Intraabdominal Infections, Part 2

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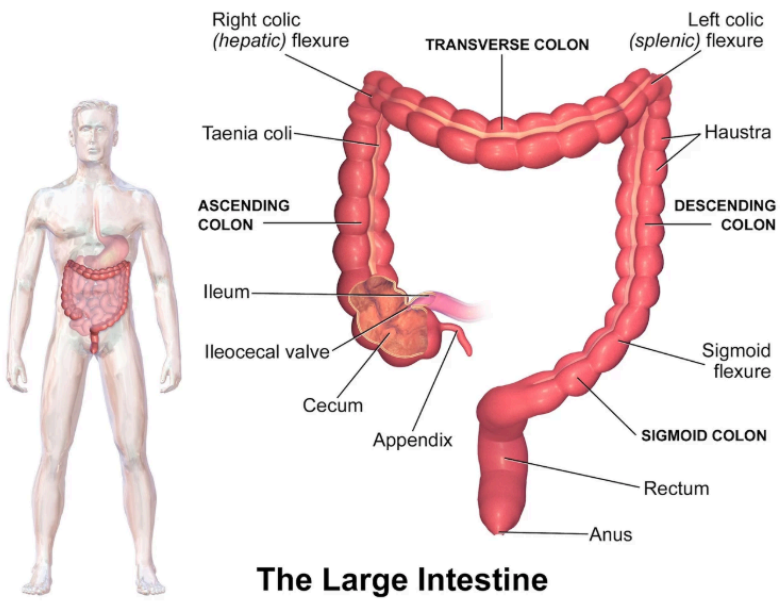
Anatomy of the appendix



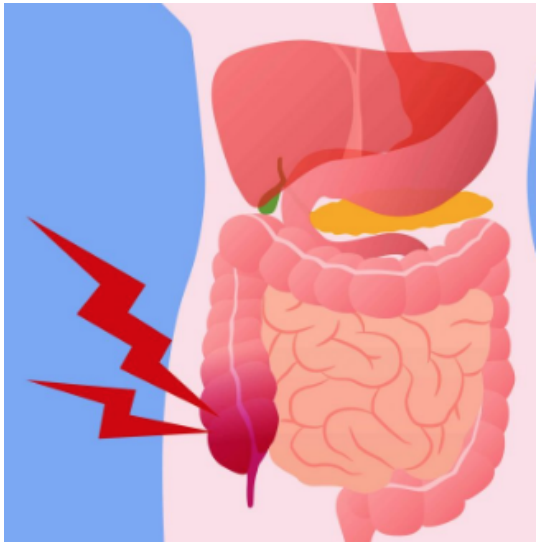
The Large Intestine

- Tube-shaped structure, 5–10 cm long
- Arises 2–3 cm below terminal ileum
- Medial posterior wall of cecum
- Common atypical positions: descending pelvic, transverse retrocecal, ascending postileal

Atypical positions are common and can significantly alter the clinical presentation, making diagnosis more challenging. A retrocecal

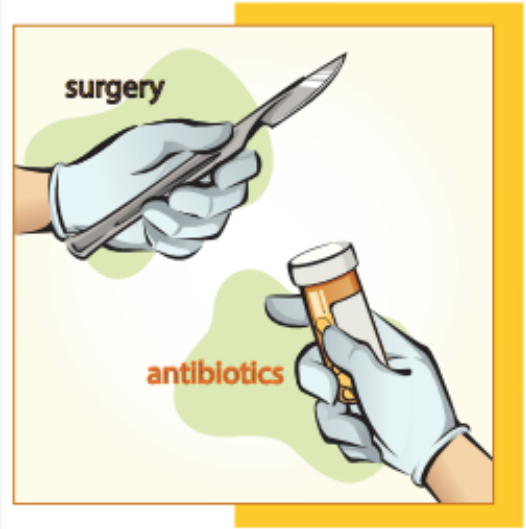
What is appendicitis?

- Acute inflammation of the vermiform appendix
- Often related to obstruction
- Complicated by polymicrobial infection
- Complications: perforation, peritonitis, intra-abdominal abscesses



Epidemiology: By the Numbers

- Lifetime risk: **8.6% in men**, **6.7% in women**
- ~108 cases per 100,000 person-years (US)
- **190,000** appendectomies in the US (2018)
- 42% decrease in appendectomies since 2011
- Mortality <1% (but ≥5% in elderly)



The decline in appendectomies reflects the growing adoption of antibiotic-first strategies for uncomplicated appendicitis. This represents a

Age and Sex Distribution

- Peak incidence: **15–25 years**
- Rare in infants
- Declines after age 45 (<25% of cases)
- Male-to-female ratio: **1.4:1**
- Most common surgery for individuals ≤17 years

Geographic Variation

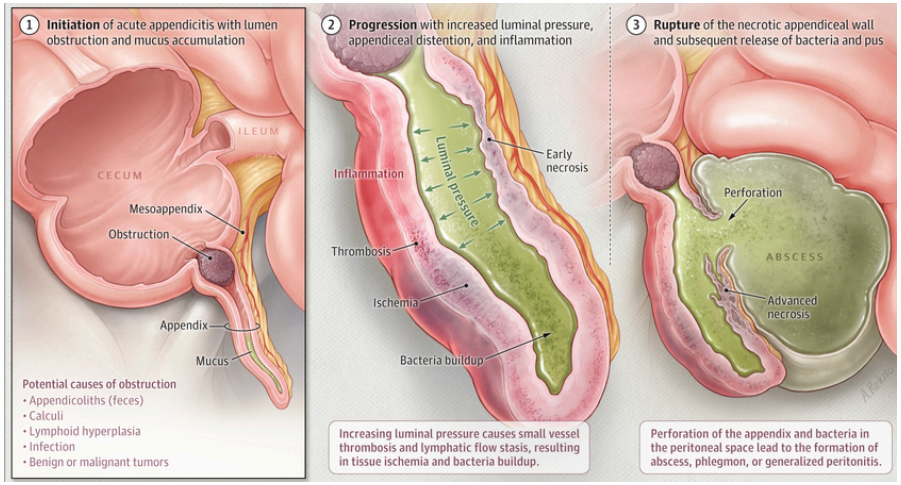
- Lower incidence in rural, nonindustrialized areas
- Incidence increases with industrialization
- Similar pattern seen with diverticulitis
- Estimated lifetime risk <1% in rural sub-Saharan Africa

The Appendix: Not just vestigial

- Contributes to gut microbiome homeostasis
- Mucus-rich biofilm houses resident bacteria
- Acts as a microbial “sanctuary”
- Repopulates gut after diarrheal illness
- Sheds bacteria at 2–3 mL/day

Recent studies have shifted our understanding of the appendix from a useless vestige to an important component of the gut immune system.

