

Bacterial, Fungal, and Parasitic Infections

Liver and biliary tree • morphology and clinical correlations

Dr Abu Suraih Sakhri

MBBS, MD (Pathology)

www.abusuraihsakhri.com

Learning objectives

Recognize

Major pathogen groups and entry routes

Explain

Abscess, cholangitis, granuloma, and fibrosis

Differentiate

Pyogenic, amebic, schistosomal, and hydatid patterns

Integrate

Morphology, clinical presentation, and organism detection

Hepatic infections: Four recurring lesions

Suppurative

Ascending cholangitis and bacterial abscess

Granulomatous

Disseminated fungal and mycobacterial infection

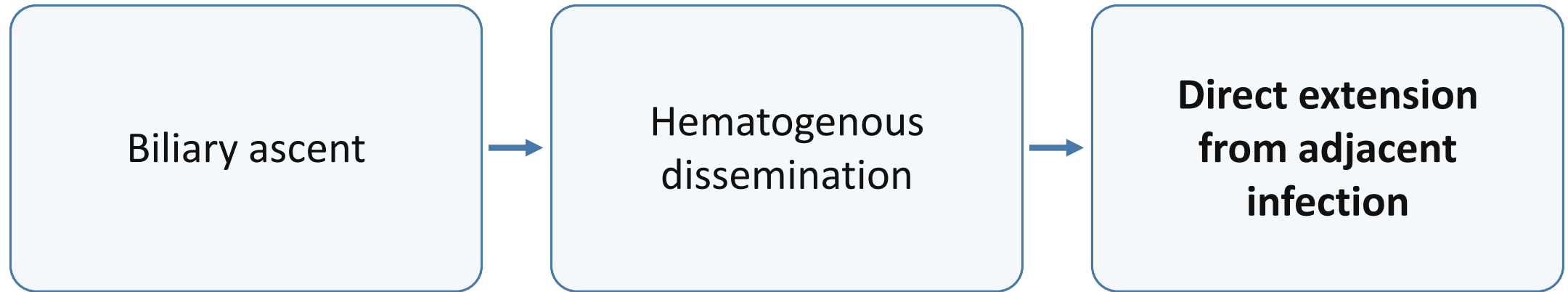
Fibrosing

Schistosomal egg-induced portal injury

Cystic / necrotic

Echinococcosis and amebiasis

Infection reaches the liver by different routes



Distinct routes converge on hepatic injury

Local infection versus systemic disease

Pattern	Typical context	Pathologic consequence
Localized	Obstructed infected bile duct	Cholangitis ± hepatic abscess
Disseminated	Systemic bacterial, fungal or mycobacterial disease	Inflammation ± granulomas
Parasite-associated	Eggs, cysts, or trophozoites	Fibrosis, cysts, or necrosis

Host reaction helps narrow the pathogen class

Neutrophilic

Suppuration and microabscesses

Granulomatous

Aggregated epithelioid macrophages

Fibrosing

Persistent inflammatory injury and scar

Liquefactive

Extensive enzymatic tissue destruction

Bacterial involvement: Systemic exemplars

Agent	Associated syndrome	Hepatic context
Staphylococcus aureus	Toxic shock syndrome	Systemic bacterial disease
Salmonella Typhi	Typhoid fever	Systemic bacterial disease
Treponema pallidum	Secondary/tertiary syphilis	Systemic bacterial disease
Bartonella henselae	Cat-scratch disease	Systemic or focal infection

Staphylococcus aureus: Systemic context

- The liver may participate in a generalized bacterial illness.
- Toxic shock syndrome is one textbook example of S. Aureus-associated systemic infection.
- A clinical infectious syndrome cannot be identified from a nonspecific liver inflammatory pattern alone.

**Systemic illness ≠
primary biliary
infection**

Typhoid fever: Systemic bacterial infection

- Salmonella Typhi is included among bacteria that can involve the liver.
- The hepatic component occurs in the context of a systemic disease.
- Organism identification and clinical context matter when the biopsy lacks a distinctive signature.

**Do not infer the
species from
nonspecific hepatitis**

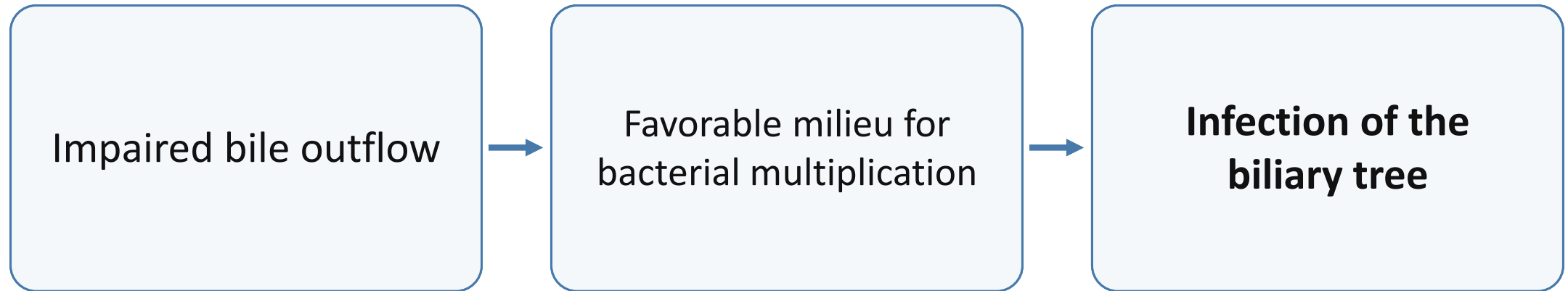
Syphilis: Spirochetal infection

- *Treponema pallidum* may involve the liver in secondary or tertiary syphilis.
- Infectious-disease histology recognizes plasma-cell-rich inflammation in syphilitic lesions.
- Hepatic evaluation must be integrated with systemic findings and organism-directed testing.

Cat-scratch disease: *Bartonella henselae*

- *Bartonella henselae* is another example of bacterial hepatic involvement.
- Its importance here is recognition of a potential systemic infectious etiology.
- A hepatic lesion should be interpreted with exposure and extrahepatic disease history.

