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performed, empirical therapy chosen in light of the most common infecting agents and the type of clinical syndrome should be given. In a controlled therapeutic trial of diabetic patients in whom no infected bone needed to be resected, the outcome of a 6-week course of antibiotics was not different from a 12-week course. In patients undergoing surgical debridement, a 3-week course of sys temic antibiotics was noninferior to a 6-week course in a prospec tive, randomized pilot trial. Wound debridement combined with antibiotics renders amputation unnecessary in about two-thirds of patients. For the diagnosis and treatment of diabetic foot infec tions, the following management strategies should be considered. If a foot ulcer is clinically infected, prompt empirical antimicrobial therapy may prevent progression to osteomyelitis. When the risk of methicillin-resistant S. aureus is considered high, an agent active against these strains (e.g., vancomycin) should be chosen. If the patient has not recently received antibiotics, the spectrum of the selected antibiotic must include gram-positive cocci (e.g., clindamycin, ampicillin-sulbactam). If the patient has received antibiotics within the past month, the spectrum of empirical anti biotics should include gram-negative bacilli (e.g., clindamycin plus a fluoroquinolone). If the patient has risk factors for Pseudomonas infection (previous colonization, residence in a warm climate, frequent exposure of the foot to water), an empirical antipseudo monal agent (e.g., piperacillin-tazobactam, cefepime) is indicated. If osteomyelitis is suspected either on clinical grounds (probe to bone) or on the basis of imaging procedures (MRI), bone biopsy should be performed. If infected bone is not entirely removed by surgery, the patient should be treated for 4–6 weeks in line with the identified pathogen(s) and their susceptibility. Treatment should initially be given by the IV route. Whether therapy can later be administered by the oral route depends on the bioavailability of oral drugs that cover the infecting agents. In the future, phage therapy may be a valuable additive therapeutic option. ■ ■GLOBAL CONSIDERATIONS The number of multiresistant microorganisms causing diabetic foot infection is increasing. The prevalence of methicillin-resistant S. aureus is 5–43% in various countries. Gram-negative pathogens are more common than gram-positive bacteria in Asia. In a study of 102 patients with diabetic foot infection from India, 69% of aerobic gram-negative bacilli produced extended-spectrum β-lactamase and 43% of S. aureus isolates were methicillin resistant. Risk factors for multidrug-resistant microorganisms are poor glycemic control, prolonged duration of infection, and large ulcer size. ■ ■FURTHER READING Depypere M et al: Pathogenesis and management of fracture-related infection. Clin Microbiol Infect 26:572,

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Intraabdominal Infections

and Abscesses Intraperitoneal infections generally arise because a normal anatomic barrier is disrupted. This disruption may result from a variety of causes—e.g., when the appendix, a diverticulum, or an ulcer ruptures; when the bowel wall is weakened by ischemia, tumor, or inflammation (e.g., in inflammatory bowel disease); or with adjacent inflammatory processes, such as pancreatitis or pelvic inflammatory disease, in which enzymes (in the former case) or organisms (in the latter) may leak into the peritoneal cavity. Whatever the inciting event, once inflammation develops and organisms usually contained within the bowel or another organ enter the normally sterile peritoneal space, a knowable series of events takes place. Intraabdominal infections occur in two stages: peritonitis and—if the patient survives this stage and goes untreated— abscess formation. The types of microorganisms predominating in each stage of infection are responsible for the pathogenesis of disease. PERITONITIS Peritonitis is a life-threatening event that is often accompanied by bacteremia and sepsis syndrome (Chap. 315). The peritoneal cavity is large but is divided into compartments. The upper and lower peri toneal cavities are divided by the transverse mesocolon; the greater omentum extends from the transverse mesocolon and from the lower pole of the stomach to line the lower peritoneal cavity. The pancreas, duodenum, and ascending and descending colon are located in the anterior retroperitoneal space; the kidneys, ureters, and adrenals are found in the posterior retroperitoneal space. The other organs, includ ing the liver, stomach, gallbladder, spleen, jejunum, ileum, transverse and sigmoid colon, cecum, and appendix, are within the peritoneal cavity. The cavity is lined with a serous membrane that can serve as a conduit for fluids—a property exploited in peritoneal dialysis (Fig. 137-1). A small amount of serous fluid is normally present in the CHAPTER 137 Intraabdominal Infections and Abscesses Left subphrenic Right subphrenic Lesser sac Subhepatic Left paracolic Right paracolic Pelvic FIGURE 137-1 Diagram of the intraperitoneal spaces, showing the circulation of fluid and potential areas for abscess formation. Some compartments collect fluid or pus more often than others. These compartments include the pelvis (the lowest portion), the subphrenic spaces on the right and left sides, and Morrison’s pouch, which is a posterosuperior extension of the subhepatic spaces and is the lowest part of the paravertebral groove when a patient is recumbent. The falciform ligament separating the right and left subphrenic spaces appears to act as a barrier to the spread of infection; consequently, it is unusual to find bilateral subphrenic collections. (Reproduced with permission from B Lorber [ed]: Atlas of Infectious Diseases, vol VII: Intra-abdominal infections, hepatitis, and gastroenteritis. Springer; 1996.)

peritoneal space, with a protein content (consisting mainly of albumin) of <30 g/L and <300 white blood cells (WBCs, generally mononuclear cells) per microliter. In bacterial infections, leukocyte

recruitment into the infected peritoneal cavity consists of an early influx of polymor phonuclear leukocytes (PMNs) and a prolonged subsequent phase of mononuclear cell migration. The phenotype of the infiltrating leu kocytes during the course of inflammation is regulated primarily by resident-cell chemokine synthesis.