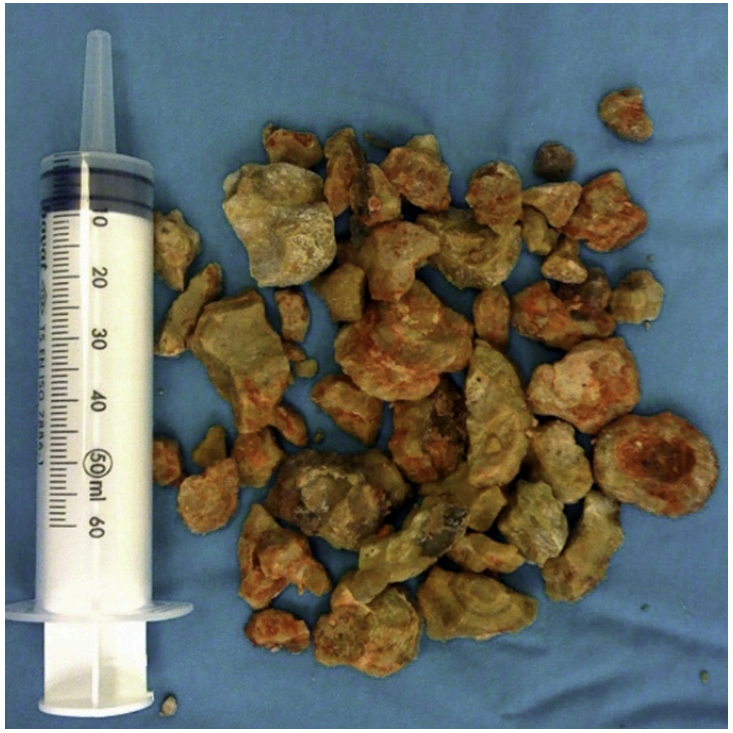
Classic pathogenesis model



1. Luminal obstruction (fecaliths, foreign bodies, tumor)
2. Mucus accumulation → increased intraluminal pressure
3. Lymphatic/vascular compression
4. Ischemic mucosal damage
5. Microbial invasion and inflammation
6. Gangrene → perforation (if untreated)

The pathologic hallmark is polymorphonuclear cells within the appendiceal wall with edema and vascular congestion. This model has been

Challenging the classic pathology model



- Fecaliths found in only a **minority** of cases
- Medical (antibiotic) treatment alone is effective in many patients
- Alternative etiologies proposed:
  - Infectious agents
  - Dietary fiber deficiency
  - Microbiome dysbiosis
  - Genetic susceptibility
  - Environmental factors

The dietary fiber hypothesis

- Short's observation (early 20th century): UK vs. Africa comparison
- Burkitt's hypothesis: fiber as bulking agent reduces risk
- Fiber prevents fecalith formation and impaction
- Microbiome influenced by dietary fiber content
- Interest in diet–microbiome–appendicitis axis growing

The observation that appendicitis rates increase with industrialization and the adoption of low-fiber Western diets has persisted for over a

Microbiology: Culture-based

- 10–14 organisms typically can be cultured from inflamed appendix
- Reflects colonic microbiota
- Key organisms:
  - *Escherichia coli*
  - *Bacteroides fragilis* group
  - *Prevotella* spp.
  - *Peptostreptococcus* spp.
  - *Streptococcus anginosus* group

Pseudomonas aeruginosa found in 4–15% of cases.

Microbiology: 16S rRNA Insights

- Appendiceal microbiome is **remarkably diverse**
- Dozens of phyla, hundreds of species
- Distinct from other GI tract locations
- Enriched in inflamed appendix:
  - *Fusobacterium*
  - *Peptostreptococcus*
  - *Parvimonas*
- Dysbiosis may drive appendicitis (not obstruction)

These oral-type bacteria enriched in the inflamed appendix has led to the dysbiosis hypothesis—a paradigm shift in understanding

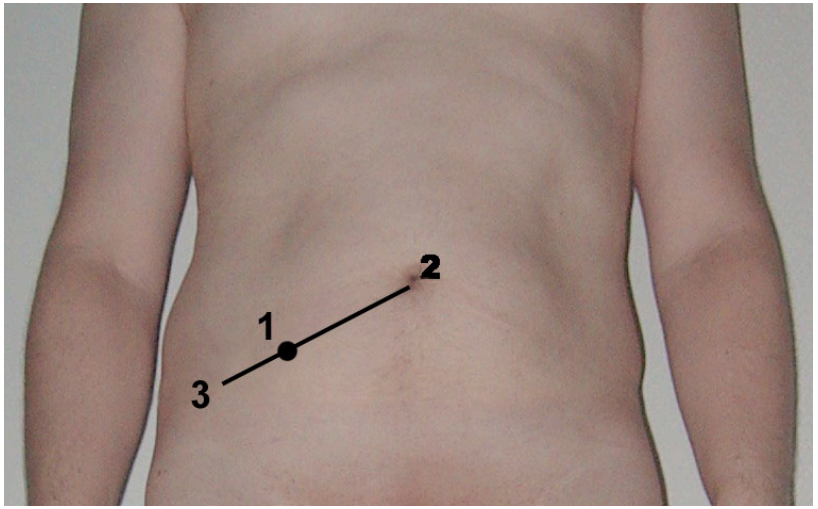
Specific pathogens to know

Organism	Presentation
Yersinia spp.	Ileocecitis, mesenteric adenitis → mimics appendicitis
Campylobacter	Pseudoappendicitis with ileocecitis
Salmonella	Pseudoappendicitis with mesenteric adenitis
E. histolytica	True appendicitis (rare)
Viruses (EBV, CMV, measles)	Mesenteric adenitis

Most cases of mesenteric adenitis have an unidentified infectious cause. The disorder is usually discovered at surgery for suspected

Classic clinical presentation

1. **Early**: Colicky, visceral periumbilical pain
2. **6–24 hours**: Migration to RLQ (somatic pain)
3. Pain at **McBurney point** (anterior appendix)
4. Associated: low-grade fever, anorexia, nausea, vomiting



McBurney point is located two to three fingerbreadths above the right anterior superior iliac spine on a line drawn to the umbilicus.