Biliary obstruction creates susceptibility



Ascending cholangitis

Common adult causes of large-duct obstruction

Stones

Choledocholithiasis

Neoplasms

Biliary tree or pancreatic head

Strictures

Postoperative or ischemic

Inflammatory

Primary sclerosing cholangitis

Ascending cholangitis: Usual microbial source

Entry setting

Biliary obstruction

Common pathogens

Enteric coliforms and enterococci

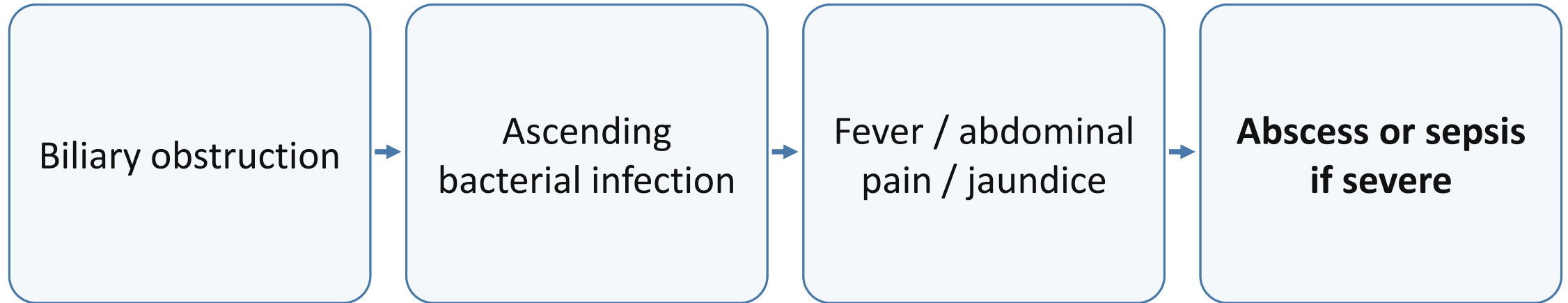
Principal site

Biliary duct lumen and wall

Extension

Intrahepatic abscess in severe infection

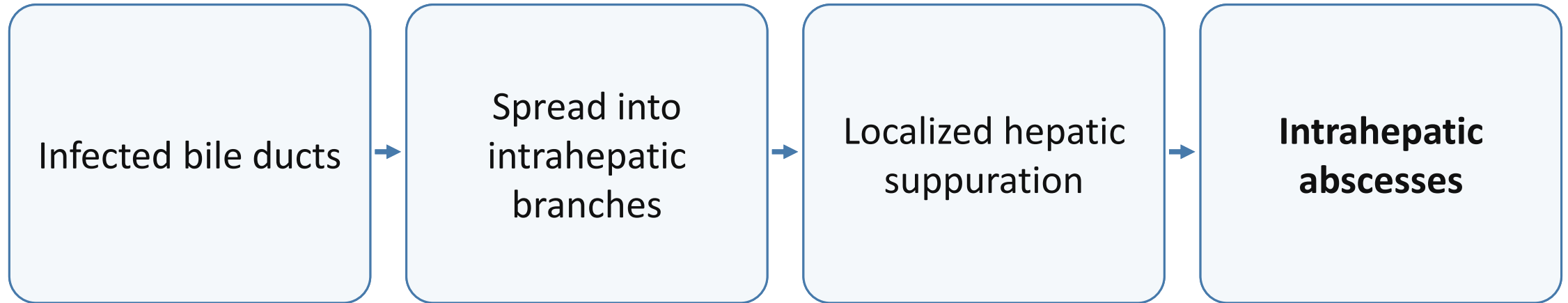
Cholangitis: Clinicopathologic sequence



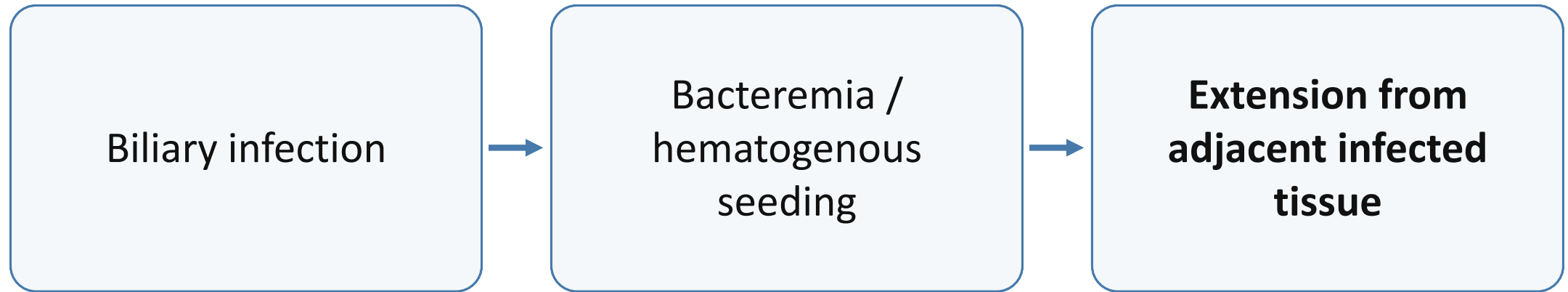
Clinical pattern of ascending cholangitis

- Fever and chills indicate an acute infectious process.
- Abdominal pain accompanies biliary inflammation or obstruction.
- Jaundice reflects impaired bile drainage.
- Severe disease may progress to abscess, sepsis, or death.

Ascending cholangitis may extend into the liver

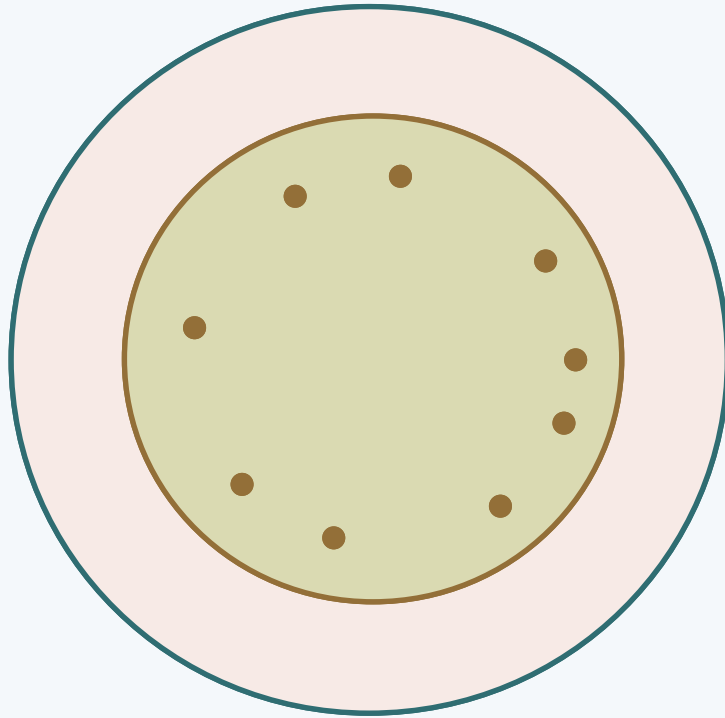


Three routes to bacterial liver abscess



All may culminate in a hepatic abscess

Suppurative injury: Abscess as tissue destruction



Schematic — not a histologic specimen

Pathologic interpretation

- Localized collection of necrotic, inflamed tissue
- Bacterial infection can produce suppurative foci
- Larger lesions may require intervention beyond antibiotics

Liver abscess: Clinical triad

Fever

Systemic inflammation

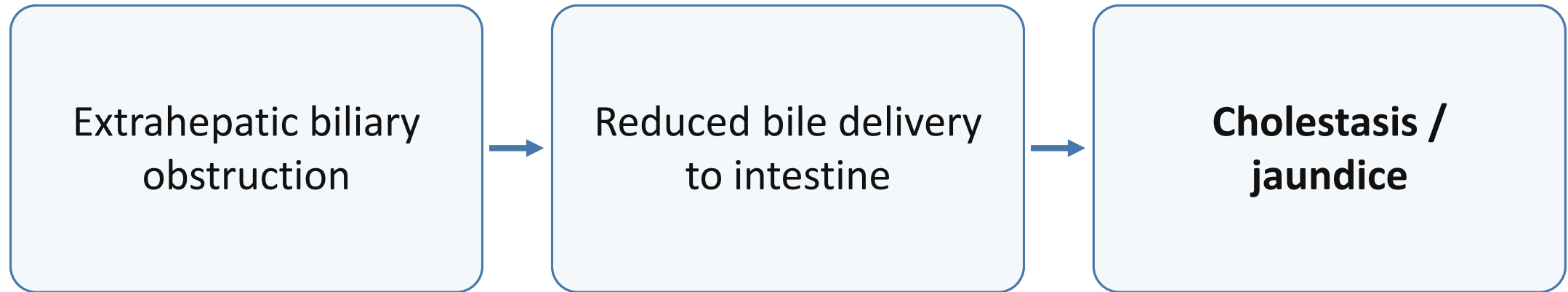
Right upper quadrant pain

Local hepatic involvement

Tender hepatomegaly

Inflamed, enlarged liver

Why jaundice may occur with liver abscess



Abscess alone does not establish obstruction

Management concepts in bacterial abscess

Antibiotics

Generally effective for bacterial infection

Drainage

May be necessary with large lesions

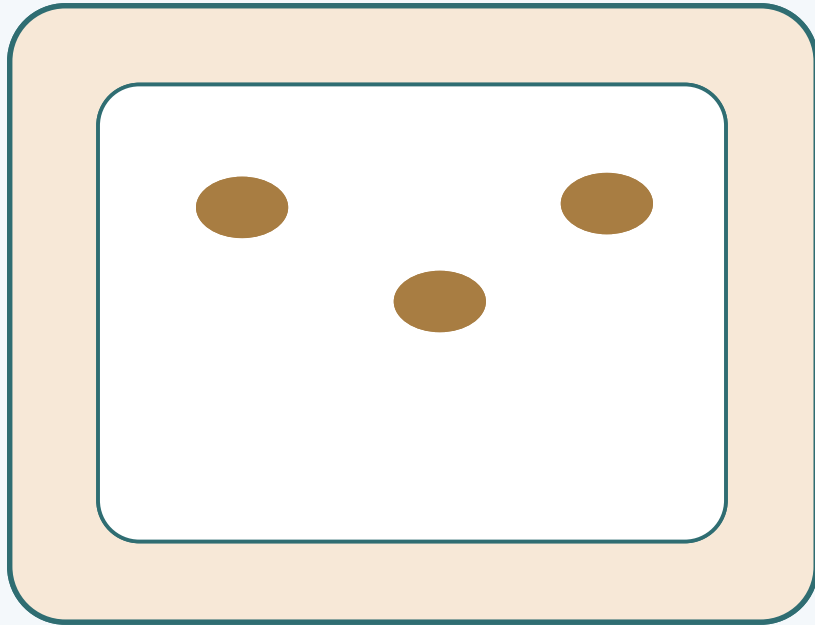
Source control

Obstruction or adjacent infection remains relevant

Extrahepatic sepsis can alter liver histology

- Intraabdominal infections may cause reactive hepatic inflammation.
- The liver changes can be nonspecific despite a serious extrahepatic infection.
- Sepsis can produce ductular reaction with cholestasis.

