Ascites

Investigation

Treatment

General

The treatment of ascites is directed at the underlying pathogenesis.

Common cirrhotic ascites can often be managed with diuretics and sodium restriction. The most successful diuretic regimen is a combination of spironolactone and furosemide. The goal of sodium restriction should be to limit intake to 2000mg/day.

- Whereas rapid diuresis can precipitate hepatorenal syndrome and should be avoided, large-volume paracentesis (>5L) has been shown to be safe and effective, regardless of the cause of ascites.
- When performing large-volume paracentesis in patients with cirrhosis, an infusion of 6-8 g of albumin per liter removed prevents the development of paracentesis-induced circulatory dysfunction often associated with large fluid shifts.
  - If there are >250 neutrophils/mm³ in an ascitic fluid sample, empirical antibiotics should be administered expeditiously.
  - In the setting of spontaneous bacterial peritonitis, intravenous albumin at 1.5 g/kg of body weight at the time of diagnosis, followed by 1 g/kg on day 3, was effective in preventing hepatorenal syndrome in one unblinded, randomized study.
- The insertion of a transjugular intrahepatic portosystemic shunt can be employed for treatment of refractory ascites. Although fairly successful in the treatment of ascites, a recent meta-analysis concluded that it can also be associated with the development of increased encephalopathy and offers no survival benefit.

- Ascites is the most common complication of cirrhosis.
- Hepatic hydrothorax is usually right sided, but may be bilateral, and is seen when ascitic fluid tracks up into the thorax through defects in the diaphragm, potentially causing respiratory embarrassment.

- Patients with ascites in the intensive care setting should undergo a diagnostic paracentesis to rule out infection.
- Ascitic fluid analysis should also include a cell count with differential and culture. In cirrhotic patients, spontaneous bacterial peritonitis is diagnosed when >250 neutrophils/mm³ are found in the fluid sample.
- If the ascites is new in onset, immediate ultrasound should be obtained to rule out acute thrombosis affecting the patency of the portal and hepatic veins.
- The serum-to-ascites albumin gradient, calculated by subtracting the ascitic fluid albumin level from the serum albumin level, has been shown to be effective in differentiating portal hypertensive from non-portal hypertensive ascites:
  - A serum-to-ascites albumin gradient of >1.1g/dL is seen when portal hypertension is present, as with:
    (i) cirrhosis, (ii) Budd-Chiari syndrome, (iii) cardiac disease, (iv) portal vein thrombosis, (v) myxedema, or (vi) liver metastasis.
  - A serum-to-ascites albumin gradient of <1.1 g/dL suggests nonportal hypertensive pathogeneses including:
    (i) malignancy, (ii) pancreatic disease, (iii) bile leak, (iv) infection, or (v) nephrosis.
- Additional useful tests performed on ascitic fluid include:
  - glucose, which is often elevated in the setting of malignancy or perforation;
  - amylase, which may be elevated in pancreatic ascites;
  - lactate dehydrogenase, which may be low in cirrhosis but elevated in SBP;
  - cytology.

If there is no obvious cause of ascites, a diagnostic laparoscopic examination may determine whether malignant or infectious peritoneal implantation is present.