Intraabdominal Infections

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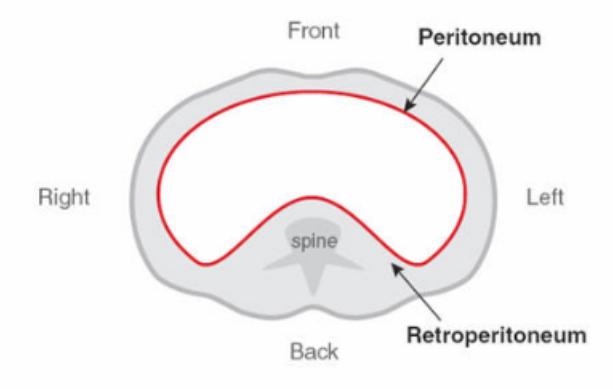
Learning objectives

1. Classify intraabdominal infections (primary vs. secondary vs. tertiary)
2. Understand epidemiology and risk factors for each infection type
3. Master the microbiology and pathogenesis of IAI
4. Apply diagnostic criteria for peritonitis variants
5. Select appropriate antimicrobial therapy based on clinical context
6. Recognize when source control is essential
7. Manage CAPD-associated peritonitis
8. Diagnose and treat intraperitoneal abscesses
9. Risk-stratify patients for prognosis and outcomes
10. Implement prevention strategies for secondary prophylaxis

Definition and classification

Peritonitis: Inflammatory response within the peritoneal cavity due to microbial or chemical contamination



By Location

- Diffuse
- Localized

By Type

- Primary (spontaneous)
- Secondary
- Tertiary

Primary vs. secondary vs. tertiary

Feature	Primary	Secondary	Tertiary
Intraabdominal source	None	Present (perforation, ischemia)	Persistent after source control
Frequency	~1% of peritonitis cases	80–90% of cases	Less common
Typical organisms	Monomicrobial gram-negative	Polymicrobial aerobic/anaerobic	MDR, gram-negative, low virulence
Mortality	5–20%	10–40%	30–64%

Community-acquired vs. Healthcare-associated

Community-acquired (~80% of IAI)

- Further subdivided: low-risk vs. high-risk
- Risk factors: drug-resistant bacteria, severity (mild/moderate/severe), comorbidities
- Common pathogens: *E. coli, Klebsiella*, anaerobes

Healthcare-associated

- Complications of elective or emergency abdominal surgery
- Nosocomial isolates (MRSA, MDR gram-negatives, *Candida*)
- Higher mortality and morbidity

Uncomplicated vs. complicated infections

Uncomplicated IAI

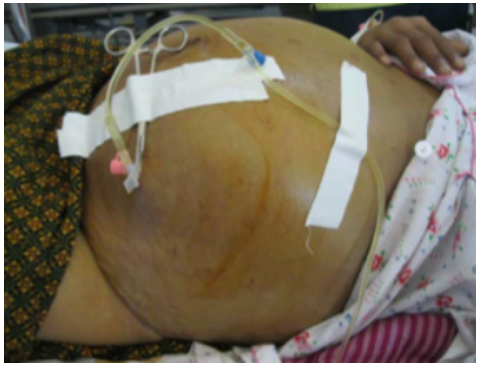
- Intramural inflammation of GI tract
- Localized to single organ (e.g., appendicitis, cholecystitis)
- No extension into peritoneal space

Complicated IAI (cIAI)

- Extension beyond organ of origin
- Peritonitis (diffuse or localized)
- Abscess formation
- Spillage into sterile peritoneal space

Primary peritonitis (SBP)

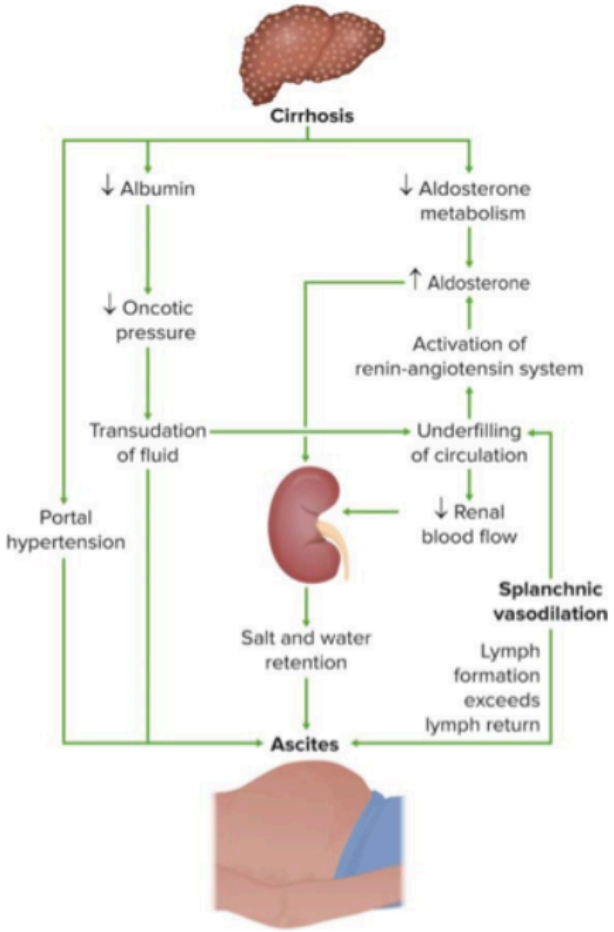
Primary peritonitis: Epidemiology



- **Prevalence** — Accounts for ~1% of all peritonitis cases
  - Present in 10–30% of hospitalized cirrhotic patients with ascites
- **High-Risk Patient Groups**
  - Cirrhosis (alcoholic, postnecrotic, cryptogenic)
  - Congestive heart failure
  - Nephrotic syndrome
  - Metastatic malignancy
  - Systemic lupus erythematosus
  - Rare in patients with no underlying disease

The key insight is that any condition producing ascites increases SBP risk. Cirrhosis is the most common underlying disease. In children, acute viral hepatitis and

Risk factors for SBP development



- **High-volume ascites** (advanced cirrhosis, Child-Pugh class C)
- **High Model for End-Stage Liver Disease (MELD) score**
- **Coexisting gastrointestinal hemorrhage**
- **Previous SBP episode**
- **Low ascitic fluid protein** (<1 g/dL)
- **Elevated serum bilirubin** (>2.5 mg/dL)
- **Proton pump inhibitor use** (reduces gastric acidity, increases translocation)

These risk factors reflect compromised host defenses and increased bacterial translocation. GI bleeding particularly increases the risk because of direct inoculation of bacteria into the peritoneal cavity and subsequent bacteremia. Low protein content in ascites correlates with reduced opsonic activity and impaired complement-

Primary peritonitis: Microbiology

Defining Characteristic: Monomicrobial infection

Most Common Organisms (in cirrhotic patients)

1. *Escherichia coli* (most frequent)
2. *Klebsiella pneumoniae*
3. Other Enterobacterales
4. *Streptococcus pneumoniae* (especially pediatrics)
5. Other streptococci (including enterococci)
6. Anaerobes (uncommon)
7. *Pseudomonas aeruginosa* (rare)

Gram-Negative Dominance — 60–69% of cases caused by gram-negative enteric bacteria; organisms presumed of gastrointestinal origin

The predominance of gram-negatives reflects bacterial translocation from the GI tract. *S. aureus* is unusual (2–4% of cases) and may indicate umbilical hernia erosion. Anaerobes are reported infrequently, possibly due to the bacteriostatic activity of ascites against *Bacteroides spp.* and the relatively high oxygen tension in peritoneal fluid. In pediatric cases, hematogenous spread of *S. pneumoniae* is more common.

Pathogenesis of primary peritonitis

Bacterial Translocation

- Passage of viable bacteria from the GI tract lumen to mesenteric lymph nodes and bloodstream
- Increased risk with portal hypertension and collateral vessel formation
- Enhanced by decreased gastric acidity (PPI use) and altered bowel flora

Hematogenous Seeding

- Bacteria reach peritoneal cavity via circulating blood
- Explains predominantly **monomicrobial pattern**
- Bacteremia present in 75% of cases with aerobic organisms

Host Factors

- Reduced opsonic activity (low complement, reduced immunoglobulins)
- Impaired polymorphonuclear cell function
- Loss of anatomic barriers in advanced cirrhosis

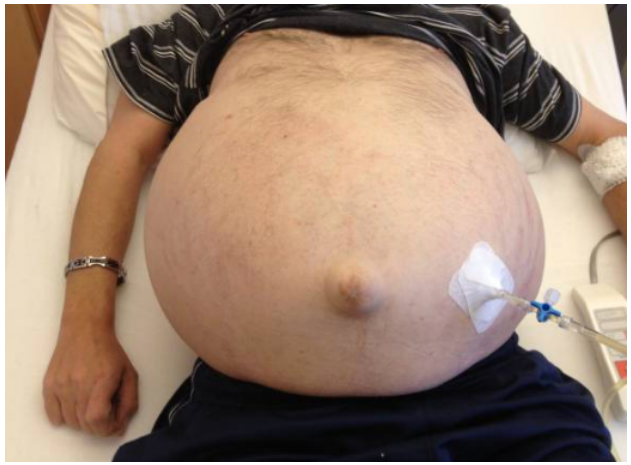
Paracentesis

Paracentesis

Diagnosis of SBP: Ascitic fluid examination

Essential Tests

- WBC count with differential
- Protein concentration
- Gram stain
- Culture (inoculate blood culture bottles)
- Glucose
- Amylase
- Lactate dehydrogenase (LDH)
- SAA (serum-ascitic albumin gradient)



