expansion with albumin (6 to 8 g/L of fluid removed), TIPS placement in appropriate candidates, and eventually liver transplantation (Fig. 43-3). Peritoneovenous shunts are not commonly used and are reserved for patients who are not candidates for paracentesis, TIPS, or transplantation.

SPONTANEOUS BACTERIAL PERITONITIS

Definition and Pathology

Cirrhotic patients may develop infection of ascitic fluid in the absence of an obvious source of contamination or surgically treatable source, a condition known as acute spontaneous bacterial peritonitis (SBP). The exact mechanism of contamination of the ascitic fluid is unclear. Factors such as bacterial overgrowth, altered motility, and increased intestinal permeability may contribute. The microbiology of SBP includes most commonly *Escherichia coli* and Enterobacteriaceae *(Klebsiella)*. Gram-positive organisms such as *Streptococcus* (viridans), *Enterococcus*, and *Pneumococcus* species may also be found. Anaerobes are uncommon, and a single organism is isolated on culture in most cases; the presence of multiple organisms suggests bowel perforation or other causes of peritonitis.

Clinical Presentation

Clinical features include fever, abdominal pain, and signs of peritoneal irritation. Often, the infection is clinically silent or manifests with worsening of HE, diarrhea, ileus, or renal insufficiency.

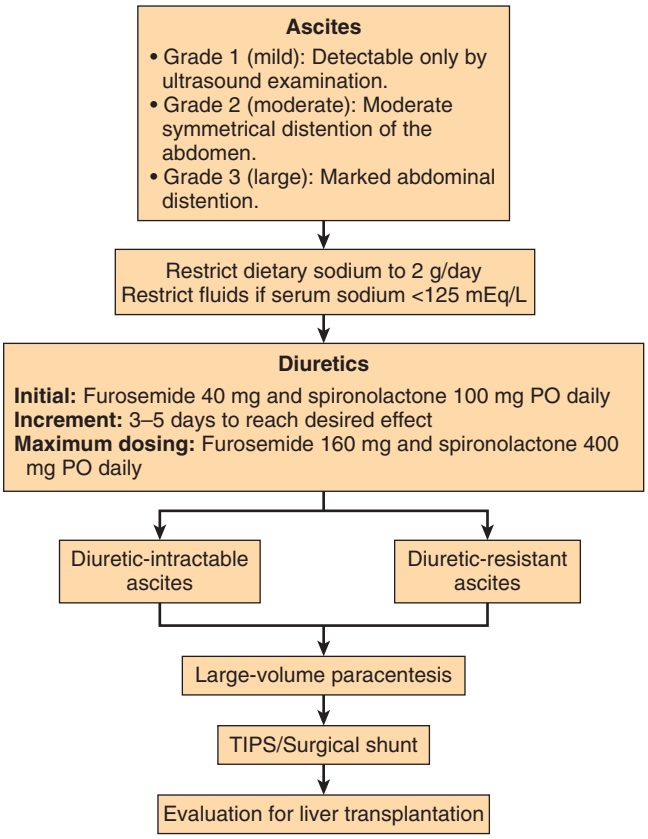

FIGURE 43-3 Management of ascites in cirrhosis. TIPS, Transjugular intrahepatic portosystemic shunt.

Diagnosis

Diagnostic paracentesis should be considered in any patient with cirrhotic ascites who deteriorates clinically. The diagnosis of SBP is highly likely if a high concentration (>250 cells/mm^3) of polymorphonuclear leukocytes (PMNs) is present in the ascitic fluid, and this finding should prompt empiric therapy while blood and ascitic fluid culture results are pending. The use of rapid bedside diagnostic methods such as leukocyte esterase reagent strips is not routinely recommended in view of their low sensitivity.

Treatment

Patients are usually treated with intravenous third-generation cephalosporin (e.g., cefotaxime, 2 g every 8 hours); quinolones are also routinely used, provided that the patient does not have prior exposure and is not in overt shock. Response to treatment is usually seen within 72 hours; therapy is continued for a minimum of 5 days and can extend up to 14 days. Repeat peritoneal fluid analysis may be done if recovery is delayed or to ensure that the ascitic fluid is sterile after treatment. The administration of intravenous albumin on day 1 (1.5 g/kg) and day 3 (1g/kg) has been shown to decrease the incidence of renal dysfunction and to improve short-term survival in SBP.

Prognosis

There is a high rate of recurrence, up to 70% within 1 year, and the 1-year mortality rate with a prior episode of SBP is 50% to 70%. Long-term antibiotic prophylaxis is indicated to reduce the recurrence rate to approximately 20%. Short-term prophylaxis should be considered for patients with cirrhosis and ascites who are hospitalized with upper gastrointestinal bleeding. Common prophylactic regimens for SBP include fluoroquinolones (ciprofloxacin, 750 mg/week; norfloxacin, 400 mg/day) and trimethoprim-sulfamethoxazole (1 double-strength tablet daily). Long-term antibiotic prophylaxis can lead to infection with resistant extended-spectrum β-lactamase (ESBL)-producing organisms or methicillin-resistant *Staphylococcus aureus* (MRSA).

HEPATORENAL SYNDROME

Definition and Pathology

Hepatorenal syndrome (HRS) is a form of functional renal failure that occurs in the presence of significant hepatic synthetic dysfunction and ascites. Three mechanisms of kidney dysfunction have been proposed: splanchnic arterial vasodilation, renal arterial vasoconstriction, and cardiac dysfunction.

Clinical Presentation and Diagnosis

Patients with HRS typically have advanced ascites and other manifestations of cirrhosis but are not otherwise symptomatic. However, some patients may notice decreased urine output or signs of encephalopathy. There is no single laboratory or imaging study that can be used alone to diagnose HRS. However, the 5-year probability of developing HRS in patients with cirrhosis and ascites is 40%, and HRS develops in approximately 30% of cirrhotic patients who are admitted with SBP. Therefore, a high