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Jaundice with associated liver dysfunction can be seen in severe cases of Plasmodium falciparum malaria. The jaundice in these cases is due to a combination of indirect hyperbilirubinemia from hemolysis and both cholestatic and hepatocellular jaundice. Weil’s disease, a severe presentation of leptospirosis, is marked by jaundice with renal failure, fever, headache, and muscle pain. Causes of extrahepatic cholestasis can be split into malignant and benign (Table 52-3). Malignant causes include pancreatic, gallbladder, and ampullary cancers as well as cholangiocarci noma. This last malignancy is most commonly associated with PSC and is exceptionally difficult to diagnose because its appear ance is often identical to that of PSC. Pancreatic and gallblad der tumors as well as cholangiocarcinoma are rarely resectable at the time of diagnosis and have poor prognoses. Ampullary carcinoma has the highest surgical cure rate of all the tumors that present as painless jaundice. Hilar lymphadenopathy due to metastases from other cancers may cause obstruction of the extrahepatic biliary tree. PART 2 Cardinal Manifestations and Presentation of Diseases Choledocholithiasis is the most common cause of extrahe patic cholestasis. The clinical presentation can range from mild right-upper-quadrant discomfort with only minimal elevations of enzyme test values to ascending cholangitis with jaundice, sepsis, and circulatory collapse. PSC may occur with clinically important strictures limited to the extrahepatic biliary tree. IgG4-associated cholangitis is marked by stricturing of the biliary tree. It is criti cal that the clinician differentiate this condition from PSC as it is responsive to glucocorticoid therapy. In rare instances, chronic pancreatitis causes strictures of the distal common bile duct, where it passes through the head of the pancreas. AIDS cholan giopathy is a condition that is usually due to infection of the bile duct epithelium with CMV or cryptosporidia and has a cholangio graphic appearance similar to that of PSC. The affected patients usually present with greatly elevated serum alkaline phosphatase levels (mean, 800 IU/L), but the bilirubin level is often near nor mal. These patients do not typically present with jaundice. Its inci dence has dropped dramatically since the introduction of potent antiretrovirals in the 1990s. ■ ■GLOBAL CONSIDERATIONS While extrahepatic biliary obstruction and drugs are common causes of new-onset jaundice in developed countries, infections remain the leading cause in developing countries. Liver involvement and jaundice are observed with numerous infections, particularly malaria, babesio sis, severe leptospirosis, infections due to Mycobacterium tuberculosis and the Mycobacterium avium complex, typhoid fever, infection with hepatitis viruses A–E, EBV, CMV, viral hemorrhagic fevers including Ebola virus, late phases of yellow fever, dengue fever, schistosomiasis, fascioliasis, clonorchiasis, opisthorchiasis, ascariasis, echinococcosis, hepatosplenic candidiasis, disseminated histoplasmosis, cryptococ cosis, coccidioidomycosis,

ehrlichiosis, chronic Q fever, yersiniosis, brucellosis, syphilis, and leprosy. Bacterial infections that do not nec essarily involve the liver and bile ducts may also lead to jaundice, as in cholestasis of sepsis. The presence of fever or abdominal pain sug gests concurrent infection, sepsis, or complications from gallstones. The development of encephalopathy and coagulopathy in a jaundiced patient with no preexisting liver disease signifies acute liver failure, which warrants urgent liver transplant evaluation. ■ ■FURTHER READING Erlinger S et al: Inherited disorders of bilirubin transport and conju gation: New insights into molecular mechanisms and consequences. Gastroenterology 146:1625, 2014. Wolkoff AW et al: Bilirubin metabolism and jaundice, in Schiff's Diseases of the Liver, 11th ed, Schiff ER et al (eds). Oxford, UK, John Wiley & Sons, Ltd, 2012, pp 120–150.

