Chapter 43: Intra-Abdominal Infections

INTRODUCTION

- Intra-abdominal infections are those contained within the peritoneum or retroperitoneal space. Two general types of intra-abdominal infections are discussed throughout this chapter: peritonitis and abscess.
- Peritonitis is defined as the acute, inflammatory response of peritoneal lining to microorganisms, chemicals, irradiation, or foreign body injury. It may be classified as either primary, secondary, or tertiary. With primary peritonitis, an intra-abdominal focus of disease may not be evident. In secondary peritonitis, a focal disease process is evident within the abdomen. Tertiary peritonitis occurs in critically ill patients and is infection that persists or recurs at least 48 hours after apparently adequate management of primary or secondary peritonitis.
- An abscess is a purulent collection of fluid separated from surrounding tissue by a wall consisting of inflammatory cells and adjacent organs. It usually contains necrotic debris, bacteria, and inflammatory cells.

PATHOPHYSIOLOGY

- **Table 43-1** summarizes many of the potential causes of bacterial peritonitis. Appendicitis is the most common cause of intra-abdominal infection. Intra-abdominal infection results from entry of bacteria into the peritoneal or retroperitoneal spaces or from bacterial collections within intra-abdominal organs. When peritonitis results from peritoneal dialysis, skin surface flora are introduced via the peritoneal catheter.
- In primary peritonitis, bacteria may enter the abdomen via the bloodstream or the lymphatic system, by transmigration through the bowel wall, through an indwelling peritoneal dialysis catheter, or via the fallopian tubes in female patients.
- In secondary peritonitis, bacteria most often enter the peritoneum or retroperitoneum as a result of disruption of the integrity of the gastrointestinal (GI) tract caused by diseases or traumatic injuries.
- When bacteria become dispersed throughout the peritoneum, the inflammatory process involves the majority of the peritoneal lining. Fluid and protein shift into the abdomen (called “third spacing”) may decrease circulating blood volume and cause shock.
- Peritonitis often results in death because of the effects on major organ systems. Fluid shifts, cytokines, and microorganism toxins may result in hypovolemia, hypoperfusion, and shock.
- An abscess begins by the combined action of inflammatory cells (eg, neutrophils), bacteria, fibrin, and other inflammatory components. Within the abscess, oxygen tension is low, and anaerobic bacteria thrive.

TABLE 43-1

Causes of Bacterial Peritonitis

Primary (spontaneous) bacterial peritonitis
Cirrhosis with ascites
Nephrotic syndrome
Peritoneal dialysis (may be secondary to catheter site infection)
Secondary bacterial peritonitis
Miscellaneous causes
Divertic ulitis
Appendicitis
Inflammatory bowel diseases
Salpingitis
Biliary tract infections
Necrotizing pancreatitis
Neoplasms
Intestinal obstruction
Perforation
Mechanical GI problems
Any cause of small bowel obstruction (adhesions, hernia)
Vascular causes
Mesenteric arterial or venous occlusion (atrial fibrillation)
Mesenteric ischemia without occlusion
Trauma
Blunt abdominal trauma with rupture of intestine
Penetrating abdominal trauma
Iatrogenic intestinal perforation (endoscopy)
Intraoperative events
Solid organ transplant in the abdomen
Peritoneal contamination during abdominal operation
Leakage from GI anastomosis

GI, gastrointestinal.

Microbiology

- Primary bacterial peritonitis is often caused by a single organism. In children, the pathogen is usually group A *Streptococcus, Escherichia coli, Streptococcus pneumoniae,* or *Bacteroides* species. When peritonitis occurs in association with cirrhotic ascites, *E. coli* is isolated most frequently.
- Peritonitis in patients undergoing peritoneal dialysis is most often caused by common skin organisms: coagulase negative staphylococci, *Staphylococcus aureus,* streptococci, and enterococci. Gram-negative bacteria associated with peritoneal dialysis infections include *E. coli, Klebsiella,* and *Pseudomonas.*
- Secondary intra-abdominal infections are often polymicrobial. The mean number of isolates of microorganisms from infected intra-abdominal sites has ranged from 2.9–3.7, including an average of 1.3–1.6 aerobes and 1.7–2.1 anaerobes. The frequencies with which specific bacteria were isolated in intra-abdominal infections are given in **Table 43-2**.