■ ■PRIMARY (SPONTANEOUS) BACTERIAL PERITONITIS Peritonitis is either primary (without an apparent source of contami nation) or secondary. The types of organisms found and the clinical presentations of these two processes are different. In adults, primary bacterial peritonitis (PBP) occurs most commonly in conjunction with cirrhosis of the liver (frequently the result of alcoholism). However, the disease has been reported in adults with metastatic malignant disease, postnecrotic cirrhosis, chronic active hepatitis, acute viral hepatitis, congestive heart failure, systemic lupus erythematosus, and lymph edema as well as in patients with no underlying disease. Although PBP virtually always develops in patients with preexisting ascites, it is, in general, an uncommon event, occurring in ≤10% of cirrhotic patients. The cause of PBP has not been established definitively but is believed to involve hematogenous spread of organisms in a patient in whom a diseased liver and altered portal circulation result in a defect in the usual filtration function. Organisms multiply in ascites, a good medium for growth. Proteins of the complement cascade are found in peritoneal fluid, with lower levels in cirrhotic patients than in patients with ascites of other etiologies. The opsonic and phagocytic properties of PMNs are diminished in patients with advanced liver disease. Cir rhosis is associated with alterations in the gut microbiota, including an increased prevalence of potentially pathogenic bacteria such as Enterobacteriaceae. Small-intestinal bacterial overgrowth is frequently present in advanced stages of liver cirrhosis and has been linked with pathologic bacterial translocation and PBP. Factors promoting these changes in cirrhosis may include deficiencies in Paneth cell defensins, reduced intestinal motility, decreased pancreatobiliary secretions, and portal-hypertensive enteropathy. PART 5 Infectious Diseases The presentation of PBP differs from that of secondary peritonitis. The most common manifestation is fever, which is reported in up to 80% of patients. Ascites is found but virtually always predates infec tion. Abdominal pain, an acute onset of symptoms, and peritoneal irritation during physical examination can be helpful diagnostically, but the absence of any of these findings does not exclude this oftensubtle diagnosis. Nonlocalizing symptoms (such as malaise, fatigue, or encephalopathy) or jaundice or acute kidney injury without another clear etiology also should prompt consideration of PBP in a susceptible patient. It is vital to sample the peritoneal fluid of any cirrhotic patient with ascites and fever. The finding of >250 PMNs/μL is diagnostic for PBP. This criterion does not apply to secondary peritonitis (see below). The microbiology of PBP also is distinctive. While enteric gram-negative bacilli such as Escherichia coli are commonly encountered, gram-posi tive organisms such as streptococci, enterococci, or even pneumococci are sometimes found. In an important development, widespread use of quinolones to prevent PBP in high-risk subgroups of patients, frequent hospitalizations, and exposure to broad-spectrum antibiotics have led to a change in the etiology of infections in patients with cirrhosis, with more gram-positive bacteria and extended-spectrum β-lactamase (ESBL)–producing Enterobacteriaceae in recent years. Risk factors for multidrug-resistant infections include nosocomial origin of infection, long-term norfloxacin prophylaxis, recent infection with multiresistant bacteria, and recent use of β-lactam antibiotics. In PBP, a single organ ism is typically isolated; anaerobes are found less frequently in PBP than in secondary peritonitis, in which a mixed flora including anaerobes is the rule. In fact, if PBP is suspected and multiple organisms including anaerobes are recovered from the peritoneal fluid, the diagnosis must be reconsidered and a source of secondary peritonitis

sought. The diagnosis of PBP is not easy. It depends on the exclusion of a primary intraabdominal source of infection. Contrast-enhanced CT

FIGURE 137-2 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.) 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 antibi otics. There is interest in identifying biomarkers in ascites that may be associated with PBP. 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 when patients have abdominal pain to exclude free air, which signals a perforation (Fig. 137-2). 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. Delays in antibiotic treatment of PBP are associated with increased mortality. Therefore, until culture results become avail able, prompt 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 if the local prevalence of multi drug resistant organisms is low. Broad-spectrum antibiotics, such as β-lactam/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), also are options, with vancomycin for patients who have had prior infection or colonization with methicillin-resistant Staphylococcus aureus. Broader empirical cov erage aimed at resistant hospital-acquired gram-negative bacteria (e.g., carbapenem or newer agents, such as ceftolozane-tazobactam or ceftazidime-avibactam) and vancomycin-resistant Enterococcus (e.g., daptomycin) may be appropriate for nosocomially acquired PBP or in patients with prior antibiotic-resistant infections or criti cal illness 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 with PBP, with greatest

benefit among those who present with serum creatinine levels ≥1 mg/dL, blood urea nitrogen levels ≥30 mg/dL, or total bilirubin levels ≥5 mg/dL. 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 bac teremia and for those whose improvement is slow. Persistence of WBCs in the ascitic fluid after therapy should prompt a search for additional diagnoses. Prognosis: PBP is associated with significant morbidity and mortality, perhaps reflecting the fact that PBP risk is highest among patients with advanced liver disease. In a 2018 study of hospitalized patients with PBP in the United States, in-hospital mortality was 17.6%. Another study in 2019 found that among patients at a U.S. tertiary academic center, mortality following PBP was 23% at 30 days and 37% at 90 days. The morbidity and mortality associated with PBP has led to interest in strategies for PBP prevention. Prevention • PRIMARY PREVENTION Several observational studies and a meta-analysis raise the concern that gastric acid sup pression may increase the risk