Diagnostic Threshold

Parameter	Significance
PMN >250/mm^3	Presumptive SBP
Ascitic culture positive	Confirms SBP
SAA >1.1 g/dL	Suggests non-peritoneal ascites

SAAG = Serum albumin − Ascitic fluid albumin

SBP variants: Monomicrobial nonneutrocytic bacterascites

Definition

- **Positive ascitic fluid culture**
- **PMN <250 cells/mm^3**
- No clinical peritonitis signs

Natural History

- Resolves spontaneously in 62–86% of cases
- Progresses to SBP in remainder (sometimes within hours)
- Mortality same as classic SBP regardless of neutrophil response

Clinical Significance

- May represent early colonization before host response
- Symptomatic patients at higher risk for progression
- Asymptomatic patients often remain colonized only
- Treat if symptomatic or if organisms are gram-positive cocci

SBP variants: Culture-negative neutrocytic ascites

Definition

- **PMN >250 cells/mm^3**
- **Negative ascitic fluid culture**
- No evident intraabdominal surgical source
- Absence of pancreatitis

Epidemiology

- Occurs in 35% of patients with clinical SBP
- Blood culture positive in one-third of cases

Reasons for Culture Negativity

- Antibiotic administration before sampling
- Inadequate culture technique
- Fastidious organisms
- Recent antibiotic exposure

Improved Detection

- Inoculate 10–20 mL ascitic fluid into blood culture bottles at bedside
- Increases culture yield significantly

SBP variants: Polymicrobial bacterascites

Definition

- Multiple bacterial species on culture or Gram stain
- PMN <250 cells/mm^3
- No elevation of ascitic protein

Etiology

- *Traumatic paracentesis (needle enters bowel)*
- Incidence: <1% of procedures
- Risk factors: ileus, abdominal scars, adhesions

Natural History

- Usually resolves spontaneously
- Peritoneal fluid protein >1 g/dL and adequate opsonic activity predict spontaneous resolution
- Treat only if symptomatic or if evolves to classic peritonitis

Clinical Pearl

> Do not treat polymicrobial bacterascites empirically unless clinical deterioration occurs.

Primary peritonitis diagnosis: Recommended paracentesis protocol

Universal Recommendation for Cirrhotic Patients

- Diagnostic paracentesis on **hospital admission** for all cirrhotic patients with ascites
- Perform regardless of admission reason
- Even asymptomatic patients may have SBP

Timing and Frequency

- At baseline assessment
- When admitted with complications (variceal bleed, etc.)
- When clinical deterioration occurs
- Repeat if initial cultures negative but high clinical suspicion

Specimen Handling

- Inoculate blood culture bottles at bedside (10–20 mL)
- Obtain cell count, differential, protein, glucose, amylase
- Gram stain for rapid assessment

Treatment of SBP: Empiric therapy

First-Line Agent

- **Cefotaxime** 2 g IV every 6–8 hours (or 3 g every 6 hours for high-risk patients)
- Covers gram-negative enteric bacteria and streptococci
- Can transition to oral after initial response
- If recent history of MDR colonization or infection, may require broader-spectrum therapy

Alternative Regimens

Condition	Agent
Non-severe	Cefixime 400 mg PO BID
Renal impairment	Adjust cephalosporin dosing
β-lactam allergy	Fluoroquinolone (ciprofloxacin or norfloxacin)

Albumin supplementation

Indication

- All patients with SBP
- Particularly important in hemodynamically unstable patients

Albumin Dosing

- **1.5 g/kg at diagnosis** (maximum 100 g)
- **1 g/kg on day 3**

Mechanism

- Expands plasma volume
- Improves renal perfusion
- Reduces variceal bleeding risk
- Decreases renal dysfunction (hepatorenal syndrome)

Outcome Benefit

- Reduces in-hospital mortality
- Decreases kidney injury
- Standard of care in SBP management

Treatment of SBP: Duration and outcomes

Duration of Therapy

- **5 days of IV cefotaxime** is sufficient for uncomplicated SBP (ineffective for ESBL producing isolates)
- May transition to oral fluoroquinolone or cephalosporin
- Repeat paracentesis not needed if clinical improvement occurs

Response Assessment

- Clinical improvement (fever resolution, abdominal tenderness improves)
- Laboratory response (declining WBC, improving renal function)

Prognosis After SBP

- **In-hospital mortality:** 5–20%
- **Long-term survival:** Poor—survivors warrant liver transplant assessment
- Liver disease severity (MELD, bilirubin) predicts outcomes

Prevention of SBP: Primary prophylaxis

Indicated When

- **Low ascitic protein** (<1.5 g/dL) +
  - Impaired renal function (creatinine >1.2 mg/dL) OR
  - Impaired liver function (bilirubin >2.5 mg/dL) OR
  - Low platelet count (<40,000/μL)

Agents for Primary Prophylaxis

Agent	Dosing	Notes
Norfloxacin	400 mg daily PO	First-line
Ciprofloxacin	750 mg weekly PO	Alternative
TMP-SMX	1 DS tablet daily	Alternative

Efficacy

- Reduces SBP incidence by 50–60%
- Improves short-term survival
- Cost-effective in high-risk patients

Prevention of SBP: Secondary prophylaxis

Indicated

- All patients after first SBP episode
- Reduces recurrence by 80%

Agents

- Norfloxacin 400 mg daily (first-line)
- Ciprofloxacin 750 mg weekly
- TMP-SMX 1 DS tablet daily

Duration

- Lifelong or until liver transplantation
- Continue indefinitely to prevent recurrence

Additional Measures

- Referral for liver transplant evaluation (poor prognosis after SBP)
- Alcohol cessation counseling
- Variceal screening and beta-blocker therapy

Secondary peritonitis

Secondary peritonitis: Epidemiology

Prevalence

- Most common intraabdominal infection (80–90% of cases)
- Results from visceral perforation or intraabdominal pathology

Common Causes

Surgical Emergencies

- Perforated peptic ulcer
- Ruptured appendicitis
- Perforated diverticulitis
- Acute cholecystitis/perforation

Other Sources

- Ischemic bowel necrosis
- Traumatic GI perforation
- Gynecologic pathology (ruptured ovarian cyst, tubo-ovarian abscess)
- Biliary or pancreatic disease

Etiologies of secondary peritonitis (representative)

Organ System	Common Causes
Upper GI	Perforated peptic ulcer, perforated gastric ulcer
Small bowel	Meckel's diverticulitis, small bowel perforation, ischemic necrosis
Appendix	Perforated appendicitis
Colon	Diverticulitis (perforation), toxic megacolon, ischemic colitis
Biliary	Perforation of gallbladder, cholangitis
Gynecologic	Ruptured tubo-ovarian abscess, perforated ovarian cyst
Trauma	Iatrogenic or penetrating perforation

This table emphasizes that secondary peritonitis can originate from nearly any abdominal organ. The location of the primary disease influences which organisms are present and the strategy for source control. For example, perforated peptic ulcer involves mostly upper GI flora (gram-positives and anaerobes), while colonic

GI tract microbiota and bacterial density

Bacterial Population Gradient

- **Stomach**: 10^3–10^4 CFU/mL (mostly anaerobes suppressed by acid)
- **Small intestine**: 10^4–10^7 CFU/mL (increasing distally)
- **Terminal ileum**: 10^7–10^9 CFU/mL (mixed aerobic/anaerobic)
- **Colon**: 10^{11}–10^{12} CFU/mL (predominantly anaerobes > aerobes by 1000:1)

Clinical Implication

- **Upper GI perforation**: Fewer organisms, less anaerobic load
- **Lower GI perforation**: Heavy polymicrobial inoculum with anaerobic dominance

This density gradient is critical for understanding secondary peritonitis microbiology. A perforated duodenal ulcer introduces mostly aerobic organisms and some anaerobes, while colonic perforation introduces massive inocula of obligate anaerobes, facultative gram-negatives, and enterococci. **This explains why colonic**

Causes of secondary peritonitis

Organ	Cause of Peritonitis
Distal esophagus	Boerhaave syndrome
	Malignancy
	Trauma
	Iatrogenic
Stomach	Peptic ulcer perforation
	Malignancy
	Trauma
	Iatrogenic
Duodenum	Peptic ulcer perforation
	Trauma
	Iatrogenic[a]
Biliary tract	Cholecystitis
	Stone perforation from gallbladder or common duct
	Malignancy
	Trauma
	Iatrogenic
Pancreas	Pancreatitis (e.g., alcohol, drugs, gallstones)
	Trauma

Organ	Cause of Peritonitis
	Iatrogenic
Small bowel	Ischemic bowel
	Incarcerated hernia
	Crohn disease
	Malignancy
	Meckel diverticulum
	Trauma
Large bowel and appendix	Ischemic bowel
	Diverticulitis
	Malignancy
	Ulcerative colitis and Crohn disease
	Appendicitis
	Volvulus
	Trauma (mostly penetrating)
	Iatrogenic

Secondary peritonitis: Microbiology

Key Features

- **Polymicrobial** (2–5 organisms typical, up to 10+)
- Reflects normal GI flora
- Aerobic-anaerobic polymicrobial common (not all may grown in culture)