Cholangiolar cholestasis in sepsis



Bile plugs in ductular lumen

Simplified schematic; Not a biopsy image

- Ductular reaction
- Bile plugs within ductules
- Cholestatic pattern associated with sepsis

Septic cholestasis versus obstructive cholangitis

Feature	Sepsis-related hepatic change	Ascending cholangitis
Primary process	Systemic infection	Infected obstructed bile ducts
Characteristic clue	Ductular bile plugs	Fever, pain and jaundice
Complication	Cholestatic dysfunction	Hepatic abscess / sepsis

Applied case: Obstruction and fever

Hypothetical teaching vignette

- A patient with biliary obstruction develops fever, chills, abdominal pain, and jaundice.
- What route of infection and principal complication should be considered?

Ascending cholangitis; Severe infection can extend to intrahepatic abscesses

Fungal and mycobacterial infections: Common pattern

Clinical setting

Disseminated infection

Tissue response

Epithelioid granulomas

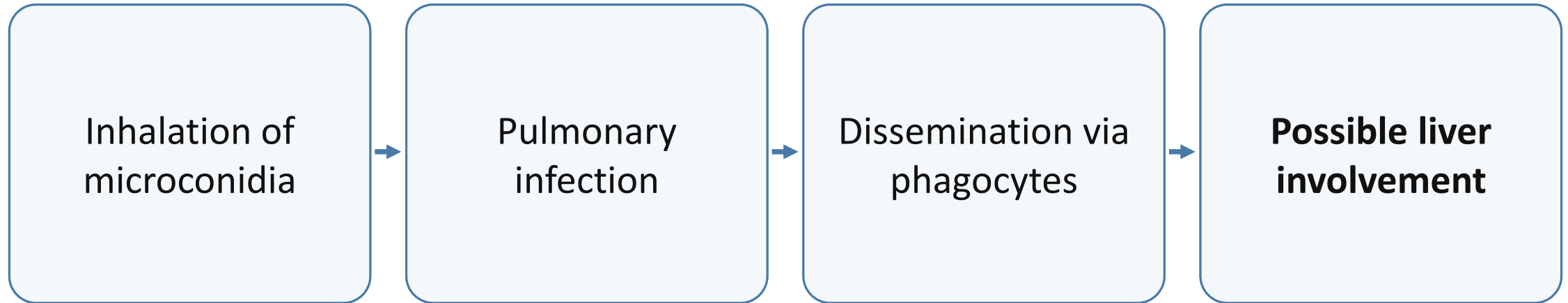
Morphologic variability

With or without necrosis

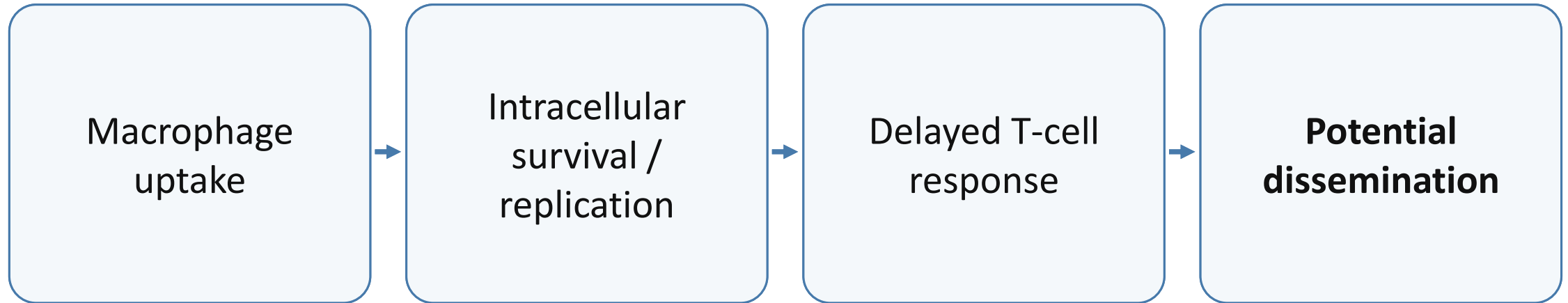
Confirmation

Serology, cultures, or organisms in biopsy

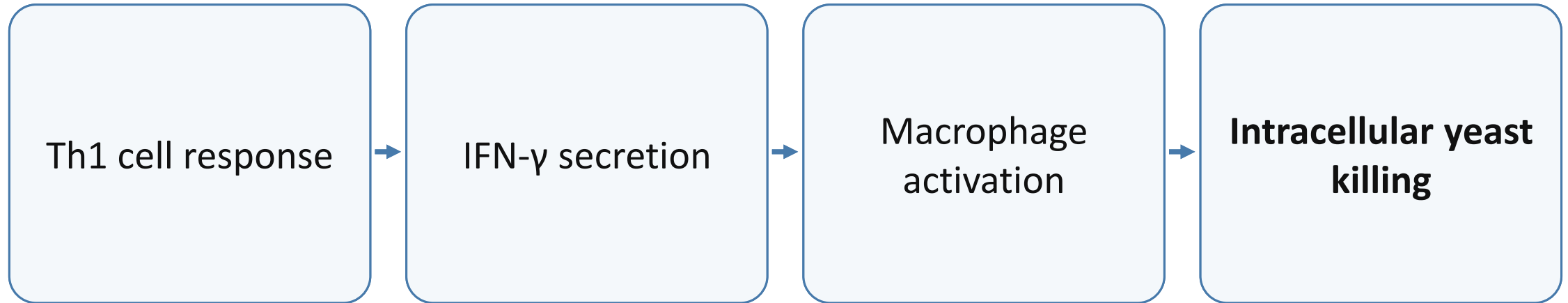
Histoplasma: Pulmonary entry to hepatic spread



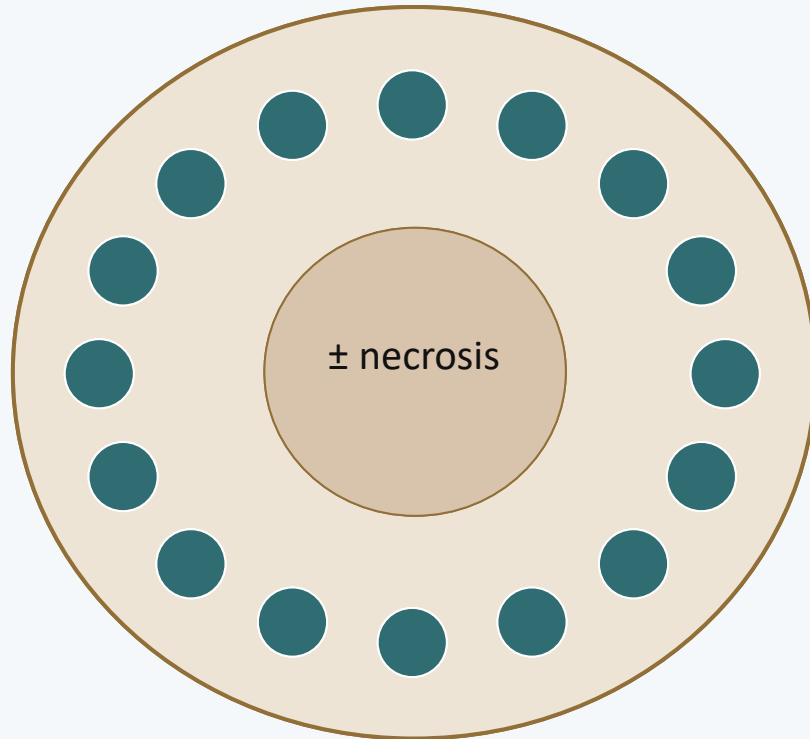
Histoplasma survives in host phagocytes



Cell-mediated immunity limits histoplasmosis



Hepatic granuloma: An immune containment lesion



Conceptual granuloma — not a micrograph

- Aggregated activated epithelioid macrophages
- Can contain giant cells and central necrosis
- Seen in tuberculosis, histoplasmosis and schistosome infection

Granulomatous infection need not be necrotizing

Granuloma component	Meaning
Epithelioid macrophages	Activated macrophage aggregation
Multinucleated giant cells	May arise by macrophage fusion
Central necrosis	Possible, but not obligatory
Organism demonstration	Requires directed techniques and context

