be measured. In contrast to "spontaneous" bacterial peritonitis, which may complicate cirrhotic ascites (see "Complications," below), secondary peritonitis is suggested by an ascitic glucose level <50 mg/dL, an ascitic LDH level higher than the serum LDH level, and the detection of multiple pathogens on ascitic fluid culture. When pancreatic ascites is suspected, the ascitic amylase level should be measured and is typically >1000 mg/dL. Cytology can be useful in the diagnosis of peritoneal carcinomatosis. At least 50 mL of fluid should be obtained and sent for immediate processing. Tuberculous peritonitis is typically associated with ascitic fluid lymphocytosis but can be difficult to diagnose by paracentesis. A smear for acid-fast bacilli has a diagnostic sensitivity of only 0 to 3%; a culture increases the sensitivity to 35–50%. In patients without cirrhosis, an elevated ascitic adenosine deaminase level has a sensitivity of >90% when a cut-off value of 30–45 U/L is used. When the cause of ascites remains uncertain, laparotomy or laparoscopy with peritoneal biopsies for histology and culture remains the gold standard.

TREATMENT ASCITES

The initial treatment for cirrhotic ascites is restriction of sodium intake to 2 g/d. When sodium restriction alone is inadequate to control ascites, oral diuretics—typically the combination of spironolactone and furosemide—are used. Spironolactone is an aldosterone antagonist that inhibits sodium resorption in the distal convoluted tubule of the kidney. Use of spironolactone may be limited by hyponatremia, hyperkalemia, and painful gynecomastia. If the gynecomastia is distressing, amiloride (5–40 mg/d) may be substituted for spironolactone. Furosemide is a loop diuretic that is generally combined with spironolactone in a ratio of 40:100; maximal daily doses of spironolactone and furosemide are 400 mg and 160 mg, respectively.

Refractory cirrhotic ascites is defined by the persistence of ascites despite sodium restriction and maximal (or maximally tolerated) diuretic use. Pharmacologic therapy for refractory ascites includes the addition of midodrine, an α_1-adrenergic antagonist, or clonidine, an α_2-adrenergic antagonist, to diuretic therapy. These agents act as vasoconstrictors, counteracting splanchnic vasodilation. Midodrine alone or in combination with clonidine improves systemic hemodynamics and control of ascites over that obtained with diuretics alone. Although β-adrenergic blocking agents (beta blockers) are often prescribed to prevent variceal hemorrhage in patients with cirrhosis, the use of beta blockers in patients with refractory ascites is associated with decreased survival rates.

When medical therapy alone is insufficient, refractory ascites can be managed by repeated large-volume paracentesis (LVP) or a transjugular intrahepatic peritoneal shunt (TIPS)—a radiologically placed portosystemic shunt that decompresses the hepatic sinusoids. Intravenous infusion of albumin accompanying LVP decreases the risk of "post-paracentesis circulatory dysfunction" and death. Patients undergoing LVP should receive IV albumin infusions of 6–8 g/L of ascitic fluid removed. TIPS placement is superior to LVP in reducing the reaccumulation of ascites but is associated with an increased frequency of hepatic encephalopathy, with no difference in mortality rates.

Malignant ascites does not respond to sodium restriction or diuretics. Patients must undergo serial LVPs, transcutaneous drainage catheter placement, or, rarely, creation of a peritoneovenous shunt (a shunt from the abdominal cavity to the vena cava).

Ascites caused by tuberculous peritonitis is treated with standard antituberculosis therapy. Noncirrhotic ascites of other causes is treated by correction of the precipitating condition.

COMPLICATIONS

Spontaneous bacterial peritonitis (SBP; **Chap. 159**) is a common and potentially lethal complication of cirrhotic ascites. Occasionally, SBP also complicates ascites caused by nephrotic syndrome, heart failure, acute hepatitis, and acute liver failure but is rare in malignant ascites. Patients with SBP generally note an increase in abdominal girth; however, abdominal tenderness is found in only 40% of patients, and rebound tenderness is uncommon. Patients may present with fever, nausea, vomiting, or the new onset of or exacerbation of preexisting hepatic encephalopathy.

SBP is defined by a polymorphonuclear neutrophil (PMN) count of ffi250/ L in the ascitic fluid. Cultures of ascitic fluid typically reveal one bacterial pathogen. The presence of multiple pathogens in the setting of an elevated ascitic PMN count suggests *secondary peritonitis* from a ruptured viscus or abscess **(Chap. 159)**. The presence of multiple pathogens without an elevated PMN count suggests bowel perforation from the paracentesis needle. SBP is generally the result of enteric bacteria that have translocated across an edematous bowel wall. The most common pathogens are gram-negative rods, including *Escherichia coli* and *Klebsiella*, as well as streptococci and enterococci.

Treatment of SBP with an antibiotic such as IV cefotaxime is effective against gram-negative and gram-positive aerobes. A 5-day course of treatment is sufficient if the patient improves clinically. Nosocomial or health care–acquired SBP is frequently caused by multidrug-resistant bacteria, and initial antibiotic therapy should be guided by the local bacterial epidemiology.

Cirrhotic patients with a history of SBP, an ascitic fluid total protein concentration <1 g/dL, or active gastrointestinal bleeding should receive prophylactic antibiotics to prevent SBP; oral daily norfloxacin is commonly used. Diuresis increases the activity of ascitic fluid protein opsonins and may decrease the risk of SBP.

Hepatic hydrothorax occurs when ascites, often caused by cirrhosis, migrates via fenestrae in the diaphragm into the pleural space. This condition can result in shortness of breath, hypoxia, and infection. Treatment is similar to that for cirrhotic ascites and includes sodium restriction, diuretics, and, if needed, thoracentesis or TIPS placement. Chest tube placement should be avoided.