Dominant Organisms

Category	Examples	Prevalence
Gram-negative rods	*E. coli, Klebsiella, Proteus, Enterobacter*	60–80%
Anaerobes	*Bacteroides fragilis*, anaerobic cocci, *Clostridium*	60–90%
Gram-positive cocci	*Streptococcus, Enterococcus, Staphylococcus*	40–60%

The polymicrobial nature of secondary peritonitis contrasts sharply with primary peritonitis. Multiple organisms allow synergistic pathogenesis: gram-negatives cause early sepsis and toxemia, while anaerobes (especially *B. fragilis*) promote abscess formation. Enterococci are present in 30–60% of cases; their treatment

Aerobic-anaerobic coverage in secondary peritonitis

Early Infection (First Hours)

- Gram-negative rod dominance (*E. coli, Klebsiella*)
- Rapid multiplication and toxin production
- Systemic toxemia and early sepsis
- Clinical: fever, tachycardia, hypotension

Late Infection (Days)

- Anaerobic bacteria proliferate (*Bacteroides fragilis* group)
- Reduced oxygen tension favors anaerobic growth
- Enhanced abscess formation (fibrin encapsulation)
- Clinical: loculation, persistent fever despite initial therapy

Clinical Consequences

- Single-agent therapy (e.g., cephalosporin alone) inadequate
- Requires dual coverage: aerobic + anaerobic agents
- Source control essential to disrupt both populations

This synergy concept explains why inadequate anaerobic coverage leads to treatment failure, recurrent fever, and abscess formation even when gram-negative coverage is appropriate. It reinforces the need for combination therapy in secondary peritonitis—β-lactam/β-lactamase inhibitor combinations,

Pathogenesis and virulence factors in secondary peritonitis

Initial Contamination

- Spillage of GI flora into sterile peritoneal space
- Magnitude of inoculum determines early severity
- Bacterial adherence and LPS/endotoxin trigger inflammation

Host Response Activation

- Complement activation (local and systemic)
- Cytokine release (TNF-α, IL-1, IL-6, IL-8)
- Polymorphonuclear recruitment to peritoneum
- Increased vascular permeability

Bacterial Virulence Factors

- Lipopolysaccharide (gram-negative endotoxin)
- Capsule and fimbriae (adherence, invasion)
- Toxins and enzymes (tissue invasion, abscess formation)
- Antibiotic resistance (β-lactamase, efflux pumps)

Pathophysiology: Local response to secondary peritonitis

Pathophysiology: Systemic response to secondary peritonitis

SIRS (Systemic Inflammatory Response Syndrome)

- Fever, tachycardia, tachypnea, leukocytosis
- Results from TNF-α, IL-1, IL-6 release
- May progress to sepsis and multiorgan failure

Hemodynamic Changes

- Initial phase: vasoconstriction (compensatory)
- Late phase: vasodilation and increased capillary permeability
- Hypovolemia and hypotension (septic shock)

Organ Dysfunction

- Renal hypoperfusion → acute kidney injury
- Pulmonary capillary leak → ARDS
- Hepatic dysfunction → coagulopathy
- GI hypomotility → ileus

Mortality Correlation

- Extent of organ dysfunction predicts outcome
- Reflected in APACHE II[1] score, Mannheim Peritonitis Index[2]
- Delayed recognition/treatment increases mortality

Clinical manifestations of secondary peritonitis

Symptoms

- **Acute abdominal pain** (sudden onset if perforation)
- Pain localized initially, generalizes with diffuse peritonitis
- Nausea and vomiting (may be present or absent)
- May have antecedent symptoms (dyspepsia before ulcer rupture, diarrhea before diverticulitis)

Physical Findings

- Rebound tenderness and guarding (peritoneal irritation)
- Absent or diminished bowel sounds (ileus)
- Abdominal distention (third-spacing of fluid)
- Hypotension and tachycardia (sepsis)
- Fever (usually present but may be absent in elderly or immunocompromised)

Severity Indicators

- Hemodynamic instability
- Acute kidney injury
- Leukocytosis >15,000 or left shift
- Metabolic acidosis

Diagnostic workup: Laboratory studies

Complete Blood Count

- WBC elevation (typically 12,000–20,000)
- Left shift (immature bands) indicates severity
- Absence of leukocytosis does not exclude peritonitis

Inflammatory Markers

Test	Utility
C-reactive protein (CRP)	Elevated; reflects severity
Procalcitonin	>2 ng/mL suggests bacterial peritonitis; guides de-escalation
Lactate	Elevated in sepsis; correlates with severity

Diagnostic workup: Laboratory studies, cont.

Chemistry

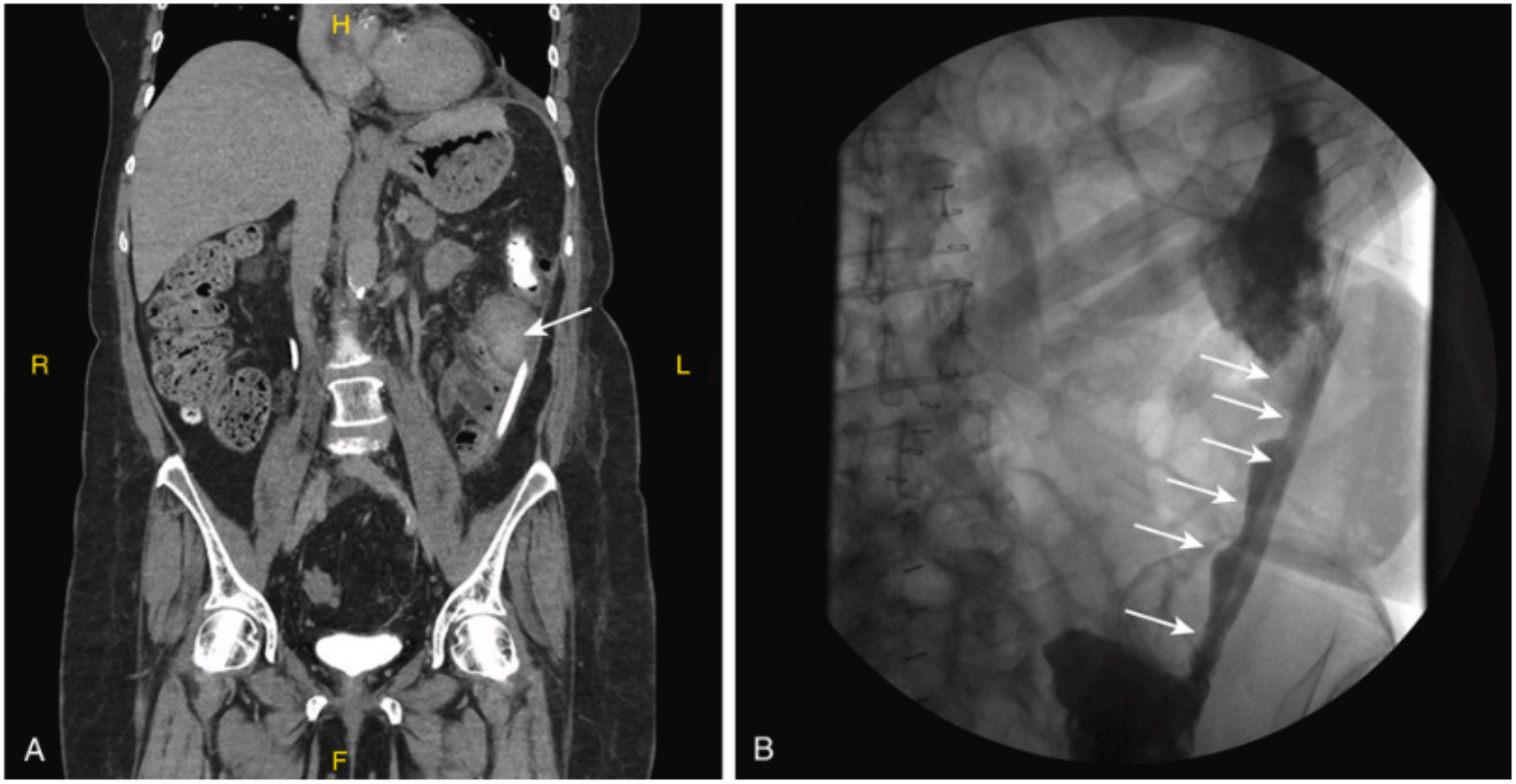
- Renal function (creatinine, BUN)
- Electrolytes (hypokalemia common from GI losses)
- Liver function tests
- Glucose (hyperglycemia or hypoglycemia possible)

Blood Cultures

- Obtain before antibiotics if possible
- Positive in 20–40% of secondary peritonitis
- Guide specific organism coverage

Laboratory findings support but do not prove peritonitis. WBC and CRP can be falsely normal in elderly or immunocompromised. Procalcitonin >2 ng/mL supports bacterial infection and helps differentiate from non-infectious peritonitis (e.g., spontaneous rupture of abdominal aortic aneurysm). Lactate elevation indicates

Diagnostic imaging: CT abdomen/pelvis



Intraperitoneal abscess (arrow) following a posthemicolectomy anastomosis leak for diverticulitis, with a percutaneous drainage catheter (CT scan of abdomen and pelvis, coronal view). (B) Evidence extravasation of contrast (arrows) in the surgical site of Hartmann pouch procedure (Gastrografin enema). *Courtesy Thomas*

