BMJ Best Practice

Intra-abdominal abscess

Straight to the point of care

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Summary

Intra-abdominal abscess (IAA) is an intra-abdominal collection of pus or infected material, usually due to a localized infection inside the peritoneal cavity. It can involve any intra-abdominal organ or be located in between bowel loops, or be free within the peritoneal cavity itself.

Commonly presents with abdominal pain, fever, and leukocytosis.

Usually secondary to inoculation, commonly from complicated intra-abdominal infection (i.e., bowel perforation, anastomotic leak, trauma).

Diagnosis can be confirmed by radiologic studies such as ultrasound or computed tomography (CT) scan. CT scan of the abdomen and pelvis is often more reliable, and provides better delineation of anatomic location and size of the IAA.

Treatment involves adequate source control (abscess drainage, whether percutaneous or surgical) as well as early appropriate and effective antimicrobial therapy.

If untreated, may lead to clinical deterioration including sepsis or septic shock.

Definition

Intra-abdominal abscess (IAA), also known as intraperitoneal abscess, is an intra-abdominal collection of pus or infected material and is usually due to a localized infection inside the peritoneal cavity. It can involve any intra-abdominal organ or can be located freely within the abdominal or pelvic cavities, including in between bowel loops. IAA is almost always secondary to a preexisting disease process, or concomitant intra-abdominal process.[1] It can be caused by one or multiple bacterial, fungal, or parasitic infectious agents.

Epidemiology

The majority of IAAs develop from appendicitis, diverticulitis, or following a surgical procedure.[1] It is estimated that 6% of patients undergoing colorectal surgery will develop a postoperative IAA.[6] [7]

Etiology

The etiology of IAA varies according to the source of the infection and the status of the patient's immune system.

IAA that develops secondary to a localized peritonitis is usually due to a perforated viscus or a direct inoculation after trauma or recent surgery. IAA are commonly secondary to appendicitis (59%), diverticulitis (26%), and surgical procedures (11%).[1] Abscess in solid organs may be secondary to hematogenous seeding, whether through the portal system in the case of hepatic abscess or from various extra-abdominal locations when bacteremia occurs. Infections associated with intraperitoneal sepsis are polymicrobial in half of patients, and caused by a single isolate 25.7% of the time.[8] Although most IAA are thought to be secondary to infection, microbiologic confirmation of IAA is inconsistent, and bacterial growth is absent in about 26% of cases.[1]

IAA are also classified as intraperitoneal, retroperitoneal, or visceral. Intraperitoneal (subphrenic, right or left lower quadrant, interloop, paracolic, pelvic) IAA are caused by bowel flora and are often polymicrobial.[9] They can occur postoperatively or as a result of perforation of a hollow viscus, appendicitis, diverticulitis, tumor, Crohn disease, pelvic inflammatory disease, or generalized peritonitis of any etiology. Retroperitoneal IAA can be either pancreatic, as a result of trauma or pancreatitis, or perinephric secondary to the spread of a renal parenchymal abscess, a complication of pyelonephritis or, rarely, due to hematogenous spread from a remote source. Pancreatic and perinephric abscesses are usually caused by bowel flora (often polymicrobial) and aerobic gram-negative bacilli respectively.[10] [11]

Visceral IAA involve the liver or spleen. Splenic abscesses occur as a result of trauma, hematogenous spread, or infarction secondary to sickle cell disease or malaria. They are caused by staphylococci, streptococci, anaerobes, aerobic gram-negative bacilli (e.g., *Salmonella*), and *Candida* in immunosuppressed patients.[12]

Intra-abdominal abscesses		
Location	**Etiology**	**Organisms**
Intraperitoneal		
Subphrenic Right or left lower quadrant Interloop Paracolic Pelvic	Postoperative; perforation of hollow viscus, appendicitis, diverticulitis, or tumour; Crohn disease; pelvic inflammatory disease; generalised peritonitis of any etiology	Bowel flora, often polymicrobial
Retroperitoneal		
Pancreatic	Trauma; pancreatitis	Bowel flora, often polymicrobial
Perinephric	Spread of renal parenchymal abscess (complication of pyelonephritis or rarely hematogenous from remote source)	Aerobic gram-negative bacilli
Visceral		
Hepatic	Trauma, ascending cholangitis, portal bacteremia	Aerobic gram-negative bacilli origin; polymicrobial bowel flora if portal bacteremia; amebic infection may occur
Splenic	Trauma, hematogenous, infarction (as in sickle cell disease and malaria)	Staphylococci, streptococci, anaerobes, aerobic gram-negative bacilli including *Salmonella*, *Candida* in immunocompromised patients

Classification of intra-abdominal abscesses (intraperitoneal, retroperitoneal, or visceral)

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Pathophysiology

IAA formation follows the same course as that of any other abscess. In a localized peritonitis, the host defense mechanism isolates the inflammatory reaction, but the debris and the edema form a collection that grows progressively and becomes walled off and isolated.[9] Due to the nonvascularized structure, as well as the significantly acidotic medium, antibiotics are ineffective.[13] Active mechanical drainage of the abscess is the necessary treatment when the abscess increases in size.

Many IAA are polymicrobial, and aerobic and anaerobic gastrointestinal organisms such as *Escherichia coli* and *Bacteroides* often predominate.[14] The abscess environment often presents special challenges for antimicrobial therapy.[14] Abscesses have a low oxidation-reduction potential and low pH as a consequence of limited vascularity and poor perfusion, anaerobic conditions, and dying tissue. High bacterial concentrations tend to depress oxygen-dependent phagocytosis and killing of bacteria by neutrophils, and to suffuse the confined space with high concentrations of beta-lactamase enzymes. Antibiotic penetration into the abscess is limited not only by poor perfusion but also by mechanical barriers such as fibrin clots and the abscess wall.[14] [15] Abscesses can be drained by percutaneous, laparoscopic, or open surgical techniques. The decision for the optimal approach for drainage often depends on the accessibility of the abscess percutaneously, as well as the degree of patient systemic illness.

Classification

Clinical anatomic classification

IAA can be classified based on the location of the abscess (such as interloop, subdiaphragmatic, paracolic, or pelvic abscess), or in relation to the involved abdominal organ (such as hepatic, splenic, pancreatic, appendiceal, or diverticular abscess).

Assessing risk of adverse outcome and treatment failure[2]

- Assess phenotypic and physiologic factors:
  - Signs of sepsis
  - Extremes of age
  - Comorbidities
  - Extent of abdominal infection and adequacy of source control
  - Presence of resistant or opportunistic pathogen.
- Characterize patients as either low or high risk for treatment failure or mortality.
- Assess for community-acquired or health care-acquired infection.
- Patients with Surviving Sepsis Campaign criteria for sepsis or septic shock, and those with APACHE II score greater than or equal to 10, are at higher risk.
- Prolonged length of hospitalization prior to surgery for intra-abdominal infection.
- Patients with diffuse peritonitis.
- Patients with delayed source control.

Case history

Case history #1

A 75-year-old man with type 2 diabetes mellitus presents to the emergency department with a 5-day history of abdominal pain that started in the left lower quadrant and was associated with obstipation, nausea and vomiting, and weakness. He has no prior history of abdominal disease or abdominal surgery. The patient is febrile (102.1°F [39.0°C]), tachycardic (heart rate 110 bpm), and hypotensive (systolic blood pressure 80 mmHg). He says he has generalized abdominal pain that is more localized to the left lower quadrant. He has not had any food in the last 24 hours.

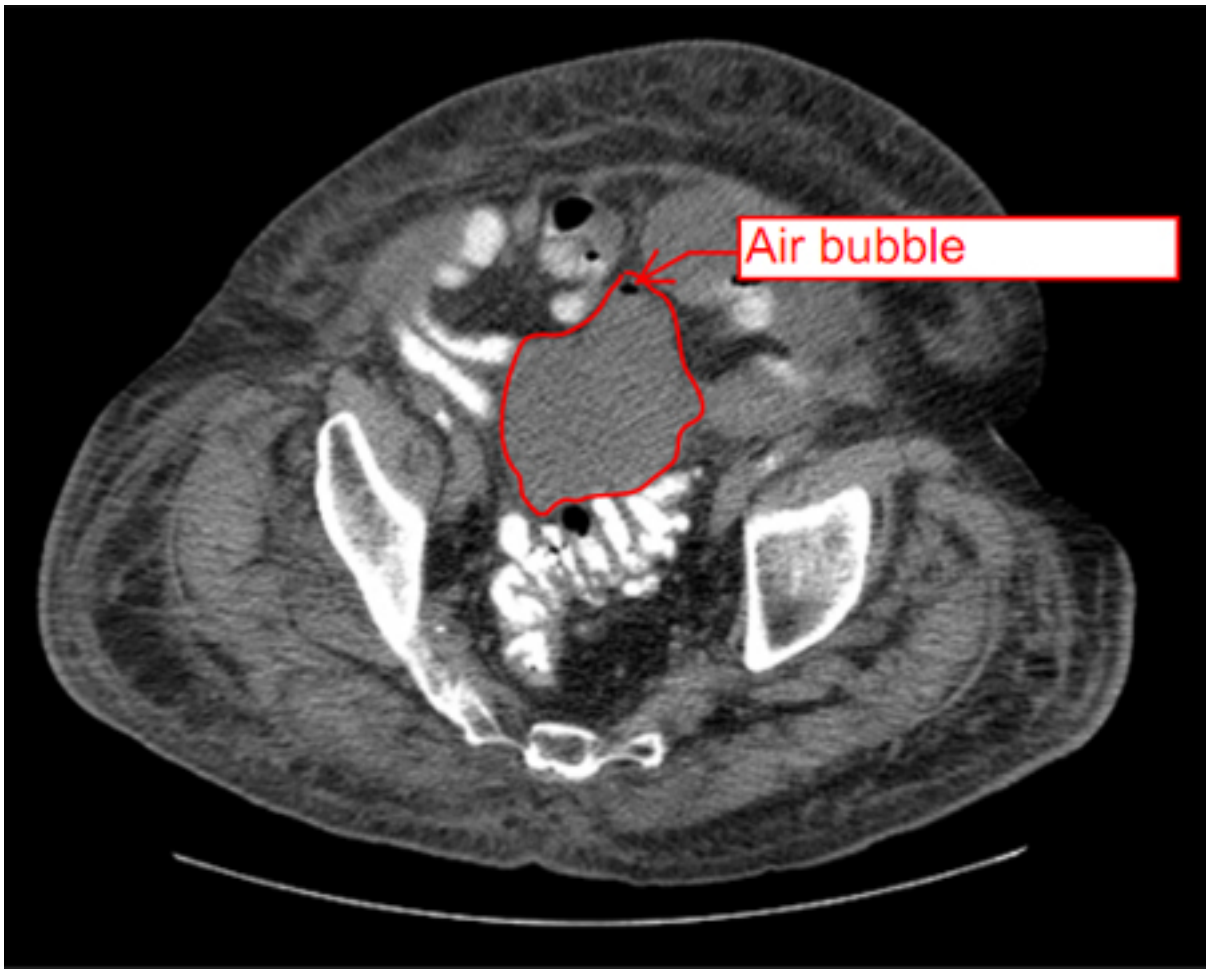
Case history #2

A 45-year-old woman has a recent diagnosis of Crohn disease. Nine days before presentation, she underwent a right hemicolectomy with ileocolic anastomosis for Crohn disease stricture with small-bowel obstruction. She had an uneventful postoperative course except for intestinal ileus. Her symptoms started 2 days before presentation, when she had to stop eating due to excessive nausea. She now has nausea, anorexia, high-grade fever (104.9°F [40.0°C]), and night sweats. Her abdomen is significantly tender on physical exam, with diffuse tenderness, and she clearly shows peritoneal signs. Her surgical incision has surrounding erythema and is draining purulent fluid.

Other presentations

IAA is commonly related to perforated appendicitis and perforated diverticulitis.[1] It usually presents with fever, abdominal pain, and leukocytosis. IAA can also present without fever or significant abdominal pain, depending on the size, degree of containment, and patient immune system integrity.[3] At the other extreme, the patient may present with clinical signs and symptoms of sepsis or septic shock, especially if very old or young, or immunocompromised. While leukocytosis is usually present, the patient may have leukopenia or even a normal white blood cell count. Primary IAA is extremely rare, but some cases have been reported.[4]

A palpable mass can be the presenting symptom.[5] Untreated IAA can track to the chest, lower extremity/inguinal region, and even scrotum/perineum.