- The combination of aerobic and anaerobic organisms appears to greatly the severity of infection. In intra-abdominal infections, facultative bacteria may provide an environment conducive to the growth of anaerobic bacteria.
- Aerobic enteric bacteria and anaerobic bacteria are both pathogens in intra-abdominal infection. Aerobic bacteria, particularly *E. coli*, appear responsible for the early mortality from peritonitis, whereas anaerobic bacteria are major pathogens in abscesses, with *Bacteroides fragilis* predominating.
- The role of *Enterococcus* as a pathogen is not clear. Enterococcal infection occurs more commonly in postoperative peritonitis, in the presence of specific risk factors indicating failure of the host defenses, or with the use of broad-spectrum antibiotics.

TABLE 43-2

Pathogens Isolated from Patients with Intra-Abdominal Infection

	Secondary Peritonitis (%)	Community-Acquired Infection (%)	Nosocomial Infection (%)
Gram-negative bacteria			
Escherichia coli	32–61	29	22.5
Enterobacter	8–26	5.2	8.0
Klebsiella	6–26	2.8	4.5
Proteus	4–23	1.7	2.4
Pseudomonas	5–13	5	13
Gram-positive bacteria			
Enterococcus	18–24	10.6	18
Streptococcus	6–55	13.7	10
Staphylococcus	6–16	3.1	4.8
Anaerobic bacteria			
Bacteroides	25–80	13.7	10.3
Clostridium	5–18	3.5	3.4
Fungi	2–5	3	4

CLINICAL PRESENTATION

- Intra-abdominal infections have a wide spectrum of clinical features often depending on the specific disease process, the location and the magnitude of bacterial contamination, and concurrent host factors. Patients with primary and secondary peritonitis present quite differently (**Table 43-3**).
- If peritonitis continues untreated, the patient may experience hypovolemic shock from fluid loss into the peritoneum, bowel wall, and lumen. This

may be accompanied by sepsis. Intra-abdominal abscess may pose a diagnostic challenge, as the symptoms are neither specific nor dramatic.

- The overall outcome from intra-abdominal infection depends on five key factors: inoculum size, virulence of the organisms, the presence of adjuvants within the peritoneal cavity that facilitate infection, the adequacy of host defenses, and the adequacy of initial treatment.

TABLE 43-3

Clinical Presentation of Peritonitis

Primary Peritonitis
General The patient may not be in acute distress, particularly with peritoneal dialysis
Signs and symptoms The patient may complain of loss of appetite, bloating, nausea, vomiting (sometimes with diarrhea), and abdominal tenderness Temperature may be only mildly elevated or not elevated in patients undergoing peritoneal dialysis Bowel sounds are hypoactive The cirrhotic patient may have worsening encephalopathy Cloudy dialysate fluid with peritoneal dialysis
Laboratory tests The patient's WBC count may be only mildly elevated Ascitic fluid usually contains greater than 250 leukocytes/mm^3 (0.25×10^9/L), and bacteria may be evident on gram stain of a centrifuged specimen In 60%–80% of patients with cirrhotic ascites, the gram stain is negative
Other diagnostic tests Culture of peritoneal dialysate or ascitic fluid should be positive, particularly if collected prior to initiation of antibiotics Procalcitonin in conjunction with clinical findings is a sensitive test for bacterial peritonitis
Secondary Peritonitis
Signs and symptoms Generalized abdominal pain Tachypnea Tachycardia Nausea and vomiting Temperature is normal initially then increases to 37.8–38.9°C (100–102°F) within the first few hours and may continue to rise for the next several hours Hypotension, hypoperfusion, and shock if volume is not restored Decreased urine output due to vascular volume depletion
Physical examination Voluntary abdominal guarding changing to involuntary guarding and a "board-like abdomen" Abdominal tenderness and distension Faint bowel sounds that cease over time
Laboratory tests

	Leukocytosis (15,000–20,000 WBC/mm^3 [15×10^9 to 20×10^9/L]), with neutrophils predominating and an elevated percentage of immature neutrophils (bands) Elevated hematocrit and blood urea nitrogen because of dehydration Patient progresses from early alkalosis because of hyperventilation and vomiting to metabolic acidosis
	Other diagnostic tests Abdominal radiographs may be useful because free air in the abdomen (indicating intestinal perforation) or distension of the small or large bowel is often evident

WBC, white blood cell.