Diagnostic imaging: CT performance

Sensitivity and Specificity

- **CT with IV contrast: 97% sensitivity for complicated IAI**
- Best imaging modality for secondary peritonitis and abscesses

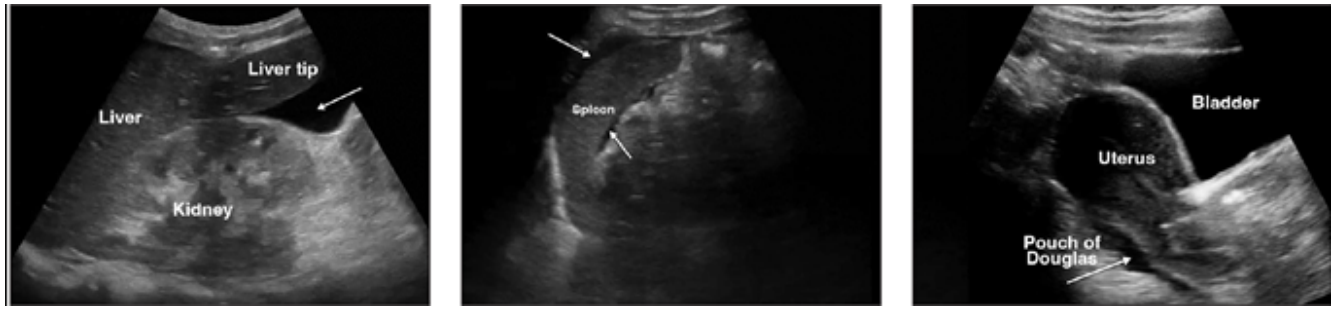
Key CT Findings

- Free air (pneumoperitoneum) → indicates perforation
- Free fluid with inflammatory changes
- Focal abscess collections
- Source organ pathology (dilated colon, appendiceal thickening, etc.)
- Evidence of ischemia (pneumatosis, portal venous gas)

Contraindications

- Hemodynamic instability (may necessitate OR before imaging)
- Renal insufficiency (contrast nephropathy risk)
- Iodine allergy

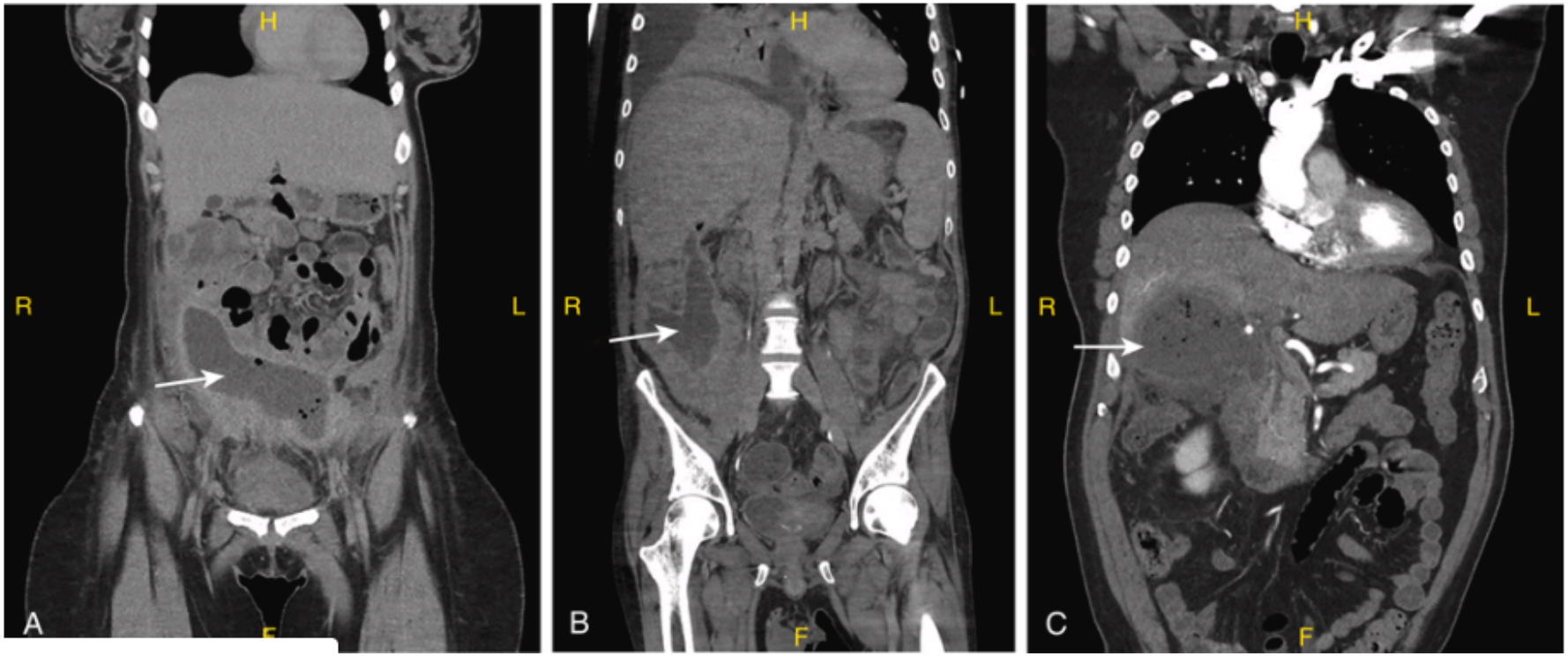
Alternative imaging



- **Ultrasound**: Portable, real-time, good for fluid assessment; lower sensitivity than CT
- **MRI**: Excellent soft tissue detail; limited by cost and time; useful in renal insufficiency

CT is the gold standard for imaging secondary peritonitis and guides source control decisions. However, clinical deterioration with shock may necessitate immediate

Secondary peritonitis



The three panel CT scan of abdomen and pelvis with coronal views. Panel A shows a large complex fluid collection, marked with an arrow, containing air in the pelvis following a hysterectomy. Panel B shows a computed tomography scan of the abdomen and pelvis in a coronal view with an arrow pointing to a right lower quadrant air and fluid collection caused by leakage from a primary anastomosis after colon cancer surgery. Panel C presents a computed tomography scan of the abdomen

Paracentesis and peritoneal cultures

Indications

- Confirm diagnosis when imaging equivocal
- Obtain organisms for culture and susceptibility
- Obtain cell count and fluid analysis

Technique

- Sterile procedure (surgical preparation)
- Avoid areas of adhesion, stomas, surgical wounds
- Obtain 20–30 mL in sterile container
- Send for Gram stain, culture, cell count

Fluid Analysis

Parameter	Finding
Appearance	Turbid, purulent, bloody
PMN count	Usually >50,000/mm^3 in secondary peritonitis
Gram stain	May show gram-negative rods, gram-positive cocci, anaerobes
Culture	Polymicrobial growth expected

Clinical Utility of paracentesis in secondary peritonitis

- Gram stain may guide initial therapy
- Culture confirms organisms and guides de-escalation
- Lower yield than in primary peritonitis (polymicrobial, fastidious anaerobes)

Paracentesis is less often done in secondary peritonitis than primary (diagnosis usually clinical + imaging), but cultures are valuable for guiding therapy.

Prognosis: Risk stratification in secondary peritonitis

APACHE II Score

- Predicts mortality based on physiology, age, comorbidities
- APACHE II >15 associated with 50% mortality
- APACHE II >25 associated with >80% mortality

Mannheim Peritonitis Index (MPI)

Factor	Points
Age >50	5
Female gender	5
Organ failure	7
Malignancy	4
Duration of peritonitis >24h	4
Origin (non-colonic)	4
Diffuse peritonitis	6
Exudate (purulent)	6

MPI Interpretation

- MPI <21: 0% mortality
- MPI 21–29: 11% mortality
- MPI >29: 47% mortality

Risk scores help counsel patients and families on prognosis and guide intensity of care. However, these are population-based estimates and individual patient factors (comorbidities, immune status, speed of source control) also matter. The best prognostic factor is often how quickly source control is achieved and how

Tertiary peritonitis

Definition

- Persistent or recurrent peritonitis despite successful source control of primary infection
- Diagnosis: Peritonitis with signs of sepsis >48 hours after adequate surgery and source control

Epidemiology

- Occurs in 3–10% of secondary peritonitis cases
- Associated with delayed source control
- Higher mortality: 30–64%

Microbiology

- Less virulent organisms (coagulase-negative staphylococci, Candida)
- Multidrug-resistant gram-negatives (Enterobacter, Pseudomonas)
- MRSA
- Often monomicrobial or sparse growth

Pathogenesis

- Host immune dysfunction (exhaustion of cytokine response, impaired opsonization)
- Biofilm-forming organisms
- Inadequate source control or recurrent leak

Tertiary peritonitis: Management principles

Diagnostic Challenge

- Distinguish from inadequately treated secondary peritonitis
- Consider recurrent leak, anastomotic dehiscence, ischemia

Management Approach

- **Repeat imaging** (CT) to identify new source
- **Selective repeat surgery** only if surgically correctable source identified
- **Avoid routine re-exploration** (may worsen outcomes)
- **Maximize supportive care**: vasopressors, ECMO if needed, nutritional support
- **Broad-spectrum antimicrobials**: carbapenem ± anti-Candida ± vancomycin pending cultures
- **Consider antifungal therapy** (Candida common)