Intra-abdominal abscess with small air bubble, secondary to perforated diverticulitis

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Approach

The diagnosis of an IAA is usually suspected from the patient's clinical history, physical exam, and laboratory data. It is confirmed by radiologic studies such as computed tomography (CT) scan.[19]

The diagnosis is highly suggested based on timing, when clinical symptoms persist for more than 5 days after abdominal surgery. Many postoperative patients report a recovery period after surgery, followed by a return of general malaise accompanied by fever, abdominal pain, and in some cases, nausea, vomiting, diarrhea, or severe obstipation.

The location of the abscess is sometimes related to the inciting event. When anastomotic leak or bowel perforation occurs, the abscess usually starts in close proximity to the affected bowel, then propagates depending on the size of the perforation, the severity of the leak, and the time to diagnosis.

History

Patients with IAA have usually had surgery or a recent trauma. They may report a prolonged ileus or abnormal bowel function, either obstipation or diarrhea. Patients may occasionally relate a period of feeling better after surgery followed by a gradual or sudden decline.

Symptoms of IAA include rapid-onset abdominal pain, loss of appetite, nausea, vomiting, bloating, obstipation, and/or fever.[19] Pain may be masked either by the effects of the surgical incision, or by postoperative pain control. Pain will worsen with time if the patient does not obtain appropriate medical or surgical care.

IAA can also present without fever or significant abdominal pain, depending on the size, degree of containment, and patient immune system integrity.[3] Patients with a suppressed immune system, caused by disease or medication, frequently present with atypical symptoms that are often mild, despite significant disease burden.

Pelvic abscesses can cause direct irritation to nearby organs, often resulting in dysuria, increased urinary frequency, diarrhea, and tenesmus. Urinalysis in the setting of pelvic abscesses often demonstrates sterile pyuria secondary to external irritation from the adjacent abscess.

Physical exam

IAA is often associated with fever.[3] Physical exam usually reveals some tenderness overlying the abdominal abscess or a generalized tenderness when multiple abscesses are present. Rebound tenderness may or may not be present. A mass can sometimes be palpated and could be the presenting sign of an appendiceal perforation with abscess formation.[5]

Suspected sepsis

Depending on the individual systemic inflammatory response, patients may present with sepsis or septic shock. Sepsis may also occur early after drainage of an IAA. In those who delay presentation for medical evaluation, intraperitoneal abscesses have been noted to spread out through the skin and subcutaneous tissues. This is often the case where there is an area of soft tissue already traumatized by prior instrumentation, surgical drains, fistula tracts, or tumor involvement.

Early recognition of sepsis is essential because early treatment - when sepsis is suspected but is yet to be confirmed - is associated with significant short- and long-term benefits in outcome.[20] The key to

early recognition is the systematic identification of any patient who has signs or symptoms suggestive of infection and is at risk of deterioration due to organ dysfunction. Several risk stratification approaches have been proposed. All rely on a structured clinical assessment and recording of the patient's vital signs.[20] [21] [22] It is important to check local guidance for information on which approach your institution recommends.

Laboratory studies

Laboratory studies are not very helpful in the diagnosis of an IAA. Although most commonly IAA are associated with an elevated leukocyte count, a normal white blood cell count should not exclude the diagnosis of an IAA. It is important to consider radiologic diagnostic imaging if clinical suspicion of IAA is high based on the patient's clinical presentation.

Erythrocyte sedimentation rate and C-reactive protein are both nonspecific markers of inflammation and could be elevated in patients with IAA.

Once IAA is identified and drained, obtaining a Gram stain and culture (aerobic and anaerobic) is particularly important in higher-risk patients, and patients with hospital-acquired IAA, to identify potential resistant or opportunistic pathogens.[2] Patients with IAA have early empiric antimicrobial therapy initiated after diagnosis, but culture and antimicrobial susceptibility information obtained from cultures of the IAA at the time of drainage permits de-escalation of antimicrobial therapy.

Serum glucose test is useful for the management of diabetic patients with IAA, though it may be difficult to treat the hyperglycemia before draining the abscess. In addition, when patients become increasingly hyperglycemic with increasing insulin demands, systemic infection should be suspected, including intra-abdominal infection and IAA.

Radiologic studies

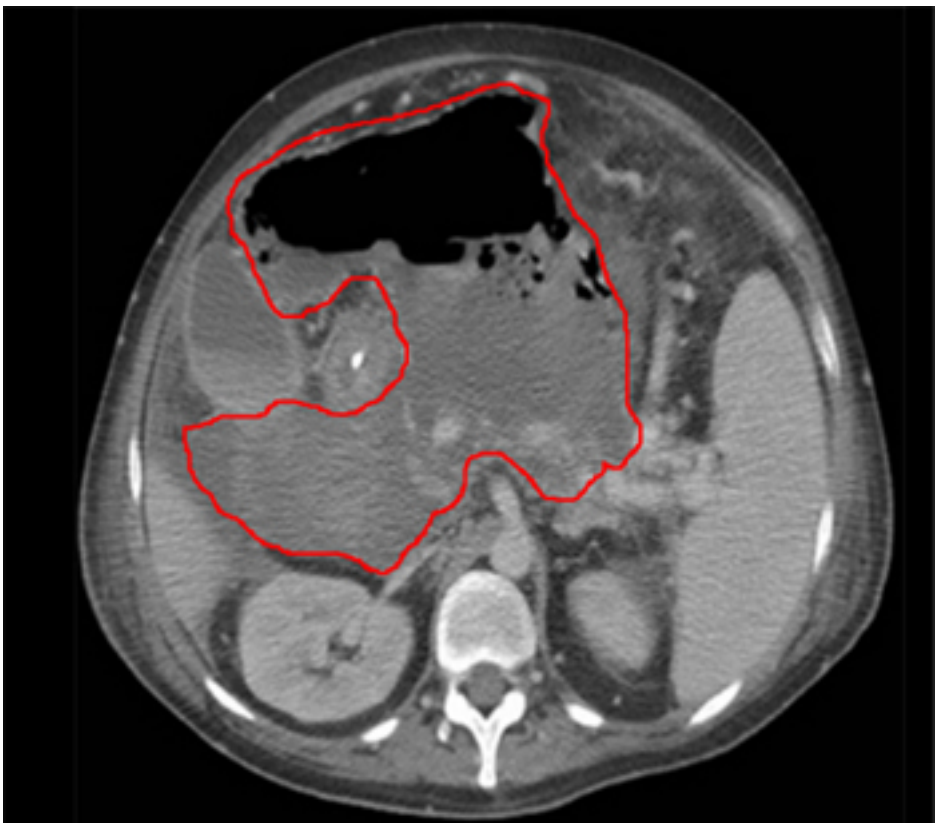
CT scan of the abdomen and pelvis is the preferred imaging modality.[19] CT can determine the size and anatomic location of the IAA.

Contrast enhancement (intravenous and enteral contrast) can help differentiate between intestinal loops and an interloop abdominal abscess, and to determine abscess proximity to vascular structures. This is particularly important in identifying whether a percutaneous approach to drainage of the IAA is possible. Rim enhancement, as well as the presence of debris or contrast leak inside the collection, raises suspicion of the diagnosis of IAA but cannot differentiate completely between an abscess and a sterile fluid collection in the peritoneal cavity. In this circumstance, percutaneous aspiration of the fluid collection and evaluation with Gram stain and culture are recommended to confirm a diagnosis of IAA. The presence of free air inside a fluid collection is diagnostic of an IAA but could also signify connection with the gastrointestinal tract. Avoid the use of noncontrast-enhanced CT imaging alongside contrast-enhanced imaging; the addition of unenhanced CT does not provide additional diagnostic information and exposes patients to unnecessary radiation.[23] [24]

Magnetic resonance imaging is useful to show the impact of an abscess on adjacent structures, especially muscle; hence, it is more useful in low pelvic abscesses.[14] Ultrasonography may be a useful aid to the diagnosis, especially when the patient cannot be transported. However, as it is user-dependent, small abscesses may be missed. In addition, ultrasonography does not demonstrate the abscess and its relation to other intra-abdominal viscera. It can also be limited by the presence of a surgical wound, and

can be affected by the size of the patient. False positives for IAA in patients with Crohn disease have also been reported.[25]

Endoscopic ultrasound has been used for evaluating and draining IAA adjacent to the gastrointestinal tract, with the largest experience being with pancreatic fluid collections. Other accessible areas include the pelvis and perirectal space, and the subphrenic and perihepatic spaces. Endoscopic ultrasound requires specialized expertise; data regarding its safety and effectiveness in draining IAA are preliminary. However, endoscopic ultrasound may be useful in critically ill patients requiring bedside procedures or for IAA not amenable to other conventional therapies.[26]



Abscess completely replacing pancreas and extending into portal hilum, with multiple gas bubbles and large air/fluid level

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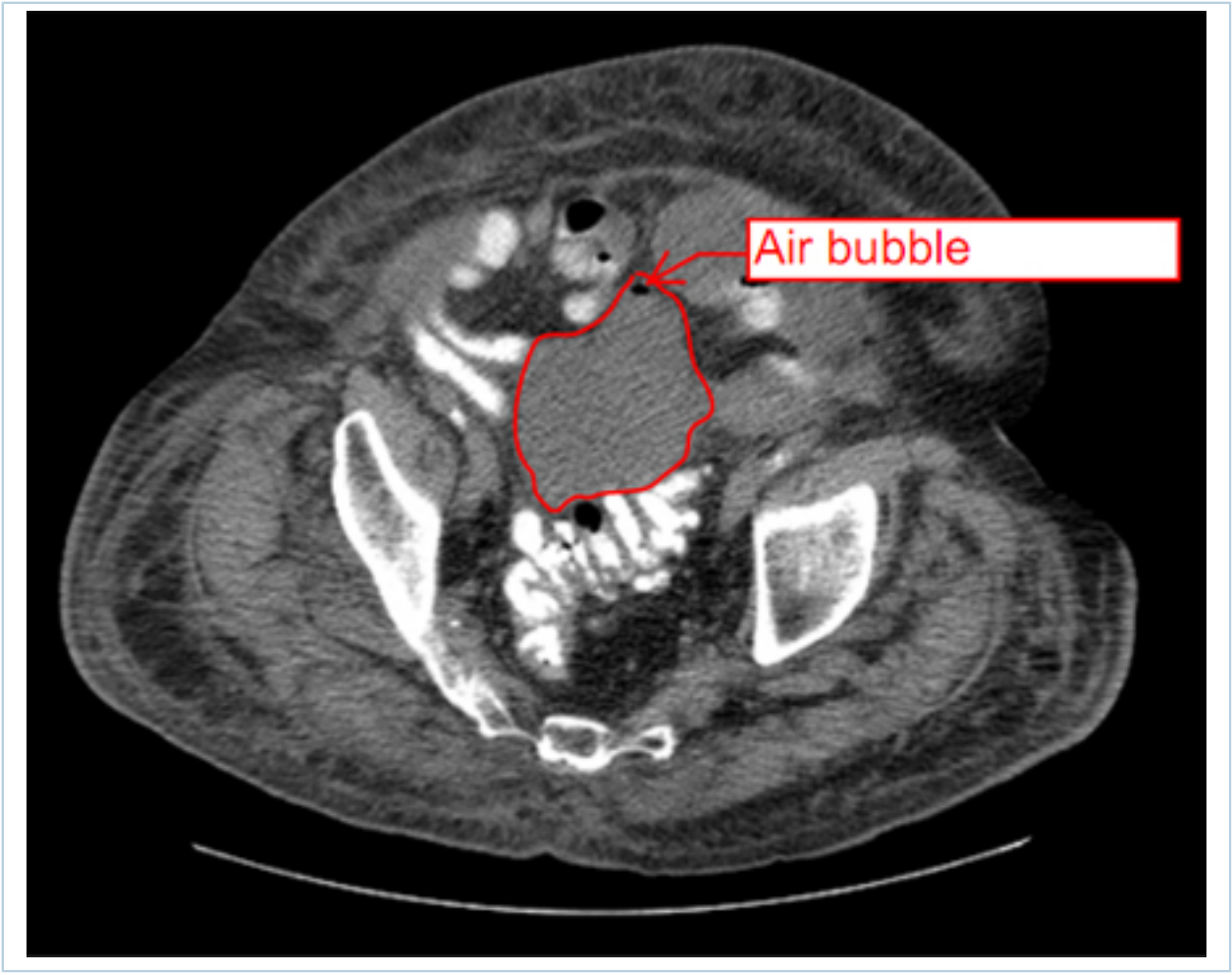

Intra-abdominal abscess with small air bubble, secondary to perforated diverticulitis