Lawrence S. Friedman

Abdominal Swelling

and Ascites ABDOMINAL SWELLING Abdominal swelling is a manifestation of numerous diseases. Patients may complain of bloating or abdominal fullness and may note increas ing abdominal girth on the basis of increased clothing or belt size. Abdominal discomfort is often reported, but pain is less frequent. When abdominal pain does accompany swelling, it is frequently the result of an intraabdominal infection, peritonitis, or pancreatitis. Patients with abdominal distention from ascites (fluid in the abdomen) may report the new onset of an inguinal or umbilical hernia. Dyspnea may result from pressure against the diaphragm and the inability to expand the lungs fully. ■ ■CAUSES The causes of abdominal swelling can be remembered conveniently as the six Fs: flatus, fat, fluid, fetus, feces, or a "fatal growth" (often a neoplasm). Flatus Abdominal swelling may be the result of increased intes tinal gas. The normal small intestine contains ~200 mL of gas made up of nitrogen, oxygen, carbon dioxide, hydrogen, and methane. Nitrogen and oxygen are consumed (swallowed), whereas carbon dioxide, hydrogen, and methane are produced intraluminally by bacte rial fermentation. Increased intestinal gas can occur in a number of conditions. Aerophagia, the swallowing of air, can result in increased amounts of oxygen and nitrogen in the small intestine and lead to abdominal swelling. Aerophagia typically results from gulping food; chewing gum; smoking; or as a response to anxiety, which can lead to repetitive belching. Celiac disease, or gluten-sensitive enteropathy, and gastroparesis may be associated with bloating and distention. In some cases, increased intestinal gas is the consequence of bacterial metabo lism of excess fermentable substances such as lactose, fructose, and other fermentable oligosaccharides, disaccharides, monosaccharides, and polyols (FODMAPs), which can lead to production of hydrogen, carbon dioxide, or methane, as occurs also in small intestinal bacterial overgrowth or intestinal methanogen overgrowth. In some persons, particularly those with irritable bowel syndrome and bloating, the subjective sense of abdominal pressure is attributable to impaired intestinal transit of gas (rather than increased gas volume) or to vis ceral hypersensitivity. Abdominal distention—an objective increase in girth—may result from a lack of coordination between diaphragmatic contraction and anterior abdominal wall relaxation ("abdomino phrenic dyssynergia"), a response in some cases to intraluminal bowel stimuli; dietary alterations, manipulation of the intestinal microbiota, or biofeedback may be effective therapy. Occasionally, increased lum bar lordosis accounts for apparent abdominal distention. Fat Weight gain with an increase in abdominal fat can result in an increase in abdominal girth and can be perceived as abdominal swell ing. Abdominal fat may be caused by an imbalance between caloric intake and

energy expenditure associated with a poor diet and seden tary lifestyle; it also can be a manifestation of certain diseases, such as Cushing’s syndrome. Excess abdominal fat has been associated with an increased risk of insulin resistance and cardiovascular disease. Fluid The accumulation of fluid within the abdominal cavity (asci tes) often results in abdominal distention and is discussed in detail below. Grade 1 ascites is detectable only by ultrasonography; grade 2 ascites is detectable by physical examination; and grade 3 ascites results in marked abdominal distention. Fetus Pregnancy results in increased abdominal girth. Typically, an increase in abdominal size is first noted at 12–14 weeks of gestation,