Prognosis

- Mortality 30–64% despite appropriate management
- Poor prognostic factors: organ failure, delayed recognition, immunosuppression
- Consider goals of care discussion early

Tertiary peritonitis represents a transition to palliative care in many cases. Aggressive re-exploration without a clear target often worsens outcomes. The focus

Antimicrobial therapy for secondary peritonitis: An overview

Goal

- Cover aerobes (gram-negative rods, gram-positive cocci) and anaerobes
- Account for severity (mild-moderate vs. high-risk)
- Consider prior antibiotic exposure (resistance risk)

Risk Stratification

Risk Category	Features	Typical Organisms
Low-risk	Community-acquired, no recent hospitalization, no immunosuppression	Susceptible gram-negatives, anaerobes
High-risk	Healthcare-associated, recent surgery, immunosuppressed, prolonged hospitalization	MDR gram-negatives, MRSA, *Candida*, enterococci

Timing

- Initiate empiric therapy immediately (within 1 hour)
- Source control should be initiated in parallel (not sequential)

Antimicrobial therapy for secondary peritonitis: Drug classes for aerobic coverage

Beta-Lactams with beta-Lactamase Inhibitors

Agent	Dosing	Notes
Amoxicillin-clavulanate	875–125 mg TID	Oral; limited spectrum
Ampicillin-sulbactam	3 g IV Q6H	Good anaerobic coverage
Piperacillin-tazobactam	4.5 g IV Q6–8H	Excellent coverage; pseudomonal

Cephalosporins (typically combined with metronidazole)

Agent	Dosing	Notes
Ceftriaxone	2 g IV Q12H + metronidazole 500 mg TID	Lower pseudomonal coverage
Ceftazidime	2 g IV Q8H + metronidazole 500 mg TID	Better *Pseudomonas* coverage

Carbapenems (broad spectrum, both Gram negative and anaerobes- reserve use)

- Meropenem 1 g IV Q8H (or extended infusion)
- Imipenem-cilastatin 500 mg IV Q6H
- Ertapenem 1 g daily (does not cover P*seudomonas*)
- Excellent gram-negative and anaerobic coverage

Antimicrobial therapy for secondary peritonitis: Anaerobic coverage

Metronidazole

- Dosing: 500 mg IV Q6–8H or 400–500 mg PO TID
- Excellent anaerobic coverage
- Minimal aerobic gram-negative coverage
- *Always combine with aerobic agent*

Clindamycin

- Dosing: 600 mg IV Q6–8H
- Good anaerobic coverage
- Some gram-positive aerobes covered
- Emerging resistance in *Bacteroides*
- Less preferred than metronidazole + cephalosporin

Carbapenems (cover both aerobes and anaerobes)

- Single agent sufficient
- Reserved for β-lactam-resistant organisms or severe disease

New antibiotics active against resistant gram-negative bacilli

Agent	ESBLs	AmpC	KPC	OXA-48	MBL	CRAB	CRPA
Plazomicin (not available in EU)	✓	✓	✓	✓	±	✗	✗
Eravacycline	✓	✓	✓	✓	✓	✓	✗
Tigecycline	✓	✓	✓	✓	✓	✓	✗
Temocillin	✓	✓	✓	✗	✗	✗	✗
Cefiderocol	✓	✓	✓	✓	✓	✓	✓
Ceftazidime/avibactam	✓	✓	✓	✓	✗	✗	±
Ceftolozane/tazobactam	✓	✗	✗	✗	✗	✗	✓
Meropenem/vaborbactam	✓	✓	✓	✗	✗	✗	✗
Imipenem/relebactam	✓	✓	✓	✗	✗	✗	±
Ampicillin/sulbactam + ceftazidime/avibactam	✓	✓	✓	✓	✓	✗	±

ESBL- extended spectrum beta lactamase, AmpC- inducible extended beta lactamase, KPC, OXA-48 MBL are carbapenemases, CRAB- carbapenem-resistant Acinetobacer baumanii, CRPA- carbapenem-resistant *Pseudomonas*

To treat ESBL- or AmpC-producing Enterobacterales, all above antibiotics can be used except for ceftolozane/tazobactam against hyperproducing AmpC Enterobacterales. To treat KPC-producing Enterobacterales, all can be used but ceftolozane/tazobactam. To treat OXA-48 producers, all but temocillin, ceftolozane/tazobactam, meropenem/vaborbactam, and imipenem/relebactam. To treat MBL producers, only eravacycline, tigecycline, cefiderocol, and

New agents for resistant organisms in secondary peritonitis

Extended-Spectrum Agents

Agent	Organism Coverage	When to Use
Ceftolozane-tazobactam	Pseudomonas, resistant GN	Healthcare-associated, MDR risk
Ceftazidime-avibactam	Carbapenem-resistant organisms, ESBLs	Suspected resistance, prior carbapenems
Meropenem-vaborbactam	Metallo-β-lactamases, carbapenem-resistant	Last-resort therapy

Anti-Candida Agents

Agent	Dosing	When to Use
Fluconazole	400–800 mg/day	Upper GI perforation, prolonged hospitalization
Anidulafungin	200 mg loading, then 100 mg/day	Suspected azole-resistant Candida
Caspofungin	70 mg loading, then 50 mg/day	Alternative to fluconazole, suspected azole-resistant Candida
Micafungin	100 mg daily	Alternative to fluconazole, suspected azole-resistant Candida
Liposomal amphotericin B	3–5 mg/kg/day	Suspected azole-resistant Candida, nephrotoxic

Vancomycin

- Dosing: 15–20 mg/kg IV Q8–12H (goal trough 15–20 µg/mL)
- For MRSA or severe penicillin allergy
- Reserve use to avoid resistance

Source Control in Secondary Peritonitis

Principles of Source Control

1. **Identify and eliminate source** (perforation, necrotic tissue, abscess)
2. **Operative debridement**: Remove devitalized tissue, purulent material
3. **GI decompression**: NG tube, venting catheter if ileus
4. **Peritoneal lavage**: Saline irrigation to reduce bacterial load
5. **Drainage** of dependent recesses (pelvis, paracolic gutters, Morrison's pouch)
6. **Repair or resection** of primary pathology

Timing

- **Emergent** (within 1–2 hours): Perforation with peritonitis, uncontrolled sepsis
- **Urgent** (within 6–12 hours): Contained abscess, clinical deterioration
- **Delayed** (>24 hours): Selected patients with localized collection responding to antibiotics

Operative Techniques

- Primary repair when possible (peptic ulcer perforation)
- Resection with anastomosis vs. colostomy (depends on contamination, blood supply)
- Avoid contamination of clean peritoneal surfaces

Source control is often ESSENTIAL in secondary peritonitis. Antibiotics alone fail in cases of inadequate source control. The timing of surgery depends on presentation (shock vs. hemodynamically stable) and anatomical factors. Percutaneous drainage under CT guidance is increasingly used for select abscess collections, delaying or avoiding surgery.

Supportive Care in Secondary Peritonitis

Hemodynamic Management

- Aggressive fluid resuscitation (often requires 5–10 L in first 24 hours)
- Vasopressors if hypotension persists after fluids (norepinephrine first-line)
- Goal: Restore tissue perfusion, prevent organ failure

Respiratory Support

- Mechanical ventilation if acute respiratory distress syndome (ARDS) develops
- Positive end-expiratory pressure (PEEP) to improve oxygenation
- Careful fluid management to balance resuscitation and pulmonary edema

Nutritional Support

- Enteral nutrition when possible (preserves gut mucosa)
- Total parenteral nutrition if unable to tolerate enteral feeds
- Early nutrition improves outcomes and reduces infection risk

Organ Support

- Renal replacement therapy if acute kidney injury develops
- Correcting coagulopathy (fresh frozen plasma, vitamin K, platelets)
- Managing hyperglycemia (insulin therapy, tight control)

Prevention of Postoperative Peritonitis

Preoperative Measures

- Appropriate patient selection and optimization
- Preoperative antibiotics (within 60 minutes of incision)
- Hair clipping (not shaving) to prevent microabrasions

Intraoperative Measures

- Strict aseptic technique
- Avoid contamination during bowel manipulation
- Gentle tissue handling to minimize ischemia
- Adequate hemostasis
- Maintain normothermia and normocapnia

