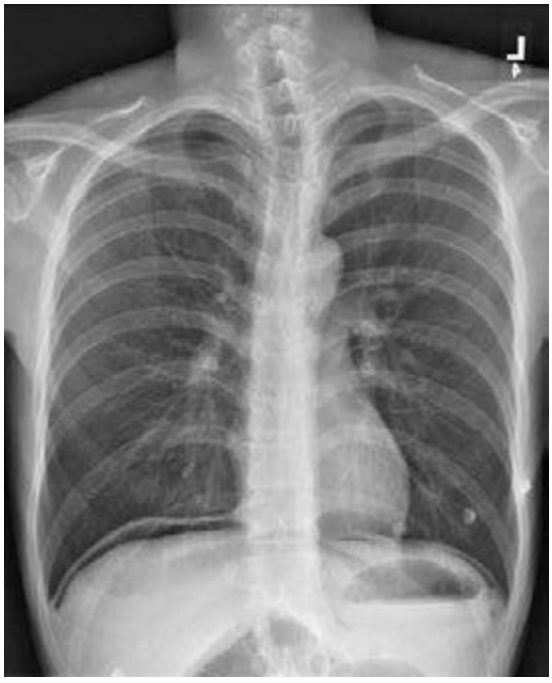

FIGURE 159-2 Pneumoperitoneum. Free air under the diaphragm on an upright chest film suggests the presence of a bowel perforation and associated peritonitis. *(Image courtesy of Dr. John Braver; with permission.)*

The diagnosis of PBP is not easy. It depends on the exclusion of a primary intraabdominal source of infection. Contrast-enhanced CT is useful in identifying an intraabdominal source for infection. It may be difficult to recover organisms from cultures of peritoneal fluid, presumably because the burden of organisms is low. However, the yield can be improved if 10 mL of peritoneal fluid is placed directly into a blood culture bottle. Because bacteremia frequently accompanies PBP, blood should be cultured simultaneously. To maximize the yield, culture samples should be collected prior to administration of antibiotics. No specific radiographic studies are helpful in the diagnosis of PBP. A plain film of the abdomen would be expected to show ascites. Chest and abdominal radiography should be performed in patients with abdominal pain to exclude free air, which signals a perforation **(Fig. 159-2)**.

TREATMENT PRIMARY BACTERIAL PERITONITIS

Treatment for PBP is directed at the isolate from blood or peritoneal fluid. Gram's staining of peritoneal fluid often gives negative results in PBP. Therefore, until culture results become available, therapy should cover gram-negative aerobic bacilli and gram-positive cocci. Third-generation cephalosporins such as cefotaxime (2 g q8h, administered IV) provide reasonable initial coverage in moderately ill patients. Broad-spectrum antibiotics, such as penicillin/β-lactamase inhibitor combinations (e.g., piperacillin/tazobactam, 3.375 g q6h IV for adults with normal renal function) or ceftriaxone (2 g q24h IV), are also options. Broader empirical coverage aimed at resistant hospital-acquired gram-negative bacteria (e.g., treatment with carbapenem) may be appropriate for nosocomially acquired PBP until culture results become available. Empirical coverage for anaerobes is not necessary. A mortality benefit from albumin (1.5 g/kg of body weight within 6 h of detection and 1.0 g/kg on day 3) has been demonstrated for patients who present with serum creatinine levels ≥1 mg/dL, blood urea nitrogen levels ≥30 mg/dL, or total bilirubin levels ≥4 mg/dL but not for patients who do not meet these criteria. After the infecting organism is identified, therapy should be narrowed to target the specific pathogen. Patients with PBP usually respond within 72 h to appropriate antibiotic therapy. Antimicrobial treatment can be administered for as little as 5 days if rapid improvement occurs and blood cultures are negative, but a course of up to 2 weeks may be required for patients with bacteremia and for those whose improvement is slow. Persistence of WBCs in the ascitic fluid after therapy should prompt a search for additional diagnoses.

Prevention • ***PRIMARY PREVENTION*** Several observational studies and a meta-analysis raise the concern that proton pump inhibitor therapy may increase the risk of PBP. No prospective studies have yet addressed whether avoidance of such therapy may prevent PBP. Nonselective beta blockers may prevent secondary bacterial peritonitis. A 2012 guideline from the American Association for the Study of Liver Diseases recommends chronic antibiotic prophylaxis with a regimen described in the next section for patients who are at highest risk for PBP—that is, those with an ascitic-fluid total protein level <1.5 g/dL along with impaired renal function (creatinine, ≥1.2 mg/dL; blood urea nitrogen, ≥25 mg/dL; or serum sodium, ≤130 mg/dL) and/or liver failure (Child-Pugh score, ≥9; and bilirubin, ≥3 mg/dL). A 7-day course of antibiotic prophylaxis is recommended for patients with cirrhosis and gastrointestinal bleeding.

SECONDARY PREVENTION PBP has a high rate of recurrence. Up to 70% of patients experience a recurrence within 1 year. Antibiotic prophylaxis is recommended for patients with a history of PBP to reduce this rate to <20% and improve short-term survival rates. Prophylactic regimens for adults with normal renal function include fluoroquinolones (ciprofloxacin, 750 mg weekly; norfloxacin, 400 mg/d) or trimethoprim-sulfamethoxazole (one double-strength tablet daily). However, long-term administration of broad-spectrum antibiotics in this setting has been shown to increase the risk of severe staphylococcal infections.

SECONDARY PERITONITIS

Secondary peritonitis develops when bacteria contaminate the peritoneum as a result of spillage from an intraabdominal viscus. The organisms found almost always constitute a mixed flora in which facultative gram-negative bacilli and anaerobes predominate, especially when the contaminating source is colonic. Early in the course of infection, when the host response is directed toward containment, exudate containing fibrin and PMNs is found. Early death in this setting is attributable to gram-negative bacillary sepsis and to potent endotoxins circulating in the bloodstream **(Chap. 325)**. Gram-negative bacilli, particularly *E. coli*, are common bloodstream isolates, but *Bacteroides fragilis* bacteremia also occurs. The severity of abdominal pain and the clinical course depend on the inciting process. The organisms isolated from the peritoneum also vary with the source of the initial process and the normal flora at that site. Secondary peritonitis can result primarily from chemical irritation and/or bacterial contamination. For example, as long as the patient is not achlorhydric, a ruptured gastric ulcer will release low-pH gastric contents that will serve as a chemical irritant. The normal flora of the stomach comprises the same organisms found in the oropharynx but in lower numbers. Thus, the bacterial burden in a ruptured ulcer is negligible compared with that in a ruptured appendix. The normal flora of the colon below the ligament of Treitz contains ~10^{11} anaerobic organisms/g of feces but only 10^{8} aerobes/g; therefore, anaerobic species account for 99.9% of the bacteria. Leakage of colonic contents (pH 7–8) does not cause significant chemical peritonitis, but infection is intense because of the heavy bacterial load.

Depending on the inciting event, local symptoms may occur in secondary peritonitis—for example, epigastric pain from a ruptured gastric ulcer. In appendicitis **(Chap. 356)**, the initial presenting symptoms are often vague, with periumbilical discomfort and nausea followed in a number of hours by pain more localized to the right lower quadrant. Unusual locations of the appendix (including a retrocecal position) can complicate this presentation further. Once infection has spread to the peritoneal cavity, pain increases, particularly with infection involving the parietal peritoneum, which is innervated extensively. Patients usually lie motionless, often with knees drawn up to avoid stretching the nerve fibers of the peritoneal cavity. Coughing and sneezing, which increase pressure within the peritoneal cavity, are associated with sharp