when the uterus moves from the pelvis into the abdomen. Abdominal distention may be seen before this point as a result of fluid retention and relaxation of the abdominal muscles. Feces In the setting of severe constipation or intestinal obstruction, increased stool in the colon leads to increased abdominal girth. These conditions are often accompanied by abdominal discomfort or pain, nausea, and vomiting and can be diagnosed by imaging studies. Fatal Growth An abdominal mass can result in abdominal swelling. Neoplasms (including ovarian cancer in women), abscesses, or cysts can grow to sizes that lead to increased abdominal girth. Enlargement of the intraabdominal organs, specifically the liver (hepatomegaly) or spleen (splenomegaly), or an abdominal aortic aneurysm can result in abdominal distention. Bladder distention also may result in abdominal swelling. APPROACH TO THE PATIENT Abdominal Swelling HISTORY Determining the etiology of abdominal swelling begins with history-taking and a physical examination. Patients should be questioned regarding the onset and timing of bloating and disten tion, the relationship to food or bowel movements, prior surgery, dietary habits, and medications, as well as symptoms suggestive of malignancy, including weight loss, night sweats, and anorexia. Inability to pass stool or flatus together with nausea or vomiting suggests bowel obstruction, severe constipation, or an ileus (lack of peristalsis). Increased eructation and flatus may point toward aero phagia or increased intestinal production of gas. Patients should be questioned about risk factors for or symptoms of chronic liver disease, including excessive alcohol use and jaundice, which sug gest ascites. Patients should also be asked about symptoms of other medical conditions, including heart failure and tuberculosis, which may cause ascites. PHYSICAL EXAMINATION Physical examination should include an assessment for signs of sys temic disease. The presence of lymphadenopathy, especially supra clavicular lymphadenopathy (Virchow’s node), suggests metastatic abdominal malignancy. Care should be taken during the cardiac examination to evaluate for elevation of jugular venous pressure (JVP); Kussmaul’s sign (elevation of the JVP during inspiration); a pericardial knock, which may be seen in constrictive pericarditis and heart failure; or a murmur of tricuspid regurgitation. Spider angiomas, palmar erythema, dilated superficial veins around the umbilicus (caput medusae), and gynecomastia suggest liver disease. The abdominal examination should begin with inspection for the presence of uneven distention or an obvious mass. Auscultation should follow. The absence of bowel sounds or the presence of highpitched localized bowel sounds points toward an intestinal obstruc tion or ileus. An umbilical venous hum may suggest the presence of portal hypertension, and a harsh bruit over the liver is heard rarely in patients with hepatocellular carcinoma or alcohol-associated hepatitis. Abdominal swelling caused by intestinal gas can be differentiated from swelling caused by fluid or a solid mass by percussion; an abdomen filled with gas is tympanic, whereas an abdomen contain ing a mass or fluid is dull to percussion. The absence of abdominal dullness, however, does not exclude ascites, because a minimum of 1500 mL of ascitic fluid is required for detection on physical exami nation. The abdomen should be palpated to assess for tenderness, a mass, enlargement of the spleen or liver,

or presence of a nodular liver suggesting cirrhosis or tumor. Light palpation of the liver may detect pulsations suggesting retrograde vascular flow from the heart in patients with right-sided heart failure, particularly tricuspid regurgitation. A rectal examination may help identify an evacuation disorder in patients with constipation.

Abdominal Swelling and Ascites CHAPTER 53 FIGURE 53-1 CT of a patient with a cirrhotic, nodular liver (white arrow), splenomegaly (yellow arrow), and ascites (arrowheads). ■ ■IMAGING AND LABORATORY EVALUATION Abdominal x-rays can be used to detect dilated loops of bowel suggest ing intestinal obstruction or ileus. Motility studies may be considered in patients with severe constipation. Abdominal ultrasonography can detect as little as 100 mL of ascitic fluid, hepatosplenomegaly, a nodular liver, or a mass. Ultrasonography is often inadequate to detect retro peritoneal lymphadenopathy or a pancreatic lesion because of overly ing bowel gas. If malignancy or pancreatic disease is suspected, CT can be performed. CT may also detect changes associated with advanced cirrhosis and portal hypertension (Fig. 53-1). Laboratory evaluation should include liver biochemical testing, serum albumin level measurement, and prothrombin time determina tion (international normalized ratio) to assess hepatic function as well as a complete blood count to evaluate for the presence of cytopenias that may result from portal hypertension or of leukocytosis, anemia, and thrombocytosis that may result from systemic infection. Serum amylase and lipase levels should be checked to evaluate the patient for acute pancreatitis. Urinary protein quantitation is indicated when nephrotic syndrome, which may cause ascites, is suspected. Hydrogen and methane absorbed from the intestine are not metabolized by the host and are excreted in expired air, and detection of increased amounts of these gases in expired breath is the basis for tests used to diagnose carbohydrate (e.g., lactose) malabsorption and small intes tinal bacterial overgrowth, although the reliability of the test results for the diagnosis of small intestinal bacterial overgrowth has been questioned. In selected cases, the hepatic venous pressure gradient (pressure across the liver between the portal and hepatic veins) can be measured via cannulation of the hepatic vein to confirm that ascites is caused by cirrhosis. In some cases, noninvasive tests for liver fibrosis, including liver stiffness measurement by elastography, or a liver biopsy may be necessary to confirm cirrhosis (Chap. 355). ASCITES ■ ■PATHOGENESIS IN THE PRESENCE OF CIRRHOSIS Ascites in patients with cirrhosis is the result of portal hypertension and renal salt and water retention. Similar mechanisms contribute to ascites formation in heart failure. Portal hypertension signifies eleva tion of the pressure within the portal vein. According to Ohm’s law,

