Alubumin Gradient

<table>
<thead>
<tr>
<th>Serum-Ascites Albumin Gradient (SAAG)</th>
<th>Low Gradient (&lt; 1.1 g/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>High Gradient (≥ 1.1 g/dl)</td>
<td></td>
</tr>
<tr>
<td>Cirrhosis</td>
<td>Peritoneal carcinomatosis</td>
</tr>
<tr>
<td>Alcoholic hepatitis</td>
<td>Peritoneal tuberculosis</td>
</tr>
<tr>
<td>Cardiac failure</td>
<td>Pancreatic ascites</td>
</tr>
<tr>
<td>Massive liver metastases</td>
<td>Biliary ascites</td>
</tr>
<tr>
<td>Fulminant hepatic failure</td>
<td>Nephrotic syndrome</td>
</tr>
<tr>
<td>Budd-Chiari syndrome</td>
<td>Serositis</td>
</tr>
<tr>
<td>Portal-vein thrombosis</td>
<td>Bowel obstruction or infarction</td>
</tr>
<tr>
<td>Veno-occlusive disease</td>
<td></td>
</tr>
<tr>
<td>Fatty liver of pregnancy</td>
<td></td>
</tr>
<tr>
<td>Myxedema</td>
<td></td>
</tr>
<tr>
<td>&quot;Mixed&quot; ascites</td>
<td></td>
</tr>
</tbody>
</table>


Treatment of Ascites

- **Portal Hypertension**
  - No ascites: No specific therapy. Consider salt restriction.
  - Uncomplicated ascites
  - Refractory ascites
  - Hepatorenal syndrome

- **Portal Hypertension**
  - No ascites: No specific therapy. Consider salt restriction.
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Treatment of Ascites

- **Portal Hypertension**
  - No ascites

- **Uncomplicated ascites**

- **Refractory ascites**

- **Hepatorenal syndrome**

Management of Uncomplicated Ascites

- **Sodium Restriction**: 2 g (or 5.2 g of dietary salt) a day
  - Fluid restriction is not necessary unless there is hyponatremia (<125 mmol/L)

- **Diuretics**
  - Spironolactone 100-400 mg/day
  - Furosemide (40-160 mg/d) for inadequate weight loss or if hyperkalemia develops

- **Increase diuretics if weight loss <1 kg in the first week and < 2 kg/week thereafter**

- **Side effects**
  - Renal dysfunction, hyponatremia, hyperkalemia, encephalopathy, gynecomastia
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Treatment of Ascites

- Portal Hypertension
  - No ascites

- Uncomplicated ascites

- Refractory ascites

- Hepatorenal syndrome

Definition and Types of Refractory Ascites

- Diuretic-intractable ascites  80%
  Therapeutic doses of diuretics cannot be achieved because of diuretic-induced complications

- Diuretic-resistant ascites  20%
  No response to maximal diuretic therapy (400 mg spironolactone + 160 mg furosemide/day)

Arroyo et al. Hepatology 1996; 23:164
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### Treatment of Ascites

- **Portal Hypertension No Ascites**
- **Uncomplicated Ascites**
- **Refractory Ascites**
- **Hepatorenal Syndrome**

1. LVP + albumin
2. TIPS
3. PVS (in non-TIPS, non-transplant candidates)

**Characteristics of Hepatorenal Syndrome**

- Renal failure in patients with cirrhosis, advanced liver failure and severe sinusoidal portal hypertension
- Absence of significant histological changes in the kidney ("functional" renal failure)
- Marked arteriolar vasodilation in the extra-renal circulation
- Marked renal vasoconstriction leading to reduced glomerular filtration rate

LVP = large volume paracentesis
TIPS = transjugular intrahepatic portosystemic shunt
Two Types of Hepatorenal Syndrome

Type 1
- Rapidly progressive renal failure (2 weeks)
- Doubling of creatinine to >2.5 or halving of creatinine clearance (CrCl) to <20 ml/min

Type 2
- More slowly progressive
- Creatinine >1.5 mg/dL or CrCl < 40 ml/min
- Associated with refractory ascites

Arroyo et al., Hepatology 1996; 23:164

Survival in Different Types of Hepatorenal Syndrome (HRS)

Gines et al., Lancet 2003; 362:1819
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**Natural History of Hepatorenal Syndrome (HRS)**

