

# Peritonitis and Abdominal Sepsis

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## Introduction

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Peritonitis is defined as inflammation of the serosal membrane that lines the abdominal cavity and the organs contained therein. Peritonitis is often caused by introduction of an infection into the otherwise sterile peritoneal environment through perforation of the bowel, such as a ruptured appendix or colonic diverticulum. The disease may also be caused by introduction of a chemically irritating material, such as gastric acid from a perforated ulcer or bile from a perforated gall bladder or a lacerated liver. In women, localized peritonitis most often occurs in the pelvis from an infected fallopian tube or a ruptured ovarian cyst.

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## History of the Procedure

Untreated cases of acute peritonitis may be fatal. In 1926, the fundamental role of operative therapy in the treatment of peritonitis was documented. Kirschner (1926) reported that the mortality rate from intra-abdominal infections decreased from more than 90% to less than 40% during the period from 1890-1924 with the introduction of operative management as an effective therapeutic modality.

Current treatment of peritonitis and peritoneal abscesses consists of a multimodality approach directed at correction of the underlying cause, administration of systemic antibiotics, and supportive therapy to prevent or limit secondary complications due to organ system failure.

## Problem

Inflammation and/or infection of the peritoneal cavity are commonly encountered problems in the practice of clinical medicine today. In general, the term peritonitis refers to a constellation of signs and symptoms, which includes abdominal pain and tenderness on palpation, abdominal wall muscle rigidity, and systemic signs of inflammation. Patients may present with an acute or insidious onset of symptoms, limited and mild disease, or systemic and severe disease with septic shock.

The peritoneum reacts to a variety of pathologic stimuli with a fairly uniform inflammatory response. Depending on the underlying pathology, the resultant peritonitis may be infectious or sterile (ie, chemical or mechanical).

Peritoneal infections are classified as primary (ie, spontaneous), secondary (ie, related to a pathologic process in a visceral organ), or tertiary (ie, persistent or recurrent infection after adequate initial therapy).

The intra-abdominal infections are usually divided into generalized (peritonitis) and localized (intra-abdominal abscess). This article focuses on the diagnosis and management of infectious peritonitis and abdominal abscesses.

## Frequency

The overall incidence of peritoneal infections and abscess formation is difficult to establish and varies with underlying abdominal disease processes.

The most common etiology of primary peritonitis is spontaneous bacterial peritonitis (SBP) due to chronic liver disease. Approximately 10-30% of all patients with liver cirrhosis who have ascites develop bacterial peritonitis over time.

The common etiologic entities of secondary peritonitis (SP) include perforated appendicitis; perforated gastric and duodenal ulcer disease; perforated (sigmoid) colon caused by diverticulitis, volvulus, or cancer; and strangulation of the small bowel (see Table 1).

Table 1. Common Causes of Secondary Peritonitis

Source Regions	Causes
Esophagus	Boerhaave syndrome Malignancy Trauma (mostly penetrating) iatrogenic*
Stomach	Peptic ulcer perforation Malignancy (eg, adenocarcinoma, lymphoma, gastrointestinal stromal tumor) Trauma (mostly penetrating) iatrogenic*
Duodenum	Peptic ulcer perforation Trauma (blunt and penetrating) iatrogenic*
Biliary tract	Cholecystitis Stone perforation from gallbladder (ie, gallstone ileus) or common duct Malignancy Choledochal cyst (rare) Trauma (mostly penetrating) iatrogenic*
Pancreas	Pancreatitis (eg, alcohol, drugs, gallstones) Trauma (blunt and penetrating) iatrogenic*
Small bowel	Ischemic bowel Incarcerated hernia (internal and external) Closed loop obstruction Crohn disease Malignancy (rare) Meckel diverticulum Trauma (mostly penetrating)
Large bowel and appendix	Ischemic bowel Diverticulitis Malignancy Ulcerative colitis and Crohn disease Appendicitis Colonic volvulus Trauma (mostly penetrating) iatrogenic
Uterus, salpinx, and ovaries	Pelvic inflammatory disease (eg, salpingo-oophoritis, tubo-ovarian abscess, ovarian cyst) Malignancy (rare) Trauma (uncommon)

\*Iatrogenic trauma to the upper GI tract, including the pancreas and biliary tract and colon, often results from endoscopic procedures; anastomotic dehiscence and inadvertent bowel injury (eg, mechanical, thermal) are common causes of leak in the postoperative period.

After elective abdominal operations for noninfectious etiologies, the incidence of SP (caused by anastomotic disruption, breakdown of enterotomy closures, or inadvertent bowel injury) should be less than 2%. Operations for inflammatory disease (ie, appendicitis, diverticulitis, cholecystitis) without perforation carry a risk of less than 10% for the development of SP and peritoneal abscess. This risk may rise to greater than 50% in gangrenous bowel disease and visceral perforation.

After operations for penetrating abdominal trauma, SP and abscess formation is observed in a small number of patients. Duodenal and pancreatic involvement, as well as colon perforation, gross peritoneal contamination, perioperative shock, and massive transfusion, are factors that increase the risk of infection in these cases.

### Etiology

SBP occurs in the absence of an apparent intra-abdominal source of infection and is observed almost exclusively in patients with ascites formation from chronic liver disease. Contamination of the peritoneal cavity is thought to result from translocation of bacteria across the gut wall or mesenteric lymphatics and, less frequently, via hematogenous seeding in the presence of bacteremia.

Approximately 10-30% of patients with cirrhosis and ascites develop this problem over time. The incidence rises with ascitic fluid protein contents less than 1 g/dL (which occurs 15% of patients) to more than 40%, presumably because of decreased ascitic fluid opsonic activity.

More than 90% of cases of SBP are caused by a monomicrobial infection. The most common pathogens include gram-negative organisms (eg, *Escherichia coli* [40%], *Klebsiella pneumoniae* [7%], *Pseudomonas* species, *Proteus* species, other gram-negative species [20%]) and gram-positive organisms (eg, *Streptococcus pneumoniae* [15%], other *Streptococcus* species [15%], *Staphylococcus* species [3%]) (see Table 2). Anaerobic microorganisms are found in less than 5% of cases, and multiple isolates are found in less than 10%.

Table 2. Microbiology of Primary, Secondary, and Tertiary Peritonitis

Peritonitis (Type)	Etiologic Organisms		Antibiotic Therapy (Suggested)
	Class	Type of Organism	
Primary	Gram-negative	<i>E coli</i> (40%) <i>K pneumoniae</i> (7%) <i>Pseudomonas</i> species (5%) <i>Proteus</i> species (5%) <i>Streptococcus</i> species (15%) <i>Staphylococcus</i> species (3%) Anaerobic species (<5%)	Third-generation cephalosporin
Secondary	Gram-negative	<i>E coli</i> <i>Enterobacter</i> species <i>Klebsiella</i> species <i>Proteus</i> species	Second-generation cephalosporin Third-generation cephalosporin Penicillins with anaerobic activity Quinolones with anaerobic activity Quinolone and metronidazole Aminoglycoside and metronidazole
	Gram-positive	<i>Streptococcus</i> species <i>Enterococcus</i> species	
	Anaerobic	<i>Bacteroides fragilis</i> Other <i>Bacteroides</i> species <i>Eubacterium</i> species <i>Clostridium</i> species Anaerobic <i>Streptococcus</i> species	
Tertiary	Gram-negative	<i>Enterobacter</i> species <i>Pseudomonas</i> species	Second-generation cephalosporin Third-generation cephalosporin Penicillins with anaerobic activity

		<i>Enterococcus</i> species	Quinolones with anaerobic activity Quinolone and metronidazole
	Gram-positive	<i>Staphylococcus</i> species	Aminoglycoside and metronidazole Carbapenems
	Fungal	<i>Candida</i> species	Triazoles or amphotericin (considered in fungal etiology) (Alter therapy based on culture results.)

SP is, by far, the most common form of peritonitis encountered in clinical practice today. It is caused by perforation or necrosis (transmural infection) of a hollow visceral organ with bacterial inoculation of the peritoneal cavity. The list of potential differential diagnoses is complex (see Table 1).

The spectrum of pathogens depends to some degree on the site of the original disease. Gram-positive organisms predominate in the upper GI tract; however, a shift toward gram-negative organisms may be noticed in patients on long-term gastric acid suppressive therapy. Contamination from a distal small bowel or colon source initially may result in the release of several hundred bacterial species (and fungi); host defenses quickly eliminate most of these organisms. The resulting peritonitis is almost always polymicrobial, containing a mixture of aerobic and anaerobic bacteria with a predominance of gram-negative organisms (see Table 2).

As many as 15% of patients who have cirrhosis with ascites who were initially presumed to have SBP have secondary peritonitis. In many of these patients, clinical signs and symptoms alone are not sensitive or specific enough to reliably differentiate the 2 entities. A thorough history, evaluation of the peritoneal fluid, and additional diagnostic tests are needed to establish the correct diagnosis and treatment in these patients.

Peritoneal abscess describes the formation of an infected fluid collection encapsulated by fibrinous exudate, omentum, and/or adjacent visceral organs. The overwhelming majority of abscesses occurs subsequent to SP. Approximately half of patients develop a simple abscess without loculation, whereas the other half of patients develop complex abscesses secondary to fibrinous septation and organization of the abscess material. Abscess formation occurs most frequently in the subhepatic area, the pelvis, and the paracolic gutters, but it may also occur in the perisplenic area, the lesser sac, and between small bowel loops and their mesentery.