Postoperative Measures

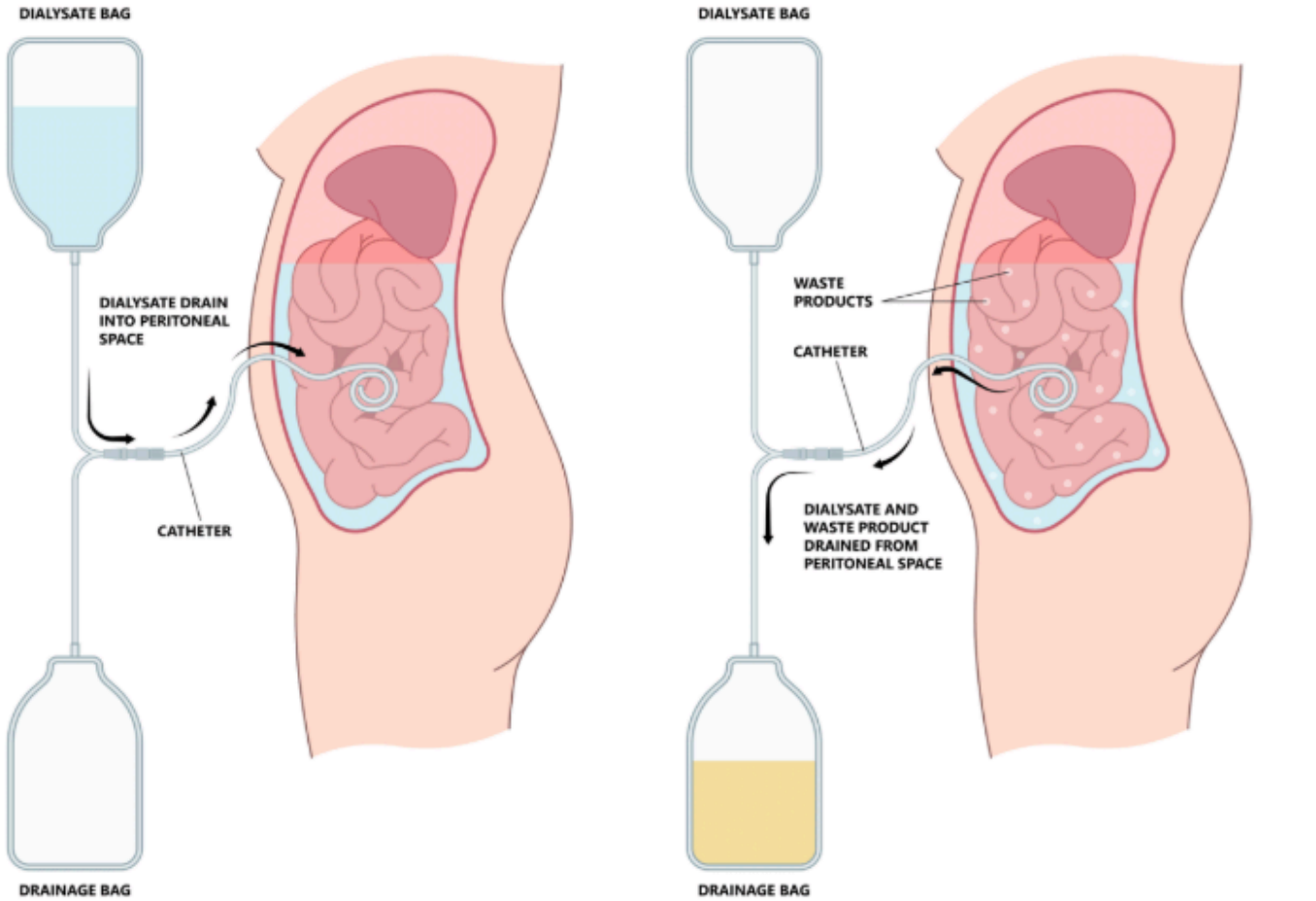
- Remove drains/catheters as soon as possible
- Monitor for signs of sepsis (fever, tachycardia, leukocytosis)
- Early mobilization and feeding to promote GI function
- Recognize anastomotic leaks early (CT imaging if deterioration)

Surgical Technique Factors

- Primary vs. staged repair
- Choice of anastomosis (mechanical, hand-sewn)
- Drain placement (controversial; generally avoid unless high contamination)

Peritoneal-dialysis associated peritonitis

Peritoneal dialysis



PD-Associated peritonitis: Epidemiology

PD Peritonitis: Microbiology

Organism Distribution

Category	Prevalence	Examples
Gram-positive	60–80%	*S. epidermidis, S. aureus, Streptococcus spp.*
Gram-negative	15–30%	*E. coli, Klebsiella*, Enterobacterales
Fungi	5–10% (increasing)	*Candida spp.*, rare molds
Mycobacteria	<5%	*M. tuberculosis* (geographic dependent)

Common Pathogens

- Coagulase-negative staphylococci (*S. epidermidis*): Most common gram-positive
- *S. aureus*: Often from patient's skin flora, more virulent
- *Streptococcus* spp.: May indicate bowel translocation
- Enterococci: Rare but significant if present
- P*seudomonas*: Often healthcare-associated, poor prognosis

Biofilm Considerations

- *S. epidermidis* forms biofilm on catheter, protects from immune response and antibiotics

PD Peritonitis: Diagnosis

ISPD[3] Criteria (International Society for PD)

Diagnosis requires 2 of 3:

1. **Cloudy peritoneal effluent** (visual inspection)
2. **Peritoneal WBC >100 cells/mm³** (typically >50% PMN)
3. **Positive dialysate culture or Gram stain**

Typical Laboratory Findings

Parameter	Finding
Appearance	Cloudy/turbid (vs. clear in health)
WBC count	>100/mm³, usually 500–10,000
PMN predominance	>50% (polymorphonuclear)
Bacteria	Gram stain positive in 40–50%
Culture yield	Positive in 90–95% with proper technique

Specimen Collection

- Obtain specimen during fresh exchange
- Use sterile technique
- Send for cell count, differential, Gram stain, culture (aerobic and anaerobic)

PD Peritonitis: Treatment Principles

Empiric Therapy

- **Intraperitoneal (IP) antibiotics preferred**
- Cover gram-positives initially (cefazolin or vancomycin)
- Add gram-negative coverage (ceftazidime or aminoglycoside)

Standard IP Regimens

Agent	Dosing (Bolus / Maintenance)	Target
Cefazolin	500 mg/2 L exchanges / 125 mg/2 L	Gram-positive coverage
Ceftazidime	500 mg/2 L exchanges / 125 mg/2 L	Gram-negative, Pseudomonas
Vancomycin	30 mg/kg loading / 15 mg/kg per exchange	MRSA, resistant gram-positive

Duration

- **ISPD guidelines**: 14–21 days of IP antibiotics, assess response at 2–5 days (peritoneal fluid should clear)
- Culture results guide de-escalation

Dialysis catheter management

- Continue CAPD during treatment (antibiotics via dialysate)
- Remove catheter if no improvement by day 4–5
- Remove catheter for fungal peritonitis (usually)
- Remove if exit-site or tunnel infection present

IP administration achieves high local concentrations and is preferred to IV therapy. Continuous IP exposure (via each exchange) maintains levels better than intermittent dosing. Patient tolerance of continued CAPD during treatment is variable; some require brief hospitalization with IV therapy and temporary

PD Peritonitis: Fungal and Mycobacterial Infection

Fungal PD Peritonitis

- Incidence: 5–10% of all CAPD peritonitis: Most common: Candida spp. (>90% of fungal cases)
- Risk factors: prior antibiotics, immunosuppression, diabetes

Management of Fungal Peritonitis

- **Antifungal agents** (fluconazole, amphotericin B): Often requires IV therapy in addition to IP
- **Mandatory catheter removal** (biofilm barrier limits drug penetration)

Mycobacterial PD Peritonitis

- *M. tuberculosis*: Geographic variation (high in endemic areas)
- **Nontuberculous mycobacteria** (NTM): Rising incidence

Management of Mycobacterial Peritonitis

- Long course of anti-TB drugs (6+ months for TB)
- Continue CAPD during treatment (if possible)
- Catheter removal for persistent infection (NTM)
- Diagnosis delay common (insidious presentation)

PD Peritonitis: Outcomes and Catheter Removal

Outcomes with Treatment

- **Resolution**: 90–95% of bacterial peritonitis cured with appropriate therapy
- **Recurrence**: 20–30% of patients experience repeat episodes
- **Modality failure**: 30–40% eventually switch to hemodialysis

Indications for Catheter Removal

- **Fungal peritonitis** (almost universally)
- **Failure to respond** by 4–5 days of therapy
- **Exit-site or tunnel infection** with peritonitis
- **Polymicrobial peritonitis** (suggests bowel perforation; need imaging)
- **Refractory peritonitis** (multiple episodes with same organism)
- **Patient choice** (prefer hemodialysis)

Prevention of Recurrence

- Patient education (proper exchange technique, hand hygiene)
- Catheter modification (Y-set or disconnect systems)
- Use of prophylactic topical antibiotics (mupirocin)
- More frequent exchanges during acute peritonitis
- Prompt treatment of exit-site infections

Intraperitoneal Abscess

Intraperitoneal Abscesses: Definition and Epidemiology

Definition

- Focal collections of pus within the peritoneal cavity
- Walled-off by fibrin, omentum, and peritoneum
- Complications of primary or secondary peritonitis

Common Locations

Location	Frequency	Clinical Features
Pelvic	30–40%	Dependent site; difficult to examine
Paracolic gutters	20–30%	Follow colon anatomy
Subphrenic	15–20%	Upper abdomen; may irritate diaphragm
Perihepatic	10–15%	Near hepatic hilum, fissures
Morrison’s pouch	5–10%	Dependent upper abdomen

Formation Mechanism

- Peritoneal routes of drainage determine abscess site
- Right paracolic gutter → pelvis (gravity)
- Upper abdomen → subphrenic spaces
- Lesser sac collections from anterior abdominal pathology

Intraperitoneal Abscesses: Microbiology

Organism Characteristics

- **Polymicrobial** (usually 2–5 organisms)
- Reflects organisms from primary source (GI tract)
- Similar to secondary peritonitis microbiology