of PBP. No prospective studies have yet addressed whether avoidance of such therapy may prevent PBP. A 2021 guideline from the American Association for the Study of Liver Diseases recommends chronic antibiotic prophylaxis be considered, after discussing risks and benefits, 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 mEq/L) and/or liver failure (Child-Turcotte-Pugh score, ≥9; and bilirubin, ≥3 mg/dL). A course of antibiotic prophylaxis is recommended for patients with cirrhosis and gastrointestinal bleed ing until bleeding has resolved and vasoactive drugs are stopped. SECONDARY PREVENTION PBP has a high rate of recurrence. Up to 70% of patients experience a recurrence within 1 year. Antibiotic prophylaxis has been recommended for patients with a history of PBP to reduce this rate and improve short-term survival rates. Prophylactic regimens for adults with normal renal function include fluoroqui nolones (ciprofloxacin, 500 mg weekly; or norfloxacin [not available in the United States], 400 mg/d) or trimethoprim-sulfamethoxazole (one double-strength tablet daily). However, these recommendations are being reconsidered in recent years as the risks and benefits evolve. Long-term administration of broad-spectrum antibiotics in this setting has been shown to increase the risk of severe staphylococcal infec tions and of infections with multidrug-resistant organisms (MDRO). Additionally, quinolone prophylaxis is less effective in patients who are colonized with MDRO. There is increased interest in using rifaximin, a broad-spectrum antibiotic that is used already for hepatic encepha lopathy and is not absorbed, for PBP prophylaxis (1200 mg daily). ■ ■SECONDARY PERITONITIS Secondary peritonitis develops when bacteria contaminate the perito neum as a result of spillage from an intraabdominal viscus. The organ isms 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. 315). Gram-negative bacilli, particularly E. coli, are common bloodstream isolates, but Bacteroides fragilis bac teremia 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 appen dix. The normal flora of the colon below the ligament of Treitz contains ~1011 anaerobic organisms/g of feces but only 108 aerobes/g; therefore, anaerobic species account for 99.9% of the bacteria (Chap. 484). Leak age 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 sec ondary peritonitis—for example, epigastric pain from a ruptured gas tric ulcer. In appendicitis (Chap. 342), 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 usu ally 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 pain. There may or may not be pain localized to the infected or dis eased organ from which secondary peritonitis has arisen. Patients with secondary peritonitis generally have abnormal findings on abdominal examination, with marked voluntary and involuntary guarding of the anterior abdominal musculature. Later findings include tenderness, especially rebound tenderness. In addition, there may be localized findings in the area of the inciting event. In general, patients are febrile, with marked leukocytosis and a left shift of the WBCs to band forms. CHAPTER 137 While recovery of organisms from peritoneal fluid is easier in secondary than in primary peritonitis, a tap of the abdomen is rarely the procedure of choice in secondary peritonitis. An exception is in cases involving trauma, where the possibility of a hemoperitoneum may need to be excluded early. Emergent studies (such as abdominal CT) to find the source of peritoneal contamination should be under taken if the patient is hemodynamically stable; unstable patients may require surgical intervention without prior imaging. Results of cultures from chronic drain sites are not reliable for defining the etiology of infections. Intraabdominal Infections and Abscesses TREATMENT Secondary Peritonitis Treatment for secondary peritonitis includes early administration of antibiotics aimed particularly at aerobic gram-negative bacilli and anaerobes (see below). The most appropriate regimen depends on the anticipated flora and the degree of illness. Community-

acquired infections associated with mild to moderate disease can be treated with many drugs covering these organisms, including broad-spectrum β-lactam/β-lactamase inhibitor combinations (e.g., ticarcillin/clavulanate, 3.1 g q4–6h IV; or piperacillin/tazobactam, 3.375 g q6h IV) or a combination of either a fluoroquinolone (e.g., levofloxacin, 750 mg q24h IV) or a third-generation cephalosporin (e.g., ceftriaxone, 2 g q24h IV) plus metronidazole (500 mg q8h IV). Eravacycline is a newer antibiotic in the tetracycline class that has been approved by the U.S. Food and Drug Administration for treatment of complicated intraabdominal infections (1 mg/ kg q12h IV). Patients in intensive care units and/or those with health care–associated infections should receive antibiotics target ing more resistant gram-negative organisms such as Pseudomonas aeruginosa—e.g., imipenem (500 mg q6h IV), meropenem (1 g q8h IV), higher-dose piperacillin/tazobactam (4.5 g IV q6h), or drug combinations such as cefepime (2 g IV q8h) or ceftazidime (2 g IV q8h) plus metronidazole. The role of enterococci and Can dida species in mixed infections is controversial; however, because cephalosporin-based regimens lack activity against enterococci, ampicillin or vancomycin can be added to these regimens for enterococcal coverage in very ill patients until culture results are

available. For patients known to be colonized with ampicillin-resistant, vancomycin-resistant enterococci (VRE), a VRE-active agent, such as linezolid or daptomycin, should be included. Antifungal cover age is warranted if there is growth of Candida species from a sterile site. Patients who are known to be colonized with highly resistant gram-negative organisms may require treatment with a newer agent such as ceftazidime/avibactam or ceftolozane/tazobactam. Second ary peritonitis usually requires both surgical intervention to address the inciting process and antibiotics to treat early bacteremia, to decrease the incidence of abscess formation and wound infection, and to prevent distant spread of infection. Although surgery is rarely indicated in PBP in adults, it may be life-saving in second ary peritonitis. In a recent study, the urgency and success of

source control impacted the odds of survival, while the choice of empiric antibiotics did not. Recombinant human activated protein C (APC) was considered at one time for treatment of severe sepsis from causes including secondary peritonitis but was withdrawn from the market in 2011 after it was determined that the drug was associated with an increased risk of bleeding and that evidence for its benefi cial effects was inadequate. Thus APC should not be used for sepsis or septic shock outside randomized clinical trials.