- **Type 2 HRS**
  - **Creatinine (mg/dL):**
    - Months:
      - 0: 1.0, 1.5, 2.0, 3.0, 4.0, 5.0
      - Therapeutic paracenteses
    - Weeks:
      - 0: SBP, 1: 1.5, 2: 2.0, 3: 3.0
  - **Cefotaxime**

- **Type 1 HRS**

*Arroyo et al., Gastroenterology 2002; 122:1658*

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**Major Criteria in the Diagnosis of Hepatorenal Syndrome**

- Advanced hepatic failure and portal hypertension
- Creatinine > 1.5 mg/dL or creatinine clearance < 40 ml/min
- Absence of shock, bacterial infection, or nephrotoxic drugs
- Absence of excessive gastrointestinal or renal fluid loss
- No improvement in renal function after plasma volume expansion with 1.5 L of isotonic saline
- Urinary protein < 500 mg/dL and normal renal ultrasound

*Arroyo et al., Hepatology 1996; 23:164*
Ascites and Hyponatremia are Always Present in HRS

- Besides renal failure, patients with HRS have sodium and water retention
- **Ascites** is universal in patients with HRS. If ascites is absent, renal failure is more likely due to other causes
- **Hyponatremia** is almost universal in HRS. If serum sodium is normal, diagnosis of HRS is unlikely

Cirrhosis

- Arteriolar resistance (vasodilatation)
- Effective arterial blood volume
- Activation of neurohumoral systems (aldosterone, renin, angiotensin, epinephrine, antidiuretic hormone)

  - Sodium retention
  - Renal vaso-constriction
  - Water retention
  - Ascites
  - Hepatorenal syndrome
  - Hyponatremia
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Management of Hepatorenal Syndrome

Proven efficacy
- Liver transplantation

Under investigation
- Vasoconstrictor + albumin
- Transjugular intrahepatic portosystemic shunt (TIPS)
- Vasoconstrictor + TIPS
- Extracorporeal albumin dialysis (ECAD)

Ineffective
- Renal vasodilators (prostaglandin, dopamine)
- Hemodialysis
Benefits of Vasoconstrictors Plus Albumin in Hepatorenal Syndrome

- Vasoconstrictors should always be used in combination with albumin (for two days as an IV bolus (1 g/kg per day [100 g maximum]), followed by 25 to 50 grams per day)
  - Vasopressin analogues: terlipressin (IV bolus 1 to 2 mg every four to six hours)
  - Midodrine (starting at 7.5 and increasing the dose at eight-hour intervals up to a maximum of 15 mg by mouth three times daily) + octreotide (IV infusion 50 mcg/hr)
  - Noradrenaline continuous infusion (0.5 to 3 mg/hr) to raise mean arterial pressure by 10 mmHg

- HRS improves in ~60% of patients
- Mortality remains high (>50% in 1 month)

Treatment of Ascites

- Portal Hypertension
  - No ascites
- Uncomplicated ascites
- Refractory ascites
- Hepatorenal syndrome
  - 1) Liver transplant
  - 2) Vasoconstrictors + albumin
  - 3) TIPS?
Early Diagnosis of SBP

- Diagnostic paracentesis:
  - If symptoms / signs of SBP occur
  - Unexplained encephalopathy and / or renal dysfunction
  - At any hospital admission

- Diagnosis based on ascitic fluid
  PMN count >250/mm³

Rimola et al., J Hepatol 2000; 32:142

Treatment of Spontaneous Bacterial Peritonitis

- Recommended antibiotics for initial empiric therapy
  - i.v. cefotaxime, amoxicillin-clavulanic acid
  - oral ofloxacin (uncomplicated SBP)
  - avoid aminoglycosides

- Minimum duration: 5 days

- Re-evaluation if ascitic fluid PMN count has not decreased by at least 25% after 2 days of treatment

Rimola et al., J Hepatol 2000; 32:142
**Spontaneous Bacterial Peritonitis**

**Use of Intravenous Albumin**

Albumin (plus antibiotics) is indicated if:
- BUN > 30 mg/dL
- creatinine > 1.0 mg/dL
- bilirubin > 4 mg/dL

Albumin is not indicated in patients with a predicted 100% cure and survival:
- community-acquired SBP
- no GI hemorrhage
- no encephalopathy
- normal renal function