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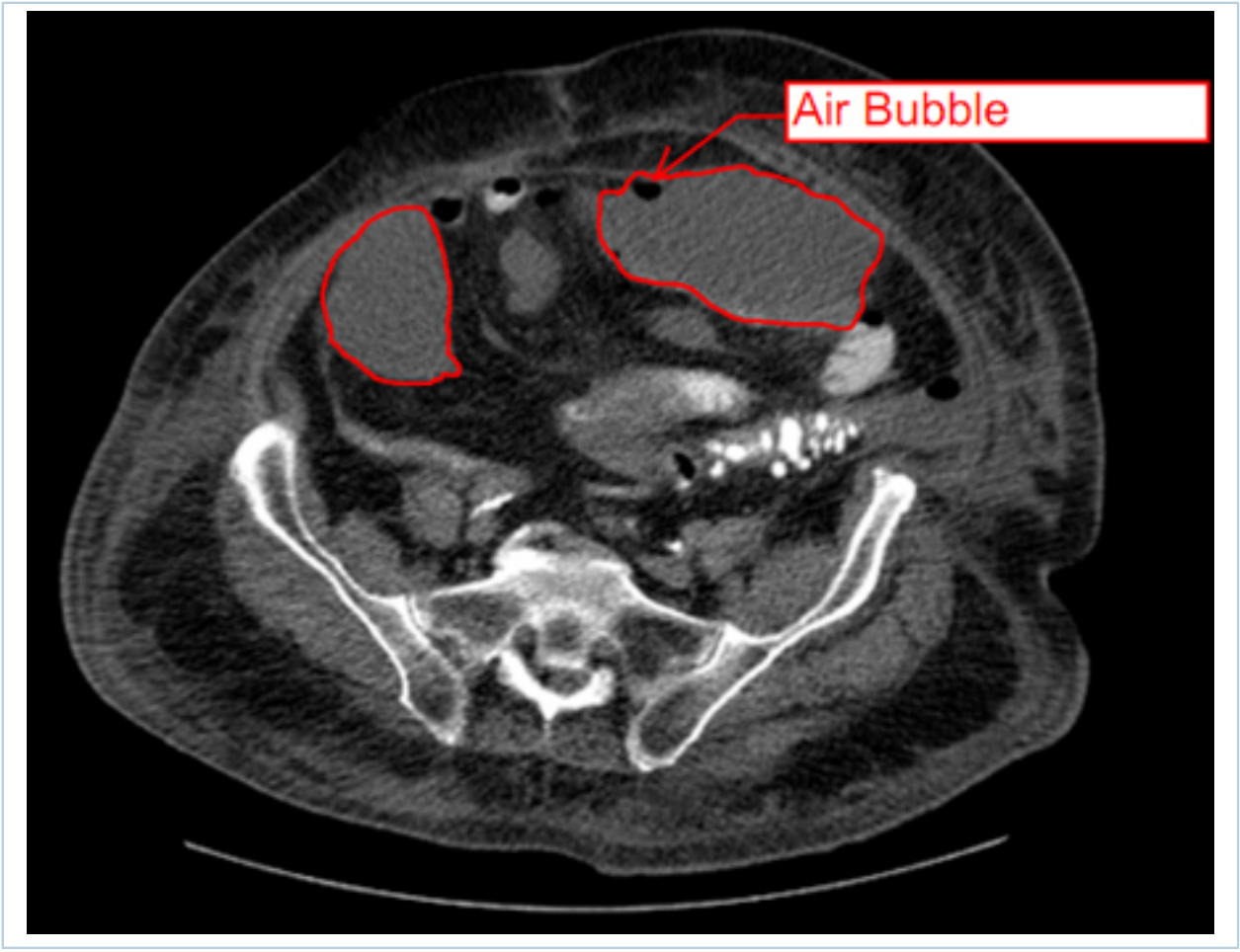

CT scan showing intra-abdominal abscess with small air bubble

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History and exam

Key diagnostic factors

recent history of surgery, trauma, or intra-abdominal infection (common)

- Patients with recent abdominal surgery, trauma requiring laparotomy, and common intra-abdominal infections (appendicitis, diverticulitis) are all at risk for IAA.[1] Patients who require intestinal resection and anastomosis are at particular risk for anastomotic leak (1.5%) and abdominal abscess formation.[16]

fever or hypothermia (common)

- A potential sign of sepsis, though nonspecific; should prompt further investigations, especially in those with known intra-abdominal pathology or recent surgery or instrumentation. In older and immunocompromised patients, hypothermia is common with IAA.

abdominal pain (common)

- Common in patients with IAA related to perforated appendicitis and diverticulitis. Pain can present as focal tenderness or generalized nonspecific abdominal pain. In postoperative patients, pain may be

masked by surgical incision or postoperative narcotic use. It could be differentiated from postsurgical or incisional pain by the fact that it does not improve with time.

rectal tenderness and fullness (uncommon)

- Pararectal abscess or low pelvic abscess may present with rectal tenderness on digital rectal exam.

Other diagnostic factors

tachycardia (common)

- Mild tachycardia is common but is usually multifactorial.

change in bowel habits/abnormal bowel function (common)

- Nonspecific symptom, but ileus, obstipation, or diarrhea may occur.

prolonged ileus (common)

- Prolonged ileus, although nonspecific, should raise suspicion of IAA.

anorexia/lack of appetite (common)

- More likely to be associated with appendicitis but could be associated with intra-abdominal infection.

nausea and vomiting (common)

- Patients may have accompanying symptoms of nausea and vomiting, as well as chills and night sweats.

palpable mass (uncommon)

- More frequent with appendicitis or diverticulitis in a thin person.

signs of sepsis (uncommon)

- Depending on individual systemic inflammatory response, patients may present with sepsis or septic shock. May also occur early after drainage of an IAA.
- Presentation ranges from subtle, nonspecific symptoms (e.g., feeling unwell with a normal temperature) to severe symptoms with evidence of multi-organ dysfunction and septic shock. Patients may have signs of tachycardia, tachypnea, hypotension, fever or hypothermia, poor capillary refill, mottled or ashen skin, cyanosis, newly altered mental state or reduced urine output.[20]

preoperative corticosteroid use (uncommon)

- Preoperative corticosteroid use has been associated with an increased risk of intra-abdominal sepsis after surgery for Crohn disease.[27]

Risk factors

Strong

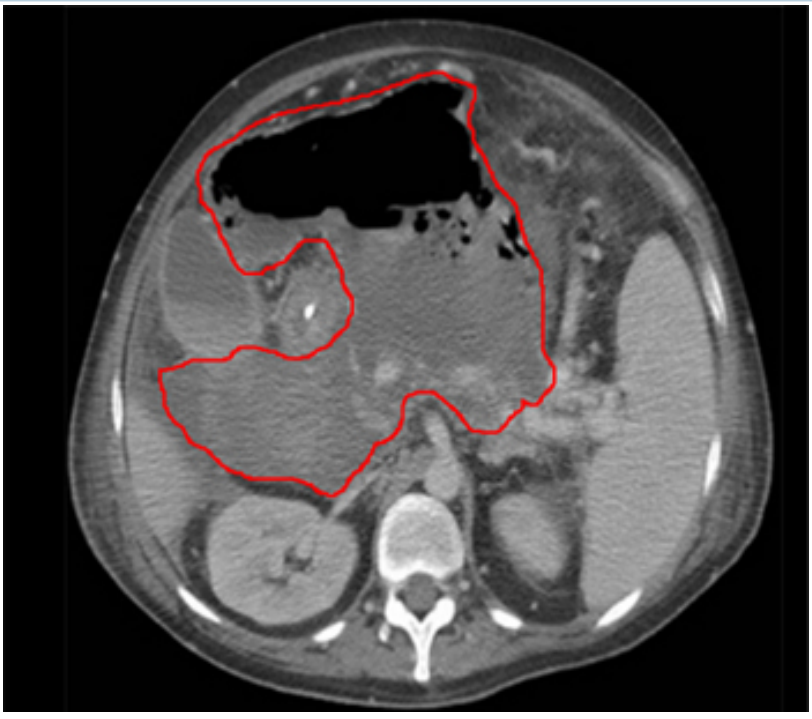
recent surgery or trauma, appendicitis, diverticulitis, or perforated ulcer

- Patients with recent abdominal surgery, trauma requiring laparotomy, and common intra-abdominal infections (appendicitis, diverticulitis) are all at risk for IAA.[1] Patients who require intestinal

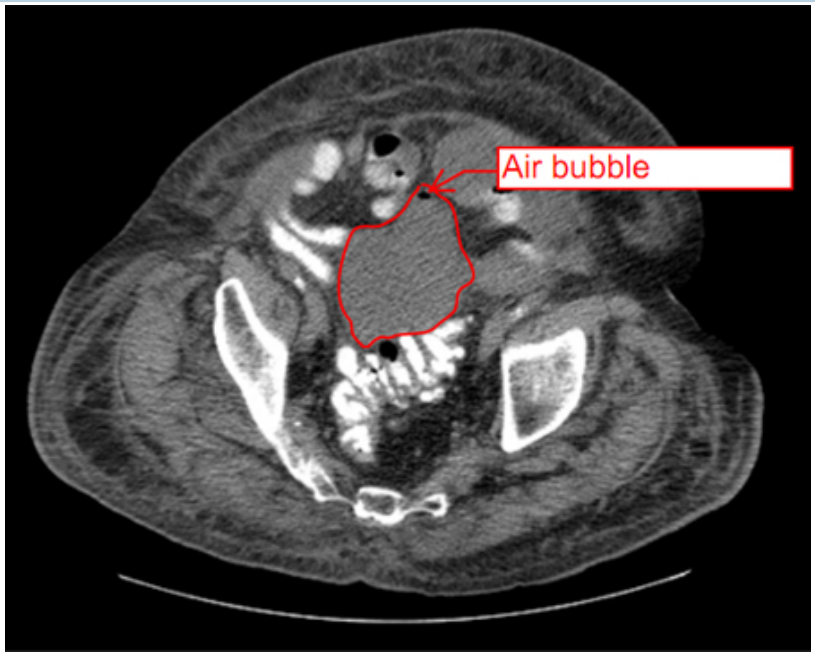
resection and anastomosis are at particular risk for anastomotic leak (1.5%) and abdominal abscess formation.[16]

Tests

1st test to order

Test	Result
WBC count • Could be useful when IAA is suspected. Although WBC count is commonly elevated, a normal count does not exclude the diagnosis. Leukopenia is more common in older and immunocompromised patients.	**elevated with increased proportion of granulocytes (left shift), persistent leukocytosis or bandemia, or leukopenia**
drainage culture • Obtaining a culture once IAA has been identified and drained is particularly important in higher-risk patients, and patients with hospital-acquired IAA, to identify potential resistant or opportunistic pathogens.[2]	**detection and confirmation of pathogenic etiologic organisms**
abdominal CT scan • Easily identifies IAA. CT scan of the abdomen and pelvis with oral and intravenous contrast is very helpful to identify IAA and anastomotic leak, and to differentiate fluid collection from a bowel loop. Identifying an air-fluid collection or visualizing a leak into the fluid collection is usually diagnostic. Rim enhancement aids the diagnosis but cannot differentiate an abscess from an organized loculated fluid collection.  *Abscess completely replacing pancreas and extending into portal hilum, with multiple gas bubbles and large air/fluid level* *From the collection of Dr Ali F. Mallat and Dr Lena M. Napolitano; used with permission*	**visualization of IAA**

Test	Result



Intra-abdominal abscess with small air bubble, secondary to perforated diverticulitis
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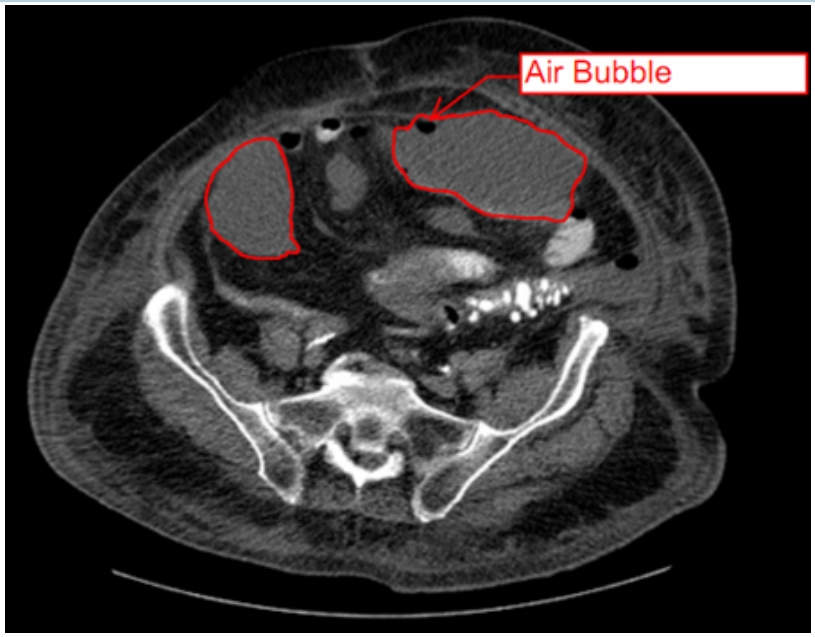