Organisms to consider in hepatic granulomas

Histoplasma

Disseminated fungal infection

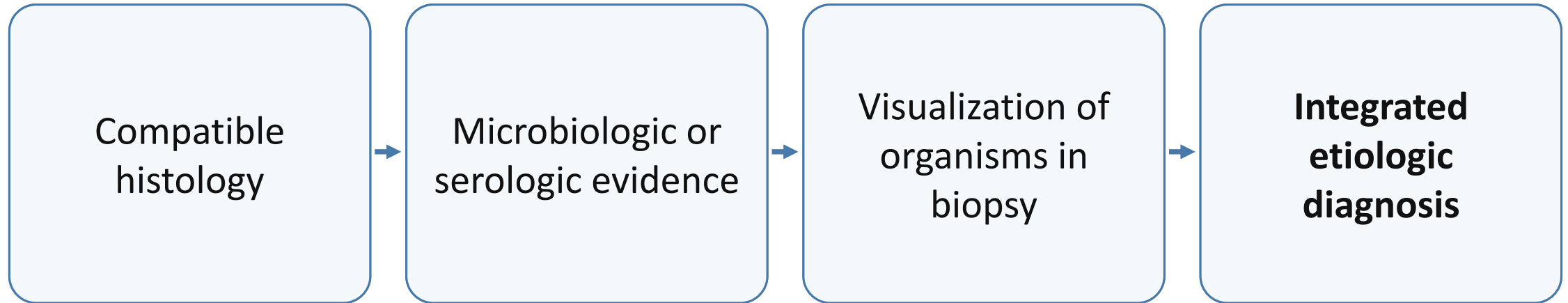
Mycobacteria

Disseminated bacterial infection

Schistosoma

Host reaction to deposited eggs

How to establish a fungal or mycobacterial cause



Impaired T-cell immunity favors disseminated histoplasmosis

- Histoplasma is an intracellular organism that depends on cellular immunity for containment.
- Immunocompromised hosts are more vulnerable to disseminated disease.
- Fungal liver disease should not be excluded because the primary portal of entry is respiratory.

Applied case: Granulomas with hepatic involvement

Hypothetical teaching vignette

- A patient with disseminated infection has hepatic epithelioid granulomas.
- Necrosis is variable. Which etiologies and confirmatory strategies are supported by the textbook?

Histoplasma or mycobacteria; Serology, cultures, and tissue organism detection

Parasitic infections: Diverse hepatic lesions

Example	Dominant hepatic consequence
Schistosoma spp.	Egg granulomas and portal fibrosis
Echinococcus spp.	Hydatid cyst formation
Entamoeba histolytica	Liquefactive necrosis and abscess
Liver flukes	Biliary disease and increased cancer risk

Parasite location predicts tissue response

Portal vessels / tracts

Schistosome eggs and fibrosis

Parenchymal cyst

Hydatid disease

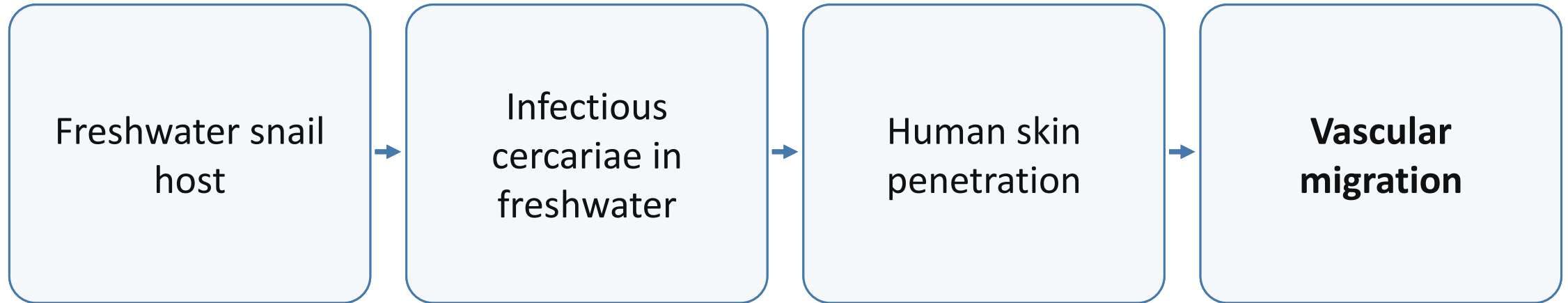
Necrotic cavity

Amebic abscess

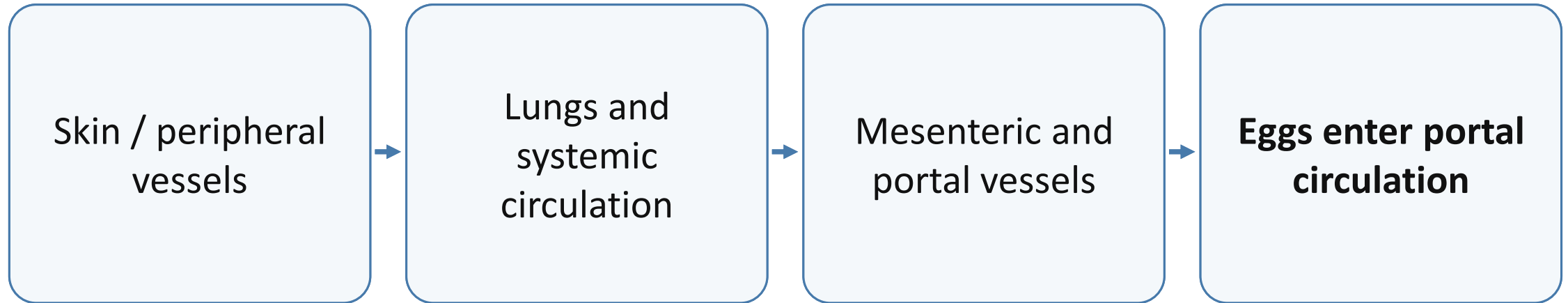
Biliary system

Clonorchis / Opisthorchis / Fasciola

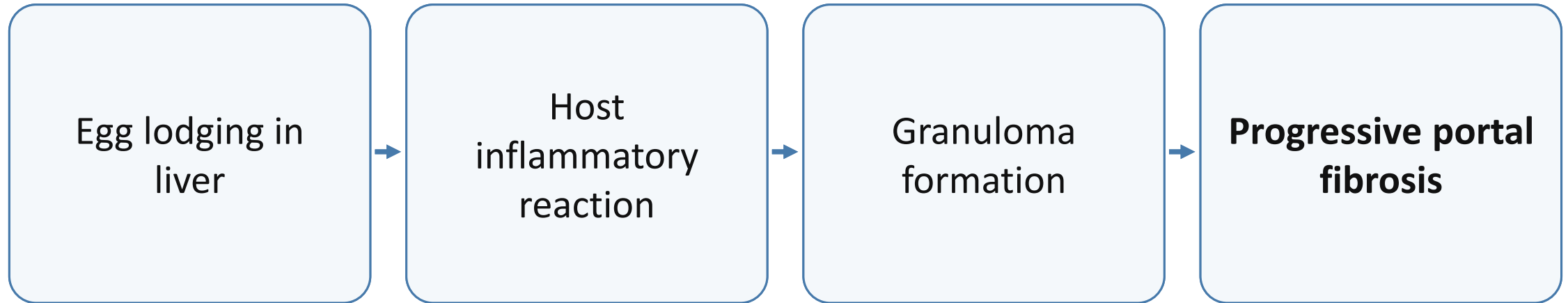
Schistosomiasis: An ecological infection



