Intra-abdominal Infections

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KEYWORDS

• Antibiotics • Complicated intra-abdominal infections • Damage control surgery
• Resuscitation • Source control • Systemic inflammatory response syndrome
• Uncomplicated intra-abdominal infections

KEY POINTS

- Intra-abdominal infections (IAI) should be suspected in a patient manifesting a systemic inflammatory response syndrome (SIRS) and gastrointestinal dysfunction.
- Uncomplicated IAI are predominantly isolated to an organ and do not involve gastrointestinal disruption, whereas complicated IAI are usually diffuse peritoneal processes that may include disruption of the gastrointestinal tract.
- Adequate treatment of IAI requires early diagnosis combined with resuscitation, appropriate antibiotic therapy, and adequate drainage/debridement of on-going infection or leaking gastrointestinal contents (ie, source control, SC).
- Appropriate and timely empiric antibiotic coverage is imperative because inappropriate or delayed coverage increases morbidity and mortality that cannot be reversed if subsequent appropriate antibiotics are added later.
- In general, β-lactam/β-lactamase antibiotics will provide adequate empiric coverage for low-risk patients; however, high-risk patients are at risk for more resistant microbiologic flora, and empiric coverage should be driven by individual hospital or unit antibiograms.
- Percutaneous drainage is preferred in stable patients with an isolated, anatomically amenable source; surgical debridement (open or laparoscopically) remains the mainstay for failed SC.

INTRODUCTION

Intra-abdominal infections (IAI) represent diverse disease processes and therapies; however, earlier diagnosis with readily available CT imaging, advanced therapeutic techniques of interventional radiology, improvement of antibiotic efficacy, and evolving critical care medicine have all combined to improve patient outcomes.

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IAI are divided into uncomplicated and complicated types. Uncomplicated IAI affect a single organ and do not spread to the peritoneum. In these cases, there is no anatomic disruption of the gastrointestinal tract. Complicated IAI describes an extension of the infection into the peritoneal space. It may be localized, as in the case of an intra-abdominal abscess. For the insult that is not contained, diffuse peritonitis may ensue.[1,2] The resultant physiologic response may develop into a systemic inflammatory response syndrome (SIRS) (**Table 1**).[3,4]

In addition to defining type of infection, patient stratification serves as an important guide for treatment and will assist with initial resuscitation, treatment options, and specifically, antimicrobial therapy. Patients are divided into low-risk and high-risk categories that take into account the patient's history, the type of infection, and the resulting physiologic derangements.

Low-risk patients typically have community-acquired infections of mild to moderate severity (perforated appendicitis or diverticulitis). The underlying physiologic status in these patients is not compromised. High-risk patients, on the other hand, are used to define patients who are at risk for multi-drug-resistant organisms,[5–7] failure of source control (SC),[8] and ultimately, increased mortality.[1,5,8–10] Predetermined patient-specific and disease-specific factors act together to determine patient morbidity and mortality (**Box 1**).[6,8,10]

PATHOPHYSIOLOGY

The inner abdomen is lined with a layer of tissue (peritoneum) innervated by the somatic nervous system. Infection begins, followed by inflammation by mast cell degranulation with subsequent increased vascular permeability. This increased vascular permeability causes an influx of complement factors and neutrophils that are responsible for both direct bacterial opsonization and release of cytokines to propagate the host response. This process may be localized to an abscess when the inflammation, chemotaxis, and fibrin formation may form sufficient physical barriers.[10]

Intra-abdominal inflammation may lead to a diffuse paralytic ileus, distention, obstipation, and vomiting.[4] When the host ability to contain the infection is overcome, the infection progresses to diffuse peritonitis. Systemic response to the release of cytokines will lead to a pro-inflammatory state, systemic vasodilation, hypotension, and myocardial depression, manifested clinically as severe sepsis and subsequently as septic shock.[3,10]

DIAGNOSIS

Diagnosis of IAI should be suspected in patients with SIRS and gastrointestinal dysfunction. Essential components of the history include any recent surgeries, and

Table 1
Systemic inflammatory response syndrome criteria[a]

Finding	Value
Temperature	<36°C or >38°C
Heart rate	>90/min
Respiratory rate	>20/min or $Paco_2$ <32 mm Hg
WBC	$<4 \times 10^9$/L, $>12 \times 10^9$/L, or 10% bands

Abbreviations: $Paco_2$, partial pressure of carbon dioxide; WBC, white blood cell count.
[a] Defined as having at least 2 of the above.

