INTRODUCTION

With intra-abdominal infection being one of the most common reasons for surgical consultation, understanding the evaluation and management of these processes becomes paramount in the day-to-day practice of the surgeon. The very broad nature of who is affected coupled with the interplay of patient comorbidities and their medications make dealing with intra-abdominal infections a challenge.

As with most complex problems in medicine, it is often useful to break them down into simpler and smaller parts. One useful way to categorize intra-abdominal infections is to divide them into those originating from previous abdominal trauma or operations and those presenting in a “virgin” abdomen. The latter group most commonly includes those patients presenting with specific organ-based infectious processes such as appendicitis, cholecystitis, or diverticulitis. These individual diseases are covered extensively in other chapters and are discussed only superficially in this chapter. The former are those patients who have sustained intra-abdominal trauma or have undergone previous abdominal interventions and are not recovering in the usual expected course. It is this group that taxes diagnostic and clinical skills and may require the most complex medical decision making.

DEFINITIONS

Intra-abdominal infections are a broad range of processes that result from bacterial invasiveness and growth in the abdominal cavity. There are several ways these types of infections have been defined or classified. One schema categorizes the infectious process into uncomplicated and complicated. The uncomplicated process is, in general, confined to the involved organ system. Examples of this include acute nonperforated appendicitis or localized acute diverticulitis. Many of these just require administration of appropriate antibiotics. Complicated intra-abdominal infections are those that extend beyond the normal confines of the organ system and diffusely invade the peritoneum. In addition to antibiotics, these infections usually require an invasive procedure in order to obtain source control.

When speaking of a complicated infection, the diffuse nature usually implies a degree of peritonitis. Peritonitis itself can be divided into primary, secondary, and tertiary. Primary peritonitis is an infection that develops in the absence of a distinct break in the structural...
integrity of the gastrointestinal (GI) tract. It is usually the result of bacterial translocation, hematogenous spread, or lymphatic seeding. The resulting infection is often monomicrobial, commonly gram-negative Enterobacteriaceae species or streptococci. Secondary peritonitis results from a break in the continuity of the GI tract. It is a polymicrobial infection often with both aerobic and anaerobic enteric bacteria. Tertiary peritonitis is often seen in immunosuppressed patients and results from failure of treatment of a secondary peritonitis. The flora seen with these types of infections are often nosocomial and include resistant gram-negative bacilli, enterococcus, staphylococcus, and yeast. This classification is somewhat more useful because primary peritonitis is treated by antimicrobial therapy alone compared to secondary and tertiary peritonitis, which almost always require some type of interventional procedures.

DIAGNOSTICS

The approach to each patient should begin with the standard history and physical, with a focus on signs of systemic illness and abdominal processes. Early recognition of systemic inflammatory response syndrome (SIRS) criteria (Box 1), especially in patients who are not in the early postoperative or posttrauma period, combined with abdominal findings such as pain, nausea, vomiting, and anorexia, are highly suggestive of a complicated intra-abdominal infection. An adequate history and physical examination in the awake patient will provide the information necessary to make a diagnosis in many cases. The severity of the illness should dictate what, if any, laboratory studies are needed. In the patient with a simple intra-abdominal process without hemodynamic or physiologic derangement, a white blood cell count with differential is sufficient. In those patients presenting with a more in-depth process, thought should be given to obtaining a complete blood count, electrolyte panel, arterial or venous blood gas, lactate, and blood cultures.

While hard to believe in today’s modern imaging world, a percentage of these patients require little if any further diagnostic studies. However, many patients with intra-abdominal infections may present with physiologic derangements, an altered mental status that precludes a complete history or physical exam. In this patient population or those in which more information is needed, further diagnostic studies are required. Standard radiography has a limited role, but the upright chest radiograph or lateral decubitus film remains very useful to help identify free air. Although ultrasound has been shown to have some utility in diagnosing acute appendicitis, with some studies reporting over 80% sensitivity and 90% specificity, it remains operator dependent, and its use has not been fully evaluated for other forms of intra-abdominal infection.

Computed tomographic (CT) scanning with oral and intravenous contrast has become the “gold standard” of diagnosing intra-abdominal infection. While the utility and added benefit of oral contrast has been questioned, the use of intravenous contrast markedly improves diagnostic accuracy. Thus, noncontrast CT scans are of limited usefulness and should be used to investigate possible intra-abdominal infections sparingly, if at all. Because new contrast agents are less nephrotoxic and allergenic, given the risk to benefit ratio, it is a rare patient who cannot tolerate one dose of intravenous contrast. Lastly, in this digital age of remote imaging and interpretation, routine direct communication between surgeons and radiologists has been often lost. In patients who have undergone complicated operations, it is incumbent on the operating surgeon to personally discuss the case with the radiologist, including what has been done and what is being searched for, so the proper study and interpretation are possible.