Physical examination signs

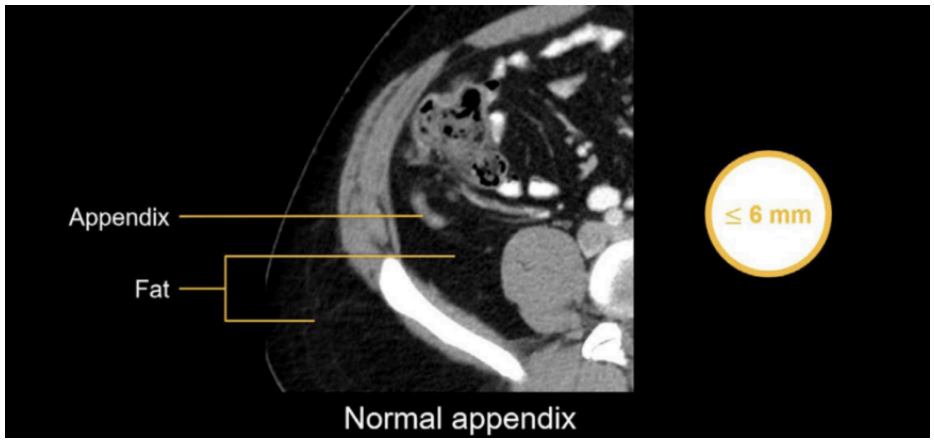
Sign	Description
Rovsing sign	RLQ pain with LLQ palpation
Psoas sign	Pain with active hip extension
Obturator sign	Pain with internal hip rotation
Guarding	Involuntary muscle contraction
Rebound tenderness	Pain on release of pressure

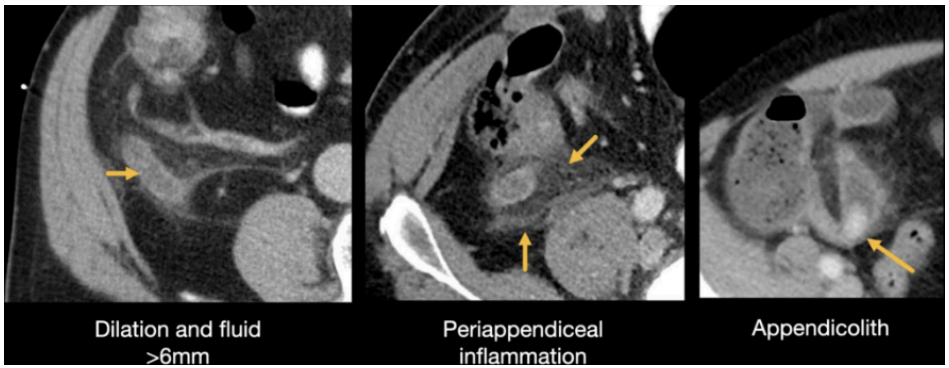
High fever or sudden pain reduction suggests perforation. Abdominal rigidity suggests diffuse peritonitis. No single finding is diagnostic, and

Diagnostic Challenges

> ⚠ **Warning**
>
> **Diagnosis is more difficult in:**
>
> - Women of childbearing age (gynecologic mimics)
> - Pelvic appendixes (pelvic/LLQ pain)
> - Third-trimester pregnancy (RUQ pain shift)
> - Elderly patients (atypical presentations)
> - Young children (inability to localize pain)

CT Imaging: Normal vs. abnormal





Imaging

- **CT with IV contrast**: Sensitivity and specificity both >95%
  - Preferred in adults
  - Dilated appendix >6 mm, periappendiceal fat stranding
- **Ultrasonography**: First-line in children and pregnant women
  - Sensitivity 70–90%
- **MRI**: Alternative in pregnancy

Scoring systems

Alvarado Score (MANTRELS): migration, anorexia, nausea, tenderness, rebound, elevated temp, leukocytosis, shift to left

Letter	Clinical Feature	Points
M	Migration of pain to right lower quadrant	1
A	Anorexia	1
N	Nausea/vomiting	1
T	Tenderness in right lower quadrant	2
R	Rebound pain	1
E	Elevated temperature (>37.3°C)	1
L	Leukocytosis (>10,000/mm^3)	2
S	Shift to left (neutrophilia >75%)	1

Alvarado Score (MANTRELS): Interpretation

Score	Risk	Recommendation
1–4	Low	Appendicitis unlikely; consider discharge with observation
5–6	Equivocal/intermediate	Further workup warranted (imaging, observation, surgical consult)
7–8	High	Likely appendicitis; surgical consultation indicated
9–10	Very high	Appendicitis highly probable; proceed to surgery

IR Score (Appendicitis Inflammatory Response):

Total: 0–12 points

Variable	Criteria	Points
Vomiting	Present	1
Right lower quadrant pain	Present	1
Rebound tenderness or muscular defense	Mild	1
	Moderate	2
	Strong	3
Temperature	≥ 38.5°C	1
Leukocytosis (×10^9/L)	10.0–14.9	1
	≥ 15.0	2
Proportion of neutrophils (%)	70–84%	1
	≥ 85%	2
CRP (mg/L)	10–49	1