TREATMENT

- Goals of Treatment: Correction of intra-abdominal disease processes or injuries that have caused infection and the drainage of collections of purulent material (eg, abscess). A secondary objective is to achieve resolution of infection without major organ system complications or adverse treatment effects.
- The three major modalities for the treatment of intra-abdominal infection are prompt surgical control and drainage of the infected site, hemodynamic resuscitation and support of vital functions, and early administration of appropriate antimicrobial therapy to treat infection not removed by surgery.
- Antimicrobials are an important adjunct to drainage procedures in the treatment of intra-abdominal infections; however, the use of antimicrobial agents without surgical source control is usually inadequate. For most cases of primary peritonitis, drainage procedures may not be required, and antimicrobial agents become the mainstay of therapy.
- In the early phase of serious intra-abdominal infections, attention should be given to the maintenance of organ system functions. With generalized peritonitis, large volumes of IV fluids are required to restore vascular volume, to improve cardiovascular function, and to maintain adequate tissue perfusion and oxygenation.
- An important component of therapy is nutrition. Enteral or parenteral nutrition as indicated facilitates improved immune function and wound healing to ensure recovery.

Nonpharmacologic Therapy

- Secondary peritonitis is treated surgically; this is called “source control,” which refers to the physical measures undertaken to eradicate the focus of infection. Abdominal laparotomy may be used to correct the cause of peritonitis. After an abscess is located, it must be drained.
- Aggressive fluid repletion and management are required for the purposes of achieving or maintaining proper intravascular volume to ensure adequate cardiac output, tissue perfusion, and correction of acidosis.
- The Surviving Sepsis Campaign recommends that resuscitation should be guided by normalization of lactate (weak recommendation, low quality of evidence). Furthermore, in patients with septic shock requiring vasopressors, an initial mean arterial pressure (MAP) of 65 mm Hg should be targeted (strong recommendation, moderate quality of evidence). Urine output should equal or exceed 0.5 mL/kg of body weight per hour.
- In patients with sepsis hypoperfusion or septic shock, IV fluids should consist of a 30 mL/kg bolus of crystalloids with additional fluids targeting predefined therapeutic goals. Initial fluid resuscitation should be completed within 3 hours of hypoperfusion recognition. Thereafter, fluids may be required at a rate of 1 L/hr or higher. Once targeted therapeutic goals are reached, judicious use of fluids should be used as a sustained positive fluid balance after initial resuscitation may be harmful.

- Maintenance fluids should be instituted with 0.9% sodium chloride and potassium chloride (20 mEq/L [mmol/L]) or 5% dextrose and 0.45% sodium chloride with potassium chloride (20 mEq/L [mmol/L]). The administration rate should be based on estimated daily fluid loss through urine and nasogastric suction, including 0.5–1 L for insensible fluid loss.
- In patients with significant blood loss (hematocrit ≤25%), blood should be given. This is generally in the form of packed red blood cells.
- Enteral or parenteral nutrition facilitates improved immune function and wound healing to ensure recovery.

Pharmacologic Therapy

- The goals of antimicrobial therapy are: (a) to eliminate the intra-abdominal infection and prevent the establishment of metastatic foci of infection or bacteremia, (b) to reduce suppurative complications (eg, abscess formation) after bacterial contamination, and (c) to prevent local spread of existing infection.
- An empiric antimicrobial regimen should be started as soon as the presence of intra-abdominal infection is suspected on the basis of likely pathogens. Antimicrobial therapy should be administered within 1 hour of the recognition of sepsis or septic shock (strong recommendation, moderate quality of evidence).