In general, the incidence of abscess formation after abdominal surgery is less than 1-2%, even when the operation is performed for an acute inflammatory process. This incidence increases with preoperative perforation of the hollow viscus, significant fecal contamination of the peritoneal cavity, bowel ischemia, delayed diagnosis and therapy of the initial peritonitis, the need for reoperation, and in the setting of immunosuppression. In these instances, the risk of abscess formation may be as high as 10-30%. Overall, abscess formation is the leading cause of persistent infection and development of tertiary peritonitis.

Tertiary peritonitis represents the persistence or recurrence of peritoneal infection following apparently adequate therapy of SBP or SP, often without the original visceral organ pathology. Patients with tertiary peritonitis usually present with an abscess, or phlegmon, with or without fistulization. Tertiary peritonitis develops more frequently in patients with significant preexisting comorbid conditions and in patients who are immunocompromised. Although rarely observed in uncomplicated peritoneal infections, the incidence of tertiary peritonitis in patients requiring ICU admission for severe abdominal infections may be as high as 50-74%.

Patients who develop tertiary peritonitis demonstrate significantly longer lengths of stay in the ICU and hospital, higher organ dysfunction scores, and higher mortality rates (50-70%). Resistant and unusual organisms (eg, *Enterococcus*, *Candida*, *Staphylococcus*, *Enterobacter*, and *Pseudomonas* species) are found in a significant proportion of cases of tertiary peritonitis. Most patients with tertiary peritonitis develop complex abscesses or poorly localized peritoneal infections that are not amenable to percutaneous drainage. Antibiotic therapy appears less effective compared to all other forms of peritonitis.

Tuberculous peritonitis (TP) is a rare disease in the United States (<2% of all causes of peritonitis), but it continues to be a significant problem in underdeveloped countries and among patients with HIV disease. The presenting symptoms are often nonspecific and insidious in onset (eg, low-grade fever, anorexia, weight loss).

More than 95% of patients have evidence of ascites on imaging studies, and more than half of these patients have clinically apparent ascites. Most patients have evidence of cirrhosis, and the diagnosis of TP may be unsuspected. Chest radiograph findings are abnormal in most patients, but active pulmonary disease is present in fewer than 30% of patients. Results on Gram stain of ascitic fluid are rarely positive, and culture results may be falsely negative in up to 80% of patients. A peritoneal fluid protein level greater than 2.5 g/dL, lactate dehydrogenase (LDH) level greater than 90 U/mL, or predominantly mononuclear cell count greater than 500 cells/ $\mu$ L should raise suspicion but has limited specificity for the diagnosis. Laparoscopy and visualization of granulomas on peritoneal biopsy specimens, as well as positive results on cultures (requires 4-6 wk) may be needed for the definitive diagnosis; however, empiric therapy should begin immediately.

Chemical (sterile) peritonitis may be caused by irritant substances such as bile, blood, barium, and other substances or by transmural inflammatory processes of visceral organs (eg, Crohn disease) without bacterial inoculation of the peritoneal cavity. Clinical signs and symptoms are indistinguishable from those of SP or peritoneal abscess, and the diagnostic and therapeutic approach should be the same.

### **Pathophysiology**

Peritonitis causes a reduction in the intra-abdominal fibrinolytic activity (increased plasminogen activator inhibitor activity) and fibrin sequestration with subsequent adhesion formation. The production of fibrinous exudates is considered an important part of the host defense, but large numbers of bacteria may be sequestered within the fibrin matrix. This may lead to retardation of spread and systemic dissemination and may decrease early mortality rates from sepsis, but it also is integral to the development of residual infection and abscess formation. As the fibrin matrix matures, the bacteria within are protected from host clearance mechanisms.

The ultimate effect (containment vs persistent infection) of fibrin may be related to the degree of peritoneal bacterial contamination. In animal studies of mixed bacterial peritonitis examining the effects of systemic defibrinogenization and those of abdominal fibrin therapy, heavy peritoneal contamination uniformly led to severe peritonitis with early death (<48 h) because of overwhelming sepsis.

Abscess formation has been viewed as a host defense strategy to contain the spread of infection; however, this process can lead to persistent infection and life-threatening sepsis.

The initiation of abscess formation involves the release of bacteria and an abscess-potentiating agent into a normally sterile environment. The host defense is unable to eliminate the infecting agent and attempts to control the spread by compartmentalization. This process is aided by a combination of factors that share a common feature, ie, impairment of phagocytotic killing. Some studies suggest that the number of bacteria present at the onset of abdominal infections is much higher than originally believed (approximately  $2 \times 10^8$  CFU/mL, much higher than the routinely used  $5 \times 10^5$  CFU/mL inocula for in vitro susceptibility testing). This bacterial load may locally overwhelm the host defense.

In minimal contamination, bacterial clearance was complete in nearly 100% of cases, and no differences in outcome were observed among fibrin-depleted, normal, and fibrin-treated groups. With moderate contamination, fibrin-treated animals demonstrated a significantly reduced early mortality rate but developed more abdominal abscesses. Finally, studies with adhesion-reducing devices (ie, bioresorbable membranes) increased the incidence of peritonitis and peritoneal infections in experimental peritonitis models.

Transient bacterial peritoneal contamination (caused by primary visceral disease and intentional or unintentional violation of the gut) is common. The resultant exposure to bacterial antigens has been

shown to alter subsequent immune responses to recurrent peritoneal inoculation. This may lead to an increased incidence of later abscess formation, alteration of the bacterial content, and increased late mortality rates. More recent studies have shown that nosocomial infections at other sites (eg, pneumonia, line sepsis, wound infections) also increase the likelihood of subsequent abdominal abscess formation.

Bacterial virulence factors that interfere with phagocytosis and neutrophil-mediated bacterial killing are important mediators leading to persistence of infections and abscess formation. Among these factors are capsule formation, facultative anaerobic growth, adhesion capabilities, and succinic acid production. Synergy between certain bacterial and fungal organisms may also play an important role in impairing the host's defense. One such synergy may exist between *B fragilis* and gram-negative bacteria, particularly *E coli*, where co-inoculation significantly increases bacterial proliferation and abscess formation.

Enterococci may be important in enhancing the severity and persistence of peritoneal infections. In animal models of peritonitis with *E coli* and *B fragilis*, the systemic manifestations of the peritoneal infection and bacteremia rates were increased, as were bacterial concentrations in the peritoneal fluid and rate of abscess formation. This is more important in light of the difficulties in eradicating *Enterococcus faecalis* with conventional antimicrobial therapy. The role of *Enterococcus* organisms in uncomplicated intra-abdominal infections remains unclear. Antibiotics that lack specific activity against *Enterococcus* organisms are often used successfully in the therapy of peritonitis, and the organism is recovered uncommonly as a blood-borne pathogen in intra-abdominal sepsis.

The role of fungi in the formation of intra-abdominal abscesses is not fully understood. Abdominal infections, particularly with *Candida* species, are becoming increasingly common in critically ill patients. Studies suggest that the microbiology of intra-abdominal infections may be inherently different in severely ill patients. *Candida albicans* was the organism most commonly isolated from the peritoneum in critically ill patients with culture-proven intra-abdominal infections and preoperative APACHE II (acute physiology and chronic health evaluation) scores greater than or equal to 15, with an associated mortality rate of 52%. Additional common peritoneal organisms in this patient population were *Enterococcus* and *Enterobacter* species and *Staphylococcus epidermidis*. These data suggest that broader antimicrobial, and possibly antifungal, coverage may be warranted in patients with severe abdominal sepsis.

Some authors suggest that bacteria and fungi exist as nonsynergistic parallel infections with incomplete competition, allowing the survival of all organisms. In this setting, treatment of the bacterial infection alone may lead to an overgrowth of fungi, which may contribute to increased morbidity. Predisposing factors for the development of abdominal candidiasis include prolonged use of broad-spectrum antibiotics, gastric acid suppressive therapy, central venous catheters and intravenous hyperalimentation, malnutrition, diabetes, and steroids and other forms of immunosuppression.

Most animal and human studies suggest that abscess formation occurs only in the presence of abscess-potentiating agents. Although the nature and spectrum of these factors has not been studied exhaustively, certain fiber analogues (eg, bran) and the contents of autoclaved stool have been identified as such abscess-potentiating agents. In animal models, these factors inhibited opsonization and phagocytotic killing by interference with complement activation.

The role of cytokines in mediation of the body's immune response and their role in the development of the systemic inflammatory response syndrome (SIRS) and multiple organ failure (MOF) have been a major focus of research over the past decade. Comparatively little data exist about the magnitude of the intraperitoneal/abscess cytokine response and implications for the host. Existing data suggest that bacterial peritonitis is associated with an immense intraperitoneal compartmentalized cytokine response. Higher levels of certain cytokines (ie, tumor necrosis factor-alpha [TNF-alpha], interleukin [IL]-6) have been associated with worse outcomes, as well as secondary (uncontrolled) activation of the systemic inflammatory cascade.

## **Presentation**

The diagnosis of peritonitis is usually clinical. Essentially, all patients present with some degree of abdominal pain. This pain may be acute or more insidious in onset. Initially, the pain is often dull and poorly localized (visceral peritoneum) and then progresses to steady, severe, and more localized pain (parietal peritoneum). If the infectious process is not contained, the pain becomes diffuse. In certain disease entities (eg, gastric perforation, severe acute pancreatitis, intestinal ischemia), the abdominal pain may be generalized from the beginning.

Anorexia and nausea are frequently present and may precede the development of abdominal pain. Vomiting may occur because of the underlying visceral organ pathology (ie, obstruction) or secondary to the peritoneal irritation.