INTRA-ABDOMINAL INFECTIONS FOLLOWING ABDOMINAL TRAUMA OR SURGERY

The recovery from abdominal trauma or abdominal operation is similar with most patients, progressing in a standardized manner. Quite often, even patients who were desperately ill preoperatively “look great” on postoperative day 1 or 2. Physiologic derangements such as tachycardia, fever, and even pressor requirements are often improved, and the abdominal pain of peritonitis is often replaced by surgically induced incisional pain. It is the astute clinician who recognizes the plateauing of improvement on postoperative days 3 and 4, the slight increase in abdominal distension, and the failure to regain full bowel function. Unless one “thinks bad thoughts,” one will never contemplate that something might be amiss. While a small fever at this time and a change in leukocyte count might be a urinary tract infection or phlebitis from an old intravenous site, the failure to entertain the possibility of an anastomotic leak following a colon resection for diverticulitis or a missed bowel injury following a thoracoabdominal gun shot wound will result in disastrous outcomes (Figure 1). One must be aware of the possibilities that can befall a patient following abdominal trauma or operative intervention. Nowhere in general surgery is the aphorism that “good clinical
judgment comes from experience and experience comes from bad judgment” more true than dealing with postoperative or posttraumatic abdominal infection. In addition, CT imaging, especially early after operation or injury, may be difficult to interpret or frankly misleading. In these cases, an astute surgeon must look at the CT as either positive or “not positive” rather than negative, and if no other explanation for the infection is identified, the surgeon should urgently reexplore the patient. Waiting until the patient shows signs of organ dysfunction will markedly increase mortality.

It is in the elective surgical population where the presence of an intra-abdominal infection can be most easily diagnosed and treated. These patients are commonly optimized prior to surgery, are not malnourished, and have a typical and well-known course. As outlined, the use of radiographic imaging with CT scanning is often unhelpful prior to postoperative day 5, so clinical judgment is required to ascertain which patients are not progressing in the expected manner. The wound should be examined by experienced individuals at least once daily, if not more frequently. While many superficial wound infections are in fact just that, not uncommonly a wound infection is the tip of the iceberg of a subfascial or intra-abdominal infection. Although with attention to surgical care improvement project (SCIP) guidelines, there has been improvement in the delivery of appropriate prophylactic antibiotics, these interventions have been less uniformly successful in decreasing overall wound infection rates. More importantly one needs to remember that these interventions are not designed to decrease intra-abdominal infections.

Bariatric surgical patients now comprise an increasing number of routine elective cases on the surgical schedule. The presentation of an intra-abdominal catastrophe in this group can be quite subtle, often limited to an unexplained mild tachycardia without abdominal pain. The usual postoperative care of these patients involves defined care plans of early mobilization and oral intake. Deviation from the normal progression, especially in the presence of tachycardia, should prompt rapid investigation for leaks or compromised bowel. Rapid reexploration in the absence of diagnostics before physiologic deterioration will be lifesaving in this group.

**Management of Intra-Abdominal Infections**

Several factors should come into play once suspicion for an intra-abdominal infection is entertained. These include resuscitation, antibiotic usage, and source control itself. Patients who present with either a suspected or diagnosed intra-abdominal infection should have some form of volume resuscitation. Even without hypotension, there are several reasons why these patients might be volume depleted. These include nausea and vomiting, fluid sequestration within the abdominal cavity or lumen of the bowel, and poor oral intake. As the process progresses, the patient may develop tachycardia, which results in an evaporative fluid loss. By this time, one can often elicit orthostatic hypotension in most patients. Fluid resuscitation should begin with the administration of isotonic crystalloid and in general be guided by evidence of end organ perfusion (adequate mental status, urine output, correction of acidosis). There is no utility using colloid such as albumin or hetastarch in these circumstances, and some data suggest a worse outcome. Should the patient present with hypotension or evidence of poor perfusion, a more aggressive approach to volume resuscitation should be employed. Our recommendation is to follow the current surviving sepsis guidelines, which include fluid challenges, monitoring/assessment of filling pressures, and the potential use of pressors and steroids.

Early administration of appropriate antibiotics in the course of an infection reduces mortality in the septic patient and should be initiated as soon as a diagnosis of intra-abdominal infection is suspected. While the absolute duration of antibiotic administration is a matter of debate, this therapy should be maintained during the interventions needed to achieve source control. As noted above, there are commonly encountered flora that will depend on the type of peritonitis as well as the presumed location of the infection in the GI tract. This can help dictate antimicrobial therapy. As one moves from the stomach, where gram-positive cocci (streptococci or lactobacilli) predominate, the number and type of bacteria change. The mid- to distal small bowel will still house gram-positive cocci, but enteric gram-negative aerobic and facultative anaerobic bacilli begin to increase in number. The colon will have large numbers of obligate anaerobes. The most common bacteria found in intra-abdominal infections are E. coli, Klebsiella sp., Enterobacter sp., Streptococci (mostly viridans), Enterococcus sp., and Bacteroides sp. It is worth noting that the “expected flora” is altered in patients with previous antibiotic administration or those coming from other health care environments such as nursing homes. In this patient population, the upper GI tract should be considered to have a higher bacterial count that includes Enterobacteriaceae, Pseudomonas, and yeast, along with a potential for antimicrobial resistance.