Schistosome migration connects gut and liver



The schistosome egg drives much of the injury



Early schistosomiasis: Inflammatory mediators

Th1 response

IFN- γ -associated
macrophage activation

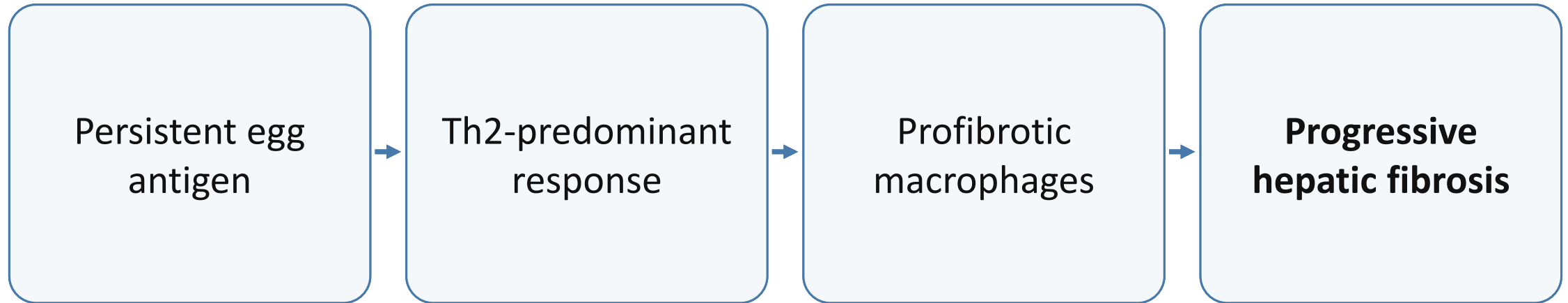
Macrophage cytokines

TNF, IL-1, IL-6

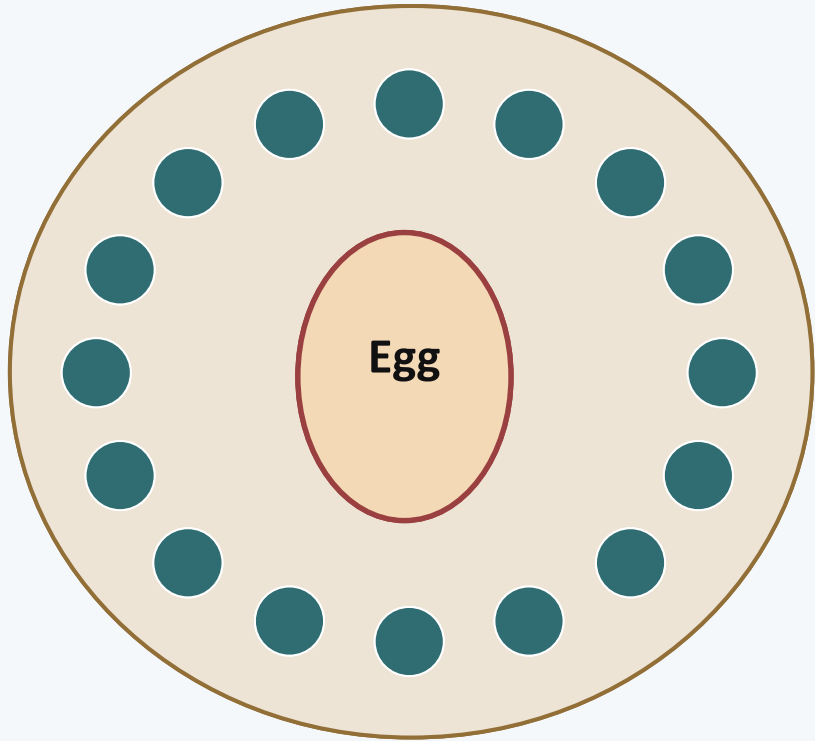
Acute effects

Inflammatory and febrile
illness

Chronic schistosomiasis: Profibrotic immunity



Schistosome granuloma: Diagnostic constituents



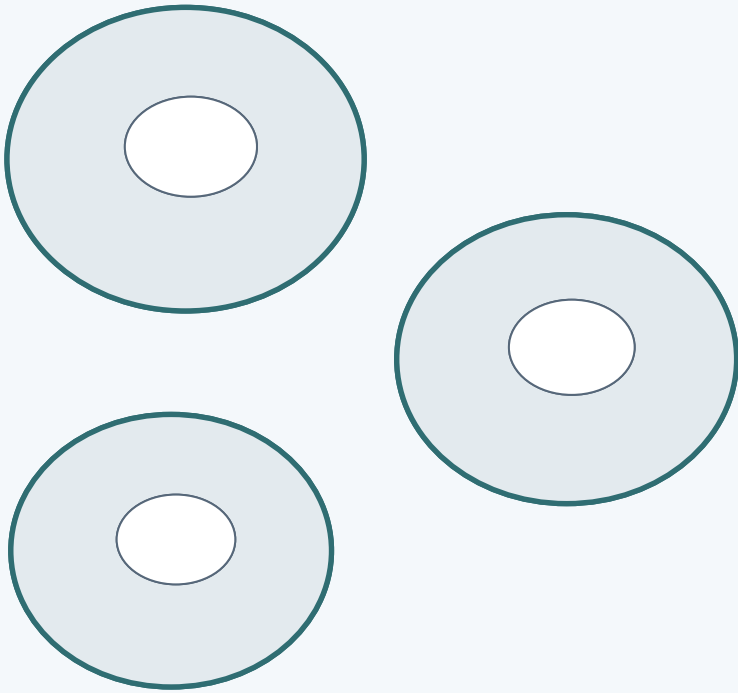
Conceptual granuloma — not a micrograph

- Egg at lesion center
- Macrophages, lymphocytes, neutrophils
- Eosinophils are conspicuous in helminth-associated lesions

Early hepatic schistosomiasis morphology

- Minute pale granulomas are scattered through the liver.
- The central egg contains a miracidium, which can degenerate and calcify.
- Liver macrophages may contain iron-free heme-derived pigment.

Pipestem fibrosis: Portal expansion



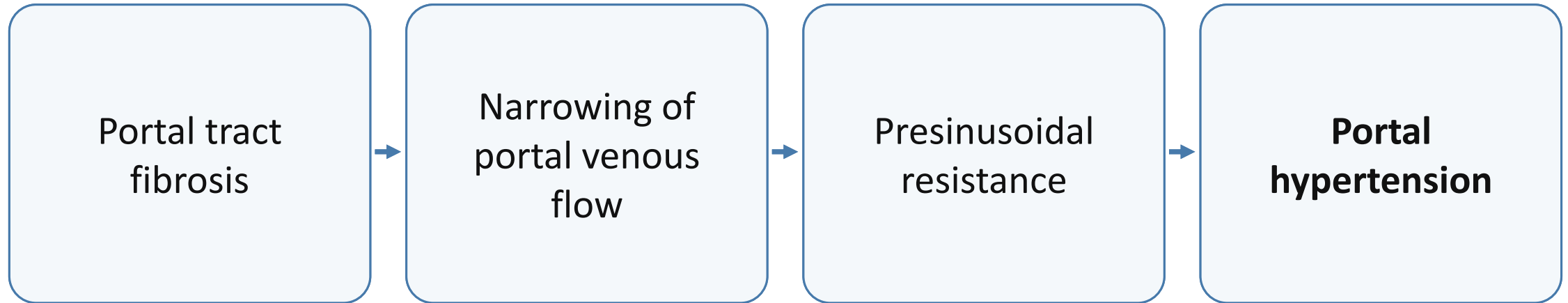
Portal-tract widening: Schematic, not histology

- Expanded fibrotic portal tracts
- Relative preservation of intervening parenchyma
- Portal vein narrowing and obliteration

Why pipestem fibrosis differs from cirrhosis

Feature	Hepatosplenic schistosomiasis	Established cirrhosis
Fibrosis	Marked portal-tract widening	Fibrous septa surround nodules
Regenerative nodules	Typically absent in pipestem lesion	Present
Portal pressure	May become markedly elevated	Often elevated

Presinusoidal obstruction produces portal hypertension



Consequences of schistosomal portal hypertension

Congestive splenomegaly

Back-pressure in portal system

Esophageal varices

Portosystemic collateral formation

Ascites

Portal circulatory disturbance

Schistosomiasis can affect more than the liver

- Eggs shunted to the lung can cause granulomatous pulmonary arteritis.
- Advanced pulmonary vascular disease may lead to cor pulmonale.
- Hepatosplenic disease is associated with certain glomerulopathies.