On physical examination, patients with peritonitis most often appear unwell and in acute distress. Fever with temperatures that can exceed 38°C is usually present, but patients with severe sepsis may present with hypothermia. Tachycardia is caused by the release of inflammatory mediators and intravascular hypovolemia caused by anorexia and vomiting, fever, and third-space losses into the peritoneal cavity. With progressive dehydration, patients may become hypotensive, they may demonstrate decreased urine output, and, with severe peritonitis. They may present in overt septic shock.

On abdominal examination, essentially all patients demonstrate tenderness to palpation. (When examining the abdomen of a patient with peritonitis, the patient should be supine. A roll or pillows underneath the patient's knees may allow for better relaxation of the abdominal wall.) In most patients (even with generalized peritonitis and severe diffuse abdominal pain), the point of maximal tenderness or referred rebound tenderness roughly overlies the pathologic process (ie, the site of maximal peritoneal irritation).

Nearly all patients demonstrate increased abdominal wall rigidity. The increase in abdominal wall muscular tone may be voluntary in response to or in anticipation of the abdominal examination or involuntary because of the peritoneal irritation. Patients with severe peritonitis often avoid all motion and keep their hips flexed to relieve the abdominal wall tension. The abdomen is often distended, with hypoactive-to-absent bowel sounds. This finding reflects a generalized ileus and may not be present if the infection is well localized. Occasionally, the abdominal examination reveals an inflammatory mass.

Rectal examination often elicits increased abdominal pain, particularly with inflammation of the pelvic organs but rarely indicates a specific diagnosis. A tender inflammatory mass toward the right may indicate appendicitis, and anterior fullness and fluctuation may indicate a cul de sac abscess.

In female patients, vaginal and bimanual examination may lead to the differential diagnosis of pelvic inflammatory disease (eg, endometritis, salpingo-oophoritis, tubo-ovarian abscess), but the findings are often difficult to interpret in severe peritonitis.

When evaluating the patient with suspected peritoneal infection, performing a complete physical examination is important. Thoracic processes with diaphragmatic irritation (eg, empyema), extraperitoneal processes (eg, pyelonephritis, cystitis, acute urinary retention), and abdominal wall processes (eg, infection, rectus hematoma) may mimic certain signs and symptoms of peritonitis. Always examine the patient carefully for the presence of external hernias to rule out intestinal incarceration.

Remember that the presentation and the findings on clinical examination may be entirely inconclusive or unreliable in patients with significant immunosuppression (eg, severe diabetes, steroid use, posttransplant status, HIV), in patients with altered mental state (eg, head injury, toxic encephalopathy, septic shock, analgesic agents), in patients with paraplegia, and in patients of advanced age. With localized deep peritoneal infections, fever and/or an elevated WBC count may be the only signs present. As many as 20% of patients with SBP demonstrate very subtle signs and symptoms. New onset or deterioration of existing encephalopathy may be the only sign of the infection at the initial presentation. Most patients with TP demonstrate only vague symptoms and may be afebrile.

## Indications

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Early control of the septic source is mandatory and can be achieved by operative and nonoperative means. Nonoperative interventional therapies include percutaneous drainage of abscesses and percutaneous and endoscopic stent placements. If an abscess is accessible to percutaneous drainage and the underlying visceral organ pathology does not clearly require an operative approach, percutaneous drainage can be used safely and effectively as the primary treatment modality.

Operative management addresses the need to control the infectious source and to purge bacteria and toxins. The type and extent of surgery depends on the underlying disease process and the severity of intra-abdominal infection. Open treatment allows for thorough drainage of the intra-abdominal infection, but the specific indications are not clearly defined.

## Relevant Anatomy

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The peritoneum is the largest and most complex serous membrane in the body. It forms a closed sac (ie, coelom) by lining the interior surfaces of the abdominal wall (anterior and lateral), by forming the boundary to the retroperitoneum (posterior), by covering the extraperitoneal structures in the pelvis (inferior), and by covering the undersurface of the diaphragm (superior). This parietal layer of the peritoneum reflects onto the abdominal visceral organs to form the visceral peritoneum. It thereby creates a potential space between the 2 layers (ie, the peritoneal cavity).

The peritoneum consists of a single layer of flattened mesothelial cells over loose areolar tissue. The loose connective tissue layer contains a rich network of vascular and lymphatic capillaries, nerve endings, and immune-competent cells, particularly lymphocytes and macrophages. The peritoneal surface cells are joined by junctional complexes, thus forming a dialyzing membrane that allows passage of fluid and certain small solutes. Pinocytotic activity of the mesothelial cells and phagocytosis by macrophages allow for clearance of macromolecules.

Normally, the amount of peritoneal fluid present is less than 50 mL, and only small volumes are transferred across the considerable surface area in a steady state each day. The peritoneal fluid represents a plasma ultrafiltrate, with electrolyte and solute concentrations similar to that of neighboring interstitial spaces and a protein content of less than 30 g/L, mainly albumin. In addition, peritoneal fluid contains small numbers of desquamated mesothelial cells and various numbers and morphologies of migrating immune cells (reference range is <300 cells/ $\mu$ L, predominantly of mononuclear morphology).

The peritoneal cavity is divided incompletely into compartments by the mesenteric attachments and secondary retroperitonealization of certain visceral organs. A large peritoneal fold, the greater omentum, extends from the greater curvature of the stomach and the inferior aspect of the proximal duodenum downward over a variable distance to fold upon itself (with fusion of the adjacent layers) and ascends back to the taenia omentalis of the transverse colon. This peritoneal fold demonstrates a slightly different microscopic anatomy, with fenestrated surface epithelium and a large number of adipocytes, lymphocytes, and macrophages, and it functions as a fat storage location and a mobile immune organ.

The compartmentalization of the peritoneal cavity, in conjunction with the greater omentum, influences the localization and spread of peritoneal inflammation and infections.

## Workup

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### Laboratory Studies

- CBC with differential, serum electrolytes with renal function
  - Most patients with intra-abdominal infections demonstrate leukocytosis (>11,000 cells/ $\mu$ L) with a shift to the immature forms on the differential cell count. Patients in severe sepsis, patients who are immunocompromised, and patients with certain

types of infections (eg, fungal, cytomegaloviral) may demonstrate absence of leukocytosis or leucopenia.

- Blood chemistry may reveal dehydration and acidosis.
- PT, PTT, and INR
- Liver function tests if clinically indicated
- Amylase and lipase if pancreatitis is suspected
- Urinalysis (UA) is essential to rule out urinary tract diseases (eg, pyelonephritis, renal stone disease); however, patients with lower abdominal and pelvic infections often demonstrate WBCs in the urine and microhematuria.
- In patients with diarrhea, evaluate a stool sample for *Clostridium difficile* toxin assay, WBC count, and specific culture (ie, *Salmonella*, *Shigella*, cytomegalovirus [CMV]) if the patient's history suggests infectious enterocolitis.
- Aerobic and anaerobic blood cultures
- Peritoneal fluid (ie, paracentesis, aspiration of abdominal fluid collections, intraoperative peritoneal fluid cultures)
  - When assessing a peritoneal fluid sample for peritoneal infection, evaluate the sample for pH, glucose, protein, lactate dehydrogenase (LDH), cell count, Gram stain, and aerobic and anaerobic cultures.
  - Obtain a peritoneal fluid amylase analysis if pancreatitis or pancreatic leak is suspected. Obtain a fluid bilirubin analysis when a biliary leak is suspected and evaluate the fluid creatinine level when a urinary leak is suspected. Compare the peritoneal levels to the respective serum levels.
  - The fluid in bacterial peritonitis generally demonstrates low pH and glucose as well as elevated protein and LDH levels. A fluid pH lower than 7.1 (and partial pressure of oxygen [PO<sub>2</sub>] <49 mm Hg) has demonstrated positive and negative predictive values of greater than 98% in some studies (median pH of 6.75 versus 7.49 for elective surgery, with PO<sub>2</sub> 28 versus 144 mm Hg). The drop in peritoneal fluid pH (and PO<sub>2</sub>) is more pronounced in mixed infections and severe bacterial contamination, with increased numbers of anaerobic bacteria in these circumstances.
- In SBP, a WBC count of more than 250 cells/μL (>500 in some studies), with more than 50% polymorphonuclear leukocytes (PMNs) is an indication to begin antibiotic therapy. Although up to 30% of culture findings remain negative in these patients, most of these patients are presumed to have bacterial peritonitis; they should be treated. A significantly decreased peritoneal fluid glucose level (<50 mg/dL), a peritoneal fluid LDH level much greater than the serum LDH, a peritoneal fluid WBC count greater than 10,000 cells/μL, a pH lower than 7.0, high amylase levels, multiple organisms on Gram stain, or recovery of anaerobes from the culture raises the suspicion of SP in these patients. Some authors recommend repeating the paracentesis in 48-72 hours to monitor treatment success (decrease in neutrophil count to <50% of the original value).
- In TP, the fluid Gram stain and acid-fast stain results are rarely positive, and routine culture findings are falsely negative in as many as 80% of cases. A peritoneal fluid protein level greater than 2.5 g/dL, LDH level greater than 90 U/mL, and predominantly mononuclear cell count of more than 500 cells/μL should raise the suspicion of TP, but specificity for the diagnosis is limited. Laparoscopy with visualization of granulomas on peritoneal biopsy and specific culture (requires 4-6 wk) may be needed for definitive diagnosis.
- Routine intraoperative peritoneal fluid cultures in defined acute disease entities (ie, gastric or duodenal ulcer perforation, appendicitis, diverticulitis or perforation of the colon caused by obstruction or ischemia) are controversial. Several studies have found no significant difference in patients with appendicitis, diverticulitis, and other common etiologies for bacterial peritonitis with regard to postoperative complication rates or overall outcomes. The antibiotic regimen was altered only 8-10% of the time based on operative culture data. In patients who had previous abdominal operations or instrumentation (eg, peritoneal dialysis catheter, percutaneous stents) and patients with prolonged antibiotic therapy, critical illness, and/or hospitalization, these cultures may reveal resistant or unusual organisms that should prompt alteration of the antibiotic strategy.