Peritonitis may develop as a complication of abdominal surger ies. These infections may be accompanied by localizing pain and/ or nonlocalizing signs or symptoms such as fever, malaise, anorexia, and toxicity. As a nosocomial infection, postoperative peritonitis may be associated with organisms such as staphylococci, compo nents of the gram-negative hospital microflora, and the microbes that cause PBP and secondary peritonitis, as described above. PART 5 Infectious Diseases ■ ■PERITONITIS IN PATIENTS UNDERGOING CONTINUOUS AMBULATORY PERITONEAL DIALYSIS A third type of peritonitis arises in patients who are undergoing continuous ambulatory peritoneal dialysis (CAPD). Unlike PBP and secondary peritonitis, which are caused by endogenous bacteria, CAPD-associated peritonitis usually involves skin organisms. The pathogenesis of infection is similar to that of intravascular device– related infection, in which skin organisms migrate along the catheter, which both serves as an entry point and exerts the effects of a foreign body. Exit-site or tunnel infection may or may not accompany CAPD-

associated peritonitis. Like PBP, CAPD-associated peritonitis is usually caused by a single organism. Peritonitis is, in fact, the most common reason for discontinuation of CAPD. Improvements in equipment design, especially the Y-set connector, have resulted in a decrease from one case of peritonitis per 9 months of CAPD to one case per 24 months. Diabetes was reported to be a risk factor for CAPD-associated perito nitis in a study from Taiwan. The clinical presentation of CAPD peritonitis resembles that of sec ondary peritonitis in that diffuse pain and peritoneal signs are common. The dialysate is usually cloudy and contains >100 WBCs/μL, >50% of which are neutrophils. However, the number of cells depends in part on dwell time. According to a guideline from the International Society for Peritoneal Dialysis (2016), for patients undergoing automated peri toneal dialysis who present during their nighttime treatment and whose dwell time is much shorter than with CAPD, the clinician should use the percentage of PMNs rather than the absolute number of WBCs to diagnose peritonitis. As the normal peritoneum has very few PMNs, a proportion above 50% is strong evidence of peritonitis even if the abso lute WBC count does not reach 100/μL. Meanwhile, patients undergoing automated peritoneal dialysis without a daytime exchange who present with abdominal pain may have no fluid to withdraw, in which case 1 L of dialysate should be infused and permitted to dwell a minimum of 1–2 h, then drained, examined for turbidity, and sent for cell count with differential and culture. The differential (with a shortened dwell time) may be more useful than the absolute WBC count. In equivocal cases or in patients with systemic or abdominal symptoms in whom the effluent appears clear, a second exchange is performed, with a dwell time of at least 2 h. Clinical judgment should guide initiation of therapy.

The most common organisms are Staphylococcus species, which accounted for ~45% of cases in one series. Historically, coagulase-

negative staphylococcal species were identified most commonly in these infections, but these isolates have more recently been decreas ing in frequency. Staphylococcus aureus is more often

involved among patients who are nasal carriers of the organism than among those who are not, and this organism is the most common pathogen in overt exitsite infections. Gram-negative bacilli and fungi such as Candida species also are found. Vancomycin-resistant enterococci and vancomycin-

intermediate S. aureus have been reported to produce peritonitis in CAPD patients. The finding of more than one organism in dialysate culture should prompt evaluation for secondary peritonitis. As with PBP, culture of dialysate fluid in blood culture bottles improves the yield. To facilitate diagnosis, several hundred milliliters of removed dialysis fluid should be concentrated by centrifugation before culture. TREATMENT CAPD Peritonitis Empirical therapy for CAPD peritonitis should be directed at S. aureus, coagulase-negative Staphylococcus, and gram-negative bacilli until the results of cultures become available. Guidelines sug gest that agents should be chosen on the basis of local experience with resistant organisms. In some centers, a first-generation cepha losporin such as cefazolin (for gram-positive bacteria) and a fluoro quinolone or a third-generation cephalosporin such as ceftazidime (for gram-negative bacteria) may be reasonable; in areas with high rates of infection with methicillin-resistant S. aureus, van comycin should be used instead of cefazolin, and gram-negative coverage may need to be broadened—e.g., with an aminoglycoside, ceftazidime, cefepime, or a carbapenem. Broad coverage including vancomycin should be particularly considered for patients with septic physiology or exit-site infections. Vancomycin should also be included in the regimen if the patient has a history of colonization or infection with methicillin-resistant S. aureus or has a history of severe allergy to penicillins and cephalosporins. Loading doses are administered intraperitoneally; doses depend on the dialysis method and the patient’s renal function. Intraperitoneal antibiotics are given either continuously (i.e., with each exchange) or intermit tently (i.e., once daily, with the dose allowed to remain in the perito neal cavity for at least 6 h). If the patient is severely ill, IV antibiotics should be added at doses appropriate for the patient’s degree of renal failure. The clinical response to an empirical treatment regi men should be rapid; if the patient has not responded after 48–96 h of treatment, new samples should be collected for cell counts and cul tures, and catheter removal should be considered. For patients who lack exit-site or tunnel infection, the typical duration of antibiotic treatment is 14 days. For patients with exit-site or tunnel infection, catheter removal should be considered, and a longer duration of antibiotic therapy (up to 21 days) may be appropriate. In fungal infections, the catheter should be removed immediately. ■ ■TUBERCULOUS PERITONITIS See Chap. 183. INTRAABDOMINAL ABSCESSES ■ ■INTRAPERITONEAL ABSCESSES Abscess formation is common in untreated peritonitis if overt gramnegative sepsis either does not develop or develops but is not fatal. In experimental models of abscess formation, mixed aerobic and anaero bic organisms have been implanted intraperitoneally. Without therapy directed at anaerobes, animals develop intraabdominal abscesses. As in humans, these experimental abscesses may stud the peritoneal cavity, lie within the omentum or mesentery, or even develop on the surface of or within viscera such as the liver. Pathogenesis and Immunity There is often disagreement about whether an abscess represents a disease state or a host response. In a