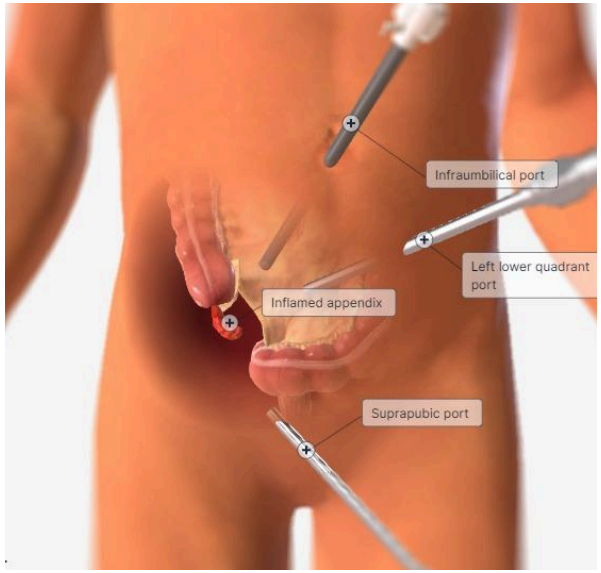
IR score interpretation

Interpretation

Score	Risk	Recommendation
0–4	Low	Appendicitis unlikely; discharge with observation
5–8	Indeterminate	Imaging, active observation, or surgical consult
9–12	High	Appendicitis highly probable; surgical intervention

Key advantages over Alvarado

Treatment: Surgical



- **Laparoscopic appendectomy**: Standard of care
  - Shorter hospital stay
  - Less postoperative pain
  - Lower wound infection rates
- Open appendectomy: reserved for complex cases
- Interval appendectomy: considered after initial conservative management of appendiceal abscess

Treatment: Antibiotic-first strategy

> **Tip**
>
> An “antibiotic first” strategy has emerged as a safe and effective option for **uncomplicated** appendicitis

- Avoids surgery in ~60–70% of patients
- Outcomes comparable to appendectomy
- Recurrence rate ~25–30% at 5 years
- Patient selection is key: uncomplicated, no fecalith

The CODA[2] and APPAC[3] trials have provided strong evidence for the antibiotic-first approach. However, patient selection and shared decision-making are critical.

Antibiotic regimens for appendicitis

- **Uncomplicated**: Piperacillin-tazobactam or ceftriaxone + metronidazole
- **Perforated**: IV antibiotics 3–5 days → oral step-down
- Duration guided by clinical response
- Ensure anaerobic coverage (metronidazole)

Broad-spectrum coverage targeting both aerobic gram-negatives and anaerobes is essential given the polymicrobial nature of the infection.

Appendicitis: Key takeaways

- Most common surgical emergency of the abdomen
- Classic: periumbilical pain → RLQ migration
- CT is the imaging gold standard in adults
- Laparoscopic appendectomy remains definitive treatment
- Antibiotic-first strategy: valid for uncomplicated cases
- Microbiome dysbiosis: emerging pathogenic concept

Infections of the liver and biliary system

Liver abscess: Two categories

Feature	Amebic	Pyogenic
Cause	*E. histolytica*	Bacterial (polymicrobial or monomicrobial)
Pathology	Hepatocyte apoptosis	Suppurative infection
“Pus” appearance	Anchovy paste (nonpurulent)	Purulent
Primary treatment	Medical (metronidazole)	Drainage + antibiotics

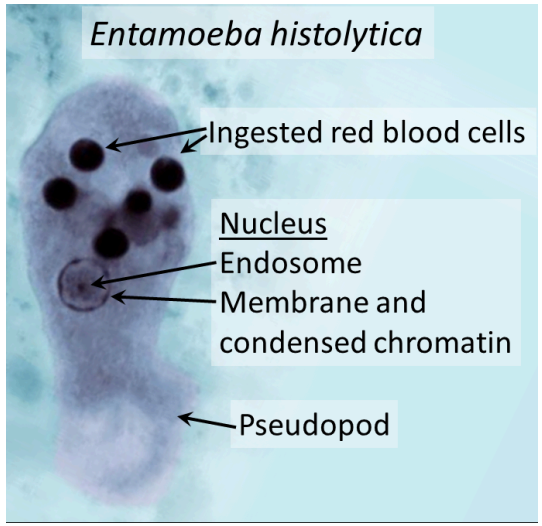
These two types of liver abscess have fundamentally different pathophysiology, microbiology, and management strategies. Distinguishing

Amebic Liver Abscess: Epidemiology

- Rare in the US (travelers, immigrants, MSM)
- 2,983 total cases of amebiasis in 1994
- Annual incidence decreasing 2.4% per year
- Worldwide: *E. histolytica* second only to malaria as cause of death from parasitic disease
- Male: female ratio = ranging from 5–18 : 1 in epidemiological studies

The epidemiology has been greatly informed by distinguishing *E. histolytica* from *E. dispar*, which is nonpathogenic. This distinction

E. histolytica vs. *E. dispar*



- *E. dispar*: nonpathogenic, colonizes 5–25% of persons
- *E. dispar* has no propensity for invasive disease
- Cannot distinguish by microscopy
- In industrialized countries: most *Entamoeba* = *E. dispar*
- In endemic regions: *E. histolytica* may predominate

The appreciation of *E. dispar* as a distinct, nonpathogenic species was a major advance in understanding the epidemiology of amebiasis. This

Pyogenic liver abscess: epidemiology

- Incidence: 1–4 per 100,000 annually (US/Europe)
- In Asia: 5–10× higher (community-acquired *K. pneumoniae*)
- Peak: 5th–6th decades of life
- 50% solitary; right lobe most common
- Biliary disease: now the leading cause

The emergence of hypervirulent *K. pneumoniae* as a cause of community-acquired liver abscess has dramatically changed the epidemiology, particularly in East and Southeast Asia. This represents one of the most significant changes in infectious disease epidemiology in recent

Routes of hepatic invasion

Route	Frequency
Biliary tree (cholangitis)	40–50%
Cryptogenic	20–40%
Portal vein (pylephlebitis)	5–15%
Hepatic artery (bacteremia)	5–10%
Direct extension	5–10%
Trauma	0–5%

Cholangitis has replaced appendicitis as the major identifiable cause. The high frequency of cryptogenic abscesses reflects our inability to