Recommendations

- For most Intra-abdominal infections, the antimicrobial regimen should be effective against both aerobic and anaerobic bacteria. **Table 43-4** presents recommended and alternative regimens for community-acquired complicated intra-abdominal infections. Guidelines for initial antimicrobial treatment of specific intra-abdominal infections are presented in **Table 43-5**.
- Evidence-based treatment principles for complicated intra-abdominal infections are given in **Table 43-6**.
- For established intra-abdominal infections, most patients are adequately treated with 4–7 days of antimicrobial therapy.
- For peritonitis that occurs from chronic peritoneal dialysis the selection of a specific agent or combination should be based on culture and susceptibility data. If microbiologic data are unavailable, empiric therapy should be initiated.
- Intraperitoneal administration of antibiotics is preferred over IV therapy in the treatment of peritonitis that occurs in patients undergoing continuous ambulatory peritoneal dialysis. For empiric intraperitoneal therapy, **cefazolin** or **vancomycin** in cases of high prevalence of methicillin-resistant *S. aureus* (MRSA) or β-lactam allergy may be used for gram-positive coverage.
- Antimicrobial therapy should typically be continued for 14–21 days.

TABLE 43-4

Recommended Agents for the Treatment of Community-Acquired Complicated Intra-Abdominal Infections in Adults

Agents Recommended for Mild-to-Moderate Infections	Agents Recommended for High Severity Infections
Single agent	
Cefoxitin[a]	Piperacillin–tazobactam
Moxifloxacin[b]	Imipenem–cilastatin,[c] meropenem,[c] doripenem[c]
Ertapenem[c]	
Combination regimens	
Cefazolin,[a] cefuroxime,[a] ceftriaxone, or cefotaxime each in combination with metronidazole	Cefepime, ceftazidime, ceftazidime/avibactam,[d] or ceftolozane/tazobactam[e] each in combination with metronidazole
Ciprofloxacin[b] or levofloxacin[b] each in combination with metronidazole	Ciprofloxacin[b] or levofloxacin[b] each in combination with metronidazole

[a]Empiric first- and second-generation cephalosporin use should be avoided unless local antibiograms show >80%–90% susceptibility of *E. coli* to these agents.

[b]Use of quinolones may be associated with treatment failure due to increasing resistance of enteric pathogens including *E. coli.* Empiric quinolone use should be avoided unless local antibiograms show >80%–90% susceptibility of *E. coli* to quinolones.

[c]Carbapenems should be reserved for settings where there is a high risk of resistance to other agents (ie, extended-spectrum β-lactamase [ESBL]-producing pathogens).

[d]Ceftazidime/Avibactam should be reserved for patients infected with *Enterobacteriaceae* that are resistant to all other β-lactams including carbapenems, and in particular, *Klebsiella pneumoniae* carbapenemase (KPC)-producing pathogens.

[e]Ceftolozane/Tazobactam should be reserved for patients infected with gram-negative bacteria that are resistant to all other β-lactams. Ceftolozane/tazobactam may have activity against multidrug-resistant *Pseudomonas,* however susceptibility must be confirmed.

TABLE 43-5

Guidelines for Empiric Antimicrobial Agents for Intra-abdominal Infections

	Primary Agents	Alternatives
Primary (spontaneous) bacterial peritonitis		
Cirrhosis	Ceftriaxone, cefotaxime	1. Piperacillin–tazobactam, carbapenems 2. Aztreonam combined with an agent active against *Streptococcus* spp. (eg, vancomycin) or quinolones with significant *Streptococcus* spp. activity (levofloxacin, moxifloxacin)