Common Organisms

- *E. coli* (gram-negative rod)
- *Bacteroides fragilis* (obligate anaerobe)
- *Streptococcus* spp. (gram-positive cocci)
- *Enterococcus* spp. (gram-positive coccus)
- Other anaerobes (*Peptostreptococcus, Clostridium*)

Culture Characteristics

- Lower culture yield than peritoneal fluid (bacteria within abscess wall)
- Gram stain may guide initial therapy
- Anaerobic cultures essential (often missed if not specifically requested)

Implications for Therapy

- Broad-spectrum empiric coverage needed
- β-lactam + anaerobic agent, or carbapenem
- Culture-guided de-escalation critical
- Drainage required in addition to antibiotics

Intraperitoneal Abscesses: Clinical Presentation

Symptoms - **Fever** (may be low-grade, intermittent) - **Localized abdominal or flank pain** (depending on location) - **GI symptoms**: nausea, vomiting, anorexia (if gastric irritation) - **Urinary symptoms**: dysuria, frequency (if bladder compression) - **Subphrenic abscess**: Shoulder pain (referred), dyspnea

Physical Findings - Localized abdominal tenderness (over abscess site) - Palpable mass (large collections only) - Fever - Hemodynamic instability (if large, with systemic toxicity)

Laboratory/Imaging Abnormalities - Leukocytosis (may be modest in chronic abscess) - Elevated CRP - CT findings: Fluid collection with enhancing rim, air-fluid level if gas-forming organism

Abscess presentation can be subtle compared to acute peritonitis. Some abscesses develop insidiously over days to weeks with low-grade fever and vague discomfort. Others present acutely with severe localized symptoms. Shoulder pain in subphrenic abscess is a classic referred pain finding. Imaging is critical for

Intraperitoneal Abscesses: Diagnosis

Imaging Modalities

Modality	Sensitivity	Utility
CT with IV contrast	95–97%	Gold standard; localization; guidance for drainage
Ultrasound	90–95%	Real-time, portable; bedside assessment
MRI	90–95%	Excellent soft tissue; limited in acute care

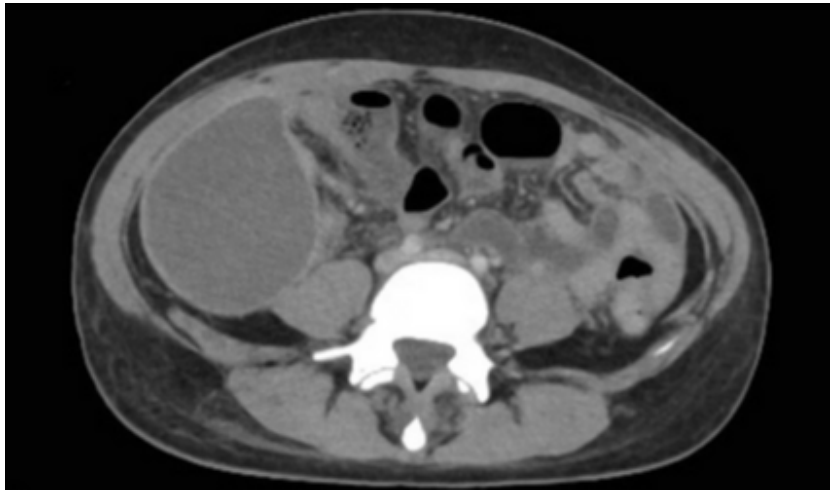
CT Findings

- Fluid collection with enhancing rim
- Air-fluid level (gas-forming organisms)
- Loculation (multiloculated abscesses)
- Associated organ pathology
- Size and location relative to surrounding structures

Paracentesis (if not already done)

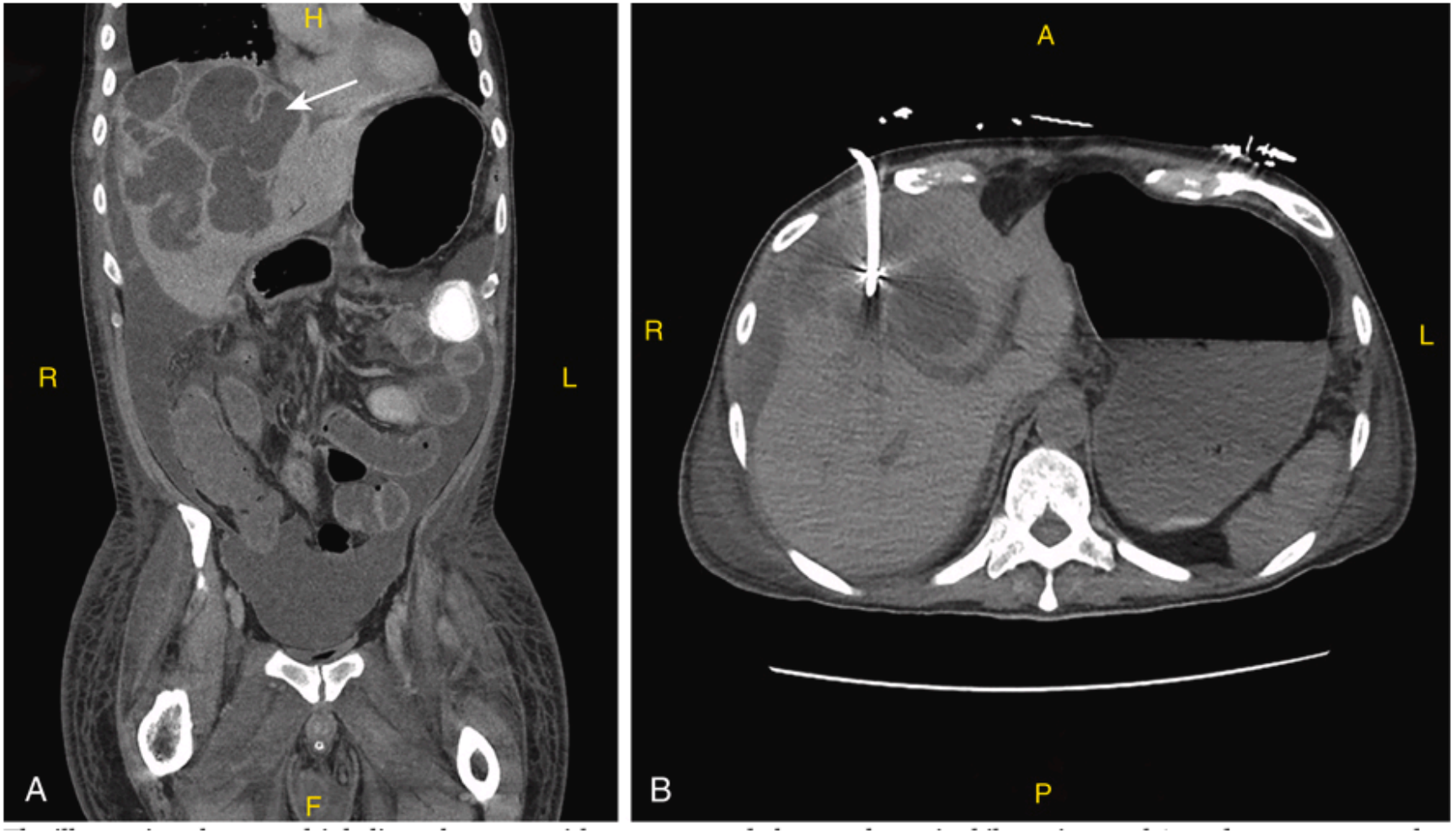
- May be diagnostic if fluid obtained
- Often done as precursor to drainage
- Culture crucial for organism identification

CT imaging: intraperitoneal abscess



1. At least two large loculated fluid collections with rim enhancement measuring up to 10.2 cm in the right paracolic gutter and 13.6 cm in the pelvis. These may represent seromas in the postsurgical setting versus abscesses.
2. Mild mesenteric fat stranding in the lower abdomen and pelvis, likely related to postsurgical changes within acute inflammatory process not excluded.
3. Additional cystic focus in the left pelvis measuring approximately 2.4 cm which may represent an additional loculated fluid collection versus cyst arising from the left ovary.
4. Small to moderate left pleural effusion with adjacent atelectasis.
5. Minimal subcutaneous emphysema along the right lower ventral abdominal wall, may represent postsurgical changes versus infection.