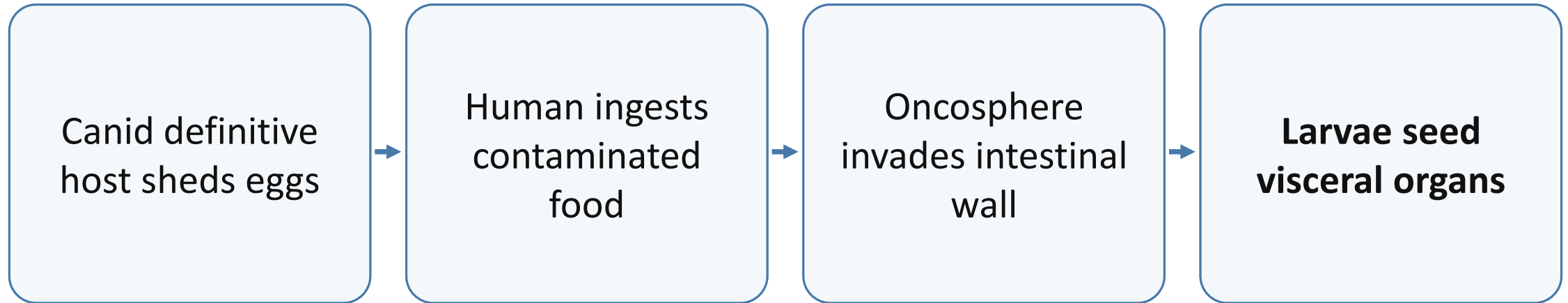
Applied case: Portal hypertension without nodules

Hypothetical teaching vignette

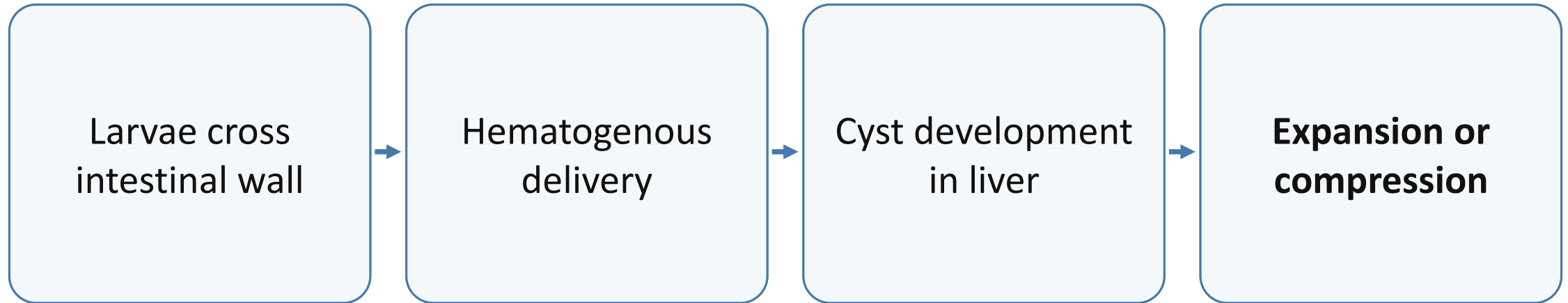
- A patient from an endemic region has portal hypertension and markedly expanded fibrotic portal tracts without regenerative nodules.
- Which infection and morphologic label fit this pattern?

Chronic hepatosplenic schistosomiasis — pipestem fibrosis

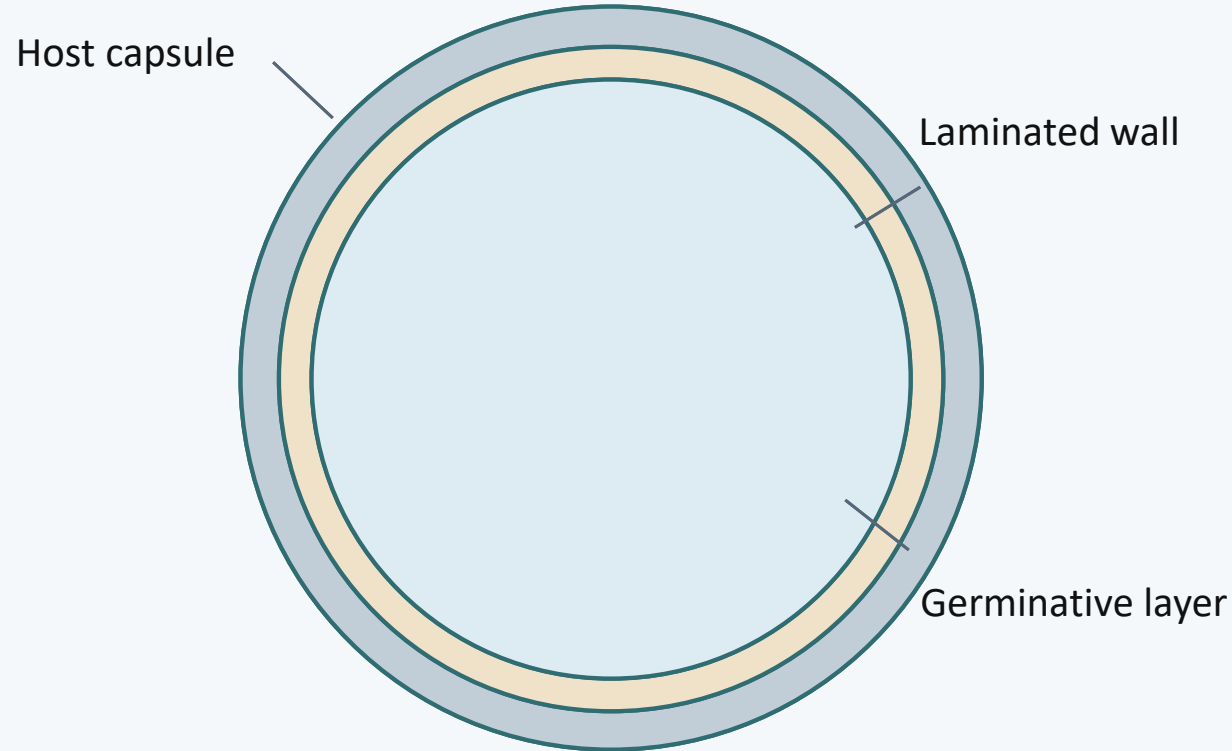
Echinococcus: Hydatid disease transmission



Hydatid cysts: Why the liver is involved



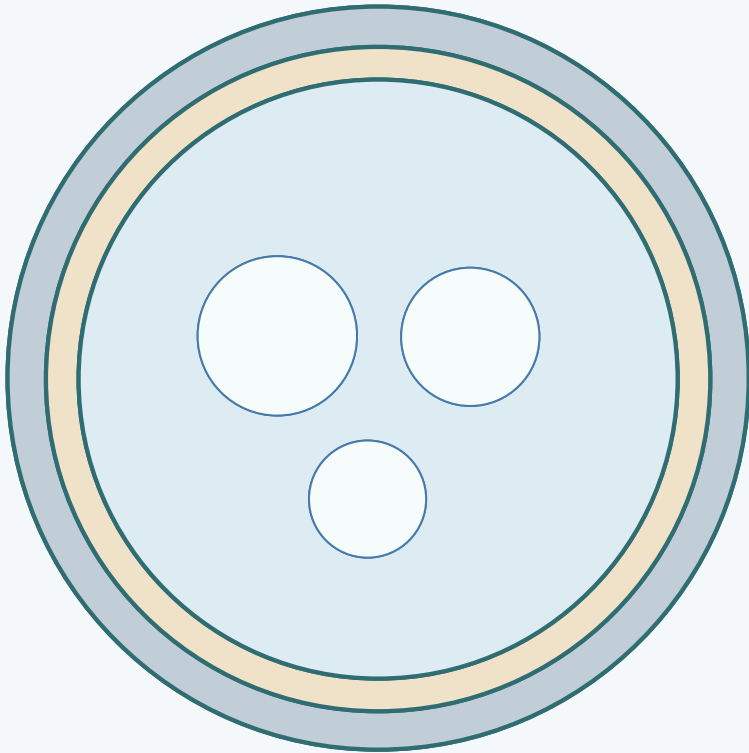
Hydatid cyst: Layered architecture



Simplified cyst anatomy — not a specimen image

- Inner germinative layer
- Outer laminated, non-nucleated layer
- External host fibrous and inflammatory capsule

Daughter cysts and hydatid sand



Simplified cyst anatomy — not a specimen image

- Daughter cysts can form inside the mother cyst.
- Brood capsules arise from the germinative lining.
- Degenerating scolices yield sediment termed hydatid sand.