Box 1
Characteristics of high-risk intra-abdominal infection patients

Patient-specific factors

- Advanced age (>70 y)
- Immunosuppression
  - Poor nutritional status
  - Corticosteroid therapy
  - Organ transplantation
- Presence of malignancy
- Pre-existing chronic conditions
  - Liver disease
  - Renal disease

Disease-specific factors

- High APACHE II score (>15)
- Health care–associated infection
- Delay in initial intervention (>24 h)
- Inability to obtain source control

the presence of vomiting, diarrhea, and obstipation. Although physical examination findings are notoriously nonspecific, particular findings may give insight.[11] Pain out of proportion to examination is classically associated with acute mesenteric ischemia. Inguinal and umbilical hernia examinations are important to rule out the source of obstruction or incarceration. Although minimally invasive surgery is increasingly common, abdominal scars are always important to note.

Laboratory workup begins with the assessment of a complete blood count and serum electrolytes. Liver function tests, amylase, and lipase may be added if clinical concern includes hepatobiliary or pancreatic pathologic abnormality. In patients with SIRS and a concern for sepsis, further assessment of end-organ perfusion or signs of oxygen debt should be assessed (ie, serum lactic acid, superior vena caval/mixed venous oxygenation saturations, arterial blood gas for base deficit).[3,10]

Initial radiographic imaging should include a CT scan with oral and intravenous (IV) contrast to maximize sensitivity and specificity.[4,8,11,12] Oral contrast helps to differentiate bowel loops from adjacent fluid collections and may help guide subsequent drainage procedures.[4] IV contrast helps delineate inflammation, identify hemorrhage, and visualize abscess walls. CT is useful in identifying small areas of free intra-abdominal air (pneumoperitoneum) associated with hollow viscous perforation, air in the biliary tree, and air within the intestinal walls (pneumotosis intestinalis). The exception to this is if biliary pathologic abnormality is suspected (right upper quadrant pain, nausea, and vomiting), then right upper quadrant ultrasound is the higher yield.[11]

Microbiologic diagnosis is not important in community-acquired IAI because empiric antibiotic therapy is initiated based on clinical impression and risk factors.[8,10,12] In the case of high-risk patients, blood and intra-abdominal cultures are necessary to guide antimicrobial therapy due to the higher risk for multi-drug-resistant organisms.

One of the most urgent clinical circumstances is the patient who presents with peritonitis (abdominal rigidity, guarding, and rebound tenderness). These signs are concerning for pending hemodynamic collapse, and urgent evaluation and disposition are necessary. Early hemodynamic assessment is a priority; if adequate (systolic blood pressure >90 mm Hg), there may be time for further workup. On the other hand, unstable patients (systolic blood pressure <90 mm Hg) and the need for vasopressor support indicate the need emergent laparotomy for diagnostic and therapeutic purposes with the understanding that the risk of mortality is higher than in a stable patient.[3]

TREATMENT

The principles of treatment require simultaneous resuscitation, SC, and antimicrobial therapy. If not aggressively managed, IAI may progress to severe sepsis, septic shock, and death.[13]

Resuscitation

Intravascular volume depletion should be expected in patients with IAI. A thorough history and physical examination may aid with guiding resuscitation. Severe nausea and vomiting will cause metabolic alkalosis with relative hypokalemia, whereas a high-volume diarrhea will cause a nonanion gap metabolic acidosis. With peritonitis, the cytokine inflammation causes fluid sequestration both locally and systemically, which may be profound, further contributing to intravascular volume depletion. Fluid accumulation is noted with an ileus by both bowel wall edema and ascites. In addition, patients with fever and tachypnea have more than 700 mL/d of excess fluid loss.[10] These abnormal fluid shifts place patients at risk for intravascular volume depletion, hypotension, and decreased end-organ perfusion. With an increasing severity of illness, more invasive hemodynamic monitoring is indicated (central venous and arterial pressure catheter placement and monitoring).