Liver abscess



The illustration shows multiple liver abscesses with an arrow and abnormal terminal ileum in panel A, and a percutaneously placed drain in one abscess in panel B. Panel A shows a computed tomography scan of the abdomen and pelvis in a coronal view, indicating multiple liver abscesses, marked with an arrow, and an abnormal terminal ileum located caudal to the liver. The abscesses are surrounded by secondarily infected ascites. Panel B presents a computed tomography scan

Intraperitoneal Abscesses: Treatment — Drainage Decisions

Percutaneous vs. Surgical Drainage

Factor	Percutaneous	Surgical
Access	Image-guided (CT/US)	Direct visualization
Invasiveness	Minimally invasive	Formal operation
Morbidity	Lower	Higher (general anesthesia, incisions)
Efficacy	80–90% success	Near 100% success
Best used in	Accessible collections, stable patient	Inaccessible collections, unstable patient

Selection Criteria for Percutaneous Drainage

- Accessible collection (not multiloculated or complex)
- > 3–4 cm in diameter (smaller often respond to antibiotics alone)
- Hemodynamically stable patient
- Expertise available for image-guided drainage

When Surgery is Preferred

Intraperitoneal Abscesses: Antibiotic Therapy

Duration and Route

- **IV therapy** initially (acute abscess)
- **Transition to oral** after clinical improvement (usually 7–10 days IV, then oral)
- **Total course**: 10–14 days (shorter if source controlled and drained)

Empiric Regimen

- **β-lactam + β-lactamase inhibitor**: Piperacillin-tazobactam 4.5 g Q6–8H
- **Or cephalosporin + metronidazole**: Ceftriaxone 2 g Q12H + metronidazole 500 mg TID
- **Or carbapenem**: Meropenem 1 g Q8H

Transition to Oral

- After clinical improvement and drain output minimal
- Options: Amoxicillin-clavulanate, fluoroquinolone + metronidazole (limited)

Culture-Directed De-escalation

- Narrow spectrum once organism susceptibilities known
- Select oral agent based on susceptibilities and drug properties
- Complete course based on clinical response (not arbitrary duration)

Intraperitoneal Abscesses: Prognosis

Outcomes with Appropriate Management

- **Cure rate**: 85–95% with combined drainage and antibiotics
- **Mortality**: 5–15% (higher in elderly, immunocompromised)

Factors Affecting Prognosis

Factor	Impact
Time to diagnosis	Delayed diagnosis → higher mortality
Size >5 cm	Larger collections → worse prognosis
Multiple organisms (esp. anaerobes)	More virulent; worse outcome
Associated peritonitis	Indicates severe primary disease
Patient age, comorbidities	Older, immunocompromised → worse

Complications of Treatment

- **Recurrent abscess**: May occur if inadequate drainage or source not controlled
- **Fistula formation**: If drain erodes into viscus
- **Drain site infection**: May progress if not managed
- **Incomplete resolution**: Imaging follow-up recommended at 4–6 weeks

SECTION 5: SUMMARY AND KEY TAKEAWAYS

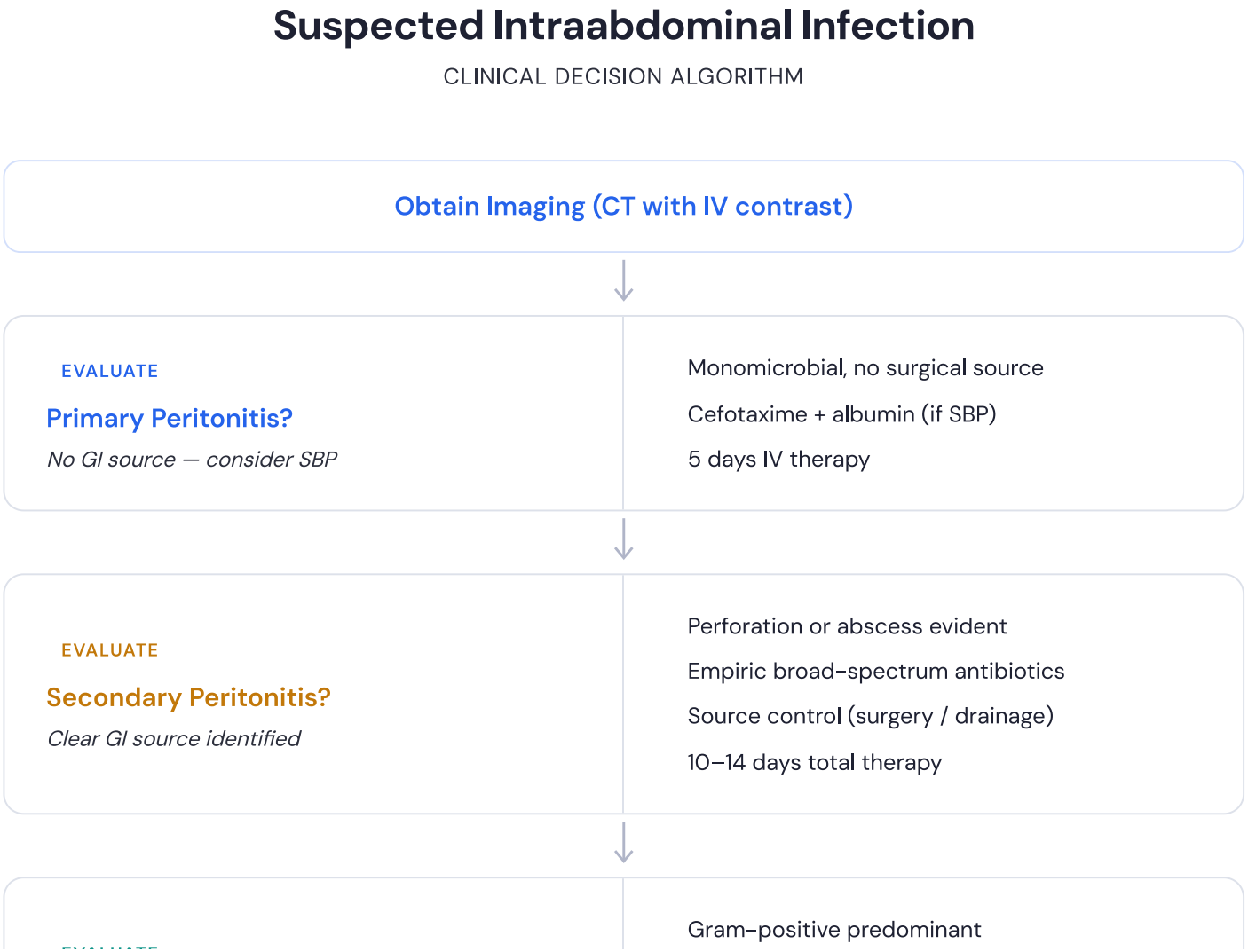
Clinical Pearl: The 10 Key Takeaways on IAI

1. **Primary peritonitis (~1% of cases)** occurs without evident abdominal source; typically monomicrobial gram-negatives in cirrhotic patients; diagnosis confirmed by PMN >250/mm^3 and positive culture.
2. **SBP prevention with antibiotics** (norfloxacin primary prophylaxis) reduces incidence 50–60%; secondary prophylaxis after first episode is lifelong.
3. **Secondary peritonitis (80–90% of cases)** is polymicrobial, requires source control, and mortality is more dependent on adequacy of drainage/surgery than antibiotic choice.
4. **Empiric therapy must cover aerobes and anaerobes**; β-lactam + β-lactamase inhibitor or cephalosporin + metronidazole are standard options for community-acquired secondary peritonitis.
5. **Tertiary peritonitis** (persistent after source control) carries 30–64% mortality; repeated surgery often worsens outcomes; supportive care and selective re-imaging are key.

Clinical Pearl: Key Takeaways (continued)

6. **CT imaging is 97% sensitive** for complicated IAI; pneumoperitoneum indicates perforation and need for urgent surgery.
7. **Fever is not always present** in elderly, immunocompromised, or on immunosuppressive therapy; maintain high suspicion with abdominal pain and leukocytosis.
8. **CAPD peritonitis is predominantly gram-positive** (touch contamination) and responds to IP antibiotics in 90–95%; fungal peritonitis requires catheter removal.
9. **Intraperitoneal abscesses require both drainage and antibiotics**; percutaneous drainage under imaging guidance is now first-line for accessible, hemodynamically stable patients.
0. **Early recognition and source control are critical** to outcome; every hour of delay in antibiotics increases mortality; source control (surgery or drainage) should proceed in parallel with antimicrobials, not sequentially.

Algorithm: Approach to Suspected IAI



When to Worry: Red Flags for MDR Organisms

High-Risk Features for Resistant Pathogens

- **Healthcare-associated infection** (recent hospitalization, surgery, invasive procedure)
- **MRSA colonization** or prior MRSA infection
- **Prior broad-spectrum antibiotics** (β-lactams, carbapenems, fluoroquinolones)
- **Immunosuppression** (chemotherapy, transplant, HIV with CD4 <200)
- **Severe illness at presentation** (septic shock, APACHE >25)
- **Candida species** in blood cultures or peritoneal fluid
- **Diabetic or renal failure** patients
- **Polymicrobial bacteremia** with unusual organisms

Modified Empiric Therapy for High-Risk Patients

- Extended-spectrum agent: Ceftazidime-avibactam or ceftolozane-tazobactam
- Add vancomycin for MRSA coverage
- Add antifungal (fluconazole or echinocandin) if high risk for Candida
- Consider consulting infectious diseases specialist

Quick Reference: Empiric Antibiotic Selection by Scenario

Clinical Scenario	First-Line Empiric	Alternative	Duration
Primary peritonitis (SBP)	Cefotaxime 2 g Q6–8H	Ciprofloxacin 400 mg PO Q12H	5 days
Secondary peritonitis, CA	Piperacillin-tazobactam 4.5 g Q6–8H	Ceftriaxone 2 g Q12H + metronidazole 500 mg TID	10–14 days
Secondary peritonitis, HA	3rd-4th gen Cephalosporin + vancomycin + anti-Candida	Carbapenem ± antifungal	10–14 days
CAPD peritonitis	Cefazolin IP + ceftazidime IP	Vancomycin IP	14–21 days
Intraperitoneal abscess	Piperacillin-tazobactam 4.5 g Q6–8H	Ceftriaxone + metronidazole	10–14 days

Abbreviations: CA = community-acquired; HA = healthcare-associated; IP = intraperitoneal

Further Learning

Key References

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