Calcification provides an imaging clue

Wall calcification

Can occur as the cyst degenerates

Imaging consequence

May assist radiologic recognition

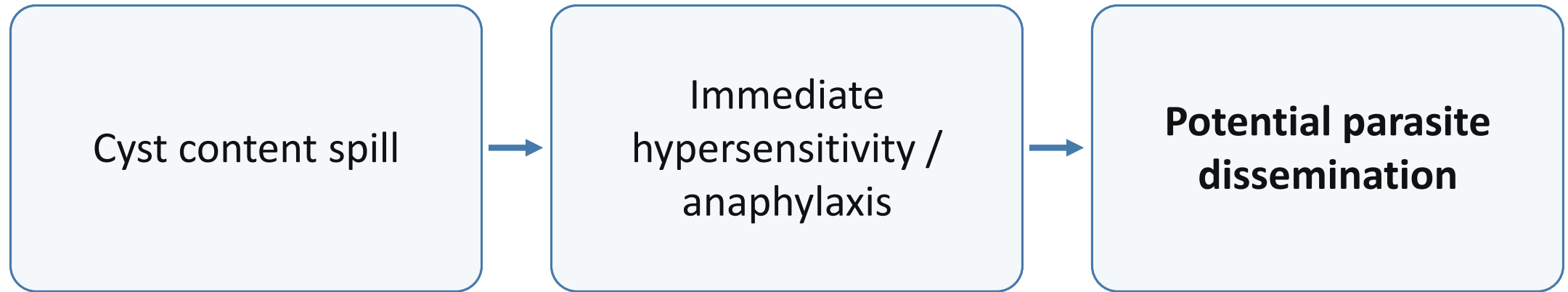
Interpretive limitation

Pattern must be placed in clinical context

Large hydatid cysts may cause obstruction

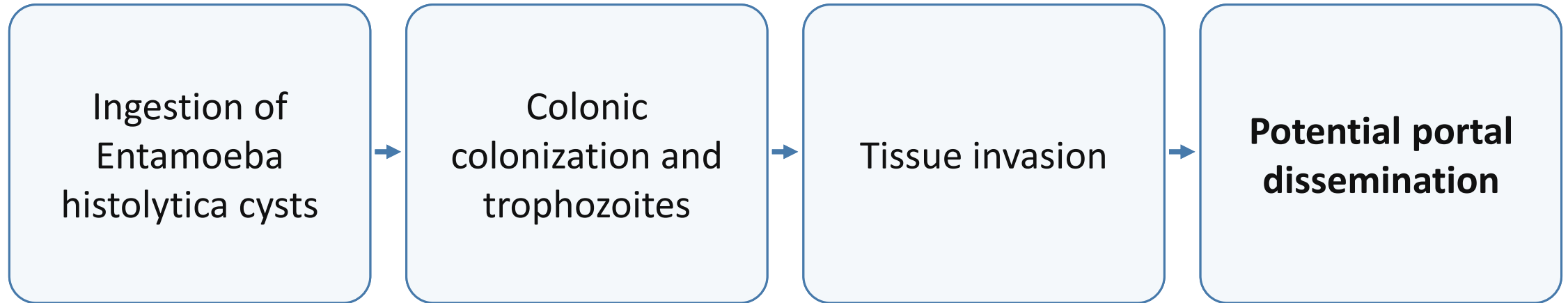
- Many infections remain clinically silent.
- Expanding hepatic cysts can cause abdominal pain.
- Large cysts may compress nearby biliary or other structures.

Hydatid cyst rupture or spill is hazardous

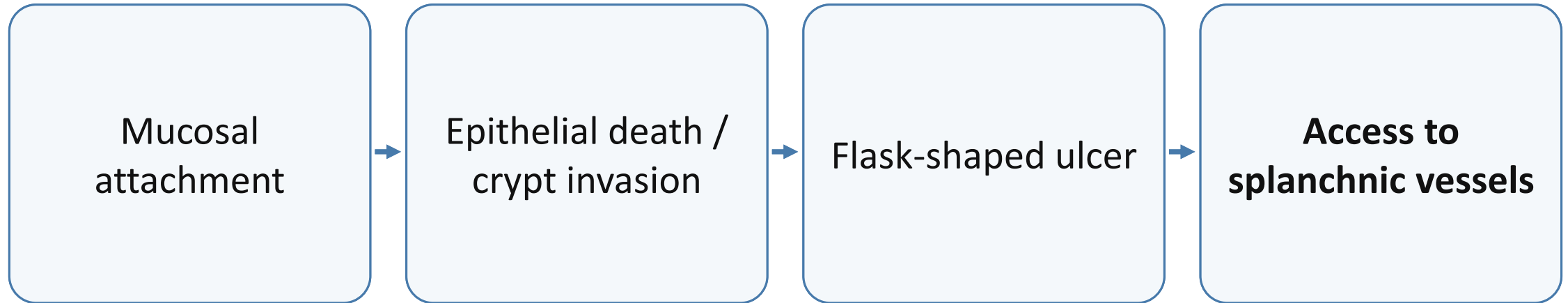


Caution during surgical manipulation

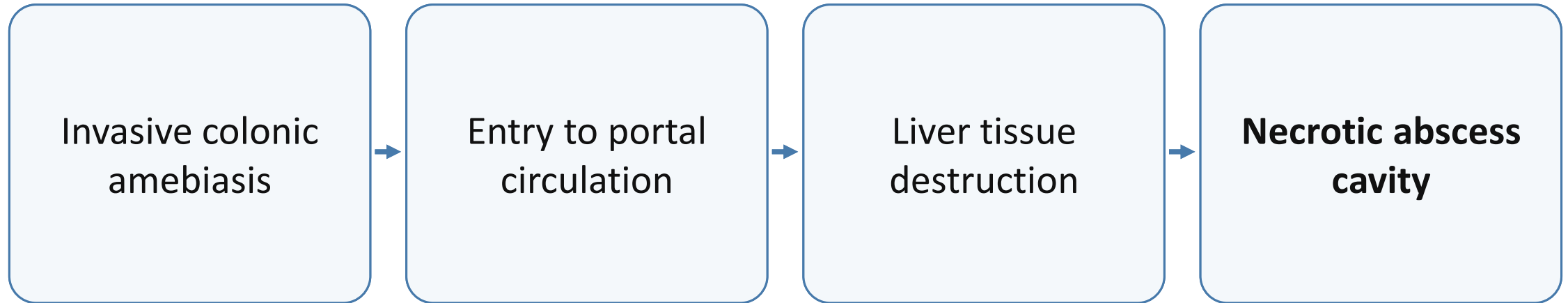
Amebiasis: Fecal–oral acquisition



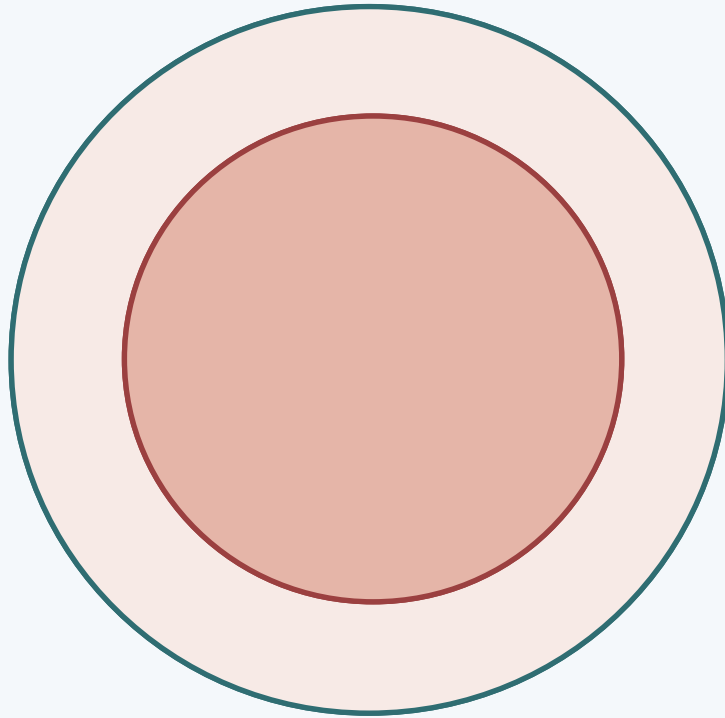
Colonic invasion precedes hepatic amebiasis



Amebic liver abscess: Portal spread



Amebic abscess: Necrosis dominates



Schematic — not a histologic specimen

Pathologic interpretation

- Extensive liquefactive tissue necrosis
- Relatively limited inflammatory reaction at the margin
- Shaggy fibrinous lining may be present

Amebic disease may outlast intestinal symptoms

- Hepatic abscess can persist after the acute intestinal illness resolves.
- Large cavities can form; Extension to contiguous thoracic organs is possible.
- The differential includes bacterial suppurative abscess, but tissue responses differ.