It has been learned from the Surviving Sepsis Campaign (SSC) that fluid resuscitation should be initiated immediately after the diagnosis of sepsis is suspected.[3] The strategy of early goal-directed therapy has been shown to decrease mortality.[14]

Source Control

SC is a fundamental surgical principle and is defined as the ability to effectively eradicate infection (ie, purulent fluid or tissue) and control leakage (ie, drainage of on-going enteric contamination) by whatever means necessary.[4,6,10] Although resuscitation and treatment with antibiotics are central to the treatment of IAI, SC is paramount. It may be accomplished in a variety of ways, ranging from percutaneous drainage to repeat operations. Timing of SC is generally undertaken as early as safely possible. Although the goal is to remove the driver of the inflammatory response, patients may be in a delicate physiologic state that puts them at high risk for immediate intervention. Nonetheless, SC is directly related to outcome, and inability to provide adequate SC is associated with increased mortality.[8,9] The exception to this rule is acute pancreatitis and pancreatic necrosis, which does not benefit from early SC (see later discussion).

In general, the least invasive procedure that is safely able to eradicate the infection is preferred. Percutaneous image-guided drainage is preferred for isolated IAI that are anatomically amenable to drainage. Surgical debridement, whether laparoscopic or open, remains the mainstay of therapy for failed percutaneous control.

Surgical intervention is required for peritonitis with hemodynamic instability, evidence of uncontrolled, on-going contamination, and/or if bowel necrosis is suspected.[4,5,8,10]

Patients may present with extreme physiologic derangements and multiorgan system failure that requires ICU resuscitation.[10,15] Unfortunately, resuscitation likely will not be successful until SC is achieved. When urgent operative intervention is indicated, intraoperative resuscitation must be continued and this requires close collaboration with anesthesia providers.[8] In these extreme circumstances, one option is to perform damage control surgery (DCS). DSC is a specific type of temporary SC originally described in the trauma setting.[15] Similar concepts are now being applied to the emergency general surgery patient who meets criteria (**Box 2**).

In these DCS patients, the priority is control of on-going contamination that directly decreases mortality.[8] In times of severe contamination and inflammation, definitive surgical treatment may not be safe; the priority is then to perform proximal diversion (if possible) and/or to allow adequate external drainage of any on-going leakage (ie, drains, sub-atmospheric/vacuum pressure dressings).

Options for temporary abdominal closure include a conventional dressing, a subatmospheric pressure dressing, or skin closure alone. The decision to not definitely close someone's abdomen should not be taken lightly. Nonclosure of the fascia is not without complications and puts patients at risk for multiple operations, prolonged intensive care unit stay, infection, fistula formation, and failure of abdominal closure. These complications may potentially negate the beneficial effects of this option if overused.[15]

Relaparotomy should be reserved for patients with specific abnormalities otherwise not recommended.[8] Planned relaparotomy as a management option was thought to be beneficial to allow for complete drainage of intra-abdominal contamination and early detection of anastomotic leaks. A randomized trial found that on-demand re-laparotomy did not have a higher risk of peritonitis-related morbidity, whereas the planned relaparotomy group had an increased use of health care services, costs, and laparotomies.[5,8,16]

Antibiotics

Although secondary to adequate SC, appropriate and timely empiric antibiotic coverage is imperative. Inappropriate coverage increases hospital stay, postoperative abscesses,[10] and mortality that cannot be reversed if subsequent appropriate antibiotics are added later in the clinical course.[2,4] In severe sepsis, appropriate coverage should be started within 1 hour as recommended by the SSC.[3,10] Just as important is the appropriate discontinuation of antibiotics (Antibiotic Stewardship). Unnecessary antibiotic use has contributed to the emergence and spread of drug-resistant microorganisms.