Recommended antimicrobial regimens for mild to moderate community acquired intra-abdominal infections include ticarcillin-clavulanate, cefoxitin, ertapenem, or moxifloxacin as single-agent drugs or combination regimens with metronidazole and cefazolin, cefuroxime, ceftriaxone, levofloxacin, or ciprofloxacin. In those with a high-severity community-acquired infection (i.e., acute physiology and chronic health evaluation [APACHE] >15), and especially those patients who have previously been treated with antibiotics, one should avoid quinolones (high resistance) and think about using single agents such as meropenem, imipenem-cilastin, piperacillin-tazobactam, or cefazidime or cefepime in combination with metronidazole. These broad-spectrum antibiotics should be tailored once culture and sensitivity reports become available. The duration of antibiotics should be limited to 4 to 7 days unless achieving source control had been difficult. Bowel injuries attributable to trauma that have been repaired in a timely fashion should be treated for no more than 24 hours.

**Source Control: Operation Versus Percutaneous Drainage Alone**

Source control, the single or multiple interventional processes by which one attains control or elimination of an infection, can potentially be achieved through a nonoperative or operative approach depending on the nature of the disease. There is little if any literature offering Level 1 advice as to the optimum technique under all circumstances. Globally, then, we recommend an appropriate source-control procedure based on the principles of control of ongoing peritoneal contamination, draining infection, and providing restoration of anatomic and physiologic parameters. This procedure can include resection/debridement of nonviable tissue, drainage, diversion, or a combination of each. As detailed below, intervention will then depend on the extent of the underlying process, how sick the patient presents, and resources available. The risk of failure for source control increases in the elderly (>70 years), higher illness severity (APACHE ≥15), delays to intervention, comorbidities, poor nutritional status, and extent of peritoneal involvement.

Once a need for intervention has been established, time to intervention becomes critical. Source control should proceed without delay. While many patients require at least some resuscitation, this should not delay intervention for more than a short time. Patients with severely altered physiology will likely never be resuscitated to “normal” values without source control. In these patients, operative source control may be the only way to halt an ongoing process, and resuscitation should be concomitant with the procedure.

The introduction of image-guided percutaneous drainage of intra-abdominal abscesses in the 1980s revolutionized the treatment
of that disease to the point that it is now the standard of care and the first and best option for many infections. Despite three decades of improvements in imaging technology, the general guidelines for successful drainage have not changed. Absolute necessities include a window in which to drain the collection and material that can be drained though a catheter. Well-localized collections, especially in those patients without diffuse peritonitis, can be managed with a percutaneous drain and appropriate antibiotics. This can even include those with small amounts of localized free air. Examples of infections that are usually amenable and well handled by percutaneous drainage include subphrenic abscesses after splenectomy, pelvic abscess following perforated appendicitis, and diverticulitis with localized abscess (Hinchey class 1b or 2) (Figure 2). Loculated or multiple abscesses are not necessarily contraindications to percutaneous drainage, but these authors have observed patients being treated with three and four and more percutaneous drainages over several weeks when one open operation would have taken care of the problem easier, more expeditiously, and at a lower cost. Patients with poorly localized or diffuse collections, necrotic tissue, or inaccessible collections require open operative intervention. Those patients with diffuse peritonitis or massive amounts of free air require immediate surgery.

The operation performed should be based on the extent of the infection, the organs involved, and the patient’s physiologic status. Despite the myriad possibilities in treating intra-abdominal infection, there are several concepts that apply across all diagnoses. The first is that complete identification of the pathology is an absolute necessity to affect proper therapy. It does no one any good to drain the abscess only to miss the necrotic piece of intestine or anastomotic disruption. The groups of patients that require operative intervention are often sicker and more complicated. These cases can be exceedingly challenging, and even senior surgeons can often benefit from another experienced pair of hands in the operating room. Consultation and collaboration with senior partners can also help in the overall intraoperative decision making. Once the anatomic problem is completely identified, the goal is now to achieve source control. As an injured trauma patient, the extent and breadth of the procedure should be dictated by the patient’s physiologic status.

