ACUTE PERITONITIS

Acute peritonitis, or inflammation of the visceral and parietal peritoneum, is most often but not always infectious in origin, resulting from perforation of a hollow viscus. This is called *secondary peritonitis*, as opposed to *primary* or *spontaneous peritonitis*, when a specific intraabdominal source cannot be identified. In either instance, the inflammation can be localized or diffuse.

ETIOLOGY

Infective organisms may contaminate the peritoneal cavity after spillage from a hollow viscus, because of a penetrating wound of the abdominal wall, or because of the introduction of a foreign object like a peritoneal dialysis catheter or port that becomes infected. Secondary peritonitis most commonly results from perforation of the appendix, colonic diverticuli, or the stomach and duodenum. It may also occur as a complication of bowel infarction or incarceration, cancer, inflammatory bowel disease, and intestinal obstruction or volvulus. Conditions that may cause secondary bacterial peritonitis and their mechanisms are listed in Table 356-5. Over 90% of the cases of primary or spontaneous bacterial peritonitis occur in patients with ascites or hypoproteinemia (<1 g/L).

TABLE 356-5 CONDITIONS LEADING TO SECONDARY BACTERIAL PERITONITIS

Bowel Perforation	Perforation or Leakage of Other Organs
Appendicitis trauma (blunt or penetrating)	Biliary leakage (e.g., after liver biopsy)
Anastomotic leakage	Cholecystitis
Adhesion	Intraperitoneal bleeding
Diverticulitis	Pancreatitis
Iatrogenic (including endoscopic perforation)	Salpingitis
Ingested foreign body	Traumatic or other rupture of urinary bladder
Inflammation	**Loss of peritoneal integrity**
Intussusception	Intraperitoneal chemotherapy
Neoplasms	Iatrogenic (e.g., postoperative foreign body)
Obstruction	Perinephric abscess
Peptic ulcer disease	Peritoneal dialysis or other indwelling devices
Strangulated hernia	Trauma
Vascular (including ischemia or embolus)	

Aseptic peritonitis is most commonly caused by the abnormal presence of physiologic fluids like gastric juice, bile, pancreatic enzymes, blood, or urine. It can also be caused by the effects of normally sterile foreign bodies like surgical sponges or instruments. More rarely, it occurs as a complication of systemic diseases like lupus erythematosus, porphyria, and familial Mediterranean fever. The chemical irritation caused by stomach acid and activated pancreatic enzymes is extreme and secondary bacterial infection may occur.

CLINICAL FEATURES

The cardinal signs and symptoms of peritonitis are acute, typically severe, abdominal pain with tenderness and fever. How the patient's complaints of pain are manifested depends on their overall physical health and whether the inflammation is diffuse or localized. Elderly and immunosuppressed patients may not respond as aggressively to the irritation. Diffuse, generalized peritonitis is most often recognized as diffuse abdominal tenderness with local guarding, rigidity, and other evidence of parietal peritoneal irritation. Physical findings may only be identified in a specific region of the abdomen if the intraperitoneal inflammatory process is limited or otherwise contained as may occur in patients with uncomplicated appendicitis or diverticulitis. Bowel sounds are usually absent to hypoactive.

Most patients present with tachycardia and signs of volume depletion with hypotension. Laboratory testing typically reveals a significant leukocytosis, and patients may be severely acidotic. Radiographic studies may show dilatation of the bowel and associated bowel wall edema. Free air, or other evidence of leakage, requires attention and could represent a surgical emergency. In stable patients in whom ascites is present, diagnostic paracentesis is indicated, where the fluid is tested for protein and lactate dehydrogenase and the cell count is measured.

THERAPY AND PROGNOSIS

Whereas mortality rates can be less than 10% for reasonably healthy patients with relatively uncomplicated, localized peritonitis, mortality rates >40% have been reported for the elderly or immunocompromised. Successful treatment depends on correcting any electrolyte abnormalities, restoration of fluid volume and stabilization of the cardiovascular system, appropriate antibiotic therapy, and surgical correction of any underlying abnormalities.

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SECTION 2 LIVER AND BILIARY TRACT DISEASE

357 Approach to the Patient with Liver Disease

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A diagnosis of liver disease usually can be made accurately by careful elicitation of the patient's history, physical examination, and application of a few laboratory tests. In some circumstances, radiologic examinations are helpful or, indeed, diagnostic. Liver biopsy is considered the criterion standard in evaluation of liver disease but is now needed less for diagnosis than for grading and staging of disease. This chapter provides an introduction to diagnosis and management of liver disease, briefly reviewing the structure and function of the liver; the major clinical manifestations of liver disease; and the use of clinical history, physical examination, laboratory tests, imaging studies, and liver biopsy.

LIVER STRUCTURE AND FUNCTION

The liver is the largest organ of the body, weighing 1–1.5 kg and representing 1.5–2.5% of the lean body mass. The size and shape of the liver vary and generally match the general body shape—long and lean or squat and square. This organ is located in the right upper quadrant of the abdomen under the right lower rib cage against the diaphragm and projects for a variable extent into the left upper quadrant. It is held in place by ligamentous attachments to the diaphragm, peritoneum, great vessels, and upper gastrointestinal organs. The liver receives a dual blood supply; ~20% of the blood flow is oxygen-rich blood from the hepatic artery, and 80% is nutrient-rich blood from the portal vein arising from the stomach, intestines, pancreas, and spleen.