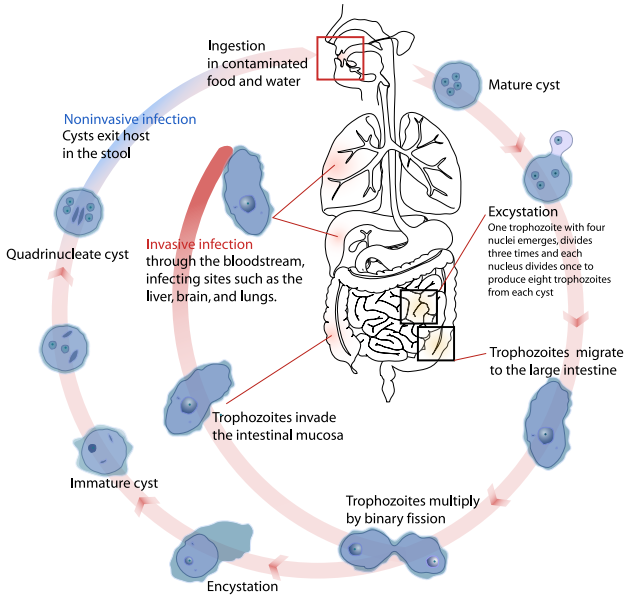
Risk factors for pyogenic liver abscess

- Diabetes mellitus: >3× risk
- Biliary disease (gallstones, obstruction)
- Hepatobiliary procedures
- Chronic granulomatous disease
- Hemochromatosis (especially *Yersinia*)
- Malignancy
- Cirrhosis
- Immunosuppression

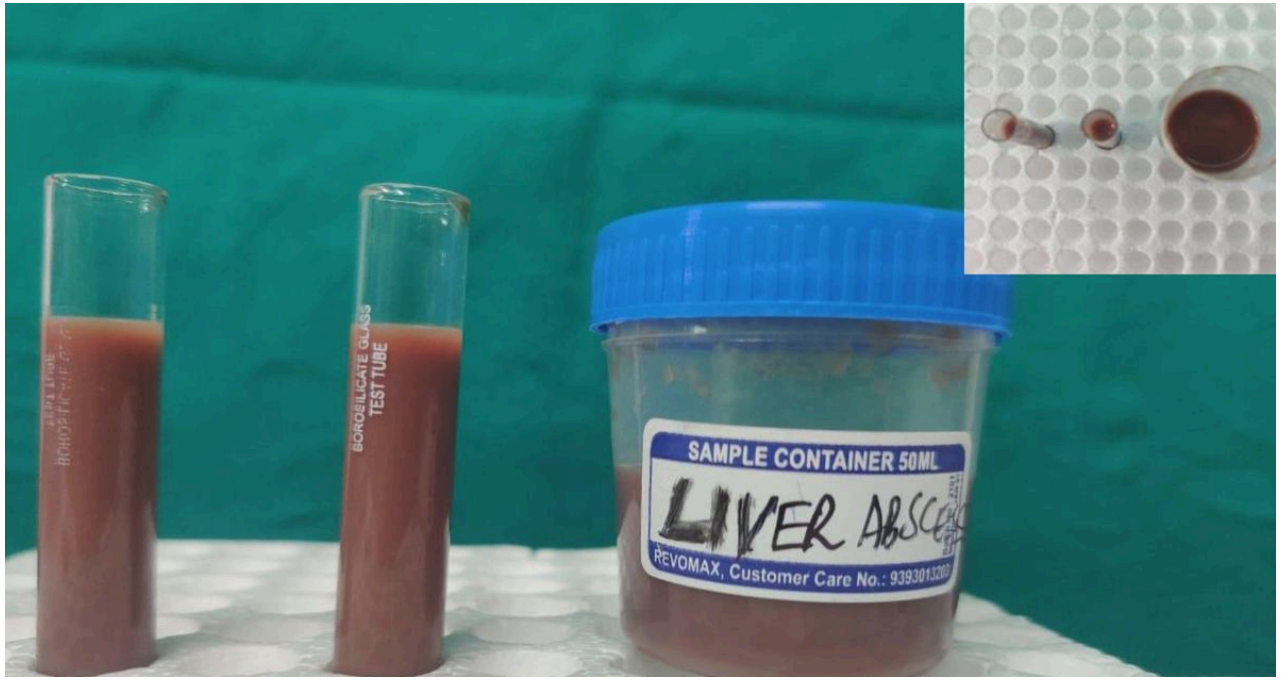
Diabetes is particularly important as a risk factor and is strongly associated with *K. pneumoniae* liver abscess, particularly in Asian

Pathogenesis: Amebic liver abscess

1. Ingestion of *E. histolytica* cysts
2. Excystation in intestinal lumen
3. Trophozoites migrate to colon
4. Adhere via **Gal/GalNAc lectin**
5. ~10% develop symptomatic colitis
6. Portal spread to liver in <1% of cases
7. **Apoptosis** of hepatocytes → abscess formation



“Anchovy paste” from liver abscess



Virulence Factors of *E. histolytica*

Factor	Function
Gal/GalNAc lectin	Epithelial adherence
Amoebapores	Pore formation in target cell membranes
Cysteine proteases	Tissue invasion, immune evasion
Apoptosis induction	Hepatocyte and neutrophil killing

Caspase-3-deficient mice are resistant to amebic liver abscess formation, underscoring the importance of host cell apoptosis in

Host Factors in Amebic Liver Abscess

- HLA-DR3: increased susceptibility
- Testosterone: risk factor (10× higher in men)
- Host defense mechanisms:
  - Complement
  - Neutrophils
  - Interferon-γ
  - Nitric oxide
  - Adaptive immunity

The dramatic male predominance is partly explained by the role of testosterone in experimental models.

Microbiology of Pyogenic Liver Abscess

- Cultures positive in 80–90% of cases
- Polymicrobial in 20–50% , key organisms:
  - *E. coli* and *K. pneumoniae* (most common gram-negatives)
  - *Streptococcus anginosus* group (most common gram-positive) -
  - *Bacteroides* spp. (most common anaerobe)
  - Anaerobes recovered in 15–30%

The source of the abscess guides the microbiology: biliary abscesses tend to be polymicrobial; cryptogenic abscesses are more often

Epidemic *K. pneumoniae* liver abscess

- First noted in Taiwan in mid-1980s
- Monomicrobial, often in diabetics
- No biliary tract disease
- Now accounts for 80% of cases in Asia
  - Spreading globally - Capsular serotypes K1 and K2

Mucoid phenotype demonstrated by loop test

This is a truly distinctive clinical syndrome that has emerged as a global concern.