pressure is the product of resistance and flow. Increased hepatic resis tance occurs by several mechanisms. First, the development of hepatic fibrosis, which defines cirrhosis, disrupts the normal architecture of the hepatic sinusoids and impedes normal blood flow through the liver. Second, activation of hepatic stellate cells, which mediate fibrogenesis, leads to smooth-muscle contraction and fibrosis. Finally, cirrhosis is associated with a decrease in endothelial nitric oxide synthetase (eNOS) production, which results in decreased nitric oxide production and increased intrahepatic vasoconstriction.

The development of cirrhosis is also associated with increased systemic circulating levels of nitric oxide (in contrast to the decrease seen intrahepatically), as well as increased levels of vascular endothe lial growth factor and tumor necrosis factor, that result in splanchnic arterial vasodilation. Vasodilation of the splanchnic circulation results in pooling of blood and a decrease in the effective circulating volume, which is perceived by the kidneys as hypovolemia. Compensatory

vasoconstriction via release of antidiuretic hormone ensues; the con sequences are free water retention and activation of the sympathetic nervous system and the renin-angiotensin-aldosterone system, which lead in turn to renal sodium and water retention. PART 2 Cardinal Manifestations and Presentation of Diseases ■ ■PATHOGENESIS IN THE ABSENCE OF CIRRHOSIS Ascites in the absence of cirrhosis generally results from peritoneal carcinomatosis, peritoneal infection, pancreatic disease, or marked hypoalbuminemia. Peritoneal carcinomatosis can result from primary peritoneal malignancies such as mesothelioma or sarcoma, abdominal malignancies such as gastric or colonic adenocarcinoma, or metastatic disease from breast or lung carcinoma or melanoma (Fig. 53-2). The tumor cells lining the peritoneum produce a protein-rich fluid that contributes to the development of ascites. Fluid from the extracellular space is drawn into the peritoneum, further contributing to the devel opment of ascites. Tuberculous peritonitis causes ascites via a similar mechanism; tubercles deposited on the peritoneum exude a protein aceous fluid. Pancreatic ascites results from leakage of pancreatic enzymes into the peritoneum. Marked hypoalbuminemia may result from nephrotic syndrome, protein-losing enteropathy, or malnutrition. ■ ■CAUSES Cirrhosis accounts for 84% of cases of ascites. Cardiac ascites, perito neal carcinomatosis, and “mixed” ascites resulting from cirrhosis and a second disease account for 10–15% of cases. Less common causes of ascites include massive hepatic metastasis, infection (tuberculo sis, Chlamydia infection), pancreatitis, and renal disease (nephrotic FIGURE 53-2 CT of a patient with peritoneal carcinomatosis (white arrow) and ascites (yellow arrow).