Pyogenic and amebic liver abscesses: Contrast

Characteristic	Bacterial/pyogenic	Amebic
Mechanism	Bacterial seeding or ascending biliary infection	Portal dissemination of trophozoites
Dominant reaction	Suppurative inflammation	Liquefactive necrosis; Scant marginal infiltrate
Clinical overlap	Fever, right upper quadrant pain	Hepatic abscess may follow colitis

Visceral leishmaniasis: Hepatic component

Systemic syndrome

Fever, weight loss,
hepatosplenomegaly

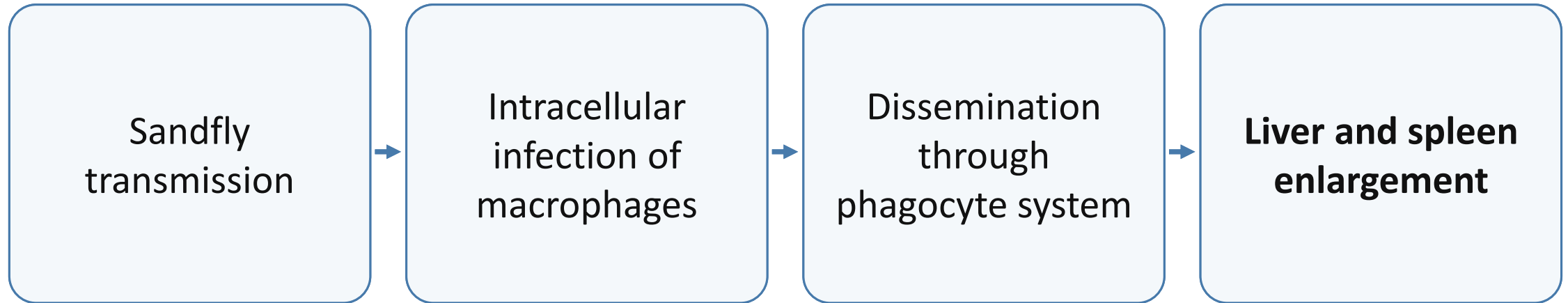
Cellular location

Parasites within phagocytic
cells

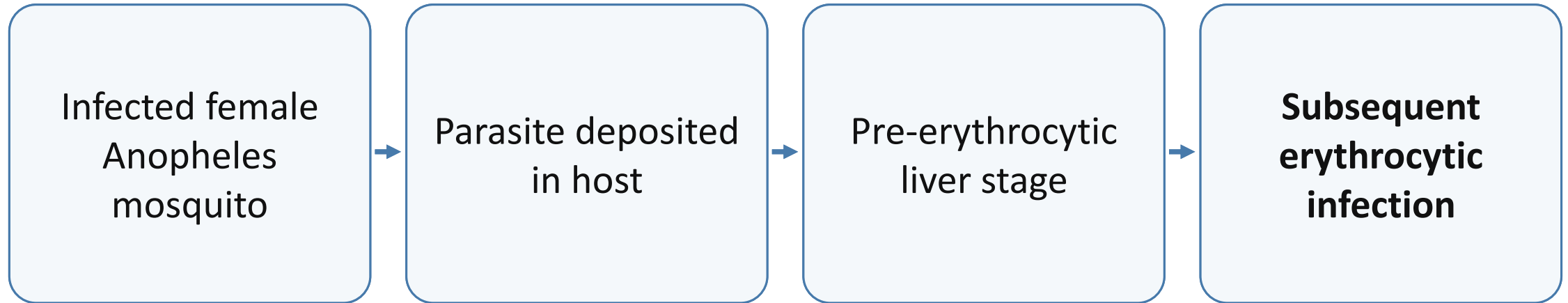
Late hepatic outcome

Increasing hepatic fibrosis

Leishmania and the mononuclear phagocyte system



Malaria: Hepatic phase is part of the life cycle



Other parasites named in the hepatic differential

Strongyloides

Helminthic infection listed in hepatobiliary section

Cryptosporidium

Protozoal infection listed in hepatobiliary section

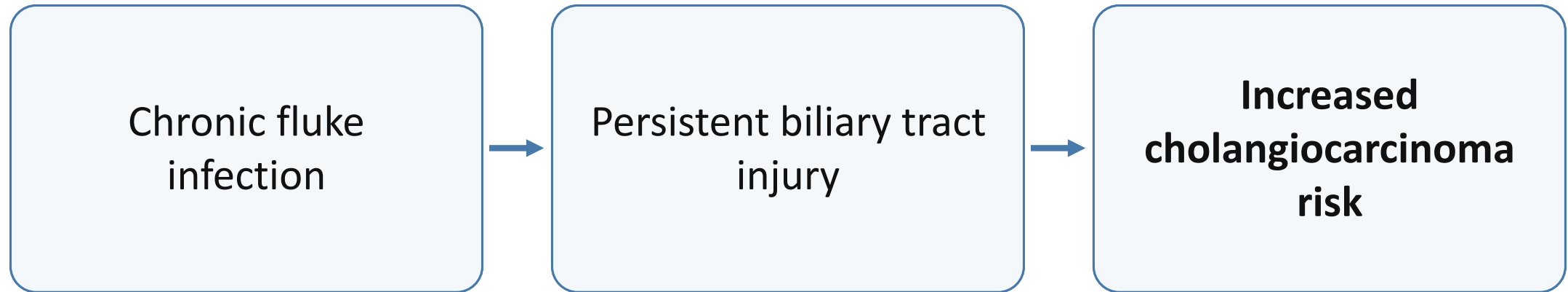
Fasciola hepatica

Liver fluke

Opisthorchis / Clonorchis

Liver flukes associated with biliary disease

Liver flukes are a biliary cancer risk context

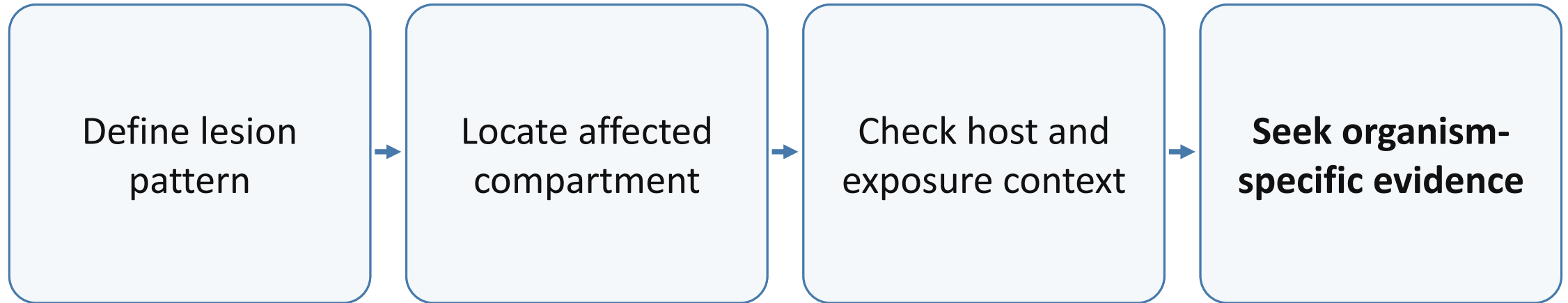


Association highlighted for Opisthorchis and Clonorchis

Hepatobiliary infection can affect different compartments

Compartment	Examples	Major lesion
Bile ducts	Ascending bacteria; Liver flukes	Cholangitis or chronic biliary injury
Portal tracts	Schistosome eggs	Granulomas and fibrosis
Parenchyma	Bacteria; Entamoeba	Abscess and tissue necrosis
Mononuclear phagocytes	Histoplasma; Leishmania	Granulomatous or systemic disease

An integrated pattern-based diagnostic approach



Microbiologic confirmation remains essential

Fungal / mycobacterial

Serology, blood culture, or biopsy organisms

Parasite-associated

Organism or characteristic tissue lesion

Bacterial obstruction

Clinical syndrome and biliary source

Any pattern

Interpret tests with clinical context

Three high-yield clinicopathologic distinctions

Observation	Strongest concept
Fever + obstructive jaundice	Consider ascending cholangitis
Granulomas $\pm$ necrosis	Consider disseminated fungal / mycobacterial disease
Portal fibrosis without regenerative nodules	Consider schistosomal pipestem fibrosis
Laminated cyst with daughter cysts	Consider hydatid disease

Applied case: Choose the dominant lesion

Hypothetical teaching vignette

- A liver specimen contains a laminated cyst wall, germinative lining, and daughter cysts.
- Which organism group is implicated, and why must spillage be avoided?

Echinococcus; Cyst contents can cause anaphylaxis or disseminate parasites

Integrated review: Organism → lesion → consequence

Cause	Dominant lesion	Clinical consequence
Ascending bacteria	Cholangitis / abscess	Sepsis; Jaundice
Histoplasma / mycobacteria	Granuloma	Systemic disseminated infection
Schistosoma	Egg granulomas / portal fibrosis	Portal hypertension
Echinococcus	Hydatid cyst	Mass effect / spill risk
Entamoeba	Liquefactive abscess	Persistent hepatic disease

Core references and scope

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th ed. 2026. Chapter 18: Liver and Gallbladder, pp. 762–763.
- Related primary-text sections: Chapter 8 (infectious response; histoplasmosis; schistosomiasis; echinococcosis; malaria; leishmaniasis).
- Additional related text within source: Chapter 17 (amebiasis), Chapter 18 (large bile duct obstruction, portal hypertension, cholangiocarcinoma).