Intra Abdominal Abscess

Intra-abdominal abscess is one of the most important and serious problem in surgical practice. Appropriate treatment is often delayed because of the obscure nature of many conditions resulting in abscess formation, which can make diagnosis and localization difficult. Associated pathophysiologic effects may become life threatening or lead to extended periods of morbidity with prolonged hospitalization. Delayed diagnosis and treatment can also lead to increased mortality rates; thus, the economic impact of delaying treatment is significant. A better understanding of intra-abdominal abscess pathophysiology and a high clinical index of suspicion should allow earlier recognition, definitive treatment, and reduced morbidity and mortality.

The 6 functional compartments in the peritoneal cavity include the following: (1) pelvis, (2) right paracolic gutter, (3) left paracolic gutter, (4) infradiaphragmatic spaces, (5) lesser sac, and (6) interloop potential spaces of the small intestine.

The paracolic gutters slope into the subhepatic and subdiaphragmatic spaces superiorly and over the pelvic brim inferiorly. In a supine patient, the peritoneal fluid tends to collect under the diaphragm, under the liver, and in the pelvis.

More localized abscesses tend to develop anatomically in relation to the affected viscous. For example, abscesses in the lesser sac may develop secondary to severe pancreatitis, or periappendiceal abscesses from a perforated appendix may develop in the right lower quadrant. Small bowel interloop abscesses may develop anywhere from the ligament of Treitz to the ileum. An understanding of these anatomic considerations is important for the recognition and drainage of these abscesses.

Pathophysiologic

Intra-abdominal abscesses are localized collections of pus that are confined in the peritoneal cavity by an inflammatory barrier. This barrier may include the omentum, inflammatory adhesions, or contiguous viscera. The abscesses usually contain a mixture of aerobic and anaerobic bacteria from the gastrointestinal (GI) tract.

Bacteria in the peritoneal cavity, in particular those arising from the large intestine, stimulate an influx of acute inflammatory cells. The omentum and viscera tend to localize the site of infection, producing a phlegmon. The resulting hypoxia in the area facilitates the growth of anaerobes and impairs the bactericidal activity of granulocytes. The phagocytic activity of these cells degrades cellular and bacterial debris, creating a hypertonic milieu that expands and enlarges the abscess cavity in response to osmotic forces. If untreated, the process continues until bacteremia develops, which then progresses to generalized sepsis with shock.
Etiology

Although multiple causes of intra-abdominal abscesses exist, the following are the most common:

- Perforation of a diseased viscus, which includes peptic ulcer perforation
- Perforated appendicitis and diverticulitis
- Gangrenous cholecystitis
- Mesenteric ischemia with bowel infarction
- Pancreatitis or pancreatic necrosis progressing to pancreatic abscess

Other causes include untreated penetrating trauma to the abdominal viscera and postoperative complications, such as anastomotic leakage or missed gallstones during laparoscopic cholecystectomy.

Microbiology includes a mixture of aerobic and anaerobic organisms. The most commonly isolated aerobic organism is *Escherichia coli*, and the most commonly observed anaerobic organism is *Bacteroides fragilis*. A synergistic relationship exists between these organisms. In patients who receive prolonged antibiotic therapy, yeast colonies (eg, candidal species) or a variety of nosocomial pathogens may be recovered from abscess fluids.

Skin flora may be responsible for abscesses after a penetrating abdominal injury. *Neisseria gonorrhoeae* and chlamydial species are the most common organisms involved in pelvic abscesses in females as part of pelvic inflammatory disease. The type and density of aerobic and anaerobic bacteria isolated from intra-abdominal abscesses depend upon the nature of the microflora associated with the diseased or injured organ.

Microbial flora of the GI tract shifts from small numbers of aerobic streptococci, including enterococci and facultative gram-negative bacilli in the stomach and proximal small bowel, to larger numbers of these species, with an excess of anaerobic gram-negative bacilli (particularly *Bacteroides* species) and anaerobic gram-positive flora (streptococci and clostridia) in the terminal ileum and colon. Differences in microorganisms observed from the upper portion of the GI tract to the lower portion partially account for differences in septic complications associated with injuries or diseases to the upper and lower gut. Sepsis occurring after upper GI perforations or leaks causes less morbidity and mortality than does sepsis after leaks from colonic insults.

Prognosis

The introduction of computed tomography (CT) scanning for the diagnosis and drainage of intra-abdominal abscesses has led to a dramatic reduction in mortality rates. (See Computed Tomography.) Sequential, multiple organ failure is the main cause of death. Incidence of death is correlated to the severity of the underlying cause, a delayed diagnosis, inadequate drainage, and unsuspected foci of infection in the peritoneal cavity or elsewhere.

Risk factors for morbidity and mortality include multiple Surgical procedures, age older than 50 years, multiple organ failure, and complex, recurrent, or persistent abscesses.
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