CT scan showing intra-abdominal abscess with small air bubble
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Avoid the use of noncontrast-enhanced CT imaging alongside contrast enhanced imaging; the addition of unenhanced CT does not provide additional diagnostic information and exposes patients to unnecessary radiation.[23] [24]

Other tests to consider

Test	Result
serum CRP • Elevated CRP is consistent with an inflammatory state.	**may be elevated**
serum erythrocyte sedimentation rate (ESR) • Elevated ESR is consistent with an inflammatory state.	**may be elevated**
Gram stain of abscess fluid • Obtaining a Gram stain once IAA has been identified and drained is particularly important in higher-risk patients, and patients with hospital-acquired IAA, to identify potential resistant or opportunistic pathogens.[2]	**positive for pathogenic organism**
serum glucose • Useful for managing diabetic patients with IAA, although treating the hyperglycemia before draining the abscess may be difficult. In addition, when patients become increasingly hyperglycemic with increasing insulin demands, systemic infection should be suspected, including intra-abdominal infection and IAA.	**elevated if diabetes present**
abdominal ultrasound • May be a useful aid in characterizing an intrahepatic abscess but its sensitivity is less than that of CT in IAA. Ultrasound may be useful as an initial diagnostic evaluation or when a patient cannot be moved to a CT scanner due to hemodynamic instability. Some studies have reported false positives for IAA using ultrasonography in patients with Crohn disease.[25]	**visualization of IAA**
abdominal MRI scan • May be useful in evaluating pregnant patients with acute abdominal and pelvic pain, and patients with hepatic pathology. CT scan should be the first-line diagnostic test in all other patients.[28]	**visualization of IAA**

Emerging tests

Test	Result
endoscopic ultrasound • Has been used for evaluating and draining IAA adjacent to the gastrointestinal tract, with the largest experience being with pancreatic fluid collections. Other accessible areas include the pelvis and perirectal space, and the subphrenic and perihepatic spaces. Endoscopic ultrasound requires specialized expertise; data regarding its safety and effectiveness in draining IAA are preliminary. However, endoscopic ultrasound may be useful in critically ill patients requiring bedside procedures or for IAA not amenable to other conventional therapies.[26] The experience remains limited to case reports.	**visualization of IAA**

Differentials

Condition	Differentiating signs / symptoms	Differentiating tests
Loculated intra-abdominal hematoma	• A loculated hematoma can be very challenging to differentiate from an IAA.	• Hemoperitoneum is usually accompanied by a drop in hematocrit. • Fluid sampling and culture is usually diagnostic. Measurement of fluid density by CT scan may be helpful.
Pancreatic pseudocyst	• Usually accompanied by a recent history of pancreatitis. A challenging part in the workup of a pancreatic pseudocyst is the exclusion of an infection.	• Air-fluid level on CT scan will help identify an infected pancreatic pseudocyst. Fluid sampling usually confirms the diagnosis. • Absence of debris on CT scan is helpful to exclude an abscess.
Diverticular or appendiceal phlegmon	• A phlegmon is an inflammatory mass with some associated exudates, and therefore not an actual abscess. Phlegmon may become an abscess if not treated. Both may have the same clinical picture.	• Sometimes very hard to differentiate from an abscess. CT scan may be helpful.
Intra-abdominal serum or lymph collection	• Other fluids that collect in the peritoneal cavity are lymph or serum (seroma). Sometimes very hard to differentiate from an abscess.	• CT scan may be helpful to evaluate the character of the fluid in the collection. Fluid sampling and culture is usually diagnostic.
Normal postoperative changes or postoperative fluid collections	• Differentiation of an early IAA from normal postoperative changes and fluid collections can be challenging. Most postoperative fluid collections are not infected and resolve spontaneously. However, infected fluid collections are associated with significant morbidity and mortality, and require percutaneous or surgical drainage.	• Imaging features that indicate infection include the presence of gas where none was seen previously, the development of a discrete abscess wall, and rim-shaped enhancement in the abscess wall, with either single- or double-ring signs.

Criteria

Assessing risk of adverse outcome and treatment failure[2]

It is important to determine the severity of the IAA infection and stratify the patient's risk at diagnosis, as this influences treatment.

- Assess phenotypic and physiologic factors:
  - Signs of sepsis
  - Extremes of age
  - Comorbidities
  - Extent of abdominal infection and adequacy of source control
  - Presence of resistant or opportunistic pathogen.
- Characterize patients as either low or high risk for treatment failure or mortality.
- Assess for community-acquired or health care-acquired infection.
- Patients with Surviving Sepsis Campaign criteria for sepsis or septic shock, and those with APACHE II score greater than or equal to 10, are at higher risk.
- Prolonged length of hospitalization prior to surgery for intra-abdominal infection.
- Patients with diffuse peritonitis.
- Patients with delayed source control.

Approach

IAA treatment is summarized by two steps: source control and effective antimicrobial therapy.[29] The source is usually controlled by either surgical or percutaneous drainage to completely evacuate the abscess cavity. Adequate source control, in addition to early appropriate effective antimicrobial therapy, is usually sufficient.

When the leak is large and therefore not contained, further surgical treatment is warranted to wash out the abdominal cavity and establish source control, usually by repairing the perforation or diverting the bowel proximal to the leak. Inadequate source control at the time of the initial surgery is associated with an increased mortality.[30]

Assessing risk of adverse outcome and treatment failure

- Assess phenotypic and physiologic factors:
    - Signs of sepsis: patients may present with shock; sepsis may also occur early after drainage of an IAA. Sepsis treatment guidelines have been produced by the Surviving Sepsis Campaign and remain the most widely accepted standards.[31] Current best practice is based upon evidence for care bundles in sepsis.[31] [32] [33] [34]
    - Extremes of age
    - Comorbidities
    - Extent of abdominal infection and adequacy of source control
    - Presence of resistant or opportunistic pathogen.
- Characterize patients as either low or high risk for treatment failure or mortality.
- Assess for community-acquired or health care-acquired infection.
- Patients with Surviving Sepsis Campaign criteria for sepsis or septic shock, and those with APACHE II score greater than or equal to 10, are at higher risk.
- Prolonged length of hospitalization prior to surgery for intra-abdominal infection.
- Patients with diffuse peritonitis.
- Patients with delayed source control.[2]

Percutaneous drainage

Percutaneous drainage is a successful modality in most cases.[35] For simple abscesses that are not associated with suspected malignancy or large anastomotic leaks, percutaneous drainage, if feasible, could be the first-line therapy. Percutaneous drainage can be performed using guidance from either ultrasonography or computed tomography (CT) scan.[36] Although very useful where there are only one or two IAA, percutaneous drainage is limited when the trajectory to the abscess requires cross-contaminating a different cavity, such as the pleura, or when the source of contamination is not sufficiently controlled, such as a large anastomotic breakdown.

Percutaneous drainage can be used as part of a staged surgical procedure such as in diverticulitis, Crohn disease, or appendicitis.[37] The overall success rate of staged surgical procedures using percutaneous catheter drainage is about 76%, and as high as 94% in appendicitis.[38] Appendiceal abscess is usually managed with percutaneous drainage, which is a sufficient treatment in many cases. Abscesses related to Crohn disease can often be managed initially with antibiotics and percutaneous drainage, thus avoiding emergency operations and multistage procedures. In highly selected cases, surgery might be avoided entirely.[39]

In one multicenter prospective study, a pancreatic origin of the abscess or positive yeast culture was a negative predictor of a successful percutaneous outcome, and postoperative abscesses were a favorable indicator of this outcome.[38] Although open surgical drainage may seem to have a higher mortality, this is likely due to patient selection bias.[14] In a large series of 95 patients with 107 abscesses, image-guided percutaneous drainage was performed with ultrasound in 71 procedures and with CT scan guidance in 36 procedures.[35] Immediate technical success was achieved in 107 of 107 fluid collections with the use of 8F to 14F pigtail drainage catheters, and no major complications occurred. Overall, the drainage catheter was left in place for a mean of 14.2 days. In 9 of 107 cases, percutaneous drainage was unable to resolve the fluid collection. Although percutaneous drainage is less invasive than surgery, this procedure has its own disadvantages and morbidities. Complications of percutaneous drainage include displacement or obstruction of the catheter, postprocedural septicemia, and insufficient drainage.[35] Other complications may include bleeding and inadvertent injury to surrounding structures.

The catheter can be removed when clinical findings disappear and drainage is <10 mL in 24 hours. Before removing the catheter, it should be ensured that cessation of drainage is not due to catheter blockage.[40]

Surgical source control

The surgical procedure depends on the cause of the IAA.[29]

- In the case of a gastric or duodenal perforation, repair with a Graham patch with unroofing and drainage of the associated abscess may suffice.
- A small-bowel perforation may require a primary repair or resection, along with either a primary anastomosis or, at times, a double barrel ostomy.
- Diverticulitis may require resection of the diseased colon and either end colostomy (a Hartmann procedure) or primary anastomosis with or without diverting ileostomy.[41] Laparoscopic lavage with drainage has also been reported to be feasible in patients with purulent peritonitis, but is controversial as trials have shown conflicting results.[42] [43] [44]
- Colonic anastomotic leaks may be treated with proximal diversion and drainage, without taking down the anastomosis.[45] When anastomotic leaks occur, the clinical picture usually dictates the course: a patient in septic shock should be resuscitated, then re-explored to determine where the leaking anastomosis can be taken down, and a proximal diversion should be performed. A single operation may not sufficiently control the source, and a 2-stage or multistage operation may be required. The presence of hemodynamic instability may contraindicate reestablishing bowel continuity, and a second laparotomy is usually planned within 24 to 48 hours. Negative pressure wound therapy can be considered if the abdomen is left open.[46]

Antimicrobial therapy selection

Early parenteral empiric antimicrobial therapy is critically important in treating IAA. Appropriate antimicrobial therapy is defined as the use of an antimicrobial that is effective against all of the pathogenic organisms isolated from the IAA. In patients with sepsis or septic shock, parenteral empiric antibiotics with broad-spectrum coverage should be initiated immediately after the diagnosis is made, as outcome worsens with each hour delay of antimicrobial therapy.[47] See Sepsis in Adults (Management Approach) .

Two meta-analyses have demonstrated reduced short term mortality using an (off-label) extended-duration infusion of beta-lactams after the initial bolus.[48] [49]

Appropriate cultures should be obtained before initiating antibiotic therapy, but should not prevent prompt administration of antimicrobial therapy.[2] [31] Antibiotics should be given before surgical or percutaneous drainage.

The frequently isolated pathogens in intra-abdominal infections are as follows.

- Gram-negative bacteria such as *Escherichia coli* , *Enterobacter* , *Klebsiella* , *Proteus* , or *Pseudomonas* .
- Gram-positive bacteria such as *Streptococcus* , *Staphylococcus aureus* , or *Enterococcus* .
- Anaerobes such as *Bacteroides* and *Clostridium* . The most prevalent anaerobic organism in intra-abdominal infections is *Bacteroides fragilis* , likely present in one third to one half of these infections.
- *Candida* . The incidence of *Candida* infections depends on the presence of predisposing factors such as immunodeficiency, prior antimicrobial treatment, and peritoneal dialysis. It is more common in tertiary peritonitis (recurrent intra-abdominal infection after initial surgical and antimicrobial therapy of secondary bacterial peritonitis) and in abscesses related to duodenal pathology.

The breadth of the empiric coverage for these pathogens can depend upon the severity of illness, medical comorbidities, and adequacy of source control.