syndrome). Rare causes of ascites include hypothyroidism and familial Mediterranean fever. ■ ■EVALUATION Once the presence of ascites has been confirmed, the etiology of the ascites is best determined by paracentesis, a bedside procedure in which a needle or small catheter is passed transcutaneously to extract ascitic fluid from the peritoneum. The lower quadrants are the most frequent sites for paracentesis. The left lower quadrant is preferred because of the greater depth of ascites and the thinner abdominal wall. Paracen tesis is a safe procedure even in patients with coagulopathy; complica tions, including abdominal wall hematomas, hypotension, hepatorenal syndrome, and infection, are infrequent. Once ascitic fluid has been extracted, its gross appearance should be examined. Turbid fluid can result from the presence of infection or tumor cells. White, milky fluid indicates a triglyceride level >200 mg/ dL (and often >1000 mg/dL), which is the hallmark of chylous ascites. Chylous ascites results from lymphatic disruption that may occur with trauma, cirrhosis, tumor, tuberculosis, or certain congenital abnor malities. Dark brown fluid can reflect a high bilirubin concentration and indicates biliary tract perforation. Black fluid may indicate the presence of pancreatic necrosis or metastatic melanoma. The ascitic fluid should be sent for measurement of albumin and total protein levels, cell and differential counts, and, if infection is sus pected, Gram’s stain and culture, with inoculation into blood culture bottles at the patient’s bedside to maximize the yield. A serum albumin level should be measured simultaneously to permit calculation of the serum-ascites albumin gradient (SAAG). The SAAG is useful for distinguishing ascites caused by portal hypertension from nonportal hypertensive ascites (Fig. 53-3). The SAAG reflects the pressure within the hepatic sinusoids and correlates with the hepatic venous pressure gradient. The SAAG is calculated by subtracting the ascitic albumin concentration from the serum albumin level and does not change with diuresis. A SAAG ≥1.1 g/dL reflects the presence of portal hypertension and indicates that the ascites is due to increased pressure in the hepatic sinusoids. According to Starling’s law, a high SAAG reflects the oncotic pressure that counterbalances the portal pressure. Possible causes include cirrhosis, cardiac ascites, hepatic vein thrombosis (Budd-Chiari syndrome), sinusoidal obstruc tion syndrome (veno-occlusive disease), or

massive liver metastases. A SAAG <1.1 g/dL indicates that the ascites is not related to portal hypertension, as in tuberculous peritonitis, peritoneal carcinomatosis, pancreatic ascites, or nephrotic syndrome. For high-SAAG (≥1.1) ascites, the ascitic protein level can provide further clues to the etiology (Fig. 53-3). An ascitic protein level of ≥2.5 g/dL indicates that the hepatic sinusoids are normal and are allowing passage of protein into the ascites, as occurs in cardiac ascites, early Budd-Chiari syndrome, or sinusoidal obstruction syndrome. An ascitic protein level <2.5 g/dL indicates that the hepatic sinusoids have been damaged and scarred and no longer allow passage of protein, as occurs with cirrhosis, late Budd-Chiari syndrome, or massive liver metastases. Pro-brain-type natriuretic peptide (BNP) is a natriuretic hormone released by the heart as a result of increased volume and ventricular wall stretch. High levels of BNP in serum occur in heart failure and may be useful in identifying heart failure as the cause of high-SAAG ascites. Further tests are indicated only in specific clinical circumstances. When secondary peritonitis resulting from a perforated hollow viscus is suspected, ascitic glucose and lactate dehydrogenase (LDH) levels can 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 pancre atic 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 typi cally associated with ascitic fluid lymphocytosis but can be difficult to