Peritoneal dialysis	Initial empiric regimens should be active against both gram-positive (including *S. aureus*) and gram-negative pathogens: gram-positive agent (first-generation cephalosporin or vancomycin) plus a gram-negative agent (third-generation cephalosporin or aminoglycoside)	1. Cefepime or carbapenems may be used alone 2. Aztreonam or an aminoglycoside may be used in place of ceftazidime or cefepime as long as combined with a gram-positive agent 3. Quinolones may be used in place of gram-negative agents if local susceptibilities allow
	1. *Staphylococcus* spp.: oxacillin/nafcillin or first-generation cephalosporin	1. Vancomycin should be used for methicillin-resistant *Staphylococcus* spp. 2. May consider addition of rifampin for 5–7 days with vancomycin for methicillin-resistant *S. aureus*
	2. *Streptococcus* or *Enterococcus*: ampicillin	1. Daptomycin or linezolid should ideally be used to treat vancomycin-resistant *Enterococcus* spp. not susceptible to ampicillin
	3. Aerobic gram-negative bacilli: ceftazidime or cefepime	1. The regimen should be based on in vitro sensitivity tests
	4. *Pseudomonas aeruginosa*: two agents with differing mechanisms of action, such as an intraperitoneal ceftazidime or cefepime each combined with either tobramycin or oral ciprofloxacin	
Secondary bacterial peritonitis		
Perforated peptic ulcer	First-generation cephalosporins	1. Ceftriaxone, cefotaxime, or antianaerobic cephalosporins[a]
Other	Third- or fourth-generation cephalosporin with metronidazole, piperacillin–tazobactam or carbapenem	1. Ciprofloxacin[b] or levofloxacin[b] each with metronidazole or moxifloxacin[b] alone 2. Aztreonam with vancomycin and metronidazole 3. Antianaerobic cephalosporins[a]
Abscess		
General	Third- or fourth-generation cephalosporin with metronidazole or piperacillin–tazobactam	1. Imipenem–cilastatin, meropenem, doripenem, or ertapenem 2. Ciprofloxacin[b] or levofloxacin[b] each with metronidazole or moxifloxacin alone
Liver	As above	Use metronidazole if amoebic liver abscess is suspected
Spleen	Ceftriaxone or cefotaxime	Moxifloxacin[b] or levofloxacin[b]

Other intra-abdominal infections		
Appendicitis	Same management as for community-acquired complicated intra-abdominal infections as listed in Table 43-4	
Community-acquired acute cholecystitis	Ceftriaxone or cefotaxime	Severe infection, piperacillin/tazobactam, antipseudomonal carbapenem, aztreonam with metronidazole
Cholangitis	Ceftriaxone or cefotaxime each with or without metronidazole	Vancomycin with aztreonam with or without metronidazole
Acute contamination from abdominal trauma	Antianaerobic cephalosporins[a] or metronidazole with either ceftriaxone or cefotaxime	1. Piperacillin/tazobactam or a carbapenem 2. Ciprofloxacin[b] or levofloxacin[b] each with metronidazole or moxifloxacin alone

[a]Cefoxitin or ceftizoxime; these agents should be avoided empirically unless local antibiograms show >80%–90% susceptibility of *E. coli* to these agents.

[b]Use of quinolones may be associated with treatment failure due to increasing resistance of enteric pathogens including *E. coli.* Empiric quinolone use should be avoided unless local antibiograms show >80%–90% susceptibility of *E. coli* to quinolones.

TABLE 43-6

Evidence-Based Recommendations for Treatment of Complicated Intra-Abdominal Infections

	Grade of Recommendation[a]
Elements of appropriate intervention	
An appropriate source control procedure to drain infected foci, control ongoing peritoneal contamination by diversion or resection, and restore anatomic and physiological function to the extent feasible is recommended for nearly all patients with intra-abdominal infection	B-2
Community-acquired infections of mild-to-moderate severity in adults	
Antibiotics used for empiric treatment of community-acquired intra-abdominal infections should be active against enteric gram-negative aerobic and facultative bacilli and enteric gram-positive streptococci	A-1
For patients with mild-to-moderate community-acquired infections, regimens with substantial anti-pseudomonal activity are not required (**Table 43-4**)	A-1
Empiric coverage of *Enterococcus* is not necessary in patients with mild-to-moderate community-acquired intra-abdominal infection	A-1
The use of agents listed as appropriate for high-severity community-acquired infection and healthcare-associated infection is not	B-2