Non-high-risk patients

Non-high-risk patients with mild-to-moderate severity community-acquired IAA can be treated with either single-agent (e.g., ertapenem or moxifloxacin) or combination (e.g., metronidazole plus a cephalosporin or a quinolone) regimens, all of which are equally effective.[2]

Empiric antibiotics should cover gram-negative aerobic and facultative bacilli, and enteric gram-positive streptococci.[2] In the presence of distal small bowel, appendiceal, colonic, and proximal gastrointestinal perforations with obstruction or paralytic ileus, these antibiotics should be active against obligate anaerobic bacilli.[2]

Ampicillin/sulbactam, and cefotetan and clindamycin, should not be used due to the resistance of *E coli* and *Bacteroides fragilis* to these antibiotics, respectively.[2]

Patients may be switched to targeted antibiotic therapy once culture results are available.

High-risk patients

High-risk patients, or those with severe community-acquired IAA, should be started on broad-spectrum antimicrobial therapy with coverage of possible multidrug-resistant gram-negative bacteria, including *Pseudomonas aeruginosa* , and then commit to de-escalation of antimicrobial therapy once the culture and susceptibility results are available. Specific decisions regarding optimal antimicrobial therapy should be based, in part, on local antibiograms and knowledge of common organisms in the hospital or community. A carbapenem or piperacillin/tazobactam should be used as a single-agent therapy; if combination therapy is desired, metronidazole should be combined with a cephalosporin.[2]

Empiric coverage of *Enterococcus* should be considered in these patients. Coverage of methicillin-resistant *Staphylococcus aureus* (MRSA) and *Candida* is only recommended if there is evidence of these infections.[2]

In adult patients with health care-associated complicated intra-abdominal infection, to achieve empiric coverage of the likely etiologic pathogens, multidrug regimens, including broad-spectrum agents with activity against gram-negative aerobic and facultative bacilli, should be used.[2]

A carbapenem, piperacillin/tazobactam, a cephalosporin, or an aminoglycoside is recommended with metronidazole. Empiric antienterococcal therapy (e.g., piperacillin/tazobactam) against *Enterococcus faecalis* is recommended, especially for patients with postoperative infection or prosthetic intravascular materials, those who have previously received cephalosporins or other antienterococcal antibiotics, and for immunocompromised patients.[2]

Multidrug-resistant pathogens are becoming an increasing concern. Vancomycin-resistant enterococci (VRE) are emerging pathogens that are resistant to many standard antibiotics. Data on the efficacy and safety of specific antimicrobials are limited, particularly with regard to the treatment of intra-abdominal infections. Linezolid is approved for the treatment of VRE infections; daptomycin and tigecycline may also be used.[2] Other antimicrobials are on the horizon but have not yet been approved by the Food and Drug Administration (FDA).[50] [51]

Adjunctive vancomycin for MRSA coverage is indicated in patients known to be colonized with MRSA or those at risk of MRSA infection because of prior treatment failure or significant antibiotic exposure.[2]

Other multidrug-resistant organisms include gram-negative bacilli producing extended-spectrum beta-lactamase (ESBL). Carbapenems are the first-line option for treating ESBL bacteria, and ertapenem may be preferred for community-acquired infections.[52]

Other options are available depending upon the susceptibility of the strain. Development of new drugs is important given the emergence of carbapenem-resistant Enterobacteriaceae (CRE). Options for treatment include, but are not limited to, colistimethate (colistin) and tigecycline. Ceftazidime/avibactam has been approved by the FDA for the treatment of complicated intra-abdominal infections when used in combination with metronidazole. It is recommended for higher-risk patients with strongly suspected or proven infection with *Klebsiella pneumoniae* carbapenemase (KPC)-producing Enterobacteriaceae, for which other agents are not suitable.[2]

Antifungal therapy is only recommended if *Candida* is grown from intra-abdominal cultures. *C albicans* should be treated with fluconazole, while an echinocandin should be used for fluconazole-resistant *Candida* species and in critically ill patients.[2]

Duration of antimicrobial therapy

Duration of antimicrobial therapy depends on the adequacy of source control and the patient's response to therapy (i.e., resolution of all signs and symptoms of infection, such as fever, leukocytosis, and abdominal pain, and resolution of the IAA by repeat diagnostic imaging).

If the patient is not responding to empiric broad-spectrum antimicrobial therapy, diagnostic imaging and cultures should be repeated, and changing the antimicrobial management considered.

A successful trial of shorter-duration antimicrobial therapy has highlighted the importance of source control. One multicenter randomized trial compared the duration of antimicrobials chosen based on resolution of clinical signs and symptoms of infection versus 4 days after source control for complicated intra-abdominal infections. The study found that patients receiving 4 days of antimicrobials had a shorter duration of therapy and had no difference in the composite outcome of surgical site infection, recurrent intra-abdominal infection, and death within 30 days.[53]

Consider limiting antimicrobial therapy to 7 days in patients with secondary bacteremia due to intra-abdominal infection, who have undergone adequate source control and are no longer bacteremic.[2]

Consideration should be given to limiting antibiotic therapy to 5 to 7 days in patients in whom a definitive source control procedure cannot be performed.[2] In those who demonstrate persistent clinical signs of infection (fever, leukocytosis, changes in bowel function) after 5 to 7 days of antibiotics, reassessment of source control should be considered.[2]

Treatment algorithm overview

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute (summary)		
community-acquired intra-abdominal abscess: non-high risk, mild-to-moderate severity		
	1st	source control
	plus	empiric intravenous broad-spectrum antibiotic therapy
	plus	switch to targeted antibiotic therapy once cultures known
	2nd	further surgical treatment
community-acquired intra-abdominal abscess: high risk or high severity		
	1st	source control
	plus	empiric intravenous broad-spectrum antibiotic therapy
	adjunct	antienterococcal coverage
	adjunct	antifungal therapy
	adjunct	methicillin-resistant Staphylococcus aureus (MRSA) coverage
	adjunct	extended-spectrum beta-lactamase (ESBL)-producing bacteria coverage
	adjunct	carbapenem-resistant Enterobacteriaceae (CRE) coverage
	plus	switch to targeted antibiotic therapy once cultures known
	2nd	further surgical treatment
health care-associated intra-abdominal abscess		
	1st	source control
	plus	empiric intravenous broad-spectrum antibiotic therapy
	plus	antienterococcal coverage
	adjunct	antifungal therapy
	adjunct	methicillin-resistant Staphylococcus aureus (MRSA) coverage

Acute		(summary)
	adjunct	extended-spectrum beta-lactamase (ESBL)-producing bacteria coverage
	adjunct	carbapenem-resistant Enterobacteriaceae (CRE) coverage
	plus	switch to targeted antibiotic therapy once cultures known
	2nd	further surgical treatment

Treatment algorithm

Please note that formulations/routes and doses may differ between drug names and brands, drug formularies, or locations. Treatment recommendations are specific to patient groups: see disclaimer

Acute

community-acquired intra-abdominal abscess: non-high risk, mild-to-moderate severity

1st **source control**

» CT- or ultrasound-guided percutaneous drainage is the first-line therapy for simple abscesses not associated with suspected malignancy or large anastomotic leaks. Useful if only one or two IAA are present (e.g., appendiceal abscess) but limited when trajectory to the abscess requires cross-contaminating a different cavity (e.g., pleura) or when the source of contamination is not sufficiently controlled (e.g., large anastomotic breakdown). Complications include catheter displacement or obstruction, postprocedural septicemia, and insufficient drainage; may include bleeding and inadvertent injury to surrounding structures.[35] Catheter can be removed when clinical findings disappear and drainage is <10 mL/24 hours; before removal, catheter blockage should be excluded.[40]

» Surgical drainage procedure depends on the cause of IAA. When anastomotic leaks occur, a single operation may not suffice, and a 2-stage or multistage operation may be required. Hemodynamic instability may contraindicate reestablishing bowel continuity, and a second laparotomy is usually planned within 24 to 48 hours. Negative pressure wound therapy can be considered if the abdomen is left open.[46]

plus **empiric intravenous broad-spectrum antibiotic therapy**

Treatment recommended for ALL patients in selected patient group

Primary options

» ertapenem: 1 g intravenously every 24 hours

OR

» moxifloxacin: 400 mg intravenously every 24 hours

OR

Acute

» cefuroxime sodium: 1.5 g intravenously every 8 hours
-or-
» ceftriaxone: 1-2 g intravenously every 12-24 hours
-or-
» cefotaxime: 1-2 g intravenously every 6-8 hours
--AND--
» metronidazole: 500 mg intravenously every 8-12 hours

OR

» ciprofloxacin: 400 mg intravenously every 12 hours
-or-
» levofloxacin: 750 mg intravenously every 24 hours
--AND--
» metronidazole: 500 mg intravenously every 8-12 hours

» Patients of younger age with no comorbidities, immunosuppression, or organ dysfunction with mild-to-moderate infection and adequate source control (e.g., perforated/abscessed appendicitis), and an Acute Physiology and Chronic Health Evaluation (APACHE) II score <10, are considered non-high risk. [Surgical Infection Society: intra-abdominal infection (IAI) high versus low risk] (http://www.sisna.org/iai-high-vs-low-risk)

» Parenteral empiric antibiotics with broad-spectrum coverage should be initiated promptly. Appropriate cultures should be obtained before initiating antibiotic therapy, but should not prevent their prompt administration.[31]

» Two meta-analyses have demonstrated reduced short term mortality using an (off-label) extended-duration infusion of beta-lactams after the initial bolus.[48] [49] Doses are not reflected here.

» Antibiotics should be given before surgical or percutaneous drainage.

» Treatment can be with either single-agent or combination regimens, all of which are equally effective. Local resistance patterns should be considered.

» Duration of antimicrobial therapy depends on the adequacy of source control and the response to therapy (i.e., resolution of signs

Acute

and symptoms of infection, and IAA on repeat diagnostic imaging). A multicenter trial suggests that 4 days of antimicrobial therapy may be sufficient in the setting of adequate source control.[53]

plus **switch to targeted antibiotic therapy once cultures known**

Treatment recommended for ALL patients in selected patient group

» Patients may be switched to targeted antibiotic therapy once culture results are available.

» If the abscess is too small to drain or aspirate (for culture) or the patient has been on antibiotics prior to drainage and there is no growth of organisms, then the patient would remain on empiric antibiotics, assuming there is clinical improvement.

2nd **further surgical treatment**

» Required if the patient is unresponsive to initial treatment or there is an uncontained leak. A single operation may not sufficiently control the source, and a 2-stage or multistage operation may be required.

» Hemodynamic instability may contraindicate reestablishing bowel continuity, and a second laparotomy is usually planned within 24 to 48 hours. Negative pressure wound therapy can be considered if the abdomen is left open.[46]

» In pyogenic hepatic abscess due to biliary sepsis, a concomitant biliary drainage procedure, whether percutaneous or endoscopic, should be contemplated.

community-acquired intra-abdominal abscess: high risk or high severity

1st **source control**

» CT- or ultrasound-guided percutaneous drainage is the first-line therapy for simple abscesses not associated with suspected malignancy or large anastomotic leaks. Useful if only one or two IAA are present (e.g., appendiceal abscess) but limited when trajectory to the abscess requires cross-contaminating a different cavity (e.g., pleura) or when the source of contamination is not sufficiently controlled (e.g., large anastomotic breakdown).

» Complications include catheter displacement or obstruction, postprocedural septicemia, and

Acute

insufficient drainage; may include bleeding and inadvertent injury to surrounding structures.[35]

» Catheter can be removed when clinical findings disappear and drainage is <10 mL/24 hours; before removal, catheter blockage should be excluded.[40]

» Surgical drainage procedure depends on the cause of IAA. When anastomotic leaks occur, a single operation may not suffice, and a 2-stage or multistage operation may be required. Hemodynamic instability may contraindicate reestablishing bowel continuity, and a second laparotomy is usually planned within 24 to 48 hours. Negative pressure wound therapy can be considered if the abdomen is left open.[46]

plus **empiric intravenous broad-spectrum antibiotic therapy**

Treatment recommended for ALL patients in selected patient group

Primary options

» piperacillin/tazobactam: 3.375 g intravenously every 6 hours
Dose consists of 3 g of piperacillin plus 0.375 g tazobactam.

OR

» imipenem/cilastatin: 500-1000 mg intravenously every 6-8 hours
Dose refers to imipenem component.