## Imaging Studies

- Radiographs
  - Plain films of the abdomen (eg, supine, upright, and lateral decubitus positions) are often the first imaging studies obtained in patients presenting with peritonitis. Their value in reaching a specific diagnosis is limited.
  - Free air is present in most cases of anterior gastric and duodenal perforation but is much less frequent with perforations of the small bowel and colon and is unusual with appendiceal perforation. Upright films are useful for identifying free air under the diaphragm (most often on the right) as an indication of a perforated viscus. Although in some cases free air can be visualized as a central round area of hyperlucency on supine films, this finding is often overlooked. Free air can also be missed easily on portable bedside films with the patient in a semirecumbent position. A left side–down lateral decubitus film might help to make the diagnosis. Remember that the presence of free air is not mandatory with visceral perforation and that small amounts of free air are missed easily on plain films.
- Ultrasound
  - Abdominal ultrasound may be helpful in the evaluation of right upper quadrant (eg, perihepatic abscess, cholecystitis, biloma, pancreatitis, pancreatic pseudocyst), right lower quadrant, and pelvic pathology (eg, appendicitis, tubo-ovarian abscess, Douglas pouch abscess), but the examination is sometimes limited because of patient discomfort, abdominal distension, and bowel gas interference.
  - Ultrasonography may detect increased amounts of peritoneal fluid (ascites), but its ability to detect quantities of less than 100 mL is limited. The central (perimesenteric) peritoneal cavity is not visualized well with transabdominal ultrasonography. Examination from the flank or back may improve the diagnostic yield, and providing the ultrasonographer with specific information of the patient's condition and the suspected diagnosis before the examination is important. With an experienced ultrasonographer, a diagnostic accuracy of greater than 85% has been reported in several series.
  - Over the past several years, ultrasound-guided aspiration and placement of drains has evolved into a valuable tool in the diagnosis and treatment of abdominal fluid collections (see Medical therapy).
- Computed tomography scan
  - Computed tomography (CT) scans of the abdomen and pelvis remain the diagnostic study of choice for peritoneal abscess and the related visceral pathology. CT scan is indicated in all cases where the diagnosis cannot be established on clinical grounds and findings on abdominal plain films. Whenever possible, the CT scan should be performed with enteral and intravenous contrast. CT scans can detect small quantities of fluid, areas of inflammation, and other GI tract pathology, with sensitivities that approach 100%.
  - Peritoneal abscesses and other fluid collections may be aspirated for diagnosis and drained under CT guidance; this technique has become a mainstay of therapy (see Medical therapy).
- Nuclear medicine scans (eg, gallium Ga 67 scan, indium In 111–labeled autologous leucocyte scan, technetium Tc 99m-iminoacetic acid derivative scan).
  - These diagnostic studies have little use in the initial evaluation of patients with suspected peritonitis or intra-abdominal sepsis. They are most frequently used in the evaluation of fever of unknown origin or in patients with persistent fever despite adequate antibiotic treatment and negative CT scan findings.
- Magnetic resonance imaging
  - Magnetic resonance imaging is an emerging imaging modality for the diagnosis of suspected intra-abdominal abscesses. Abdominal abscesses demonstrate decreased signal intensity on T1-weighted images and homogeneous or heterogeneous increased signal intensity on T2-weighted images; abscesses are observed best on gadolinium-enhanced, T1-weighted, fat-suppressed images as well-defined fluid collections with rim enhancement.

- Limited availability and high cost, as well as the need for MRI-compatible patient support equipment and the length of the examination currently limit its usefulness as a diagnostic tool in acute peritoneal infections, particularly for patients who are critically ill.
- Contrast studies
  - Conventional contrast studies (ie, Gastrografin swallow, upper GI tract study with follow-through, colorectal contrast enema, fistulogram, contrast studies of drains and stents) are reserved for specific indications in the setting of suspected peritonitis or peritoneal abscess.

## Diagnostic Procedures

- See Surgical therapy for a discussion of laparoscopy.

## Treatment

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### Medical Therapy

The general principles guiding the treatment of intra-abdominal infections are 4-fold: (1) to control the infectious source, (2) to eliminate bacteria and toxins, (3) to maintain organ system function, and (4) to control the inflammatory process.

Medical, nonoperative interventional, and operative treatment options are complimentary, not competitive, in the treatment of peritoneal infections. Medical support includes (1) systemic antibiotic therapy; (2) intensive care with hemodynamic, pulmonary, and renal replacement support; (3) nutrition and metabolic support; and (4) inflammatory response modulation therapy.

Early control of the septic source is mandatory and can be achieved by operative and nonoperative means. Nonoperative interventional therapies include percutaneous drainage of abscesses and percutaneous and endoscopic stent placements.

Treatment of peritonitis and intra-abdominal sepsis always begins with volume resuscitation, correction of potential electrolyte and coagulation abnormalities, and empiric broad-spectrum parenteral antibiotic coverage. For treatment of septic shock, see Shock, Distributive.

### Antibiotic therapy

In SBP, initiate empiric therapy with a third-generation cephalosporin and then tailor therapy according to the culture results. Avoid aminoglycosides because patients with chronic liver disease are at an increased risk for nephrotoxicity. The optimal duration of therapy is not known; traditionally, a course of 10 days has been recommended, although newer studies suggest that 5 days of therapy (with documentation of a decrease of peritoneal fluid WBC count to <250 cells/ $\mu$ L) may be sufficient in most cases.

In secondary and tertiary peritonitis, systemic antibiotic therapy is the second mainstay of therapy. Several studies suggest that antibiotic therapy is not as effective in later stages of the infection and that early (preoperative) systemic antibiotic therapy can result in significant reduction of concentration and growth rates of viable bacteria in the peritoneal fluid. Therefore, begin empiric therapy as soon as the diagnosis of peritoneal infection is suspected. The initial therapy for SP must be mainly active against gram-negative organisms (eg, *E coli*, Enterobacteriaceae species) and anaerobes (eg, *B fragilis*).

In community-acquired infections, a second- or third-generation cephalosporin or a quinolone with or without metronidazole provides adequate coverage, as do broad-spectrum penicillins with anaerobic activity (ie, ampicillin/sulbactam) and newer quinolones (ie, trovafloxacin, clinafloxacin). Most studies suggest that single-drug therapy is as effective as dual or triple combination therapy in mild-to-moderate abdominal infections.

In severe and hospital-acquired intra-abdominal infections, imipenem, piperacillin/tazobactam, and a combination of aminoglycosides and metronidazole are often effective. A recent study of nearly 400 patients documented that ertapenem, a novel carbapenem with a half-life that allows once-a-day dosing, was effective (86.7% success rate) compared to piperacillin/tazobactam (81.2% success rate) in the treatment of complicated intra-abdominal infection and was well tolerated. Additional clinical antimicrobial studies are underway investigating the efficacy of new quinolones in the treatment of intra-abdominal infection.

With persistence of the infection (ie, tertiary peritonitis) and prolonged critical illness, obtaining peritoneal fluid and/or abscess cultures with sensitivities at operation or drainage is important to properly treat unusual (eg, gram-positive organisms, fungi) and resistant organisms (eg, *Enterococcus*, *Staphylococcus*, *Pseudomonas*, resistant *Bacteroides*, and *Candida* species). Certain preexisting conditions, immunoincompetence, gastric acid suppression therapy, and recent antibiotic use may also influence the spectrum of microorganisms. Consultation with infectious disease specialists is warranted in these cases to apply all available information about prevalence and resistance patterns of these organisms in the individual institution.

The optimal duration of antibiotic therapy must be individualized and depends on the underlying pathology, severity of infection, speed and effectiveness of source control, and the patient response to therapy. In uncomplicated peritonitis with early adequate source control, a course of 5-7 days is adequate in most cases. Mild cases (eg, early appendicitis, cholecystitis) may not need more than 24-72 hours of postoperative therapy. Inadequate initial therapy has been linked to worse outcomes, and these outcomes could not be significantly changed by later specific or prolonged therapy.

Complicated persistent infections and infections in patients who are immunocompromised may warrant a prolonged course of antibiotic therapy. In these cases, continuously seeking and aggressively treating all new extraperitoneal and new or persistent intra-abdominal sources is important. The length of the individual course of treatment is variable and is often linked to signs of resolution of the inflammatory process (eg, lack of fever for >24-48 h, return of the WBC count to reference range levels).

Some patients demonstrate persistent signs of inflammation without a defined infectious focus. In these patients, continued broad-spectrum antibiotic therapy may be more harmful than beneficial (eg, emergence of resistant organisms, *C difficile* colitis), and a trial of antibiotic therapy cessation under close surveillance may be warranted.

Finally, realizing that systemic antibiotics alone are seldom sufficient to treat intra-abdominal abscesses is important, and adequate drainage of the abscess is of paramount importance. For most of the commonly used antibiotics, abscess fluid antibiotic levels are generally below the minimum inhibitory concentration-90 (MIC90) for *B fragilis* and *E coli*, and repeated dosing or high-dose therapy does not improve penetration significantly.