**Indications for Prophylactic Antibiotics to Prevent Spontaneous Bacterial Peritonitis**

- Cirrhotic patients hospitalized with GI hemorrhage (short-term)
  - Norfloxacin 400 mg p.o. BID x 7 days

- Patients who have recovered from SBP (long-term)
  - Norfloxacin 400 mg p.o. daily, indefinitely
  - Weekly quinolones not recommended (lower efficacy, development of quinolone-resistance)
Risk Factors for HCC

- Cirrhosis from any cause
  - HCV
  - HBV
  - Heavy alcohol consumption
  - Non-alcoholic fatty liver disease
- HBV
- Inherited metabolic diseases
  - Hemochromatosis
  - Alpha-1 antitrypsin deficiency
  - Glycogen storage disease
  - Porphyria cutanea tarda
  - Tyrosinemia
  - Autoimmune hepatitis

HCV Cirrhosis and Hepatoma

Arterial phase

Equilibrium
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HCC Epidemiology

- HCV Infection
  - 1% (1%-3%/year)

- Chronic Hepatitis
  - 90% (60%-95%)

- Cirrhosis
  - 15% (10%-30%)

- HCC
  - 1% (1%-3%/year)

Screening for HCC: AASLD Recommendations

- Population in which screening should be done
  - Cirrhosis (any etiology)
  - HBV: older, family history, cirrhosis

- Surveillance for HCC should be performed with ultrasonography (level II)

- AFP alone should not be used for surveillance unless ultrasonography is not available (level II)

- Screening should occur every 6-12 months (level II)

- The surveillance interval does not need to be shortened for patients at higher risk of HCC (level III)

Goodgame B, et al., Am J Gastroenterol 2003

Guidelines for Diagnosis of HCC

**Ultrasound findings**

- **< 1 cm**
  - Repeat US every 3-6 mo
- **1-2 cm**
  - Dynamic CT, contrast US or MRI, 2 tests
  - Typical = HCC
  - Atypical = biopsy
- **> 2 cm**
  - Dynamic CT, contrast US or MRI, 1 test
  - Typical = HCC
  - Atypical = biopsy

Typical features of HCC = vascular nodule on arterial phase with washout in delayed phases

Bruix J, et al, Hepatology 2005

Varices Increase in Diameter Progressively

- **No varices**
  - 7-8%/year
- **Small varices**
  - 7-8%/year
- **Large varices**

Merli et al. J Hepatol 2003;38:266
Screening for Varices

- Current recommendations for screening
  - AASLD:
    - All cirrhotic patients especially with
      - mod-severe cirrhosis (Child B/C)
      - Child A with signs of portal hypertension (plts <140,000, PV >13mm, or evidence of collaterals)
  - ACG: All patients with cirrhosis upon diagnosis of cirrhosis

Treatment of Varices / Variceal Hemorrhage

- No varices
- Varices No hemorrhage
- Variceal hemorrhage
- Recurrent hemorrhage

No specific therapy
Repeat endoscopy in 2-3 yrs*

* Sooner with cirrhosis decompensation
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Treatment of Varices / Variceal Hemorrhage

- No varices
- Varices No hemorrhage
  - Prevention of first variceal hemorrhage
- Variceal hemorrhage
  - Recurrent hemorrhage

- Management depends on the size of varices
Non-Selective Beta-Blockers Prevent First Variceal Hemorrhage

<table>
<thead>
<tr>
<th>Bleeding rate (~2 year)</th>
<th>Control</th>
<th>Beta-blocker</th>
<th>Absolute rate difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>All varices (11 trials)</td>
<td>25% (n=600)</td>
<td>15% (n=590)</td>
<td>-10% (-16 to -5)</td>
</tr>
<tr>
<td>Large varices (8 trials)</td>
<td>30% (n=411)</td>
<td>14% (n=400)</td>
<td>-16% (-24 to -8)</td>
</tr>
<tr>
<td>Small varices (3 trials)</td>
<td>7% (n=100)</td>
<td>2% (n=91)</td>
<td>-5% (-11 to 2)</td>
</tr>
</tbody>
</table>