OR

» meropenem: 1 g intravenously every 8 hours

OR

» ertapenem: 1 g intravenously every 24 hours

OR

» ceftazidime sodium: 2 g intravenously every 8 hours
-or-
» cefepime: 2 g intravenously every 8-12 hours
--AND--

Acute

» metronidazole: 500 mg intravenously every 8-12 hours

» Patients of advanced age with comorbidities (e.g., malignancy), organ dysfunction, malnutrition, low albumin, or immunosuppression, an APACHE II score ≥10, sepsis, or septic shock, and with peritoneal involvement and/or diffuse peritonitis and inadequate source control are considered high risk. [Surgical Infection Society: intra-abdominal infection (IAI) high versus low risk] (http://www.sisna.org/iai-high-vs-low-risk)

» Delay in initial intervention (>24 hours) or prolonged hospitalization prior to surgery for intra-abdominal infection also place patients at high risk.[2]

» In patients with sepsis or septic shock, parenteral empiric antibiotics with broad-spectrum coverage should be initiated immediately after diagnosis as outcome worsens with each hour delay of antimicrobial therapy.[47]

» Two meta-analyses have demonstrated reduced short term mortality using an (off-label) extended-duration infusion of beta-lactams after the initial bolus.[48] [49] Doses are not reflected here.

» Local resistance patterns should be considered.

» Appropriate cultures should be obtained before initiating antibiotic therapy, but should not prevent their prompt administration.[31]

» Antibiotics should be given before surgical or percutaneous drainage.

» Duration of antimicrobial therapy depends on the adequacy of source control and the response to therapy (i.e., resolution of signs and symptoms of infection, and IAA on repeat diagnostic imaging). A multicenter trial suggests that 4 days of antimicrobial therapy may be sufficient in the setting of adequate source control.[53]

adjunct **antienterococcal coverage**

Treatment recommended for SOME patients in selected patient group

Primary options

» ampicillin: 2 g intravenously every 4-6 hours

OR

Acute

» piperacillin/tazobactam: 3.375 g intravenously every 6 hours
Dose consists of 3 g of piperacillin plus 0.375 g tazobactam.

OR

» vancomycin: 15-20 mg/kg intravenously every 8-12 hours

Secondary options

» linezolid: 600 mg intravenously every 12 hours

OR

» daptomycin: 8-12 mg/kg intravenously every 24 hours
Higher doses than those approved for skin and soft tissue infections are usually required for vancomycin-resistant enterococci. This is an off-label use. Data advocates for the safety and efficacy of daptomycin at a dose of 8-12 mg/kg/day based on the minimum inhibitory concentration (MIC).[54] [55]
Consult specialist for further guidance on dose.

OR

» tigecycline: 100 mg intravenously initially as a loading dose, followed by 50 mg every 12 hours

» Empiric coverage of *Enterococcus* should be considered in high-risk patients.[2]

» If piperacillin/tazobactam has been used as part of the empiric broad-spectrum antibiotic coverage then *Enterococcus* is already covered.

» Vancomycin-resistant enterococci (VRE) are emerging pathogens that are resistant to many standard antibiotics. Data on the efficacy and safety of specific antimicrobials are limited, particularly with regard to the treatment of intra-abdominal infections. Linezolid is approved for the treatment of VRE infections; daptomycin and tigecycline may also be used.[2]

» Two meta-analyses have demonstrated reduced short term mortality using an (off-label) extended-duration infusion of beta-lactams after

Acute

the initial bolus.[48] [49] Doses are not reflected here.

adjunct **antifungal therapy**

Treatment recommended for SOME patients in selected patient group

Primary options

» fluconazole: 400-800 mg/day intravenously

Secondary options

» caspofungin: 70 mg intravenously on day 1, followed by 50 mg every 24 hours

OR

» anidulafungin: 200 mg intravenously on day 1, followed by 100 mg every 24 hours

OR

» micafungin: 100 mg intravenously every 24 hours

OR

» voriconazole: 6 mg/kg intravenously every 12 hours on day 1, followed by 4 mg/kg every 12 hours

» Coverage of *Candida* is only recommended if there is evidence of infection.[2]

» *C albicans* should be treated with fluconazole, while an echinocandin should be used for fluconazole-resistant *Candida* species and in critically ill patients.[2]

adjunct **methicillin-resistant Staphylococcus aureus (MRSA) coverage**

Treatment recommended for SOME patients in selected patient group

Primary options

» vancomycin: 15-20 mg/kg intravenously every 8-12 hours

» Coverage of methicillin-resistant *Staphylococcus aureus* (MRSA) is only recommended if there is evidence of infection.[2]

» Adjunctive vancomycin for MRSA coverage is indicated (if not already in use) in patients known to be colonized with MRSA, or those at risk

Acute

of MRSA infection because of prior treatment failure or significant antibiotic exposure.[2]

adjunct **extended-spectrum beta-lactamase (ESBL)-producing bacteria coverage**

Treatment recommended for SOME patients in selected patient group

Primary options

» ertapenem: 1 g intravenously every 24 hours

OR

» imipenem/cilastatin: 500-1000 mg intravenously every 6-8 hours
Dose refers to imipenem component.

OR

» meropenem: 1 g intravenously every 8 hours

Secondary options

» ceftolozane/tazobactam: 1.5 g intravenously every 8 hours
Dose consists of 1 g ceftolozane plus 0.5 g tazobactam

-and-
» metronidazole: 500 mg intravenously every 8-12 hours

» ESBL-producing bacteria are resistant to many extended-spectrum cephalosporins as well as aminoglycosides, sulfonamides, and fluoroquinolones. Carbapenems are the first-line option (if not already in use).[52] Novel combinations of cephalosporins and beta-lactamase inhibitors, such as ceftolozane/tazobactam, have shown some success against some ESBL strains in one phase 3 trial but further trials are needed.[56] Ceftolozane/tazobactam is used in combination with metronidazole.

» Two meta-analyses have demonstrated reduced short term mortality using an (off-label) extended-duration infusion of beta-lactams after the initial bolus.[48] [49] Doses are not reflected here.

adjunct **carbapenem-resistant Enterobacteriaceae (CRE) coverage**

Acute

Treatment recommended for SOME patients in selected patient group

Primary options

» tigecycline: 100 mg intravenously initially as a loading dose, followed by 50 mg every 12 hours

OR

» colistimethate: consult specialist for guidance on dose

OR

» polymyxin B: 15,000-25,000 units/kg intravenously given in divided doses every 12 hours

Secondary options

» ceftazidime/avibactam: 2.5 g intravenously every 8 hours
Dose consists of 2 g ceftazidime and 0.5 g avibactam

-and-
» metronidazole: 500 mg intravenously every 8-12 hours

» Options for treatment are limited as many of the carbapenem-resistant bacteria also harbor resistance to other antibiotics. Colistimethate (colistin) or tigecycline are recommended, and polymyxin B and ceftazidime/avibactam may also be used.

» Ceftazidime/avibactam has been approved by the Food and Drug Administration (FDA) for the treatment of complicated intra-abdominal infections when used in combination with metronidazole. It is recommended for higher-risk patients with strongly suspected or proven infection with *Klebsiella pneumoniae* carbapenemase (KPC)-producing Enterobacteriaceae, for which other agents are not suitable.[2]

plus **switch to targeted antibiotic therapy once cultures known**

Treatment recommended for ALL patients in selected patient group

» Patients may be switched to targeted antibiotic therapy once culture results are available.

Acute

» If the abscess is too small to drain or aspirate (for culture) or the patient has been on antibiotics prior to drainage and there is no growth of organisms, then the patient would remain on empiric antibiotics, assuming there is clinical improvement.

2nd **further surgical treatment**

» Required if the patient is unresponsive to initial treatment or there is an uncontained leak. A single operation may not sufficiently control the source, and a 2-stage or multistage operation may be required.

» Hemodynamic instability may contraindicate reestablishing bowel continuity, and a second laparotomy is usually planned within 24 to 48 hours. Negative pressure wound therapy can be considered if the abdomen is left open.[46]

» In pyogenic hepatic abscess due to biliary sepsis, a concomitant biliary drainage procedure, whether percutaneous or endoscopic, should be contemplated.

health care-associated intra-abdominal abscess

1st **source control**

» CT- or ultrasound-guided percutaneous drainage is the first-line therapy for simple abscesses not associated with suspected malignancy or large anastomotic leaks. Useful if only one or two IAA are present (e.g., appendiceal abscess) but limited when trajectory to the abscess requires cross-contaminating a different cavity (e.g., pleura) or when the source of contamination is not sufficiently controlled (e.g., large anastomotic breakdown). Complications include catheter displacement or obstruction, postprocedural septicemia, and insufficient drainage; may include bleeding and inadvertent injury to surrounding structures.[35] Catheter can be removed when clinical findings disappear and drainage is <10 mL/24 hours; before removal, catheter blockage should be excluded.[40]

» Surgical drainage procedure depends on the cause of IAA. When anastomotic leaks occur, a single operation may not suffice, and a 2-stage or multistage operation may be required. Hemodynamic instability may contraindicate reestablishing bowel continuity, and a second laparotomy is usually planned within 24 to 48 hours. Negative pressure wound therapy can be considered if the abdomen is left open.[46]

Acute

plus **empiric intravenous broad-spectrum antibiotic therapy**

Treatment recommended for ALL patients in selected patient group

Primary options

» meropenem: 1 g intravenously every 8 hours
-or-
» imipenem/cilastatin: 500-1000 mg intravenously every 6-8 hours
Dose refers to imipenem component.

-or-
» piperacillin/tazobactam: 3.375 g intravenously every 6 hours
Dose consists of 3 g of piperacillin plus 0.375 g tazobactam.

-or-
» ceftazidime sodium: 2 g intravenously every 8 hours
-or-
» cefepime: 2 g intravenously every 8-12 hours
-or-
» gentamicin: 5-7 mg/kg intravenously every 24 hours
-or-
» tobramycin: 5-7 mg/kg intravenously every 24 hours
-or-
» amikacin: 15-20 mg/kg intravenously every 24 hours

--AND--

» metronidazole: 500 mg intravenously every 8-12 hours

» In patients with sepsis or septic shock, parenteral empiric antibiotics with broad-spectrum coverage should be initiated immediately after diagnosis as outcome worsens with each hour delay of antimicrobial therapy.[47] Appropriate cultures should be obtained before initiating antibiotic therapy, but should not prevent their prompt administration.[31]

» Two meta-analyses have demonstrated reduced short term mortality using an (off-label) extended-duration infusion of beta-lactams after the initial bolus.[48] [49] Doses are not reflected here.

» Antibiotics should be given before surgical or percutaneous drainage.

Acute

» Duration of antimicrobial therapy depends on the adequacy of source control and the response to therapy (i.e., resolution of signs and symptoms of infection, and IAA on repeat diagnostic imaging). A multicenter trial suggests that 4 days of antimicrobial therapy may be sufficient in the setting of adequate source control.[53]

plus **antienterococcal coverage**

Treatment recommended for ALL patients in selected patient group

Primary options

» ampicillin: 2 g intravenously every 4-6 hours

OR

» piperacillin/tazobactam: 3.375 g intravenously every 6 hours
Dose consists of 3 g of piperacillin plus 0.375 g tazobactam.

OR

» vancomycin: 15-20 mg/kg intravenously every 8-12 hours

Secondary options

» linezolid: 600 mg intravenously every 12 hours

OR

» daptomycin: 8-12 mg/kg intravenously every 24 hours
Higher doses than those approved for skin and soft tissue infections are usually required for vancomycin-resistant enterococci. This is an off-label use. Data advocates for the safety and efficacy of daptomycin at a dose of 8-12 mg/kg/day based on the minimum inhibitory concentration (MIC).[54] [55]
Consult specialist for further guidance on dose.

OR

» tigecycline: 100 mg intravenously initially as a loading dose, followed by 50 mg every 12 hours

» Empiric antienterococcal therapy against *Enterococcus faecalis* is recommended,

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especially for patients with postoperative infection or prosthetic intravascular materials, those who have previously received cephalosporins or other antienterococcal antibiotics, and for immunocompromised patients.[2]

» If piperacillin/tazobactam has been used as part of the empiric broad-spectrum antibiotic coverage then *Enterococcus* is already covered.

» Vancomycin-resistant enterococci (VRE) are emerging pathogens that are resistant to many standard antibiotics.

» Data on the efficacy and safety of specific antimicrobials are limited, particularly with regard to the treatment of intra-abdominal infections. Linezolid is approved for the treatment of VRE infections, and daptomycin or tigecycline may also be used.[2]

» Two meta-analyses have demonstrated reduced short term mortality using an (off-label) extended-duration infusion of beta-lactams after the initial bolus.[48] [49] Doses are not reflected here.

adjunct **antifungal therapy**

Treatment recommended for SOME patients in selected patient group

Primary options

» fluconazole: 400-800 mg/day intravenously

Secondary options

» caspofungin: 70 mg intravenously on day 1, followed by 50 mg every 24 hours

OR

» anidulafungin: 200 mg intravenously on day 1, followed by 100 mg every 24 hours

OR

» micafungin: 100 mg intravenously every 24 hours

OR

» voriconazole: 6 mg/kg intravenously every 12 hours on day 1, followed by 4 mg/kg every 12 hours

Acute

Tertiary options

» amphotericin B deoxycholate: 0.6 to 1 mg/kg intravenously every 24 hours

» Antifungal therapy is only recommended if *Candida* is grown from intra-abdominal cultures.