sense, it represents both: while an abscess is an infection in which via ble infecting organisms and PMNs are contained in a fibrous capsule, it is also a process by which the host confines microbes to a limited space, thereby preventing further spread of infection. In any event, abscesses do cause significant symptoms, and patients with abscesses can be quite ill. Experimental work has helped to define both the host cells and the bacterial virulence factors responsible—most notably in the

case of B. fragilis. This organism, although accounting for only 0.5% of the normal colonic flora, is the anaerobe most frequently isolated from intraabdominal infections, is especially prominent in abscesses, and is the most common anaerobic bloodstream isolate. On clinical grounds, therefore, B. fragilis appears to be uniquely virulent. Moreover, B. fragilis acts alone to cause abscesses in animal models of intraabdominal infec tion, whereas most other Bacteroides species must act synergistically with a facultative organism to induce abscess formation. Of the several virulence factors identified in B. fragilis, one is critical: the capsular polysaccharide complex found on the bacterial surface. This complex comprises at least eight distinct surface polysaccharides. Structural analysis of these polysaccharides has shown an unusual motif of oppositely charged sugars. Polysaccharides having these zwitterionic characteristics, such as polysaccharide A, evoke a host response in the peritoneal cavity that localizes bacteria into abscesses. B. fragilis and polysaccharide A have been found to adhere to primary mesothelial cells in vitro; this adherence, in turn, stimulates the pro duction of tumor necrosis factor α and intercellular adhesion molecule 1 by peritoneal macrophages. Although abscesses characteristically contain PMNs, the process of abscess induction depends on the stimu lation of T lymphocytes by these unique zwitterionic polysaccharides. The stimulated CD4+ T lymphocytes secrete leukoattractant cytokines and chemokines. The alternative pathways of complement and fibrino gen also participate in abscess formation. While antibodies to the capsular polysaccharide complex enhance bloodstream clearance of B. fragilis, CD4+ T cells are critical in immu nity to abscesses. When administered experimentally, B. fragilis poly saccharide A has immunomodulatory characteristics and stimulates CD4+ T regulatory cells via an interleukin 2–dependent mechanism to produce interleukin 10. Interleukin 10 downregulates the inflamma tory response, thereby preventing abscess formation. Clinical Presentation Of all intraabdominal abscesses, 74% are intraperitoneal or retroperitoneal and are not visceral. Most intraperi toneal abscesses result from fecal spillage from a colonic source, such as an inflamed appendix. Abscesses can also arise from other processes. They usually form within weeks of the development of peritonitis and may be found in a variety of locations from omentum to mesentery, pelvis to psoas muscles, and subphrenic space to a visceral organ such as the liver, where they may develop either on the surface of the organ or within it. Periappendiceal and diverticular abscesses occur com monly. Diverticular abscesses are least likely to rupture. Infections of the female genital tract and pancreatitis also are among the more common causative events. When abscesses occur in the female genital tract—either as a primary infection (e.g., tuboovarian abscess) or as an infection extending into the pelvic cavity or peritoneum—B. fragilis figures prominently among the organisms isolated. B. fragilis is not found in large numbers in the normal vaginal flora. For example, it is encountered less commonly in pelvic inflammatory disease and endo metritis without an associated abscess. In pancreatitis with leakage of damaging pancreatic enzymes, inflammation is prominent. Therefore, clinical findings such as fever, leukocytosis, and even abdominal pain do not distinguish pancreatitis itself from complications such as pan creatic pseudocyst, pancreatic abscess (Chap. 359), or intraabdominal collections of pus. Especially in cases of necrotizing pancreatitis, in which the incidence of local pancreatic infection may be as high as 30%, needle aspiration under CT guidance is performed to sample fluid for culture. Traditionally, many centers have prescribed preemp tive antibiotics for patients with necrotizing pancreatitis. Imipenem is frequently used for this purpose because it reaches high tissue levels in the pancreas (although it is not unique in this regard). Random ized controlled studies have not demonstrated a benefit from this

practice, and guidelines no longer recommend preemptive antibiotics for patients with acute pancreatitis. If needle aspiration yields infected fluid in the setting of acute necrotizing pancreatitis, antibiotic treat ment is appropriate in conjunction with surgical and/or percutaneous drainage of infected material. Infected pseudocysts that occur remotely from acute pancreatitis are unlikely to be associated with significant amounts of necrotic tissue and may be treated with either surgical or percutaneous catheter drainage in conjunction with appropriate anti biotic therapy.