### **Nonoperative drainage**

Today, abundant literature documents the safety and efficacy of ultrasound- and CT-guided percutaneous drainage of abdominal and extraperitoneal abscesses.

The success of percutaneous drainage is generally defined as effective source control with avoidance of surgical therapy. In some instances, success also includes the ability to delay surgery until the acute process and sepsis are resolved and a definitive procedure can be performed under elective circumstances.

When considering primary percutaneous management of intra-abdominal abscesses, clearly establishing the etiology, location, and morphology of the abscess prior to drainage is important; evaluate for the presence of ongoing enteric leak or fistula formation. With proper indication, most studies have reported success rates of greater than 80% (range 33-100%) for drainage of localized nonoculated abscesses; however, the success rates depend to some degree on the underlying pathology. In these studies, no significant differences were found between operative and primary

nonoperative management with regard to the overall morbidity or length of hospital stay (mean duration of drainage 8.5 d).

Common reasons for failure of primary nonoperative management include enteric fistula (eg, anastomotic dehiscence), pancreatic involvement, infected clot, and multiple or multiloculated abscesses. Procedure-related significant complications are reported to occur in less than 10% of cases (range 5-27%), with less than a 1% attributable mortality rate with experienced physicians.

In peritoneal abscess formation caused by subacute bowel perforation (eg, diverticulitis, Crohn disease, appendicitis), primary percutaneous management with percutaneous drainage was successful in most patients. Patients with Crohn disease whose abscesses were drained percutaneously had significantly fewer associated fistulae. Failure in these patients was related to preexisting fistulization and extensive stricture formation.

Concerns regarding the transgression of small or large bowel with drainage catheters in deep abscesses or ileus have been addressed in animal studies, which have found no increase in abscess formation, independent of whether catheters remained for 5 days or longer. Similar data are not available for human patients.

In summary, percutaneous and surgical drainage should not be considered competitive but rather complementary. If an abscess is accessible to percutaneous drainage and the underlying visceral organ pathology does not clearly require an operative approach, percutaneous drainage can be used safely and effectively as the primary treatment modality. Closely monitoring the clinical progress of these patients is important. Improvement should be observed in less than 24-48 hours. With lack of improvement, patients must be reevaluated aggressively (eg, repeat CT scan) and the therapeutic strategy should be altered accordingly.

### **Surgical Therapy**

Surgery remains an important therapeutic modality for all cases of peritoneal infection. Any operation should address the first 2 principles of the treatment of intra-peritoneal infections: early and definitive source control and elimination of bacteria and toxins from the abdominal cavity. The issue of timing and adequacy of surgical source control is paramount because an improper, untimely, or incorrect operation may have an overwhelmingly negative effect on outcome (compared to medical therapy).

The operative approach is directed by the underlying disease process and the type and severity of the intra-abdominal infection. The surgeon should always strive to arrive at a specific diagnosis and delineate the intra-abdominal anatomy as accurately as possible prior to the operation. However, in severe abdominal sepsis, a delay of operative management may lead to a significantly higher need for reoperations and overall worse outcomes; early exploration (ie, prior to completion of diagnostic studies) may be indicated.

Among the causes of peritonitis, pancreatitis is unique in several ways. Patients may present with significant abdominal symptoms and a severe systemic inflammatory response, yet they may have no clear organ-specific indications for emergent exploration. Not all cases of severe (ie, necrotizing) pancreatitis and peripancreatic fluid collections are associated with a superinfection.

These patients may be served best by a period of 12-24 hours of observation and intensive medical support. Deterioration of the patient's clinical status or development of organ-specific indications (eg, intra-abdominal bleed, gas-forming infection of the pancreas) should lead to prompt operation. Percutaneous treatment is reserved for the management of defined peripancreatic fluid collections in stable patients. Pancreatic abscess or infected pancreatic necrosis generally should be treated with surgical debridement and repeated exploration. If an anastomotic dehiscence is suspected, percutaneous drainage is of limited value, and the patient should be treated surgically.

### **Open-abdomen technique and scheduled reoperation**

In certain situations, staging the operative approach to intraperitoneal infections is appropriate. Staging may be performed as a scheduled second-look operation or through open management, with or without temporary closure (eg, mesh, VAC technique).

Second-look operations may be used in a damage control fashion. In these cases, the patient at initial operation is severely ill and unstable from septic shock or coagulopathy (eg, mediator liberation, disseminated intravascular coagulation). The goal of the initial operation is to provide preliminary drainage and to remove obviously necrotic tissue. Then, the patient is resuscitated and stabilized in an ICU setting for 24-36 hours and returned to the operating room for a more definitive drainage and source control.

In conditions related to bowel ischemia, the initial operation aims to remove all frankly devitalized bowel. The second-look operation serves to reevaluate for further demarcation and decision-making regarding reanastomosis or diversion.

In severe peritonitis, particularly with extensive retroperitoneal involvement (eg, necrotizing pancreatitis), open treatment with repeat reexploration, debridement, and intraperitoneal lavage has been shown to be effective.

Temporary closure of the abdomen to prevent herniation and contamination from the outside of the abdominal contents can be achieved using gauze and large, impermeable, self-adhesive membrane dressings, mesh (eg, Vicryl, Dexon), nonabsorbable mesh (eg, GORE-TEX, polypropylene) with or without zipper or Velcrolike closure devices, and vacuum-assisted closure (VAC) devices (see Table 3). Advantages of this management strategy include avoidance of abdominal compartment syndrome (ACS) and easy access for reexploration. The disadvantages include significant disruption of respiratory mechanics and potential contamination of the abdomen with nosocomial pathogens.

Table 3. Open Abdomen Technique: Options for Temporary and Permanent Closure

Closure Technique	Description	Advantages	Disadvantages
Self-adhesive impermeable membranes	Abdominal dressing with gauze and coverage of the entire wound with impermeable membrane with and without placement of drains between the layers	Inexpensive Easy application	Difficult to maintain seal Potentially large volume losses Fistula formation
Vicryl or Dexon mesh	Suturing of the mesh to the fascial edges; different options for dressing	Can be applied directly over bowel Allows for drainage of peritoneal fluid	Rapid loss of tensile strength (in the setting of infection) Potentially large volume losses Higher incidence of later ventral hernia development No reopen and close option Fistula formation
Polypropylene mesh	Suturing of the mesh to the fascial edges; different options for dressing	Good tensile strength Allows for drainage of peritoneal fluid	Risk of intestinal erosion when applied directly over bowel Potentially large volume losses High risk of mesh infection Fistula formation
GORE-TEX mesh	Suturing of the mesh to the fascial edges; different options for	Good tensile strength	Potential fluid accumulation

	dressing	Reopen and close option	underneath the mesh Limited tissue integration and granulation tissue formation over the mesh Risk of mesh infection Fistula formation
Vacuum-assisted closure device	Sponges applied over mesh and attached to controlled low-level suction	Controlled drainage of secretions Accelerated granulation tissue formation Wound debridement Can remain in place for longer than 48 hours	Cost Risk of intestinal erosion when applied directly over bowel Fistula formation
Wittmann patch	Suturing of artificial burr (ie, Velcro) to fascia, staged abdominal closure by application of controlled tension	Good tensile strength Allows for easy reexploration and eventual primary fascial closure	Fistula formation

Multiple reoperations for abdominal sepsis may be associated with a substantial inflammatory response and hypotension and thus may be potentially harmful to the patient; however, persistent necrotic or infectious abdominal foci may have worse effects on patient outcomes. Open treatment allows for thorough drainage of the intra-abdominal infection, but the specific indications are not clearly defined. Many trials lack control groups or use historical controls; outcome variables (eg, mortality) are often not specific enough, and data on resource use are limited.

To date, no conclusive data suggest the clear advantage of the open-abdomen versus closed-abdomen technique in the treatment of severe abdominal sepsis; however, in the author's experience, bowel edema and subsequent inflammatory changes limit the use of closed-abdominal technique. Secondary abdominal compartment syndrome (secondary ACS) may ensue if abdominal closure is performed before the inflammatory process has resolved.

### Laparoscopy

Laparoscopy is gaining wider acceptance in the diagnosis and treatment of abdominal infections. Initial laparoscopic examination of the abdomen can assist in determination of the etiology of peritonitis (eg, right lower quadrant pathology in female patients). Laparoscopic diagnosis and peritoneal lavage in patients with peritonitis secondary to diverticulitis without fecal peritoneal contamination has helped to avoid operation in most patients in small clinical trials. Successful laparoscopic repair of perforated gastric and duodenal ulcers has also been reported.

No definitive guidelines have been established regarding the optimal selection of patients for successful laparoscopic repair. Recent studies are investigating scoring systems (eg, APACHE II, Boey score) for patient risk stratification to better select appropriate patients for laparoscopic repair.

The treatment of perihepatic infections via laparoscopic approach has been well established in acute cholecystitis, where laparoscopic cholecystectomy has become the mainstay of therapy. More recently, primary treatment of subphrenic abscesses and laparoscopic ultrasound-assisted drainage of pyogenic liver abscesses have been performed successfully.

Individual reports also describe successful drainage of peripancreatic fluid collections and complicated intra-abdominal abscesses that are not amenable to CT scan- or ultrasound-guided percutaneous drainage.

As minimally invasive procedures continue to advance technologically, use of these approaches is likely to increase, reducing the need for the open surgical approach for peritoneal abscess drainage.