» *C albicans* should be treated with fluconazole, while an echinocandin should be used for fluconazole-resistant *Candida* species and in critically ill patients.[2]

adjunct **methicillin-resistant Staphylococcus aureus (MRSA) coverage**

Treatment recommended for SOME patients in selected patient group

Primary options

» vancomycin: 15-20 mg/kg intravenously every 8-12 hours

» Coverage of methicillin-resistant *Staphylococcus aureus* (MRSA) is only recommended if there is evidence of infection.[2]

» Adjunctive vancomycin for MRSA coverage is indicated (if not already in use) in patients known to be colonized with MRSA, or those at risk of MRSA infection because of prior treatment failure or significant antibiotic exposure.[2]

adjunct **extended-spectrum beta-lactamase (ESBL)-producing bacteria coverage**

Treatment recommended for SOME patients in selected patient group

Primary options

» imipenem/cilastatin: 500-1000 mg intravenously every 6-8 hours
Dose refers to imipenem component.

OR

» meropenem: 1 g intravenously every 8 hours

Secondary options

» ceftolozane/tazobactam: 1.5 g intravenously every 8 hours
Dose consists of 1 g ceftolozane plus 0.5 g tazobactam

-and-

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» metronidazole: 500 mg intravenously every 8-12 hours

» ESBL-producing bacteria are resistant to many extended-spectrum cephalosporins as well as aminoglycosides, sulfonamides, and fluoroquinolones.

» Carbapenems are the first-line option (if not already in use), although ertapenem is not preferred for hospital-acquired ESBL-producing bacterial infections because of lack of significant activity against *Pseudomonas* or *Acinetobacter* .[52]

» Non-carbapenem antibiotics have been used to treat infections with ESBL-producing bacteria, but there is reluctance to recommend them because of observational studies showing clinical failure of such antibiotics, even when susceptibility in vitro has been demonstrated. Novel combinations of cephalosporins and beta-lactamase inhibitors, such as ceftolozane/tazobactam, have shown some success against some ESBL strains in one phase 3 trial but further trials are needed.[56] Ceftolozane/tazobactam is used in combination with metronidazole.

» Two meta-analyses have demonstrated reduced short term mortality using an (off-label) extended-duration infusion of beta-lactams after the initial bolus.[48] [49] Doses are not reflected here.

adjunct **carbapenem-resistant Enterobacteriaceae (CRE) coverage**

Treatment recommended for SOME patients in selected patient group

Primary options

» tigecycline: 100 mg intravenously initially as a loading dose, followed by 50 mg every 12 hours

OR

» colistimethate: consult specialist for guidance on dose

OR

» polymyxin B: 15,000-25,000 units/kg intravenously given in divided doses every 12 hours

Acute

Secondary options

» ceftazidime/avibactam: 2.5 g intravenously every 8 hours
Dose consists of 2 g ceftazidime and 0.5 g avibactam

-and-
» metronidazole: 500 mg intravenously every 8-12 hours

» Options for treatment are limited as many of the carbapenem-resistant bacteria also harbour resistance to other antibiotics. Colistimethate (colistin) or tigecycline are recommended, and polymyxin B and ceftazidime/avibactam may also be used.

» Ceftazidime/avibactam has been approved by the Food and Drug Administration (FDA) for the treatment of complicated intra-abdominal infections when used in combination with metronidazole. It is recommended for higher-risk patients with strongly suspected or proven infection with *Klebsiella pneumoniae* carbapenemase (KPC)-producing Enterobacteriaceae, for which other agents are not suitable.[2]

plus **switch to targeted antibiotic therapy once cultures known**

Treatment recommended for ALL patients in selected patient group

» Patients may be switched to targeted antibiotic therapy once culture results are available.

» If the abscess is too small to drain or aspirate (for culture) or the patient has been on antibiotics prior to drainage and there is no growth of organisms, then the patient would remain on empiric antibiotics, assuming there is clinical improvement.

2nd **further surgical treatment**

» Required if the patient is unresponsive to initial treatment or there is an uncontained leak. A single operation may not sufficiently control the source, and a 2-stage or multistage operation may be required.

» Hemodynamic instability may contraindicate reestablishing bowel continuity, and a second laparotomy is usually planned within 24 to 48 hours. Negative pressure wound therapy can be considered if the abdomen is left open.[46]

Acute

» In pyogenic hepatic abscess due to biliary sepsis, a concomitant biliary drainage procedure, whether percutaneous or endoscopic, should be contemplated.

Emerging

Endoscopic ultrasound-guided drainage

Endoscopic ultrasound-guided drainage has been suggested for internal drainage of IAAs, such as pancreatic abscesses. Other potentially accessible areas include the pelvis and perirectal space, and the subphrenic and perihepatic spaces. However, the technique has not been rigorously compared with percutaneous drainage, and risks include severe bleeding and perforation. Endoscopic ultrasound requires specialized expertise; data regarding its safety and effectiveness in draining IAA are preliminary. However, endoscopic ultrasound may be useful in critically ill patients requiring bedside procedures or for IAA not amenable to other conventional therapies. The experience remains limited to case reports; however, interest persists.[26] [57]

Eravacycline

Eravacycline, an intravenous fluorcycline antibiotic (in the tetracycline class of antibiotics), has been approved by the Food and Drug Administration and the European Medicines Agency for adults with complicated intra-abdominal infections that involve the following organisms: *Escherichia coli* , *Klebsiella pneumoniae* , *Citrobacter freundii* , *Enterobacter cloacae* , *Klebsiella oxytoca* , *Enterococcus faecalis* , *Enterococcus faecium* , *Staphylococcus aureus* , *Streptococcus anginosus* group, *Clostridium perfringens* , *Bacteroides* species, and *Parabacteroides distasonis* . Eravacycline has shown activity against organisms that express tetracycline-specific resistance mechanisms. Like other tetracycline antibiotics, it may cause discoloration of deciduous teeth.

Meropenem/vaborbactam

Meropenem/vaborbactam is a fixed combination intravenous antibiotic containing meropenem (an antibiotic in the carbapenem class of antibiotics) and vaborbactam (a beta-lactamase inhibitor). The European Medicines Agency has approved the drug to treat complicated intra-abdominal infections. The approval provides an option for organisms that are resistant to carbapenem antibiotics, which has been increasing recently, particularly in gram-negative bacteria. Meropenem/vaborbactam is available in the US, but is currently approved only for the treatment of complicated urinary tract infections.

Imipenem/cilastatin/relebactam

Imipenem/cilastatin/relebactam is a fixed combination intravenous antibiotic containing imipenem (an antibiotic in the carbapenem class of antibiotics), cilastatin (a renal dihydropeptidase inhibitor, which reduces the nephrotoxicity associated with imipenem and has no antimicrobial activity), and relebactam (a beta-lactamase inhibitor). The Food and Drug Administration has approved this drug to treat adults with complicated intra-abdominal infection when there are limited or no alternative antibacterial drugs available to treat the infection. It is not available in Europe as yet.

Primary prevention

There are no preventive strategies for primary IAA (i.e., related to perforated appendicitis and diverticulitis). One Cochrane review examined the effect of abdominal drainage on the prevention of intraperitoneal abscesses after open appendectomy for complicated appendicitis. The effectiveness of abdominal drainage was unclear, and the evidence was of low certainty. Abdominal drainage may increase the 30-day complication rate and length of hospital stay; larger studies are needed to more reliably determine the effects of drainage on morbidity and mortality outcomes.[17] There is no evidence from randomized controlled trials to confirm or refute the use of prophylactic antibiotics for penetrating abdominal trauma.[18] For patients with secondary IAA (i.e., postoperative or related to spread through bacteremia), potential strategies for prevention of IAA include adequate source control of the initial complicated intra-abdominal infection and early initiation of appropriate empiric antimicrobial therapy.

The host response to intra-abdominal infection depends on five key factors: 1) inoculum size, 2) virulence of the contaminating organisms, 3) presence of adjuvants within the peritoneal cavity, 4) adequacy of local, regional, and systemic host defenses, and 5) adequacy of initial treatment (i.e., source control). Patients are at higher risk of IAA formation after treatment of intra-abdominal infection if any of these key factors are not adequately treated.

Secondary prevention

Sound surgical technique and compliance with all standard sterile measures during surgery may decrease risk of postoperative IAA. Appropriate preoperative antimicrobial therapy as indicated by the procedure is a critical step in preventing postoperative infections.

Patient discussions

Patients at high risk should monitor for signs and symptoms of IAA and are encouraged to call their doctor early to avoid IAA complications such as septic shock.

FOLLOW UP

Monitoring

Monitoring

Abscess resolution is usually monitored clinically by following resolution of clinical signs and symptoms of infection (fever, leukocytosis, and abdominal pain), routine vitals, laboratory data, and drain output. A repeat computed tomography scan may be indicated if drainage has stopped or the patient shows persistent signs and symptoms of sepsis.

Complications

Complications	Timeframe	Likelihood
sepsis	**short term**	**medium**
Early and appropriate parenteral antimicrobial therapy is a mainstay in the treatment and prevention of this complication.		
rupture of abscess	**short term**	**low**
Ruptured IAA can be a severe complication. Patients present with generalized peritonitis, and probably with severe septic shock. Early resuscitation, antimicrobial therapy, and surgical control of the infectious source should be instituted immediately.		
abscess recurrence	**variable**	**medium**
Usually results secondary to inadequate source control. Reaccumulation of an abscess could be treated either percutaneously or surgically depending on the cause of the recurrence. In percutaneous drainage, most recurrences are due to malposition, insufficient drain size, or kink or blockage of the catheter. A communicating abscess with the bowel lumen may indicate a surgical procedure.		

Prognosis

Patients with IAA carry a mortality risk that is usually related directly to the etiology of the abscess. A patient with severe infected pancreatitis and acute respiratory distress syndrome certainly has a higher mortality than a patient with a small appendiceal abscess. Usually patients who require surgical drainage have higher morbidity and mortality. The emergence of multidrug-resistant organisms as a source of intra-abdominal infection also increases morbidity and possibly mortality. Mortality depends on initiating early appropriate treatment to restore fluid and electrolyte imbalances, supporting the function of vital organs, providing appropriate broad-spectrum antimicrobial therapy, and adequately controlling the source.