Diagnosis Scanning procedures have considerably facilitated the diagnosis of intraabdominal abscesses. Abdominal CT probably has the highest yield, although ultrasonography is particularly useful for the right upper quadrant, kidneys, and pelvis. Both indium-labeled WBCs and gallium tend to localize in abscesses and may be useful in finding a collection. Because gallium is taken up in the bowel, indiumlabeled WBCs may have a slightly greater yield for abscesses near the bowel. Neither indium-labeled WBC scans nor gallium scans serve as a basis for a definitive diagnosis, however; both need to be followed by other, more specific studies, such as CT, if an area of possible abnormality is identified. PET scanning should also be considered due to its ready availability and because it provides more resolution. Abscesses contiguous with or contained within diverticula are particu larly difficult to diagnose with scanning procedures. Although barium should not be injected if a perforation is suspected, a barium enema occasionally may detect a diverticular abscess not diagnosed by other procedures. If one study is negative, a second study sometimes reveals a collection. Although exploratory laparotomy has been less commonly used since the advent of CT, this procedure still must be undertaken on occasion if an abscess is strongly suspected on clinical grounds. CHAPTER 137 TREATMENT Intraperitoneal Abscesses Intraabdominal Infections and Abscesses An algorithm for the management of patients with intraabdominal (including intraperitoneal) abscesses by percutaneous drainage is presented in Fig. 137-3. Treatment of intraabdominal infections involves determination of the initial focus of infection, admin istration of broad-spectrum antibiotics targeting the organisms involved, and performance of a drainage procedure if one or more definitive abscesses have formed. Antimicrobial therapy, in general, is adjunctive to drainage and/or surgical correction of an underlying lesion or process in intraabdominal abscesses. Results of cultures from drain sites are not reliable for defining the etiology of infections. Unlike the intraabdominal abscesses resulting from most causes, for which drainage of some kind is generally required, Percutaneous drainage No improvement by 48 h Defervescence by 24–48 h Repeat CT scan with dilute Hypaque injection into cavity and attempt further drainage Successful drainage and defervescence No drainage or no improvement Drain out when criteria for catheter removal satisfied Surgery FIGURE 137-3 Algorithm for the management of patients with intraabdominal abscesses by percutaneous drainage. Antimicrobial therapy should be administered concomitantly. (Reproduced with permission from B Lorber [ed]: Atlas of Infectious Diseases, vol VII: Intra-abdominal infections, hepatitis, and gastroenteritis. Philadelphia, Current Medicine, 1996, p 1.30, as adapted from OD Rotstein, RL Simmons, in SL Gorbach et al [eds]: Infectious Diseases. Philadelphia, Saunders; 1992.)

abscesses associated with diverticulitis usually wall off locally after rupture of a diverticulum, so that surgical intervention is not rou tinely required.

A number of agents exhibit excellent activity against aerobic gram-negative bacilli. Because death in intraabdominal sepsis is linked to gram-negative bacteremia, empirical therapy for intra-

abdominal infection always needs to include adequate coverage of gram-negative aerobic, facultative, and anaerobic organisms. Even if anaerobes are not cultured from clinical specimens, they still must be covered by the therapeutic regimen. Empirical antibiotic therapy should be the same as that discussed above for secondary peritonitis. Most clinical treatment failures are due to failure to drain the abscess and thereby achieve source control. The appro priate duration of antibiotic treatment for abdominal abscesses depends on whether the presumptive source of the intraabdominal infection has been controlled. With adequate source control, anti biotic treatment may be limited to 4 or 5 days. ■ ■VISCERAL ABSCESSES Liver Abscesses The liver is the organ most subject to the develop ment of abscesses. In one study of 540 intraabdominal abscesses, 26% were visceral. Liver abscesses made up 13% of the total number, or 48% of all visceral abscesses. Liver abscesses may be solitary or mul tiple; they may arise from hematogenous spread of bacteria or from local spread from contiguous sites of infection within the peritoneal cavity. In the past, appendicitis with rupture and subsequent spread of infection was the most common source for a liver abscess. Currently, associated disease of the biliary tract is most common. Pylephlebitis (suppurative thrombosis of the portal vein), usually arising from infec tion in the pelvis but sometimes from infection elsewhere in the perito neal cavity, is another common source for bacterial seeding of the liver. PART 5 Infectious Diseases Fever is the most common presenting sign of liver abscess. Some patients, particularly those with associated disease of the biliary tract, have symptoms and signs localized to the right upper quadrant, includ ing pain, guarding, punch tenderness, and even rebound tenderness. Nonspecific symptoms, such as chills, anorexia, weight loss, nausea, and vomiting, also may develop. Only 50% of patients with liver abscesses, however, have hepatomegaly, right-upper-quadrant tender ness, or jaundice; thus, one-half of patients have no symptoms or signs to direct attention to the liver. Fever of unknown origin may be the only manifestation of liver abscess, especially in the elderly. Diagnostic studies of the abdomen, especially the right upper quadrant, should be a part of any workup for fever of unknown origin. The single most reliable laboratory finding is an elevated serum concentration of alka line phosphatase, which is documented in 70% of patients with liver abscesses. Other tests of liver function may yield normal results, but 50% of patients have elevated serum levels of bilirubin, and 48% have elevated concentrations of aspartate aminotransferase. Other labora tory findings include leukocytosis in 77% of patients, anemia (usually normochromic, normocytic) in 50%, and hypoalbuminemia in 33%. Concomitant bacteremia is found in one-third to one-half of patients. A liver abscess is sometimes suggested by chest radiography, especially if a new elevation of the right hemidiaphragm is seen; other suggestive findings include a right basilar infiltrate and a right pleural effusion. Imaging studies are the most reliable methods for diagnosing liver abscesses. These studies include ultrasonography, CT (Fig. 137-4), indium-labeled WBC or gallium scan, and MRI. More than one such study may be required. Organisms recovered from liver abscesses vary with the source. In liver infection arising from the biliary tree, enteric gram-negative aero bic bacilli and enterococci are common isolates. Klebsiella pneumoniae liver abscess has been well described in Southeast Asia for more than 20 years and has become an emerging syndrome in North America and elsewhere. These community-acquired infections have been linked to a virulent hypermucoviscous K. pneumoniae phenotype and to a specific genotype. The typical syndrome includes liver abscess, bacteremia, and metastatic infection in the eye(s) and lung(s). Ampicillin/amoxicillin therapy started within the previous 30 days has been associated with