recommended for patients with mild-to-moderate community-acquired infection, because such regimens may carry a greater risk of toxicity and facilitate acquisition of more resistant organisms	
High-severity community-acquired infections in adults[b]	
The empiric use of antimicrobial regimens with broad-spectrum activity against gram-negative organisms including *Pseudomonas* spp., such as meropenem, imipenem–cilastatin, doripenem, piperacillin–tazobactam, ciprofloxacin or levofloxacin in combination with metronidazole, or ceftazidime or cefepime in combination with metronidazole, is recommended for patients with high-severity community-acquired intra-abdominal infection (**Table 43-4**)	A-1
Aztreonam plus metronidazole is an alternative, but addition of an agent effective against gram-positive cocci is recommended	B-3
Healthcare-associated infections in adults[c]	
Empiric antibiotic therapy for healthcare-associated intra-abdominal infection should be driven by local microbiologic results	A-2
To achieve empiric coverage of likely pathogens, multidrug regimens that include agents with expanded spectra of activity against gram-negative aerobic and facultative bacilli may be needed. These agents include meropenem, imipenem–cilastatin, doripenem, piperacillin–tazobactam, or metronidazole combined with either cefepime or ceftazidime. For multidrug-resistant aerobic gram-negative pathogens, aminoglycosides, colistin, polymyxin B, ceftazidime/avibactam, or ceftolozane/tazobactam may be required	B-3
Antimicrobial agents not recommended	
Ampicillin–sulbactam is not recommended for use because of high rates of resistance to this agent among community-acquired *E. coli*	B-2
Quinolone-resistant *E. coli* have become common in some communities, and quinolones should not be used unless hospital surveys indicate 90% susceptibility of *E. coli* to quinolones	A-2
Cefotetan and clindamycin are not recommended for use because of increasing prevalence of resistance to these agents among *Bacteroides fragilis*	B-2
Because of the availability of less toxic agents demonstrated to be at least equally effective, aminoglycosides are not recommended for routine use in adults with community-acquired intra-abdominal infection	B-2
Oral completion therapy	
For adults recovering from intra-abdominal infection, completion of the antimicrobial course with oral forms of moxifloxacin, ciprofloxacin plus metronidazole, levofloxacin plus metronidazole, an oral cephalosporin with metronidazole, or amoxicillin–clavulanic acid is acceptable in patients able to tolerate an oral diet and in patients in whom susceptibility studies do not demonstrate resistance	B-2
Duration of therapy	
Antimicrobial therapy of established infection should be limited to 4 days, unless it is difficult to achieve adequate source control. Longer durations of therapy have not been associated with improved outcome	A-1
For acute stomach and proximal jejunum perforations, in the absence of acid-reducing therapy or malignancy and when source control is achieved within 24 hours, prophylactic anti-infective therapy directed at aerobic gram-positive cocci for 24 hours is adequate	B-2