Importance of early appropriate antibiotic therapy and mortality

Studies have identified that the initiation of early appropriate empiric antimicrobial therapy is associated with improved outcome and reduced mortality in intra-abdominal infections.[58]

Treatment guidelines

International

ACR appropriateness criteria: radiologic management of infected fluid collections (https://www.acr.org/Clinical-Resources/ACR-Appropriateness-Criteria) [36]

Published by: American College of Radiology **Last published:** 2019

Revised guidelines on the management of intra-abdominal infection (https://www.sisna.org/guidelines) [2]

Published by: Surgical Infection Society **Last published:** 2017

The management of intra-abdominal infections from a global perspective: 2017 WSES guidelines for management of intra-abdominal infections (https://www.wses.org.uk/guidelines) [29]

Published by: The World Society of Emergency Surgery **Last published:** 2017

Online resources

1. Surgical Infection Society: intra-abdominal infection (IAI) high versus low risk (http://www.sisna.org/iai-high-vs-low-risk) *(external link)*

Key articles

- Mazuski JE, Tessier JM, May AK, et al. The Surgical Infection Society revised guidelines on the management of intra-abdominal infection. Surg Infect (Larchmt). 2017 Jan;18(1):1-76. Full text (https://www.liebertpub.com/doi/full/10.1089/sur.2016.261) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/28085573?tool=bestpractice.bmj.com)
- Solomkin JS, Mazuski JE, Bradley JS, et al. Diagnosis and management of complicated intra-abdominal infection in adults and children: guidelines by the Surgical Infection Society and the Infectious Diseases Society of America. Surg Infect (Larchmt). 2010 Feb;11(1):79-109. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/20163262?tool=bestpractice.bmj.com)
- Pieracci FM, Barie PS. Intra-abdominal infections. Curr Opin Crit Care. 2007 Aug;13(4):440-9. Abstract (http://www.ncbi.nlm.nih.gov/pubmed/17599016?tool=bestpractice.bmj.com)
- Evans L, Rhodes A, Alhazzani W, et al. Surviving Sepsis Campaign: international guidelines for management of sepsis and septic shock 2021. Intensive Care Med. 2021 Nov;47(11):1181-247. Full text (https://www.doi.org/10.1007/s00134-021-06506-y) Abstract (http://www.ncbi.nlm.nih.gov/pubmed/34599691?tool=bestpractice.bmj.com)
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Images

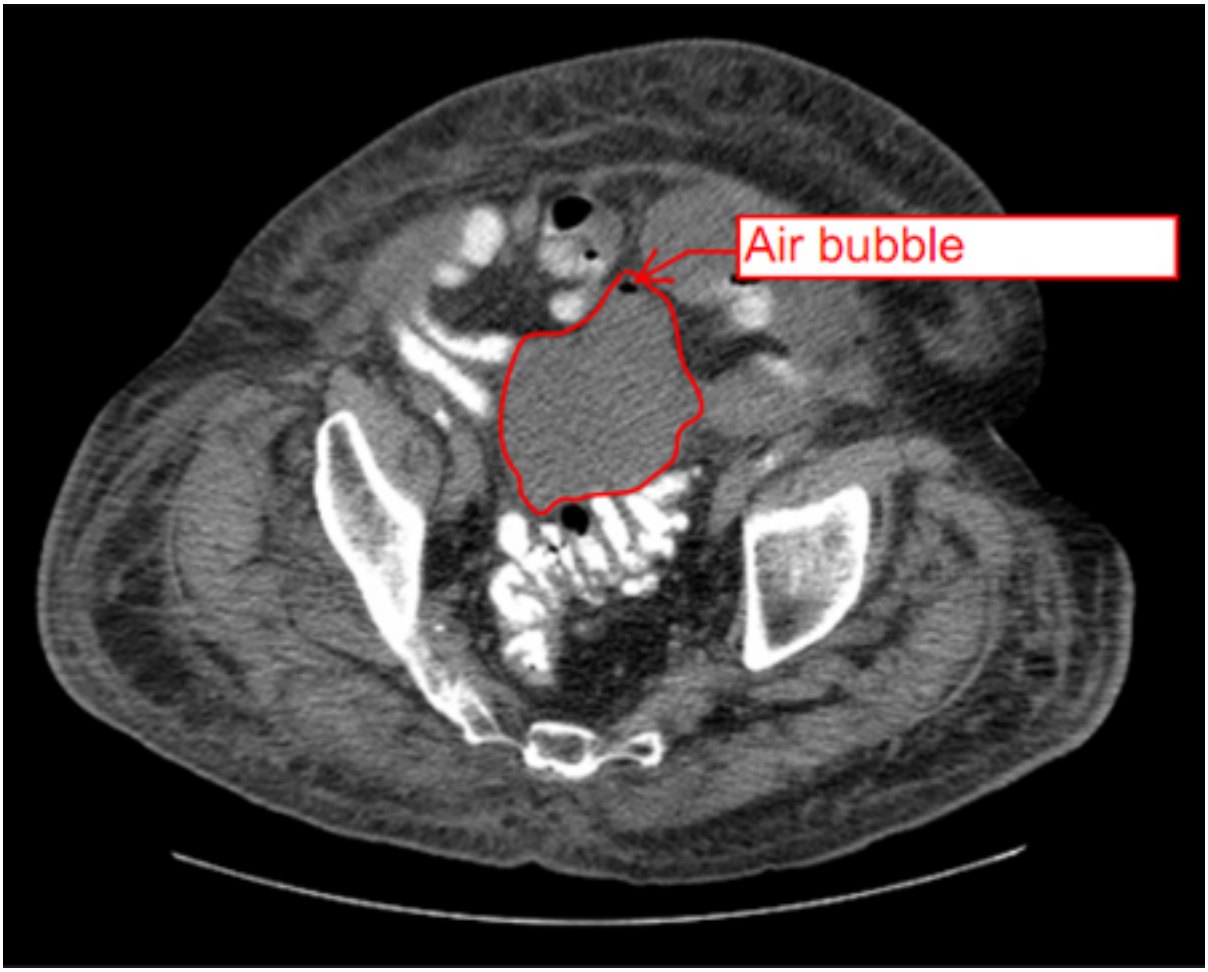

Figure 1: Intra-abdominal abscess with small air bubble, secondary to perforated diverticulitis

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Intra-abdominal abscesses		
Location	**Etiology**	**Organisms**
Intraperitoneal		
Subphrenic Right or left lower quadrant Interloop Paracolic Pelvic	Postoperative; perforation of hollow viscus, appendicitis, diverticulitis, or tumour; Crohn disease; pelvic inflammatory disease; generalised peritonitis of any etiology	Bowel flora, often polymicrobial
Retroperitoneal		
Pancreatic	Trauma; pancreatitis	Bowel flora, often polymicrobial
Perinephric	Spread of renal parenchymal abscess (complication of pyelonephritis or rarely hematogenous from remote source)	Aerobic gram-negative bacilli
Visceral		
Hepatic	Trauma, ascending cholangitis, portal bacteremia	Aerobic gram-negative bacilli origin; polymicrobial bowel flora if portal bacteremia; amebic infection may occur
Splenic	Trauma, hematogenous, infarction (as in sickle cell disease and malaria)	Staphylococci, streptococci, anaerobes, aerobic gram-negative bacilli including *Salmonella*, *Candida* in immunocompromised patients

Figure 2: Classification of intra-abdominal abscesses (intraperitoneal, retroperitoneal, or visceral)

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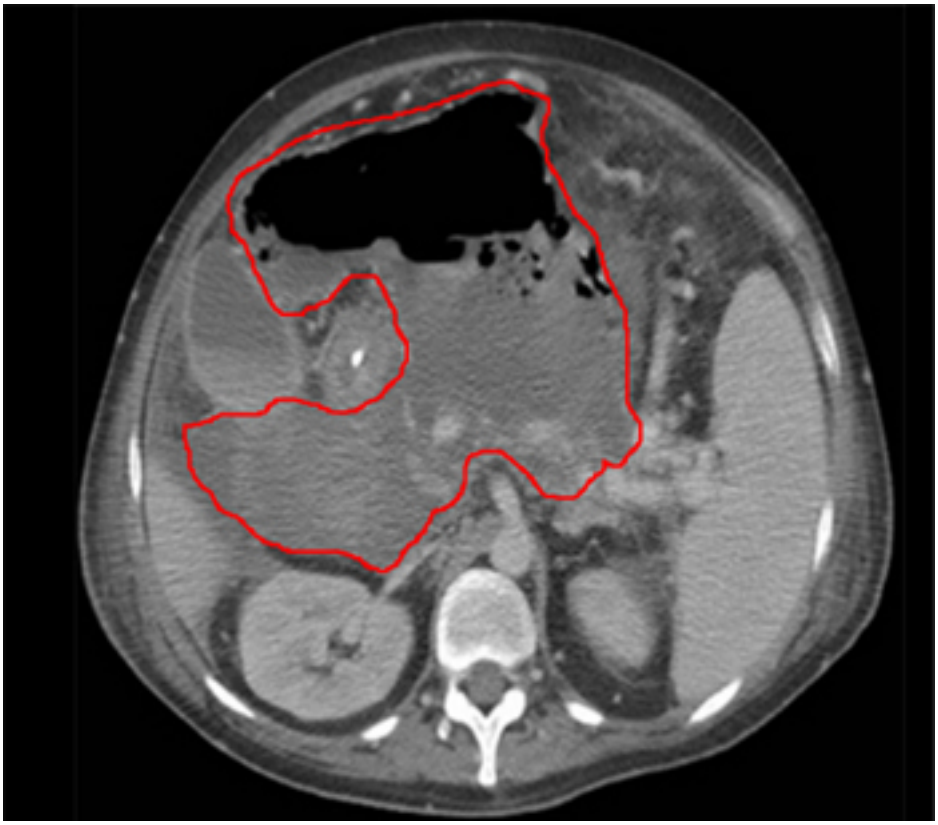

Figure 3: Abscess completely replacing pancreas and extending into portal hilum, with multiple gas bubbles and large air/fluid level

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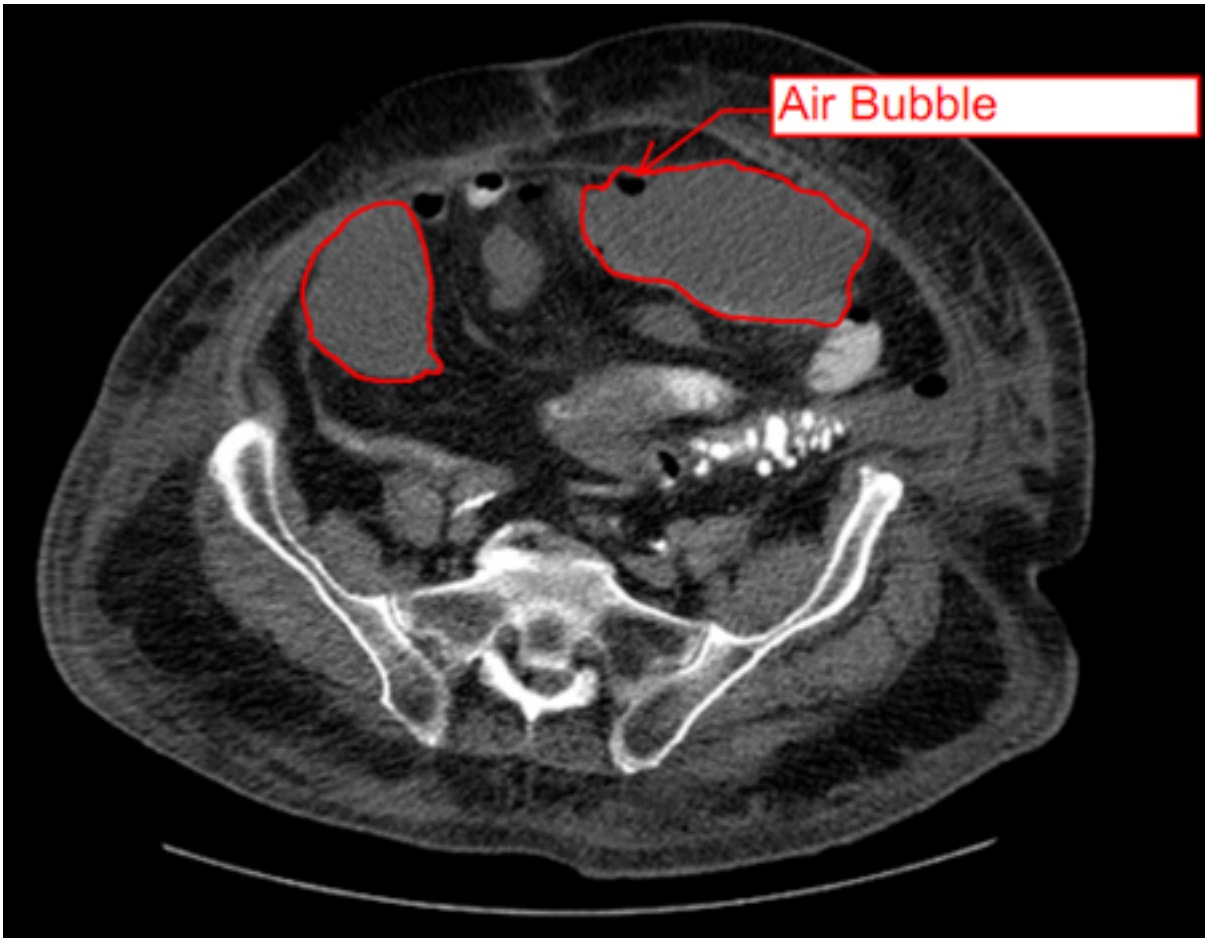

Figure 4: CT scan showing intra-abdominal abscess with small air bubble

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Regardless of the language in which the content is displayed, numerals are displayed according to the original English-language numerical separator standard. For example 4 digit numbers shall not include a comma nor a decimal point; numbers of 5 or more digits shall include commas; and numbers stated to be less than 1 shall be depicted using decimal points. See Figure 1 below for an explanatory table.

BMJ accepts no responsibility for misinterpretation of numbers which comply with this stated numerical separator standard.

This approach is in line with the guidance of the International Bureau of Weights and Measures Service.

Figure 1 – BMJ Best Practice Numeral Style

5-digit numerals: 10,000

4-digit numerals: 1000

numerals < 1: 0.25

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