K. pneumoniae virulence determinants

- **Capsular polysaccharide** (K1/K2 serotypes)
- ***magA* gene → hypermucoviscosity**
- *cps* gene cluster (25-kb chromosomal element)
- RmpA (mucoid phenotype regulator)
- Aerobactin (iron acquisition)
- Virulence plasmids (pLVPK, pK2044)

Mutagenesis of *magA* abolishes hypermucoviscosity and increases sensitivity to phagocytosis and serum-mediated lysis. This has enabled

Convergent drug-resistant *K. pneumoniae*

> ⚠ **Warning**
>
> **Public Health Threat**
>
> - Classic hypervirulent strains: typically drug **sensitive**
> - MDR/XDR strains can acquire virulence plasmids
> - Or hypervirulent strains can acquire resistance plasmids
> - Fatal ventilator-associated pneumonia outbreaks in China
> - Only a minority currently show hypervirulent phenotypes in lab

The convergence of hypervirulence and antimicrobial resistance in *K. pneumoniae* represents one of the most concerning developments in infectious diseases.

Clinical features: Amoebic liver abscess

- Fever + dull, aching RUQ pain
- Only 15–35% have GI symptoms
- Acute (<2 weeks) in ~2/3 of cases
- Can develop months to years after travel
- Risk factors: male sex, corticosteroid use
- Indistinguishable from pyogenic on clinical grounds alone

Epidemiologic risk factor assessment is crucial for differentiating amebic from pyogenic liver abscess at presentation.

Clinical Features: Pyogenic Liver Abscess

- Classic triad (fever, jaundice, RUQ tenderness) in only 10%
- Most common: **fever without localizing signs**
- General failure to thrive
- Malaise, fatigue, anorexia, weight loss
- Hematogenous abscesses present most acutely (3 days)
- Pylephlebitis-related abscesses: longest duration (42 days)

The nonspecific nature of symptoms means that a high index of suspicion is required. Before widespread imaging, liver abscess was among

Comparing amoebic vs. Pyogenic liver abscess

Feature	Amebic	Pyogenic
Male:female	5–18:1	1–2.4:1
Age	30–40	50–60
Duration (days)	<14	5–26
Mortality	10–25%	0–5%
Abdominal pain	80%	55%
RUQ tenderness	75%	25–55%

Note the striking gender difference—amebic liver abscess has a much stronger male predominance, likely related to testosterone and sex-

Diagnosis: Laboratory findings

- **Leukocytosis**: present in most patients (75–80%)
- **Elevated alkaline phosphatase**: most common abnormal LFT (~2/3)
  - Normal value does not exclude diagnosis
- Transaminases: generally mildly elevated
- Procalcitonin: typically elevated
- Albumin and PT: usually normal

Lab abnormalities may be prognostic: Hgb <10 and BUN >28 were independent predictors of mortality with odds ratios of 13 and 14 respectively.

Diagnosis: imaging

Modality	Sensitivity	Best For
Ultrasonography	70–90%	Initial assessment, biliary disease
CT (contrast-enhanced)	~95%	Definitive diagnosis, drainage guidance
MRI	High	Distinguishing from neoplasia
Fine-needle aspiration	Definitive	Diagnostic confirmation

US is the study of choice when biliary disease is suspected or in patients who cannot receive contrast.

Diagnosis: amoebic serology

- Enzyme immunoassay: sensitivity 65–92%, highly specific
- Can be negative if symptom duration <2 weeks
- Repeat serology in 1–2 weeks usually positive
- Positive serology confirms present or prior infection
- Cannot distinguish from extraintestinal disease
- ELISA for Gal/GalNAc lectin: >95% sensitivity in serum

Microscopic examination of stool for cysts is of little value because *E. histolytica* cannot be distinguished from *E. dispar* without further testing.

Emerging diagnostics

- **Molecular multiplex panels**:
  - Highly sensitive for amebic colitis
  - Patients with liver abscess usually don’t have concurrent intestinal infection
  - PCR: Potential for aspirated fluid; limited to research labs
  - cfDNA testing: Circulating cell-free DNA
    - Recent study: 90% sensitivity, 100% specificity for amoebic abscess
    - Noninvasive blood-based assay

cfDNA testing represents a potentially transformative diagnostic approach.

Treatment: Amoebic liver abscess — Medical therapy

> **Tip**
>
> Almost always treatable with **medical therapy alone**

Drug	Regimen
Metronidazole	750 mg TID × 7–10 days
Tinidazole	2 g daily × 3 days
Secnidazole/Ornidazole	Extended half-life alternatives
Paromomycin (luminal agent)	Follow-up to eliminate colonization

Decreased fever and abdominal pain are usually observed within 3–5 days of starting therapy.

Treatment: When to drain amoebic abscess

- Uncomplicated: drainage **NOT required**
- Indications for drainage: - No response to medical therapy (>5–7 days)
- Diagnostic uncertainty (rule out pyogenic)
- Large lesions at risk for rupture:
  - Left-sided abscesses (pericardial rupture risk)
  - Bacterial superinfection (1–5% of cases)
  - Percutaneous preferred over surgical drainage

One RCT found a salutary effect only in patients with large abscesses (>300 mL).

Treatment: Pyogenic Liver Abscess — Drainage

- **Percutaneous catheter drainage**: Preferred primary therapy
  - Success rate: **69–90%**
  - Can be performed at time of diagnosis
  - Catheter left in place 5–14 days until drainage resolves
  - Recent success rates with antibiotics: **80–95%** (even for >10 cm abscesses)
  - Surgical drainage if: percutaneous fails, concurrent surgical disease, multiple/loculated abscesses

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Two recent meta-analyses found percutaneous drainage superior to aspiration in recurrence rates, speed of resolution, and duration of antibiotic use. The technique is now first-line at most institutions[2]

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Two recent meta-analyses found percutaneous drainage superior to aspiration in recurrence rates, speed of resolution, and duration of antibiotic use. The technique is now first-line at most institutions[4]

Treatment: Pyogenic liver abscess — Antibiotics

Approach	Agents
Monotherapy	Piperacillin-tazobactam OR carbapenem
Combination	3rd/4th-gen cephalosporin + metronidazole OR fluoroquinolone + metronidazole

Antibiotic choice should be guided by suspected source. Biliary source suggests gram-negatives and enterococci. Colonic/pelvic source suggests gram-negatives and anaerobes.