Box 2
Clinical indications for damage control surgery

- Hemodynamic instability
- On-going contamination or need for further debridement
- Tissue/organ ischemia
- Loss of abdominal domain
- Development of/risk for abdominal compartment syndrome

Initial empiric antibiotic coverage requires both knowledge of normal enteric flora and assessment of potential risk factors. In general, proximal small bowel contains enteric gram-positive streptococcus and gram-negative bacteria, whereas anaerobic bacteria populate the distal ileum and colon (**Table 2**).[8,10,12]

To help guide the clinician, guidelines have been published that standardize the diagnosis and management of IAI.[8,12] The first guideline represents a consensus between the Surgical Infection Society and the Infectious Disease Society of America. Second, worldwide guidelines have been published by the World Society of Emergency Surgery. As previously mentioned, patients with IAI are divided into low-risk and high-risk categories to stratify their risk for developing complicated infections. In general, β-lactam/β-lactamase (penicillin, cephalosporins, carbapenems, monobactams) antibiotics will provide adequate empiric coverage for low-risk patients (**Table 3**).[8,12]

High-risk patients, on the other hand, are at risk for more resistant microbiologic flora. Specifically, this includes gram-negative *Pseudomonas aeruginosa* and *Acinetobacter* species, extended spectrum β-lactamase producing *Klebsiella* species, *Escherichia coli*, *Enterobacter* species, *Proteus* species, methicillin-resistant *Staphylococcus aureus* (MRSA), enterococci, and *Candida* species. Empiric therapies are institution-specific and should be adjusted for individual hospital/unit antibiograms (**Table 4**).

Routine coverage for *Enterococcus faecalis* is only recommended if IAI is health care–associated, if the patient had previously received cephalosporins, if the patient has a history of valvular heart disease/prosthetics, or if the patient is elderly or critically ill. *E faecalis* is seen with frequency in patients with liver disease and infections with a hepatobiliary source.[7] Antibiotics that will provide adequate coverage include ampicillin, piperacillin-tazobactam, and vancomycin. Fungal coverage is necessary in the presence of a nosocomial infection, a critically ill community-acquired infection, a patient on pharmacologic immunosuppression, or isolation of fungi from normally sterile sites. Also, coverage should be considered if there was recent exposure to broad-spectrum antimicrobials. Fluconazole is recommended unless critically ill; then echinocandin is recommended as first-line treatment. MRSA coverage is recommended

Table 2
Normal enteric flora by gastrointestinal region

Stomach and duodenum	Streptococcus Lactobacillus
Biliary	*E coli* *Klebsiella* sp. *Enterococcus* sp. (±)
Small intestines	*E coli* *Klebsiella* sp. *Enterococcus* sp. *Diptheroid* sp. *Enterococci* sp.
Distal ileum and colon	*Bacteroides fragilis* *Clostridium* sp. *Enterobacter* sp. *Enterococcus* sp. *E coli* *Klebsiella* sp. *Peptostreptococcus* sp.

Table 3
Empiric antibiotic recommendations for low-risk intra-abdominal infection patients

	Low Risk	High Risk
Single agent	Cefoxitin Ertapenem Moxifloxacin Tigecycline Ticarcillin-clavulanic acid	Imipenem-cilastin Meropenem Doripenem Pipercillin-tazobactam
Combination (with metronidazole)	Cefazolin Cefuroxime Ceftriaxone Cefotaxime Ciprofloxacin Levofloxacin	Cefepime Ceftazidime Ciprofloxacin Levofloxacin

Data from Lopez N, Kobayashi L, Coimbra R. A comprehensive review of abdominal infections. World J Emerg Surg 2011;6:7; and Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med 2001;345:1368–77.

when there is a known history of MRSA, a hospital-acquired infection, or recent, significant antibiotic exposure. Vancomycin is then recommended as treatment.[8]

Duration of antibiotic treatment is an on-going point of discussion in the literature and important to clinically reassess daily.[4,10] General consensus recommendations are a course of 4 to 7 days. Prompt discontinuation of treatment is encouraged if