### **Preoperative Details**

Volume resuscitation and prevention of secondary organ system dysfunction are of utmost importance in the treatment of patients with intra-abdominal infections. Depending on the severity of the disease, these patients should have Foley catheters placed to monitor urine output. Use invasive hemodynamic monitoring in severely ill patients to guide volume resuscitation and inotropic support. Correct existing serum electrolyte disturbances and coagulation abnormalities as best as possible before any intervention.

Begin empiric broad-spectrum systemic antibiotic therapy as soon as the diagnosis of intra-abdominal infection is suspected and tailor therapy according to the underlying disease process and culture results. Remember that patients with peritonitis often have severe abdominal pain. Provide adequate analgesia with parenteral narcotic agents as soon as possible. In the setting of significant nausea, vomiting, or abdominal distension caused by obstruction or ileus, institute nasogastric decompression as soon as possible. Consider intubation and ventilator support early in patients with evidence of septic shock or altered mental status to prevent further decompensation.

Even if patients do not appear critically ill initially, arranging for postoperative intensive care support before the operation is often wise, particularly in patients of advanced age and those with significant comorbidities.

In patients with severe infections and certain disease processes (eg, necrotizing pancreatitis, bowel ischemia), informed consent should include the potential need for several reoperations and enteric diversion. The involved physicians and surgeon should not downplay the significant morbidities associated with abdominal sepsis when discussing these issues with the patient and/or family.

### **Intraoperative Details**

A discussion of the specific details of the operative treatment of all the potential etiologies of intraperitoneal infections is beyond the scope of this article. Certain principles always apply when performing celiotomies in patients with peritonitis. The goals of operative treatment of peritonitis are to eliminate the source of contamination, to reduce the bacterial inoculum, and to prevent recurrent or persistent sepsis.

A vertical midline incision is the incision of choice in most patients with generalized peritonitis because it allows access to the entire peritoneal cavity. In patients with localized peritonitis (eg, acute appendicitis, cholecystitis), an incision directly over the site of pathology (eg, right lower quadrant, right subcostal) is usually adequate. In patients with an unclear etiology of the peritonitis, initial diagnostic laparoscopy may be useful.

The intra-abdominal anatomy may be significantly distorted because of inflammatory masses and adhesions. Normal tissue planes and boundaries may be obliterated. The inflamed organs are often very friable, and the surgeon must exercise great caution when exploring the patient with peritoneal infection.

Hemodynamic instability may occur at any time during treatment because of bacteremia and cytokine release. Patients often demonstrate significant fluid shifts with third spacing. Swelling of the bowel, retroperitoneum, and abdominal wall may preclude safe abdominal closure after prolonged cases in patients who are severely ill.

Inflammation causes regional hyperemia, and sepsis may cause coagulation deficits and platelet dysfunction, leading to increased bleeding. Careful dissection and meticulous hemostasis are of utmost importance.

When faced with extensive abdominal inflammatory disease and septic shock, draining the infection temporarily, controlling the visceral leak quickly (eg, oversewing, enteric diversion), and deferring any

definitive repair until after the patient has recovered from the initial insult (ie, damage control operation) may be better.

One of the critical decisions in the surgical treatment of patients with severe peritonitis is regarding whether to use a closed-abdomen or open-abdomen technique. The goal of the closed-abdomen technique is to provide definitive surgical treatment at the initial operation; perform primary fascial closure and perform repeat laparotomy only when clinically indicated. The goal of the open-abdomen technique is to provide easy direct access to the affected area. Source control is achieved through repeated reoperations or open packing of the abdomen. This technique may be well suited for initial damage control in extensive peritonitis.

Also consider patients who are at high risk for development of abdominal compartment syndrome (eg, intestinal distension, extensive abdominal wall and intra-abdominal organ edema) for this technique because attempts to perform primary fascial closure under significant tension in these circumstances are associated with an increased incidence of MOF (eg, renal, respiratory), necrotizing abdominal wall infections, and mortality.

### **Postoperative Details**

Postoperatively, monitor all patients closely in the appropriate clinical setting for adequacy of volume resuscitation, resolution or persistence of sepsis, and the development of organ system failure. Appropriate systemic broad-spectrum antibiotic coverage must be continued without interruption for the appropriate time (see Medical therapy).

The patient's overall condition should improve significantly and progressively within 24-72 hours of the initial treatment (ie, resolution of the signs and symptoms of infection, mobilization of interstitial fluid). This time course may be prolonged in patients who are critically ill with significant multiple organ system dysfunction. A lack of improvement should prompt an aggressive search for a persistent or recurrent intraperitoneal or new extraperitoneal infectious focus.

Patients requiring surgical intervention for peritonitis demonstrate a significantly increased risk for surgical site infections and wound healing failure; monitor patients closely for this potential complication.

All patients who are critically ill and patients receiving prolonged antibiotic therapy are at increased risk for developing secondary opportunistic infections (eg, *C difficile* colitis, fungal infections, central venous catheter infections, ventilator-associated pneumonia); monitor patients closely for signs and symptoms of these complications.

### **Nutrition**

In general, patients with peritonitis develop some degree of gut dysfunction (eg, ileus) after exploration. Consider establishing some form of nutritional support early in the course of treatment because most patients have an insufficient enteral intake for a variable amount of time preoperatively. The existing data support that enteral nutrition is superior to parenteral hyperalimentation. If enteral feeding is contraindicated or not tolerated, parenteral nutrition should be instituted.

### **Follow-up**

After resolution of peritonitis and peritoneal abscesses, follow-up care is directed mostly by specifics of the underlying disease process and the presence or absence of chronic complications (eg, enterocutaneous fistulae). Patients with simple peritoneal infections after appendicitis or cholecystitis are usually cured and do not require long-term follow-up care. Patients with peritoneal operations for perforated peptic ulcer disease, Crohn disease, pancreatitis, and others often require lifelong medical therapy and treatment of recurrent complications.

## **Complications**

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### **Surgical site infection/dehiscence**

The incidence of surgical site infection increases with the degree of contamination; therefore, surgical site infection occurs at much higher rates after operations for peritonitis and peritoneal abscess (ie, 5-15% compared to <5% for elective abdominal operations for noninfectious etiologies). Surgical site infection may be expected if the wound is closed in the setting of gross abdominal contamination (see Table 4). Perioperative systemic antibiotics, the use of wound protector devices, and lavage of the wound at the end of therapy do not reliably prevent this complication. These wounds should be left open and be treated with wet-to-dry dressing changes several times a day or VAC dressing should be applied.

Table 4. Wound Classification and Risk for Surgical Site Infection

<b>Classification</b>	<b>Examples</b>	<b>Incidence of Surgical Site Infection (%)</b>
Clean	Elective surgery without violation of the gut or infected spaces	<2
Clean contaminated	Elective bowel surgery (prepared bowel, mechanical and antibiotic)	5-15
Contaminated	Emergent bowel surgery (unprepared bowel, minor spillage), drainage of infected spaces	15-30
Dirty	Grossly contaminated traumatic wounds, significant intestinal spillage, grossly infected and devitalized tissue (necrotizing infection)	>30

### **Impaired wound healing**

The same factors that impair clearance of the abdominal infection contribute to increased problems related to wound healing (eg, malnutrition, severe sepsis, multiple organ system dysfunction, advanced age, immunosuppression) and should be addressed aggressively. Patients with severe abdominal infections demonstrate higher incidences of fascial dehiscence and incisional hernia development, requiring later reoperation.

### **Complications related to percutaneous drainage**

Percutaneous drainage procedures carry a risk of related significant complications of less than 10% (range 5-27%) depending on the underlying pathology and abscess location. These complications include bleeding, injury, erosion, transgression of small and large bowel, fistula formation, and others. Strategies to prevent these problems include correction of coagulation problems and determination of the exact etiology, location, and anatomic relationships of the abscess. Indication for percutaneous treatment of complex abscesses and patients with a persistent enteric leak should be reviewed critically, and operative treatment should not be delayed with lack of adequate patient improvement.

### **Tertiary peritonitis**

Persistence of intra-abdominal infection (ie, tertiary peritonitis) is a complication that may occur following the treatment of primary or secondary peritonitis and peritoneal abscess. The details of this problem are described in the different sections of this article.

### **Complications related to the open-abdomen technique**

One of the complications related to treatment of severe intra-abdominal infections with the open-abdomen technique and multiple reoperations is the development of enterocutaneous fistulae. A retrospective study to assess the results of open management of the abdomen in severe bacterial

peritonitis after perforation or intestinal anastomotic disruption was performed in 67 patients. The mean number of reoperations required was 9. Fistula formation and severe bleeding occurred in 16 patients (24%). The in-hospital mortality rate was 42%. Long-term morbidity, particularly the number of abdominal wall defects, was considerable.

A recent study of trauma patients shows that morbidity due to wound complications (wound infections, abscess, and/or fistula) from the open abdomen remains high at 25%.

Enterocutaneous fistulae can lead to ongoing (potentially large) volume, protein, and electrolyte losses; inability to use the gut for nutritional support; and associated long-term complications of intravenous alimentation. Patients with small, low-output, and distal fistulae often can be fed enterally with elemental diets. A proportion of these fistulae close spontaneously as the patient's overall status and nutritional status improve.

High-output and proximal fistulae often require a delayed surgical repair. Optimal timing of this repair is critical. Initial inflammatory adhesions and dense scar formation may make safe reexploration impossible. Maturation of the scar tissue occurs over 6-12 months. Close observation of the patient's overall condition and nutritional status is important during that time. Deterioration of the patient's condition may force an earlier reoperation.