D'Amico et al., Sem Liv Dis 1999; 19:475

Treatment of Varices / Variceal Hemorrhage

- No varices
- Small varices
  - No hemorrhage
- Medium/ large varices
  - No hemorrhage
- Variceal hemorrhage
- Recurrent hemorrhage

*1) β-blockers (propranolol, nadolol) indefinitely
*2) Endoscopic variceal ligation in patients intolerant to β-blockers

*β-blocker vs EBL based on expertise, availability, preference
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Treatment of Varices / Variceal Hemorrhage

- **No varices**
  - Repeat endoscopy in 1-2 years*
  - Beta-blockers+

- **Small varices No hemorrhage**

- **Medium/ large varices No hemorrhage**

- **Variceal hemorrhage**
  - * Sooner with cirrhosis decompensation
  - + if varices have high risk signs or Child-Pugh B/C patients

- **Recurrent hemorrhage**

Predictors of hemorrhage:
- Variceal size
- Red signs
- Child B/C


Variceal hemorrhage
Varix with red signs

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Treatment of Varices / Variceal Hemorrhage

- No varices
- Small varices
  - No hemorrhage
- Medium/large varices
  - No hemorrhage
- Variceal hemorrhage
  - Control of hemorrhage
- Recurrent hemorrhage

Treatment of Acute Variceal Hemorrhage

General Management:
- IV access and fluid resuscitation
- Do not overtransfuse (hemoglobin ~ 8 g/dL)
- Antibiotic prophylaxis

Specific therapy:
- Pharmacological therapy: terlipressin, somatostatin and analogues, vasopressin + nitroglycerin
- Endoscopic therapy: ligation, sclerotherapy
- Shunt therapy: TIPS, surgical shunt
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Tuesday, September 25, 2014

Treatment of Varices / Variceal Hemorrhage

- No varices
- Small varices
  - No hemorrhage
- Medium/large varices
  - No hemorrhage
- Variceal hemorrhage
  - 1) Safe vasoactive drug + endoscopic therapy + antibiotic prophylaxis
  - 2) TIPS / Shunt (rescue therapy)
- Recurrent hemorrhage
  - 1) β-blockers + EVL may be preferable
  - 2) TIPS / shunt surgery
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Evolution of Varices

<table>
<thead>
<tr>
<th>Cirrhosis with no varices</th>
<th>Level of Intervention</th>
<th>Management Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Small varices No hemorrhage</td>
<td>Pre-primary prophylaxis</td>
<td>Repeat endoscopy in 2-3 years, No specific therapy</td>
</tr>
<tr>
<td>Medium / large varices No hemorrhage</td>
<td>Primary prophylaxis</td>
<td>Small varices: Repeat endoscopy in 1-2 years, No specific therapy, ? beta-blocker to prevent enlargement</td>
</tr>
<tr>
<td>Variceal hemorrhage</td>
<td>Secondary prophylaxis</td>
<td>Medium/Large varices: Non-selective beta-blockers, EVL in those intolerant to drugs</td>
</tr>
<tr>
<td>Recurrent variceal hemorrhage</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Take Home Points

- Three Major Complications of Portal Hypertension
  - Varices
    - Primary prophylaxis
    - Management of acute bleeding
    - Secondary prophylaxis
  - Hepatic Encephalopathy
    - Clinical diagnosis
    - Identify precipitating factors
    - Identify and then treat patients with recurrent encephalopathy
Take Home Points

- Ascites Management
  - Diagnose portal hypertension related ascites using SAAG
  - Sodium restriction is important
  - Judicious use of diuretics
  - Refractory Ascites
    - Make sure definition is fulfilled before ‘giving up’
  - Spontaneous Bacterial Peritonitis
    - Diagnosis: PMN count >250/mm³
    - Secondary prophylaxis

- Hepatorenal syndrome
  - Any condition that leads to worsening of the hemodynamic status of the cirrhotic patient can lead to renal failure
  - Identify and rapidly address correctable causes of AKI
  - Once HRS develops
    - Start vasoconstrictor plus albumin
    - Liver transplant referral if a candidate
Take Home Points

- **Liver Cancer Screening**
  - Only screen appropriate candidates (mainly in cirrhotics)
  - Do not use AFP by itself
  - Once a lesion is noted on ultrasound perform an immediate dynamic imaging study
    - Triple phase CT scan
    - MRI with and without contrast
  - If a suspicious lesion is identified, refer out to a specialist