Table 4
Empiric antibiotic recommendations for high-risk IAI patients

Local Organism	Carbapenems	Piperacillin-Tazobactam	Ceftazidime or Cefepime (+Metronidazole)	Aminoglycoside	Vancomycin
<20% resistant *P aeruginosa* ESBL-producing *Enterobacter* sp. *Acinetobacter* sp. or other MDR GNR	+	+	+		
ESBL-producing *Enterobacter* sp.	+	+		+	
>20% of *P aeruginosa* resistant to ceftazidime	+	+		+	
MRSA					+

Abbreviations: ESBL, extended spectrum β-lactamase; MDR GNR, multi-drug-resistant gram-negative rod; MRSA, methacillin-resistant *Staphlococcus aureus*.

Data from Lopez N, Kobayashi L, Coimbra R. A Comprehensive review of abdominal infections. World J Emerg Surg 2011;6:7; and Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med 2001;345:1368–77.

patients show clinical response because longer treatment has not been associated with improved outcome.[8] Historically, studies have suggested antibiotics should be continued until the patient has resolved their leukocytosis or fever and is tolerating oral diet, but that may not be necessary.[10] Transition to oral antibiotics may be initiated when the patient is taking oral diet without an increased risk of treatment failure.[8,17]

If the patient continues to show signs of fever, leukocytosis, or delayed gastrointestinal function after 7 days, a persistent infection should be suspected and reimaging should be completed to search for on-going infection.[8] In this situation, it is recommended to continue antibiotics and strongly consider a change in covering antibiotic-resistant microorganisms.

SPECIFIC CONSIDERATIONS AND CONTROVERSIES

Appendicitis

Acute appendicitis is the most common source of infection in community-acquired IAI.[4] Antibiotic coverage depends on extent of disease. Prompt discontinuation of antibiotics is recommended after appendectomy if surgery reveals no perforation.[10]

Source Control

In acute appendicitis, nonoperative management has been suggested as an alternative to traditional treatment of appendectomy. Meta-analyses demonstrate antibiotic treatment alone was associated with decreased complications, less pain, and a shorter sick leave. Ultimately, antibiotics were found to have only a treatment success rate of 63% at 1 year and thus remain inferior to surgical management.[10,18,19]

Both the open laparoscopic approach and the laparoscopic approach to appendectomy continue to be accepted treatment modalities and have been extensively compared in the literature. The open approach has been associated with less cost, shorter operative times, and decreased risk of IAI in multiple studies. Alternatively, the laparoscopic approach has been found to have fewer surgical site infections, less pain, shorter hospital stays, and more rapid return to normal activity. For complicated or perforated appendicitis, the laparoscopic approach has been shown to reduce overall mortality.[10,20–22]

Patients who present with a phlegmon or periappendiceal abscess had traditionally required an operation for SC. When patients present during the peak of intra-abdominal inflammation, the safety of surgical intervention comes into question. Treatment with antibiotics and percutaneous drainage, if amenable, have been found to be associated with fewer complications and shorter hospital stay when compared with immediate appendectomy.[10,23] Treatment of periappendiceal abscesses with antibiotics alone has also been suggested but compared with percutaneous drainage has a significant recurrence rate and is therefore not recommended.[24]

For those patients who were treated with percutaneous drainage and antibiotics, generally an interval appendectomy was recommended owing to the variable rates of recurrence (5%–37%).[10,23,25] There is not enough evidence to firmly support interval appendectomy and, in fact, interval appendectomy may be unnecessary in 75% to 90% of cases.[12] Advocates for interval appendectomy argue that there is a significant risk of recurrence and, if no surgical intervention is undertaken, there is a risk of missing a diagnosis of cancer or Crohn disease. A systematic meta-analysis reviewed 61 studies from 1964 to 2005 and found a recurrence rate of only 7.4% and a 1.2% risk of malignancy.[26] Patients who underwent interval appendectomy were found to have a prolonged hospital stay.[26] Interval appendectomy is not strongly supported in the literature.

