

can result in abdominal distention. Bladder distention also may result in abdominal swelling. In addition, malignancies, abscesses, or cysts can grow to sizes that lead to increased abdominal girth.

## 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 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 aerophagia 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 suggest ascites. Patients should also be asked about other symptoms of medical conditions, including heart failure and tuberculosis, which may cause ascites.

### PHYSICAL EXAMINATION

Physical examination should include an assessment for signs of systemic disease. The presence of lymphadenopathy, especially supraclavicular 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 heart failure or constrictive pericarditis; or a murmur of tricuspid regurgitation. Spider angiomata, palmar erythema, dilated superficial veins around the umbilicus (*caput medusae*), and gynecomastia suggest chronic 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 high-pitched localized bowel sounds points toward an ileus or intestinal obstruction. 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 alcoholic 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 containing 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 examination. Finally, 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.

### IMAGING AND LABORATORY EVALUATION

Abdominal x-rays can be used to detect dilated loops of bowel suggesting intestinal obstruction or ileus. 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 retroperitoneal lymphadenopathy or a pancreatic lesion because of overlying 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. 59-1).

Laboratory evaluation should include liver biochemical testing, serum albumin level measurement, and prothrombin time determination (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



**FIGURE 59-1** CT of a patient with a cirrhotic, nodular liver (white arrow), splenomegaly (yellow arrow), and ascites (arrowheads).

for acute pancreatitis. Urinary protein quantitation is indicated when nephrotic syndrome, which may cause ascites, is suspected.

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 (Chap. 365). In some cases, a liver biopsy may be necessary to confirm cirrhosis.

### 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 elevation of the pressure within the portal vein. According to Ohm's law, pressure is the product of resistance and flow. Increased hepatic resistance 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 (contrary to the decrease seen intrahepatically) as well as increased levels of vascular endothelial 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 consequences 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.

#### PATHOGENESIS IN THE ABSENCE OF CIRRHOSIS

Ascites in the absence of cirrhosis generally results from peritoneal carcinomatosis, peritoneal infection, or pancreatic disease. 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