Management of Refractory Ascites

Michael Klein, MD
SUNY- Downstate Medical Center
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Case Presentation

74M with liver cirrhosis and hepatocellular carcinoma discovered 1 year ago presents complaining of increased abdominal distention and bilateral LE edema.
History

PMH: HBV, HTN, DM, iron deficiency anemia
PSH: paracentesis x3 over past 11 days (5L, 1.4L, 2.5L)
   right portal vein embolization 1 month ago

Medications: Lasix 40 daily, spironolactone 100 TID, atenolol, propranolol, flomax, tenofivir, pravastatin

No family history of malignancy
Denied tobacco, EtOH, illicit
Physical Exam

NAD, AAOx3, comfortable

CTAB RRR

Abdomen soft, NT, moderately distended
  (+) caput medusae, (+) fluid wave

2+ LE edema
Liver Function Tests:
6.3 / 3.2 / 58 / 52 / 170 / 1.1

PT / INR / PTT:
12.2 / 1.2 / 24.8

AFP: 3 normal

Viral Studies:
HBVSAg: > 1000 high
HBVSAb: < 3.1 normal
HBVCAb: > 8 high
HCVSAg: neg
Assessment and Plan

74M with HCC and cirrhosis, with increased ascites and suboptimal contralateral hypertrophic response to portal vein embolization.

(1) Control of ascites
(2) Control of malignancy
Hospital Course

HD 2

6L paracentesis, 50g albumin administered

HD 5

Measurement of hepatic vein pressure gradient

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<table>
<thead>
<tr>
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<tbody>
<tr>
<td>Hepatic Wedge Pressure</td>
<td>24</td>
<td></td>
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<tr>
<td>Free Wedge Pressure</td>
<td>12</td>
<td></td>
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<tr>
<td>Pressure gradient</td>
<td>12</td>
<td>high</td>
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Table 2. Prognostic Significance of Hepatic Venous Pressure Gradient Thresholds According to the Compensated or Decompensated Stage of Cirrhosis.

<table>
<thead>
<tr>
<th>Clinical setting</th>
<th>HVPG (mmHg)</th>
<th>Increased risk of threshold</th>
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<tr>
<td>Compensated cirrhosis</td>
<td>10</td>
<td>Presence [18] and development of gastroesophageal varices [2]</td>
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<td>First clinical decompensation in patients without varices [76]</td>
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<td>Development of HCC [19]</td>
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<td></td>
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<td>Decompensation after surgery for HCC [24,25]</td>
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<tr>
<td></td>
<td>12</td>
<td>Variceal bleeding [18,20,77-79]</td>
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<td></td>
<td>16</td>
<td>First clinical decompensation in patients with varices [80]; mortality [81]</td>
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<tr>
<td>Decompensated cirrhosis</td>
<td>16</td>
<td>Variceal rebleeding and mortality [21]</td>
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<tr>
<td></td>
<td>20</td>
<td>Failure to control variceal bleeding in patients actively bleeding from varices [22,50]; mortality</td>
</tr>
<tr>
<td></td>
<td>22</td>
<td>Mortality in patients with alcoholic cirrhosis and AAH [82]</td>
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<tr>
<td></td>
<td>30</td>
<td>Spontaneous bacterial peritonitis [83]</td>
</tr>
</tbody>
</table>

AAH: Acute alcoholic hepatitis; HCC: Hepatocellular carcinoma
Hospital Course

HD 5 – 10
  Medical optimization, preoperative risk stratification

HD 11
  Denver peritoneovenous shunt placement

HD 12 – 13
  Regular diet, discharge home
Postoperative Course

POD 7

Outpatient clinic followup
Denies pain, doing well
Increased scrotal and b/l LE edema
Plan to return for TACE of liver mass
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Ascites

- High hydrostatic pressure, low oncotic pressure cause transudation of fluid into the interstitial and peritoneal spaces
  - Increased vascular resistance in hepatic microcirculation
  - Increased hepatic and intestinal lymph formation
  - Peritoneal absorption: 500 mL / day
Ascites formation

↓ systemic intravascular volume

↑ renin-angiotensin-aldosterone activity

↑ intravascular volume

↑ portal hydrostatic pressure

Ascites formation

↓ systemic intravascular volume

↑ intravascular volume

↑ portal hydrostatic pressure

Nitric oxide, vasodilator release

Splanchnic vasodilatation

Respiratory distress

SBP

Hepatorenal syndrome
Medical Management

- Sodium restriction (2g/day)
  - May resolve up to 25% of cases
  - Fluid restriction no longer indicated

- Diuresis
  - Spironolactone (100 – 400mg / day)
  - Add furosemide (40 – 160mg / day) if hyperkalemic or refractory
  - Goal: ↓ body weight 1lb / day
Refractory Ascites

- Patients whose ascites cannot be controlled by diet and diuretics
- 1-year mortality: 50%

- Transplant evaluation or more invasive measures should ideally be considered prior to reaching this point
Fig. 1. Prognosis of patients with cirrhosis at the onset of ascites.
Non-surgical Interventions

- Large-volume paracentesis (LVP)
- Transjugular intrahepatic portosystemic shunt (TIPS)
Large-Volume Paracentesis (LVP)

- >= 6 liters
- Administer 6-8g albumin per liter removed to avoid systemic hypotension
- Short-term solution with multiple risks

FIGURE 1  Preferred needle entry site for paracentesis. (From Drake and Vogel: Gray’s atlas of anatomy. Philadelphia: Churchill Livingstone, 2007.)
TIPS

- Indicated if LVP required > 1x/month or for variceal bleeding
- Bridge to transplantation
- High rate of hepatic encephalopathy, shunt occlusion

**FIGURE 2** Transjugular intrahepatic portosystemic shunt placement. (From Johns Hopkins Medical Institutions, used with permission.)
Surgical Interventions

- Peritoneovenous shunt
- Liver transplantation
Peritoneovenous (Denver) Shunt

Fig. 1—Drawings show graphic representation of transjugular placement of Denver Shunt (CareFusion). Note that valve must be placed over immobile portion of thoracic cage to allow efficient valve compression.
Denver Shunt: Benefits

- Correction of hypovolemia
- Decreased peripheral vascular resistance
- Correction of sodium retention
  - Decreased renin/AT/aldosterone activation
  - Increased response to diuretics
Denver Shunt: Risks

- Obstruction (40% 1-year risk)
- Central venous thrombosis
- Intestinal obstruction
- Disseminated intravascular coagulation
  - Peritoneal fluid may contain tissue factor (factor X activator)
Liver Transplantation

- Milan Criteria
  - Used to select patients with HCC and cirrhosis for liver transplantation.
  - Must have:
    - 1 lesion smaller than 5cm OR 3 lesions smaller than 3cm
    - No extrahepatic disease
    - No vascular invasion
What about surgical shunts?

- End to side portacaval
- Side to side portacaval
- Interposition 1 – Portacaval 2 – Mesocaval 3 – Mesorenal
- Conventional splenorenal
- Distal splenorenal
References


Cameron JL. Current Surgical Therapy. 11th edition.