Antibiotic Duration: An open question

- No RCTs establishing optimal duration
- Meta-analysis: pooled mean **32.7 days**
  - oTraditional: IV 2–3 weeks → oral 4–6 weeks total
  - Some evidence for shorter courses (2–4 weeks)
- **Singapore RCT**: 4-week oral ciprofloxacin noninferior to 4-week IV ceftriaxone for *K. pneumoniae*
- Follow-up imaging to guide duration

Aspiration vs. catheter drainage

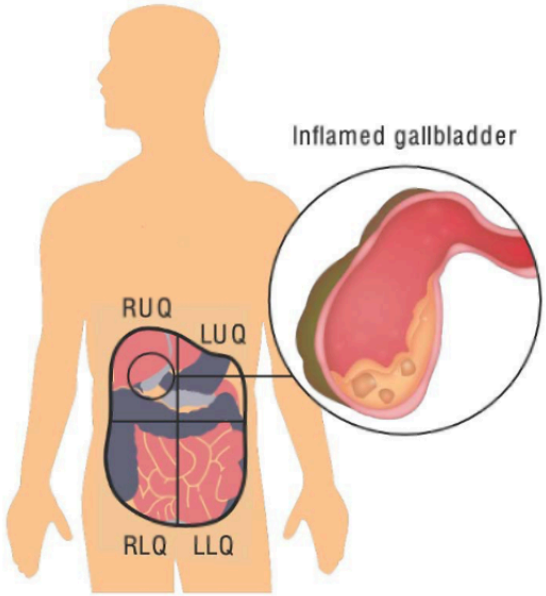
Feature	Aspiration	Catheter Drainage
Success rate	58–88% (≤5 cm)	69–90%
Recurrence	Higher	Lower
Meta-analysis evidence	Drainage superior	Drainage superior
Best for	Small, solitary abscesses	Larger, multiple abscesses

Two recent meta-analyses (>2,800 patients combined) found percutaneous drainage was superior to aspiration alone.

Biliary tract infections

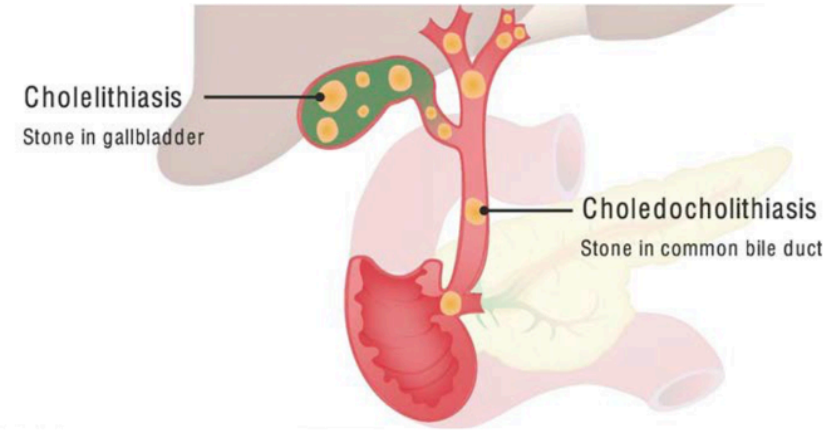
Biliary system infections: Overview

- Infections associated with obstruction to bile flow
- Gallstones: common and usually asymptomatic
- 1% to 4% complicated by acute cholecystitis
  - Over 100,000 cholecystectomies per year in Italy
  - 2–15% of cases are acalculous cholecystitis



In endemic regions, parasites such as Ascaris and Clonorchis may cause biliary disease.

Cholecystitis: Key points

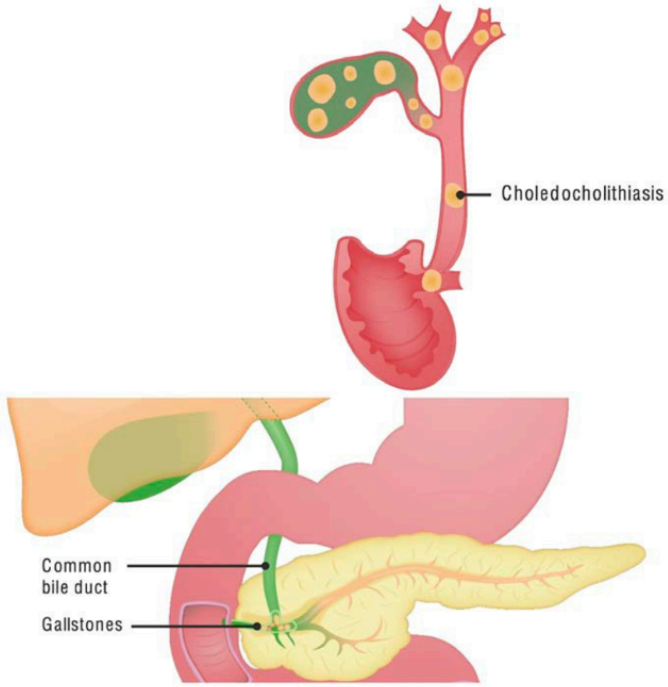


- Inflammation/bacterial infection of the gallbladder
- Usually from obstructing gallstones
- Acalculous cholecystitis: similar process without stones
- Treatment: cholecystectomy + antibiotics
- Mortality higher in acalculous cases

Acalculous cholecystitis is more common in critically ill patients and carries a higher mortality rate.