In patients with intra-abdominal infection without an obvious GI perforation, unroofing of all abscess cavities and collections is all that is required. Intraoperative cultures should be performed and transported to the laboratory with some expediency. It is always unfortunate to perform these procedures in the middle of the night only to find out that the “negative cultures” of obvious purulence did not make it to the microbiology laboratory until the next day. In patients without any definable residual abscess cavities, there is no need for drains. In fact, drains in this situation are more often associated with subsequent infection than no drainage. In our estimation, the quantity of drains placed often far outweighs the quality of their use. What we mean by this is that because almost all drains are of the closed suction variety, little thought is often given to drain placement, assuming that the suction will allow for proper drainage in all circumstances. While it may be true in concept, it is not always accurate in practice. Bulbs get filled, resulting in a loss of suction; tubing may be kinked by patient positioning or even the dressing.

Additionally, consideration should be given to how the drain is brought out through the abdomen. One must always remember that the drain exit site will ultimately end up several inches more medial when the abdomen is eventually closed. This can result in a loss of the “direct” path of the drain and can decrease efficacy. We believe and teach that placement of drains in a more “gravity-friendly” fashion with more lateral exit tracts will result in shorter and more direct drain paths and can even aid closed suction drainage. In addition, we do not advocate large sump-type drains. These drains often come with filters that are quickly clogged with serum and particulate matter, rendering the sump feature of the drains nonfunctional. If the material is too thick to be drained through a closed suction drain, it is also likely to clog a sump drain. In these circumstances, if sump drainage is employed, we have utilized a short course of continuous irrigation either through one of the sump ports or through a second drain. Lastly, all current drains on the market are made from silicone or a similar nonreactive material. As such, they induce little or no inflammation, and they will not form a tract. While in most cases this is beneficial, there are times where one is relying on the inflammation induced by a latex or rubber drain to “seal the area” and control the infection.

When the genesis of the infection is related to a break in the GI tract, source control involves eliminating or controlling the ongoing contamination. The two overriding concepts in these circumstances are not to make the hole bigger and not to create any new (unnecessary) holes. While on the surface these concepts appear obvious, the compulsion to put “just one stitch” in a leaking anastomosis in the face of gross peritonitis can sometimes be irresistible. The success of this is negligible and will most assuredly lead to further breakdown, a larger hole, and a bigger problem. The complexity of entry into the abdomen itself along with the actual dissection toward the primary pathology should not be underestimated. Creation of inadvertent enterotomies can occur but need to be recognized and treated. The care needed to do these cases cannot be underestimated. In addition, when enterotomies are created, one must critically decide how they should be handled, similarly to the primary pathology.

In most cases, intestinal perforation in the face of peritonitis should be handled by resection. One exception to this is perforated duodenal ulcers, which are treated by graham patch. Perforation in the body of the stomach can be closed primarily followed by imbrication using Lembert sutures because there is abundant gastric tissue to cover a suture line. In the proximal small bowel, resection is usually followed by anastomosis, whereas in the distal small bowel and colon,
one must consider the risk to benefit ratio of performing an anastomosis against creation of a stoma. If one chooses to restore intestinal continuity, it is necessary to use bowel that is not involved in the inflammatory process.

While there have been many studies showing equivalence between stapled and hand-sewn anastomosis in elective procedures, there is some suggestion in emergent surgery, and in multiple trauma especially, to suggest that hand-sewn anastomosis may be superior. In any event, when faced with peritonitis and bowel edema, it is our preference to perform only hand-sewn anastomosis. The decision to perform an anastomosis must also take into account the patient’s physiologic status and the risk of failing to achieve source control. There is a surgical truism that the anastomosis that is never created will never leak. While a stoma commits the patient to a subsequent operation, surviving intra-abdominal infection and sepsis should be considered a success and an acceptable trade-off. In those patients with gross peritonitis, pressor requirements, and hemodynamic instability, a truncated procedure (i.e., damage control) should be employed. This allows the surgeon to eliminate the source of infection and provide ongoing resuscitation in a more controlled environment such as the intensive care department. During the subsequent operations, hopefully in a much less hostile abdomen, one can plan a more definitive approach to control infection and restore intestinal continuity.

There are also circumstances where a bowel resection or revision of an anastomosis is not possible. Some examples include duodenal stump blowouts, associated phlegmons compromising surrounding vascular structures, or pancreatico-intestinal anastomotic leaks. In these types of cases, the goal is controlling the effluent to create a controlled fistula while allowing the inflammatory and fibrotic process to “scar” over the problem. An important part of the management is the use of latex or red rubber drains placed in the lumen of the intestine. This both allows drainage and enhances the inflammatory process. T-tubes can be particularly useful because they are not prone to inadvertent removal. Liberal use of suction drains in proximity to these processes and placed as outlined above will further capture the effluent not coming through the latex drains. This drain strategy combined with appropriate nutritional support will lead to a controlled fistula that will often allow the clinician to remove the latex drains at much later time when the infection and inflammation has subsided.

**Suggested Readings**