FIGURE 137-4 Multilocular liver abscess on CT scan. Multiple or multilocular abscesses are more common than solitary abscesses. (Reprinted with permission from B Lorber [ed]: Atlas of Infectious

Diseases, vol VII: Intra-abdominal Infections, Hepatitis, and Gastroenteritis. Philadelphia, Current Medicine, 1996, Fig. 1.22.) increased risk for this syndrome, presumably because of selection for the causative strain. Unless previous surgery has been performed, anaerobes are not generally involved in liver abscesses arising from biliary infections. In contrast, in liver abscesses arising from pelvic and other intraperitoneal sources, a mixed flora including both aerobic and anaerobic species is common; B. fragilis is the species most frequently isolated. With hematogenous spread of infection, usually only a single organism is encountered; this species may be S. aureus or a strepto coccal species such as one in the Streptococcus milleri group. Liver abscesses may also be caused by Candida species; such abscesses usu ally follow fungemia in patients receiving chemotherapy for cancer and often present when PMNs return after a period of neutropenia. Amebic liver abscesses are not an uncommon problem (Chap. 230). Amebic serologic testing gives positive results in >95% of cases. In addition, polymerase chain reaction (PCR) testing has been used in recent years. Negative results from these studies help to exclude this diagnosis. TREATMENT Liver Abscesses (Fig. 137-3) Drainage is the mainstay of therapy for intraabdomi nal abscesses, including liver abscesses; the approach can be either percutaneous (with a pigtail catheter kept in place or possibly with a device that can perform pulse lavage to fragment and evacuate the semisolid contents of a liver abscess), transluminal (with endo scopic ultrasound guidance), or surgical. However, there is growing interest in medical management alone for pyogenic liver abscesses. The drugs used for empirical therapy include the same ones used in intraabdominal sepsis and secondary bacterial peritonitis. Usu ally, blood cultures and a diagnostic aspirate of abscess contents should be obtained before the initiation of empirical therapy, with antibiotic choices adjusted when the results of Gram’s staining and culture become available. Cases treated without definitive drainage generally require longer courses of antibiotic therapy. When percu taneous drainage was compared with open surgical drainage, the average length of hospital stay for the former was almost twice that for the latter, although both the time required for fever to resolve and the mortality rate were the same for the two procedures. The mortality rate was appreciable despite treatment, averaging 15%. Several factors predict the failure of percutaneous drainage and therefore may favor primary surgical intervention. These factors include the presence of multiple, sizable abscesses; viscous abscess contents that tend to plug the catheter; associated disease (e.g., disease of the biliary tract) requiring surgery; the presence of yeast; communication with an untreated obstructed biliary tree; or the lack of a clinical response to percutaneous drainage in 4–7 days.

Treatment of candidal liver abscesses often entails initial admin istration of liposomal amphotericin B (3–5 mg/kg IV daily) or an echinocandin, with subsequent fluconazole therapy (Chap. 222). In some cases, therapy with fluconazole alone (6 mg/kg daily) may be used—e.g., in clinically stable patients whose infecting isolate is susceptible to this drug. Splenic Abscesses Splenic abscesses are much less common than liver abscesses. The incidence of splenic abscesses has ranged from 0.14 to 0.7% in various autopsy series. The clinical setting and the organ isms isolated usually differ from those for liver abscesses. The degree of clinical suspicion for splenic abscess needs to be high because this condition is frequently fatal if left untreated. Even in the most recently published series, diagnosis was made only at autopsy in 37% of cases. Although splenic abscesses may arise occasionally from contiguous spread of infection or from direct trauma to the spleen, hematogenous spread of infection is more common. Bacterial endocarditis is the most common associated infection (Chap. 133). Splenic abscesses can develop in patients who have received extensive immunosuppressive therapy (particularly those with malignancy involving the spleen)