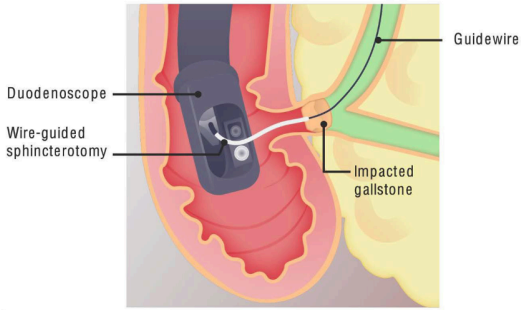
Cholangitis: Key points

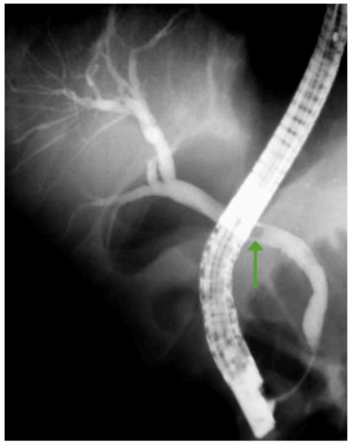
- Inflammation/infection of the bile ducts
- **Charcot triad**: fever, jaundice, RUQ pain
- **Reynolds pentad**: triad + hypotension + altered mental status (sepsis)
- Life-threatening emergency requiring urgent biliary decompression
  not an encapsulated infection and will spill over to abdomen and bloodstream
- Mortality: 5–10% with treatment; up to 90% without

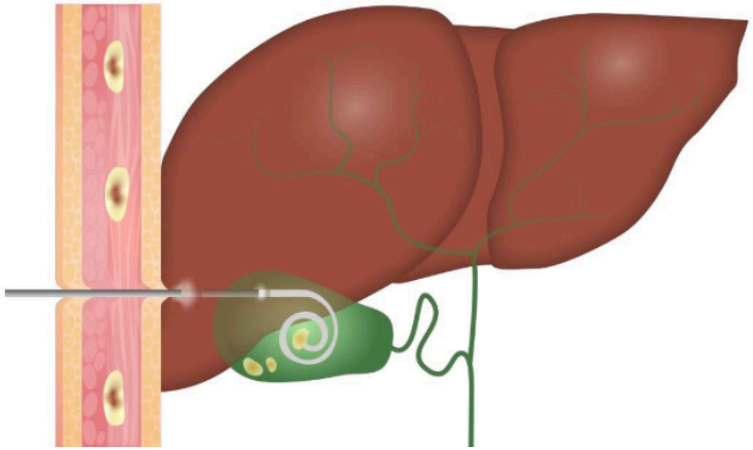


Cholangitis represents a medical emergency.

ERCP or percutaneous transhepatic drainage







Antibiotic therapy billary tract infections

Good Penetration (ABSCR ≥1)	ABSCR	Low Penetration (ABSCR <1)	ABSCR
Piperacillin/tazobactam	4.8	Ceftriaxone	0.75
Tigecycline	>10	Cefotaxime	0.23
Amoxicillin/clavulanate	1.1	Meropenem	0.38
Ciprofloxacin	>5	Ceftazidime	0.18
Ampicillin/sulbactam	2.4	Vancomycin	0.41
Cefepime	2.04	Amikacin	0.54
Levofloxacin	1.6	Gentamicin	0.30
Penicillin G	>5		
Imipenem	1.01		

ABSCR = Antibiotic Bile/Serum Concentration Ratio. Source: Ansaloni et al. PMID 27307785

Biliary tract infections: Mild–moderate severity

- **Community-acquired**
- **No prior biliary instrumentation**
- **Low resistance risk**

Regimen	Agents
3rd-gen cephalosporin	Ceftriaxone, cefotaxime
2nd-gen cephalosporin	Cefuroxime
1st-gen cephalosporin	Cefazolin *(cholecystitis only)*
Fluoroquinolone	Ciprofloxacin, levofloxacin *(if local resistance permits)*
β-lactam/β-lactamase inhibitor	Amoxicillin/clavulanate

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> **Note**
>
> Anaerobic coverage **not required** unless bilioenteric anastomosis or prior biliary intervention

Biliary Tract Infections: Severe / Healthcare-Associated

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::: callout-note Anaerobic coverage **not required** unless bilioenteric anastomosis or prior biliary intervention :::

Biliary tract infections: Severe / healthcare-associated

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- **Grade III cholangitis**
- **Prior instrumentation**
- **Healthcare-acquired**

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- **High resistance risk**

Regimen	Agents
Carbapenem	Imipenem/cilastatin, meropenem, ertapenem
β-lactam/β-lactamase inhibitor	Piperacillin/tazobactam
Fluoroquinolone + anaerobic cover	Ciprofloxacin or levofloxacin + metronidazole

Additional considerations:

Summary: Comparing hepato-biliary infections

	Appendicitis	**Amebic Abscess**	**Pyogenic Abscess**
Peak age	15–25	30–40	50–60
Cause	Obstruction/dysbiosis	*E. histolytica*	Biliary/polymicrobial
Diagnosis	CT	Serology + imaging	Culture + imaging
Treatment	Surgery or antibiotics	Metronidazole	Drainage + antibiotics

Each condition has distinct epidemiology, pathogenesis, and management. The key clinical skill is recognizing the distinctive features of each

Clinical decision algorithm: Liver abscess

1. **Suspect**: Fever + RUQ pain/tenderness + leukocytosis
2. **Image**: CT with contrast (or US if CT not available)
3. **Differentiate**: Serology, epidemiology, aspirate if needed
4. **Treat**:
   - Amebic → Metronidazole + paromomycin
   - Pyogenic → Drainage + empiric antibiotics
5. **Follow up**: Imaging to confirm resolution

This algorithm emphasizes the importance of early imaging and microbiologic diagnosis to guide appropriate therapy. Shared decision-

Take-Home Messages

1. **Appendicitis**: Most common surgical emergency; antibiotic-first is valid for uncomplicated cases
2. **Amebic liver abscess**: Think travel/endemic exposure; treat medically with metronidazole
3. **Pyogenic liver abscess**: Drainage + antibiotics; think *K. pneumoniae* in diabetics
4. ***Klebsiella pneumoniae***: Global emergence of hypervirulent strains; convergent MDR strains are a growing threat
5. **Microbiome**: Paradigm shift in understanding appendicitis pathogenesis

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