Acute Cholangitis

Acute cholangitis is defined as a biliary obstruction complicated by infection. The obstruction may be due to calculi, stricture, or a blocked biliary stent. The clinical presentation and subsequent decompensation of a patient may be quite rapid so prompt diagnosis is essential. Rates of mortality have improved over time but remain 11% to 27%.[27] Classic diagnosis is described by Charcot triad: fever, abdominal pain, and jaundice. The complicated form of cholangitis includes septic shock and mental status change (ie, Reynold pentad). The Tokyo Guidelines clarified the diagnostic criteria and in addition graded the severity of cholangitis (**Box 3**).[28]

The severity of disease increases with the presence of organ dysfunction and nonresponse to initial medical treatment. Severe acute cholangitis requires urgent biliary compression with endoscopic retrograde cholangiopancreatography (ERCP).[27]

Source control

No randomized trials have been completed that compare treatment options, but in accordance with the theme of least invasive treatment that may safely provide SC, ERCP-directed internal drainage is the first-line therapy. Recent data suggest that early ERCP ($\leq$24 hours) leads to significantly shorter hospitalization without a significant increase in intervention-related complications.[29] Percutaneous transhepatic drainage is available as a second-line therapy. Operative drainage may be indicated. Recently, endoscopic ultrasound-guided biliary drainage has emerged as an option for biliary decompression.[30]

Antibiotics

Coverage of microorganisms from the proximal bowel is usually sufficient for initial empiric treatment in biliary disease. Anaerobic therapy is added in the case of acute cholangitis and, when there is a biliary-enteric anastomosis, severe physiologic disturbance, or an immunocompromised state. *Enterococcus* species coverage is only necessary if the patient has undergone an extensive hepatic procedure or has other risk factors for enterococcus, such as immunocompromisation.[8]

Box 3
Diagnostic criteria for acute cholangitis

A. Systemic inflammation
   1. Fever and/or chills
   2. Elevated WBC or CRP

B. Cholestasis
   1. Jaundice
   2. Elevated transaminases

C. Imaging
   1. Biliary dilatation
   2. Evidence of cause on imaging (stricture, stone, stent)

Suspected diagnosis: one item in A + one item in either B or C.
Definite diagnosis: one item in A, one item in B, and one item in C.
Abbreviations: CRP, C-reactive protein; WBC, white blood cell count.
Data from Sartelli M, Viale P, Catena F, et al. 2013 WSES guidelines for management of intra-abdominal infections. World J Emerg Surg 2013;8:3; and Weber DG, Bendinelli C, Balogh ZJ. Damage control surgery for abdominal emergencies. Br J Surg 2014;101:e109–18.

Pancreatitis

Pancreatitis has a variable presentation and if not recognized and treated may result in rapid and severe patient decompensation. Ninety percent of acute pancreatitis is caused by alcohol and gallstones. Simultaneous evaluation of cause should be delineated. Ultrasound and serum alcohol level should be performed in all patients. If these are negative, less common causes should be pursued.[31]

Given the severe inflammatory response seen in these patients, resuscitation is paramount. Worsening hemoconcentration 24 hours after admission is associated with increased morbidity. Lactated Ringer solution should be run at 250 to 500 mL/h within the first 12 to 24 hours of admission, and urine output should be closely monitored.[31,32]

Many scoring systems have been proposed to predict which patients are at risk for complicated pancreatitis. The classic Ranson's criteria on admission and at 48 hours may delay recognizing severe pancreatitis. The Bedside Index for Severe Acute Pancreatitis has been described as easier to use, whereas The Revised Atlanta Classification incorporates both physiologic and radiologic findings. Unfortunately, no system has proven to be all inclusive and therefore the close evaluation of fluid losses, SIRS, and presence of organ dysfunction is absolutely imperative.[31] Radiologic evaluation of pancreatitis is best if performed at least 72 hours after presentation to get a complete evaluation of pancreatic inflammation and necrosis.[32]