For an extended time after operations for intra-abdominal infections, patients are at a several-fold increased risk of developing bowel obstruction related to intra-abdominal scar formation. While in some patients this obstruction may be partial and reversible and may improve with cessation of enteral intake and gastric decompression, most patients require reoperation over time.

**Complications related to abdominal compartment syndrome**

ACS is a well-recognized disease entity related to acutely increased abdominal pressure (ie, intra-abdominal hypertension [IAH]) and is associated with the development of multiple organ dysfunction.

Elevated intra-abdominal pressure adversely impacts pulmonary, cardiovascular, renal, splanchnic, musculoskeletal, integumentary, and central nervous system physiology. The combination of IAH and disordered physiology results in a clinical syndrome with significant morbidity and mortality. ACS can occur in a variety of surgical conditions, particularly those with major life-threatening hemorrhage, massive volume resuscitation, prolonged operation times, and coagulopathy. In patients who are severely traumatized, the incidence of ACS is reported to be as high as 15% after damage control laparotomies. The exact incidence of ACS in patients undergoing surgery for intra-abdominal infections and peritonitis is unknown. However, closure of the abdomen under tension at completion of the laparotomy is clearly associated with a much higher risk for ACS postoperatively (visceral edema and accumulation of peritoneal fluid).

The intra-abdominal pressure can be easily assessed by measuring the urine bladder pressure, which correlates well with the actual intra-abdominal pressure. Increasing bladder pressure measurements suggest increased risk for ACS and warrant more aggressive intervention (Table 5).

Table 5. Abdominal Compartment Syndrome Grading System by Transvesical (Urinary Bladder) Pressure Measurement

Grade	Bladder Pressure (mm Hg)	Therapeutic Recommendations
I	10-15	Maintain normovolemia. Observe clinically.
II	16-25	Hypervolemic resuscitation is indicated. Closely monitor bladder pressures and renal function.
III	26-35	Abdominal decompression is indicated.

IV	>35	Urgent abdominal decompression and exploration for potential surgical causes is indicated
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A more recent study determined that abdominal perfusion pressure, defined as mean arterial pressure minus intra-abdominal pressure, was statistically superior to both mean arterial pressure and intravesicular pressure in predicting patient survival from IAH and ACS. Multiple regression analysis demonstrated that abdominal perfusion pressure was also superior to other common resuscitation endpoints, including arterial pH, base deficit, arterial lactate, and hourly urinary output.

The onset of ACS requires prompt recognition and appropriately timed and staged intervention to optimize outcome. Surgical decompression of the abdomen by means of a laparotomy is the treatment of choice for ACS. Urgent laparotomy can be lifesaving in some cases. However, no single threshold of abdominal pressure can be applied universally. The best therapeutic option is decompression of the abdomen surgically if the intravesicular pressure is 25 mm Hg or higher in patients with refractory hypotension, acute renal failure, or respiratory failure caused by abdominal distension. Recent studies also suggest that abdominal decompression for ACS can be accomplished with laparoscopy in patients with increased intra-abdominal pressure postoperatively that is related to the accumulation of tense ascites and not intraperitoneal hemorrhage.

#### **Complications related to enteric insufficiency**

Extensive initial (gastrointestinal) disease, chronic recurrent infections, and associated reoperations may lead to enteric insufficiency because of short gut, pancreatic insufficiency, or hepatic dysfunction. Treatment of these problems can be quite challenging and can require a multispecialty approach to optimize gastrointestinal function and nutritional status.

## **Outcome and Prognosis**

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### **Spontaneous bacterial peritonitis**

The overall mortality rate of patients with SBP may exceed 30% if diagnosis and treatment are delayed, but the mortality rate is less than 10% in fairly well-compensated patients with early therapy. As many as 70% of patients who survive an episode of SBP have a recurrent episode within 1 year, and, for these patients, the mortality rate approaches 50%. Some studies suggest that the recurrence rate of SBP may be decreased to less than 20% with long-term antibiotic prophylaxis (eg, quinolones, trimethoprim-sulfamethoxazole); however, whether this improves long-term survival without liver transplantation is unclear.

### **Secondary peritonitis and peritoneal abscess**

Treatment success of peritoneal infections is defined as adequate source control with resolution of sepsis and clearance of all residual intra-abdominal infection. With percutaneous treatment, the definition of success includes the avoidance of further operative intervention and, in some cases, the delay of surgery until after resolution of the initial sepsis. Over the past decade, the combination of better antibiotic therapy, more aggressive intensive care, and earlier diagnosis and therapy with a combination of operative and percutaneous techniques have led to a significant reduction in morbidity and mortality related to intra-abdominal sepsis.

Uncomplicated SP and simple abscesses carry a mortality rate of less than 5%, but this rate may increase to greater than 30-50% in severe infections. The overall mortality rate related to intra-abdominal abscess formation is less than 10-20%. Factors that independently predict worse outcomes include advanced age, malnutrition, presence of cancer, a high APACHE II score on presentation, preoperative organ dysfunction, the presence of complex abscesses, and failure to improve in less than 24-72 hours after adequate therapy.

In severe intra-abdominal infections and peritonitis, the mortality rate may increase to greater than 30-50%. The concurrent development of sepsis, SIRS, and MOF can increase the mortality rate to

greater than 70%, and, in these patients, more than 80% of deaths occur with an active infection present.

Several scoring systems (eg, APACHE II, SIRS, multiple organ dysfunction syndrome [MODS], Mannheim peritonitis index) have been developed to assess the clinical prognosis of patients with peritonitis. Most of these scores rely on certain host criteria, systemic signs of sepsis, and complications related to organ failure. Although valuable for comparing patient cohorts and institutions, these scores have limited value in the specific day-to-day clinical decision-making process for any given patient. In general, the mortality rate is less than 5% with an APACHE II of less than 15 and rises to greater than 40% with scores above 15. Rising APACHE II scores on days 3 and 7 are associated with an increase of mortality rates to greater than 90%, whereas falling scores predict mortality rates of less than 20%.

The mortality rate without organ failure generally is less than 5% but may rise to greater than 90% with quadruple organ failure. A delay of more than 2-4 days of either medical therapy or surgical therapy has been clearly associated with increased complication rates, the development of tertiary peritonitis, the need for reoperation, multiple organ system dysfunction, and death.

Outcomes are worse in patients requiring emergent reoperations for persistent or recurrent infections (30-50% increase in the mortality rate); however, patients undergoing early planned second-look operations do not demonstrate this trend.

Persistent infection, recovery of enterococci, and multidrug-resistant gram-negative organisms, as well as fungal infection, are related to worse outcomes and recurrent complications.

Patients older than 65 years have a 3-fold increased risk of developing generalized peritonitis and sepsis from gangrenous or perforated appendicitis and perforated diverticulitis than younger patients and are 3 times more likely to die from these disease processes. Older patients with perforated diverticulitis are 3 times more likely than younger patients to have generalized rather than localized (ie, pericolic, pelvic) peritonitis. These findings are consistent with the hypothesis that the biologic features of peritonitis differ in elderly persons, who are more likely to present with an advanced or more severe process than younger patients with peritonitis.

Overall, studies suggest that host-related factors are more significant than the type and source of infection with regard to the prognosis in intra-abdominal infections.

## **Future and Controversies**

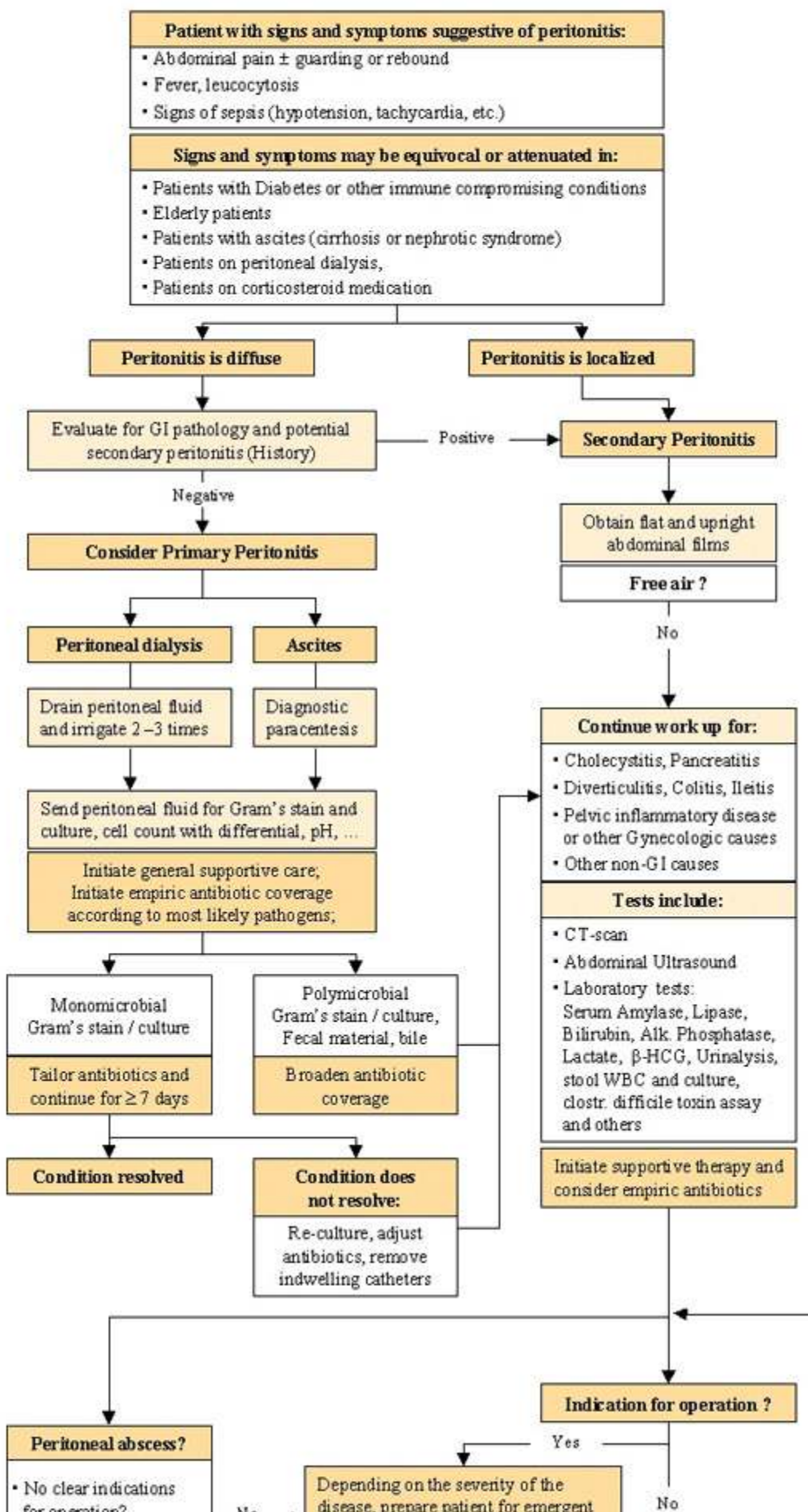
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Laparoscopy is gaining wider acceptance in the diagnosis and treatment of abdominal infections (see Laparoscopy in Surgical therapy). However, no definitive guidelines have been established regarding the optimal selection of patients for successful laparoscopic repair. As minimally invasive procedures continue to advance technologically, use of these approaches is likely to increase, reducing the need for the open surgical approach for peritoneal abscess drainage.