and in patients with hemoglobinopathies or other hematologic disorders (especially sickle cell anemia). Although ~50% of patients with splenic abscesses have abdominal pain, the pain is localized to the left upper quadrant in only one-half of these cases. Splenomegaly is found in ~50% of cases. Fever and leukocytosis are generally present; the development of fever preceded diagnosis by an average of 20 days in one series. Left-sided chest find ings may include abnormalities to auscultation, and chest radiographic findings may include an infiltrate or a left-sided pleural effusion. CT scan of the abdomen has been the most sensitive diagnostic tool. Ultra sonography can yield the diagnosis but is less sensitive. Liver–spleen scan or gallium scan also may be useful. Streptococcal species are the most common bacterial isolates from splenic abscesses, followed by S. aureus—presumably reflecting the associated endocarditis. An increase in the prevalence of gram-negative aerobic isolates from splenic abscesses has been reported; these organisms often derive from a urinary tract focus, with associated bacteremia, or from another intraabdominal source. Salmonella species are seen fairly commonly, especially in patients with sickle cell hemoglobinopathy. Anaerobic species accounted for only 5% of isolates in the largest collected series, but the reporting of a number of “sterile abscesses” may indicate that optimal techniques for the isolation of anaerobes were not used. TREATMENT Splenic Abscesses Because of the high mortality figures reported for splenic abscesses, splenectomy with adjunctive antibiotics has traditionally been con sidered standard treatment and remains the best approach for complex, multilocular abscesses or multiple abscesses. However, percutaneous drainage has worked well for single, small (<3-cm) abscesses in some studies and may also be useful for patients with high surgical risk. Patients undergoing splenectomy should be vaccinated against encapsulated organisms (Streptococcus pneu moniae, Haemophilus influenzae, Neisseria meningitidis). The most important factor in successful treatment of splenic abscesses is early diagnosis. Perinephric and Renal Abscesses Perinephric and renal abscesses are not common. The former accounted for only ~0.02% of hospital admissions and the latter for ~0.2% in Altemeier’s series of 540 intraabdominal abscesses. Before antibiotics became available, most renal and perinephric abscesses were hematogenous in origin, usually complicating prolonged bacteremia, with S. aureus most commonly recovered. Now, in contrast, >75% of perinephric and renal abscesses arise from a urinary tract infection. Infection ascends from the blad der to the kidney, with pyelonephritis preceding abscess development. Bacteria may directly invade the renal parenchyma from medulla

to cortex. Local vascular channels within the kidney may facilitate the transport of organisms. Areas of abscess developing within the parenchyma may rupture into the perinephric space. The kidneys and adrenal glands are surrounded by a layer of perirenal fat that, in turn, is surrounded by Gerota’s fascia, which extends superiorly to the diaphragm and inferiorly to the pelvic fat. Abscesses extending into the perinephric space may track through Gerota’s fascia into the psoas or transversalis muscles, into the anterior peritoneal cavity, superiorly to the subdiaphragmatic space, or inferiorly to the pelvis. Of the risk factors that have been associated with the development of perinephric abscesses, the most important is concomitant nephrolithiasis obstruct ing urinary flow. Of patients with perinephric abscess, 20–60% have renal stones. Other structural abnormalities of the urinary tract, prior urologic surgery, trauma, and diabetes mellitus also have been identi fied as risk factors.

The organisms most frequently encountered in perinephric and renal abscesses are E. coli, Proteus species, and Klebsiella species. E. coli, the aerobic species most commonly found in the colonic flora, seems to have unique virulence properties in the urinary tract, including factors promoting adherence to uroepithelial cells. The urease of Proteus species splits urea, thereby creating a more

alkaline and more hospitable environment for bacterial proliferation. Proteus species are frequently found in association with large struvite stones caused by the precipitation of magnesium ammonium sulfate in an alkaline environment. These stones serve as a nidus for recurrent urinary tract infection. Although a single bacterial species is usually recovered from a perinephric or renal abscess, multiple species may be found. If a urine culture is not contaminated with periurethral flora and is found to contain more than one organism, a perinephric or renal abscess should be considered in the differential diagnosis. Urine cultures may also be polymicrobial in cases of bladder diverticulum. CHAPTER 137 Candida species can cause renal abscesses. Fungi of this genus may spread to the kidney hematogenously or by ascension from the bladder. The hallmark of the latter route of infection is ureteral obstruction with large fungal balls. Intraabdominal Infections and Abscesses The presentation of perinephric and renal abscesses is quite non specific. Flank pain and abdominal pain are common. At least 50% of patients are febrile. Pain may be referred to the groin or leg, particularly with extension of infection. The diagnosis of perinephric abscess, like that of splenic abscess, is frequently delayed, and the mortality rate in some series is appreciable, although lower than in the past. Perinephric or renal abscess should be most seriously considered when a patient presents with symptoms and signs of pyelonephritis and remains febrile after 4 or 5 days of treatment. Moreover, when a urine culture yields a polymicrobial flora, when a patient is known to have renal stones, or when fever and pyuria coexist with a sterile urine culture, these diagnoses should be entertained. Renal ultrasonography and abdominal CT are the most useful diagnostic modalities. If a renal or perinephric abscess is diagnosed, nephrolithiasis should be excluded, especially when a high urinary pH suggests the presence of a urea-splitting organism. TREATMENT Perinephric and Renal Abscesses Treatment for perinephric and renal abscesses, like that for other intraabdominal abscesses, includes drainage of pus and antibiotic therapy directed at the organism(s) recovered. For perinephric abscesses, percutaneous drainage is usually successful. Psoas Abscesses The psoas muscle is another location in which abscesses are encountered. Psoas abscesses may arise from a hema togenous source, by contiguous spread from an intraabdominal or pelvic process, or by contiguous spread from nearby bony structures (e.g., vertebral bodies). Associated osteomyelitis due to spread from bone to muscle or from muscle to bone is common in psoas abscesses. When Pott’s disease was common, Mycobacterium tuberculosis was a frequent cause of psoas abscess. Currently, either S. aureus or a mixture

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