Source control

In the setting of gallstone pancreatitis, clearance of CBD with ERCP is strongly recommended within 24 hours. Although the evidence is not as strong as it is in the presence of cholangitis, most recommendations include ERCP.[31,33] If on-going signs of obstruction are present, surgical exploration of the common bile duct may be indicated. Early cholecystectomy (within 48 hours) is recommended in mild gallstone pancreatitis because waiting until complete symptoms and chemical resolution is unnecessary. Aboulian and colleagues[34] found in their randomized study that early cholecystectomy was not associated with increased technical difficulty or complications but resulted in a shorter hospital length of stay. In addition, offering an interval cholecystectomy was associated with a significant increase in biliary readmissions (18%) and is therefore not recommended.[35]

Antibiotics

Unlike other intra-abdominal infectious processes, this disease is not initially secondary to bacterial infection. Unless signs of cholangitis are present, routine use of antibiotics in pancreatitis is not recommended. Prophylactic antibiotics, even in severe necrotizing pancreatitis, do not prevent progression of sterile necrosis to infected necrosis and are therefore not recommended.[8] Ten percent of patients with pancreatitis will ultimately become infected. This number increases to between 30% and 70% if necrosis is present.[1,32]

Infected Pancreatic Necrosis

Infected pancreatic necrosis should be suspected if there is an acute deterioration or failure to improve over a period of 7 to 10 days. Given the underlying SIRS causes fever and tachycardia, ultimately diagnosing infected pancreatic necrosis may be challenging. The diagnostic imaging of choice is a CT scan with IV contrast. Infection is suspected if there is gas in the necrotic cavity.[31]

Source control

With a diagnosis of infected pancreatitis, traditionally this was an indication for operative intervention. Clearly, there is a need for intervention, but image-guided catheter placement with upsizing as necessary has proven to be effective and safe and may be able to successfully avoid surgery in 50% of patients. Multiple approaches have been successfully attempted, including laparoscopic anterior/retroperitoneal or percutaneous radiologic-guided catheter placement followed by endoscopy through the tract.

Ultimately, surgical debridement may be needed to remove infected, necrotic tissue if catheter drainage does not appear to be providing adequate SC. Delayed intervention in pancreatitis, unlike other acute IAI, is associated with improved morbidity. Recommended surgical approach is midline or subcostal and approaches the lesser sac through the gastrocolic ligament. The initial goal of surgery is to obtain aggressive SC and close the abdomen with closed suction drains. Open packing and planned relaparotomies are associated with significant mortality.[32]

Antibiotics

If infection is suspected, broad-spectrum antibiotics should be initiated and CT or US-guided fine-needle aspiration should be obtained for culture material. Carbapenems, fluoroquinolones, metronidazole, and high-dose cephalosporins have best penetrance into pancreatic tissue.[31]

Diverticulitis

The frequency of diverticulosis within the Western population increases with age. Thirty percent of people have diverticulosis by the age of 60. Ten to 25% of these patients will ultimately develop diverticulitis.[36] Diverticulitis is an inflammation and ultimately a microperforation of a diverticula-containing segment of colon. With the great variation in presentation and clinical course, it is imperative to appropriately classify patients. CT scan of the abdomen and pelvis with oral, IV, and rectal contrast is the examination of choice for patients with suspected diverticulitis.[36] The traditional classification was based on clinical and operative findings but, with the widespread use of CT scanning, a modified classification has been proposed (**Table 5**).

Table 5
Hinchey classification with modification

	Hinchey Classification		Modified Hinchey	
Uncomplicated	I	Pericolic abscess or phlegmon	Ia	Confined pericolonic inflammation, phlegmon
			Ib	Confined pericolonic abscess
Complicated	II	Pelvic, intra-abdominal or retroperitoneal abscess	II	Pelvic, distant intra-abdominal or retroperitoneal abscess
	III	Generalized purulent peritonitis	III	Generalized purulent peritonitis
	IV	Generalized fecal peritonitis	IV	Fecal peritonitis

Data from Sartelli M, Viale P, Catena F, et al. 2013 WSES guidelines for management of intra-abdominal infections. World J Emerg Surg 2013;8:3; and Moore LJ, Moore FA, Jones SL, et al. Sepsis in general surgery: a deadly complication. Am J Surg 2009;198:868–74.