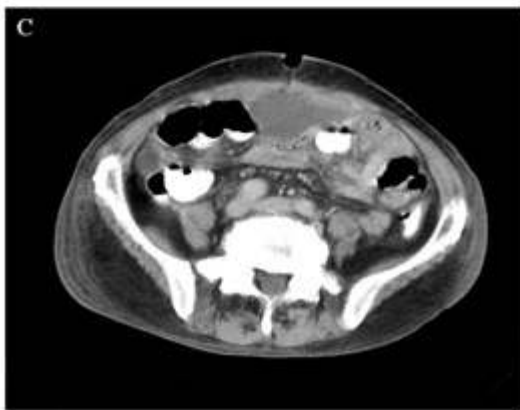
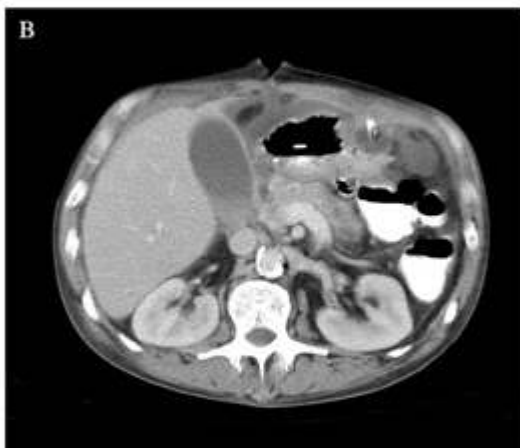
## **Multimedia**

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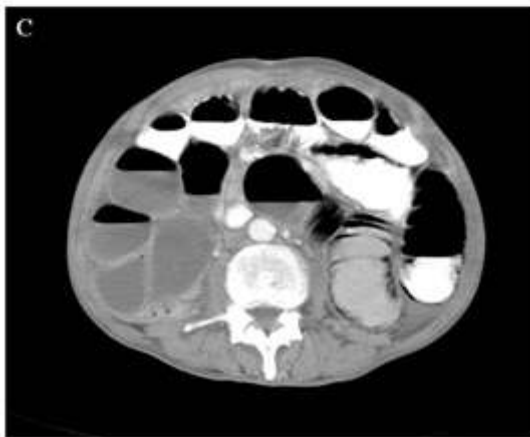
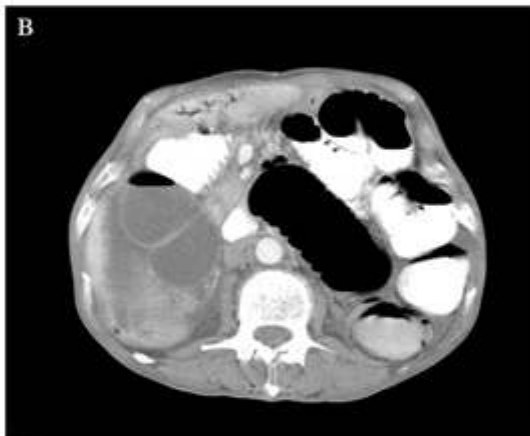
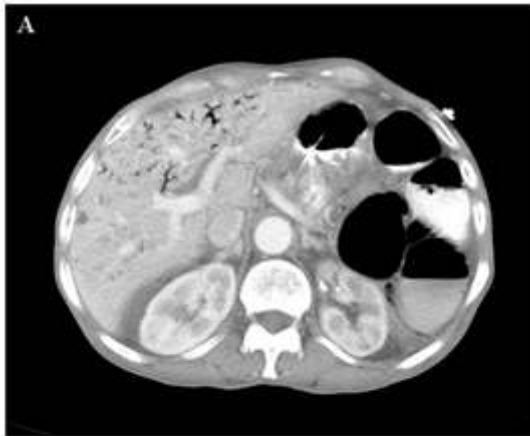
**Diagnostic and therapeutic approach to peritonitis and peritoneal abscess:**



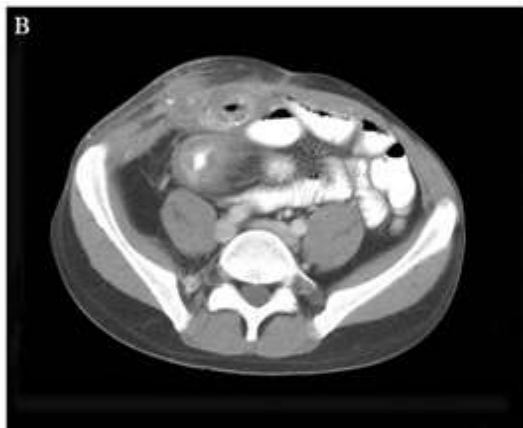
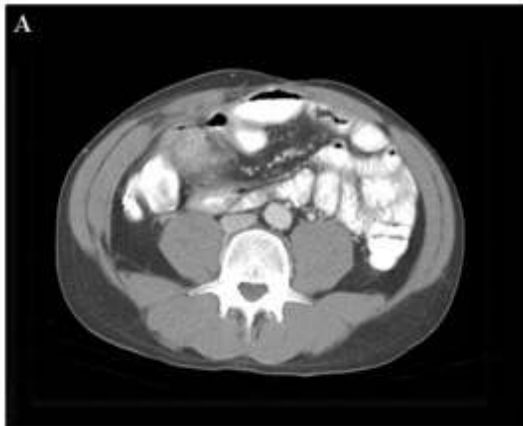
**Media file 1: Peritonitis and abdominal sepsis. Diagnostic and therapeutic approach to peritonitis and peritoneal abscess.**



**Media file 2: Peritonitis and abdominal sepsis. A 48-year-old man underwent suprapubic laparotomy, right hemicolectomy, and gastroduodenal resection for right colon cancer invading the first portion of the duodenum. He developed abdominal pain and distention. CT scan confirmed anastomotic dehiscence. Figure A shows a contrast enhanced scan of the abdomen and pelvis that reveals multiple fluid collections, perihepatic ascites, and mild periportal edema. A collection of fluid containing an air-fluid level is visible anterior to the left lobe of the liver. A second collection is anterior to the splenic flexure of the colon. In figure B, a third fluid collection is present in the inferior aspect of the lesser space and in the transverse mesocolon. Figure C shows the pelvis with a collection of free fluid in the rectovesical pouch.**



**Media file 3: Peritonitis and abdominal sepsis. A 78-year-old man was admitted with a history of prior surgery for small bowel obstruction and worsening abdominal pain, distended abdomen, nausea, and obstipation. In figure A, a marked amount of portal venous gas within the liver, mesenteric venous gas, and pneumatosis intestinalis are consistent with ischemic small intestine. The superior mesenteric artery appears patent. The liver has a nodular contour consistent with cirrhosis. In figures B and C, markedly distended loops of small intestine containing fluid and air-fluid levels are consistent with a small bowel obstruction. No focal fluid collections are identified.**



**Media file 4: Peritonitis and abdominal sepsis.** A 35-year-old man with a history of Crohn disease presented with pain and swelling in the right abdomen. In figure A, a thickened loop of terminal ileum is evident adherent to the right anterior abdominal wall. In figure B, the right anterior abdominal wall is markedly thickened and edematous with adjacent inflamed terminal ileum. In figure C, a right lower quadrant abdominal wall abscess and enteric fistula is observed and confirmed by the presence of enteral contrast in the abdominal wall.

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## Keywords

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peritoneal abscess, perforation of the bowel, perforated ulcer, perforated gall bladder, lacerated liver, infected fallopian tube, ruptured ovarian cyst, abdominal pain, spontaneous bacterial peritonitis, SBP, liver disease, liver cirrhosis, tuberculous peritonitis, TP, spontaneous peritonitis, SP, primary peritonitis, secondary peritonitis, tertiary peritonitis

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