≥1.1 g/dL Ascitic protein <2.5 g/dL Heart failure/constrictive pericarditis Cirrhosis Late Budd-Chiari syndrome Early Budd-Chiari syndrome Massive liver metastases IVC obstruction Sinusoidal obstruction syndrome FIGURE 53-3 Algorithm for the diagnosis of ascites according to the serum-ascites albumin gradient (SAAG). IVC, inferior vena cava. diagnose by paracentesis. A smear for acid-fast bacilli has a diagnostic sensitivity of only 0–3%; a culture increases the sensitivity to 35–50%. In patients without cirrhosis, an elevated ascitic adenosine deaminase level has a sensitivity of >90% for tuberculous ascites when a cut-off value of 30–45 U/L is used. When the cause of ascites remains uncer tain, laparotomy or laparoscopy with peritoneal biopsies for histology and culture remains the gold standard. TREATMENT Ascites The initial treatment for cirrhotic ascites is moderate restriction of sodium intake to 2 g/d. When sodium restriction alone is inad equate to control ascites, oral diuretics—typically the combination of spironolactone and furosemide—are used to increase urinary sodium excretion. Spironolactone is an aldosterone antagonist that inhibits sodium resorption in the distal convoluted tubule of the kidney. Use of spironolactone may be limited by hypona tremia, hyperkalemia, and painful gynecomastia. If the gyneco mastia 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 generally 400 mg and 160 mg, respectively. Fluid intake may be restricted in patients with hyponatremia (serum sodium <125 mEq/L). Nonsteroidal anti-inflammatory drugs, angiotensin-converting enzyme inhibi tors, and angiotensin receptor blockers should be avoided in patients with cirrhosis and ascites. Refractory cirrhotic ascites is defined by the persistence of asci tes despite sodium restriction and maximum diuretic use (diuretic resistant) or the development of side effects of diuretics that preclude the use of maximum doses (diuretic intractable). Phar macologic therapy for refractory ascites includes the addition of midodrine, an α1-adrenergic agonist, or clonidine, an α2-adrenergic agonist, to diuretic

therapy. These agents act as vasoconstrictors, counteracting splanchnic vasodilation. Midodrine alone or in com bination 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 may be associated with decreased survival rates. When medical therapy alone is insufficient, refractory cirrhotic ascites can be managed by repeated large-volume (>5 L) paracente sis (LVP) or a transjugular intrahepatic peritoneal shunt (TIPS)—a radiologically placed portosystemic shunt that decompresses the hepatic sinusoids. Intravenous (IV) infusion of albumin accom panying LVP decreases the risk of “postparacentesis circulatory dysfunction” and death. Patients undergoing LVP should receive IV

SAAG <1.1 g/dL Ascitic protein ≥2.5 g/dL Biliary leak Abdominal Swelling and Ascites CHAPTER 53 Nephrotic syndrome Pancreatitis Peritoneal carcinomatosis Tuberculosis albumin infusions of 6–8 g/L of ascitic fluid removed. TIPS place ment is superior to LVP in reducing the reaccumulation of ascites but is associated with an increased frequency of hepatic encepha lopathy, with no difference in mortality rates. The Alfapump sys tem, which consists of an automated pump and tunneled peritoneal catheter that transports ascites from the peritoneal cavity to the urinary bladder, has shown promise in the management of refrac tory ascites but is associated with a high frequency of technical difficulties and renal dysfunction. Malignant ascites does not respond to sodium restriction or diuretics. Patients must undergo serial LVPs, transcutaneous drain age catheter placement, or, rarely, creation of a peritoneovenous shunt (a shunt from the abdominal cavity to the vena cava) or place ment of the Alfapump system, if available. 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. 137) 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 asci tes. 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 or an exacerbation of preexisting hepatic encephalopathy. In hospitalized patients with ascites, paracentesis within 12 hours of admission reduces mortality because of early detection of SBP. SBP is defined by a polymorphonuclear neutrophil (PMN) count of ≥250/ µL in the ascitic fluid. Cultures of ascitic fluid should be performed in blood culture bottles and 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. 137). 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 translo cated 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 has traditionally been with a third-generation cephalosporin such as IV cefotaxime, 2 g every 12 hours, and is gen erally effective against gram-negative and gram-positive aerobes. A 5-day course of treatment is sufficient if the patient improves clini cally. Repeat paracentesis is recommended after 48 hours of antibiotic therapy to confirm that the ascitic PMN count has decreased by at least 25% from baseline. Increasingly, SBP, particularly if nosocomial or health care–acquired, is caused by multidrug-resistant bacteria, and

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