Classic presentation is lower abdominal pain, fever, and leukocytosis. Depending on the extent of the disease, peritonitis may be present. Most patients (75%–90%) will experience uncomplicated diverticulitis (Hinchey class I). The goal of therapy has to be tailored around the acute attack as well as the possibility of future episodes. Multiple factors must be taken into account, including underlying patient comorbidities.

Source control

The ideal approach of SC in complicated diverticulitis has been widely debated intensely studied, and ultimately, undergone significant changes in management. Traditional treatment of complicated diverticulitis was managed by surgical intervention up until the 1990s.[37] Current treatment paradigm has shifted to aggressive medical support and, if necessary, nonurgent surgical intervention. Approximately 15% of patients with acute diverticulitis will develop a pericolonic or intramesenteric abscess.[36] Abscesses less than 3 cm have been found to safely resolve with antibiotics alone. Percutaneous drainage is recommended for accessible abscesses greater than 4 cm.[10,38] The ultimate goal for those who are amenable to drainage with percutaneous catheters is to avoid emergency surgery.[36] In fact, nonoperative management has been found successful in 91% of patients with complicated diverticulitis, including patients with large pneumoperitoneum and large abscesses.[37,39] Failure of nonoperative management ultimately requires segmental colectomy. Laparoscopic approach, even for complicated diverticulitis, has been shown as safe even in the setting of longer operative times, demonstrating fewer complications, less pain, and shorter hospital stay.[40,41]

What is not debated is that emergency operative intervention is required for free perforation with peritonitis (Hinchey III or IV) or the presence of hemodynamic instability. Immunocompromised patients are more likely to present with perforation and failed medical management necessitating a lower threshold for urgent surgery. Historically, a Hartmann procedure was standard and necessary. In certain clinical scenarios, primary resection and anastomosis have been proven safe even with diffuse peritonitis.[12] A recent randomized trial compared the Hartmann procedure to resection and primary anastomosis (PA). The PA group was found to have less risk of serious complications, lower in-hospital costs, decreased operating times, and ultimately, decreased hospital length of stay.[42] Another less invasive treatment option that has been proposed is laparoscopic lavage. Laparoscopic lavage has also been proven safe and will be compared with conventional management in the upcoming "LADIES" trial.[43]

Antibiotics

Uncomplicated diverticulitis has a standard treatment with bowel rest and antibiotics for 7 to 10 days, which is successful in 70% to 100% of patients.[10,36,44] Classically, a combination of quinolone and metronidazole are used, but recently Ertapenem, a carbapenem, has been increasingly used and may provide a well-tolerated monotherapy.[44]

The length of treatment has come into question in the literature. A recent study randomized patients with uncomplicated sigmoid diverticulitis to a 4-day versus the traditional 7-day course of antibiotics and found no significant differences in recurrence rate at 1 month and at 1 year. The 4-day group had a significantly shorter hospital length of stay.[44] In the face of increased antibiotic resistance, Chabok and colleagues[45] took the next step and questioned the need for antibiotics at all in uncomplicated acute diverticulitis. In an interesting multicenter randomized trial, patients with

acute uncomplicated diverticulitis were given either no antibiotics or the standard 7-day course. Ultimately, they found no differences in complications, operations, recurrences, or hospital stay. Despite lack of adequate evidence to treat with antibiotics, current recommendation for uncomplicated acute diverticulitis is antibiotic coverage for gram-negative and anaerobic organisms for 5 to 10 days.[12]

SUMMARY

IAI arise from many sites and range from a moderate nuisance to life-threatening. Prompt identification, diagnosis, and treatment allow optimal patient outcomes. Resuscitation from shock, early appropriate antibiotic administration, and control of the source of infection are necessary components of a 3-pronged approach. Initial antibiotic administration should be broad spectrum and tailored to the most likely pathogen and then narrowed to the best agent for the appropriate duration. SC may be obtained using radiographically placed percutaneous or traditional operative drains; the choice depends on the anatomic site, site accessibility, and the patient's clinical condition. Patient-specific factors (advanced age and chronic medical conditions) as well as disease-specific factors (health care–associated infections and inability to obtain SC) combine to affect patient morbidity and mortality.

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