Bowel injuries attributable to penetrating, blunt, or iatrogenic trauma that are repaired within 12 hours and any other intraoperative contamination of the operative field by enteric contents should be treated with antibiotics for ≤24 hours	A-1
Acute appendicitis without evidence of perforation, abscess, or local peritonitis requires only prophylactic administration of narrow spectrum regimens active against aerobic and facultative and obligate anaerobes; treatment should be discontinued within 24 hours	A-1
The administration of prophylactic antibiotics to patients with severe necrotizing pancreatitis prior to the diagnosis of infection is not recommended	A-1
Anaerobic coverage	
Coverage for obligate anaerobic bacilli should be provided for distal small bowel, appendiceal, and colon-derived infection and for more proximal GI perforations in the presence of obstruction or paralytic ileus	A-1
Antifungal therapy	
Antifungal therapy for patients with high-severity community-acquired or healthcare-associated infection is recommended if *Candida* is grown from intra-abdominal cultures	B-2
Anti-MRSA therapy	
Empiric antimicrobial coverage directed against MRSA should be provided to patients with healthcare-associated intra-abdominal infection who are known to be colonized with the organism or who are at risk of having an infection due to this organism because of prior treatment failure and significant antibiotic exposure	B-2
Vancomycin is recommended for treatment of suspected or proven intra-abdominal infection due to MRSA	A-3
Antienterococcal therapy	
Antimicrobial therapy for enterococci should be given when enterococci are recovered from patients with high-severity community-acquired or healthcare-associated infection	B-III
Empiric antienterococcal therapy is recommended for patients with high-severity community-acquired infections and healthcare-associated intra-abdominal infections, particularly those with postoperative infection, those who have previously received cephalosporins or other antimicrobial agents selected for *Enterococcus* species, immunocompromised patients, and those with valvular heart disease or prosthetic intravascular materials	B-II
Initial empiric antienterococcal therapy should be directed against *Enterococcus faecalis*. Antibiotics that can potentially be used against this organism, on the basis of susceptibility testing of the individual isolate, include ampicillin, piperacillin/tazobactam, and vancomycin	B-III
Empiric therapy directed against vancomycin-resistant *Enterococcus faecium* is not recommended unless the patient is at very high risk for an infection due to this organism, such as a liver transplant recipient with an intra-abdominal infection originating in the hepatobiliary tree or a patient known to be colonized with vancomycin-resistant *E. faecium*	B-III

[a]Strength of recommendations: A, B, C, good, moderate, and poor evidence to support recommendation, respectively. Quality of evidence: 1, evidence from ≥1 properly randomized, controlled trial; 2, evidence from ≥1 well-designed clinical trial without randomization, from cohort or case-controlled analytic studies, from multiple time series, or from dramatic results from uncontrolled experiments; 3, evidence from opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert communities.

[b]Criteria for high-severity community-acquired infection: sepsis or septic shock, APACHE II score ≥15, delay in initial intervention >24 hours, advanced age, comorbidity and degree of organ dysfunction, low albumin level, poor nutritional status, degree of peritoneal involvement or diffuse peritonitis, inability to achieve adequate debridement or control of drainage, and presence of malignancy.

[c]Criteria for classification of intra-abdominal infections as healthcare-associated infection varies. However, patients who develop an infection after surgery, reside in a long-term care facility, who were recently hospitalized, or who have other significant healthcare exposure can be considered to have a healthcare-associated intra-abdominal infection.

MRSA, methicillin-resistant *Staphylococcus aureus*.

EVALUATION OF THERAPEUTIC OUTCOMES

- The patient should be continually reassessed to determine the success or failure of therapies.
- Once antimicrobials are initiated and other important therapies described earlier in the Treatment section are used, most patients should show improvement within 2–3 days. Usually, temperature will return to near normal, vital signs should stabilize, and the patient should not appear in distress, with the exception of recognized discomfort and pain from incisions, drains, and nasogastric tube.
- Within 24–48 hours, aerobic bacterial culture results should return. If a suspected pathogen is not sensitive to the antimicrobial agents being given, the regimen should typically be changed to active therapy. If the isolated pathogen is susceptible to a narrower spectrum agent, therapy should be deescalated.
- With present anaerobic culturing techniques and the slow growth of these organisms, anaerobes are often not identified until 4–7 days after culture, and sensitivity information is difficult to obtain. A report indicating that anaerobes were not isolated should not be the sole justification for discontinuing antianaerobic drugs.
- Superinfection in patients being treated for intra-abdominal infection is often due to *Candida*; however, enterococci or opportunistic gram-negative bacilli such as *Pseudomonas* may be involved.
- Treatment regimens for intra-abdominal infection can be judged successful if the patient recovers from the infection without recurrent peritonitis or intra-abdominal abscess and without the need for additional antimicrobials. A regimen can be considered unsuccessful if a significant adverse drug reaction occurs, if reoperation is necessary, or if patient improvement is delayed beyond 1 or 2 weeks.

See Chapter 132, Intra-abdominal Infections, authored by Alan E. Gross and Keith M. Olsen, for a more detailed discussion of this topic.