Liver abscesses and cystic liver diseases

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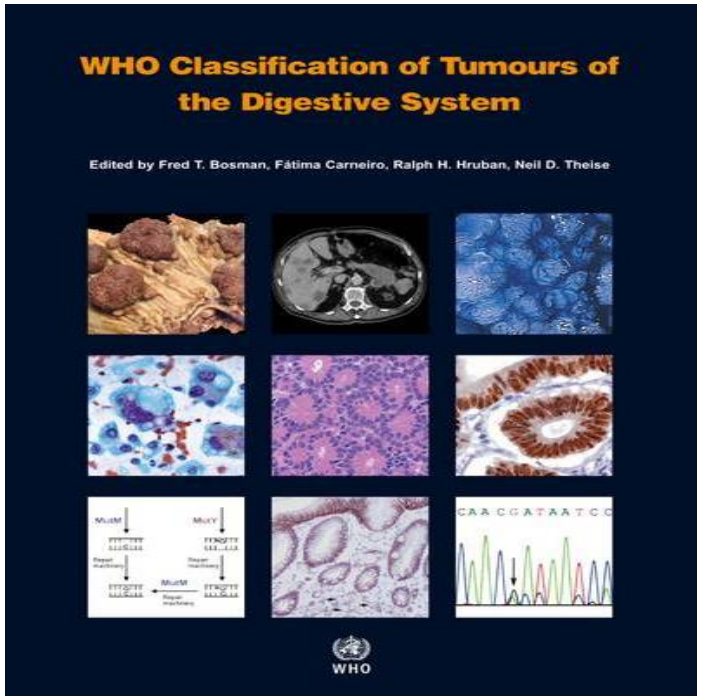
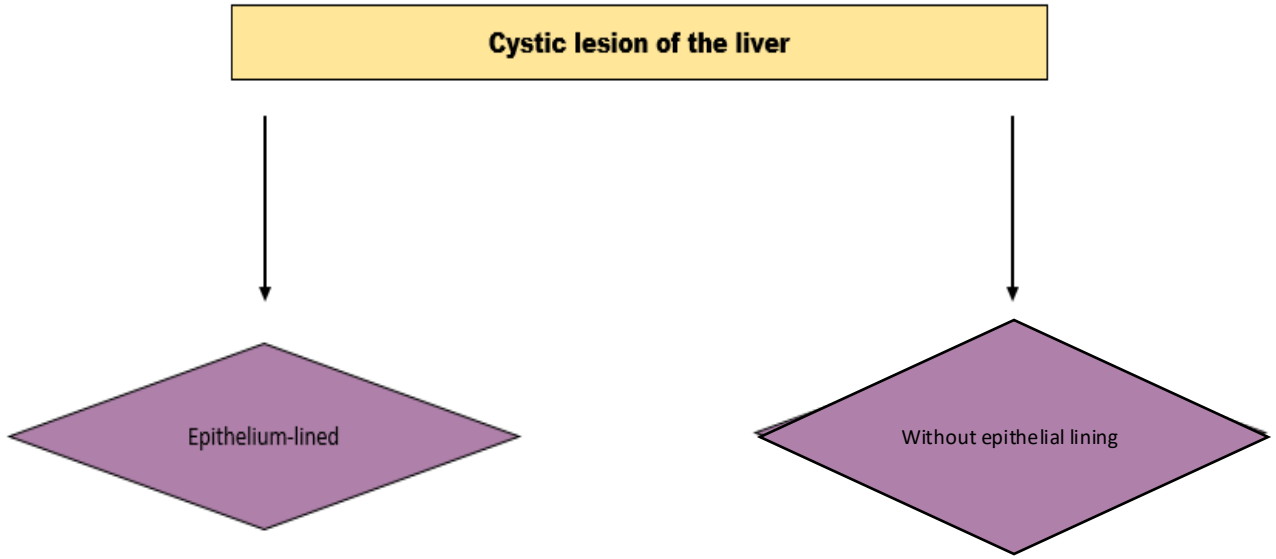
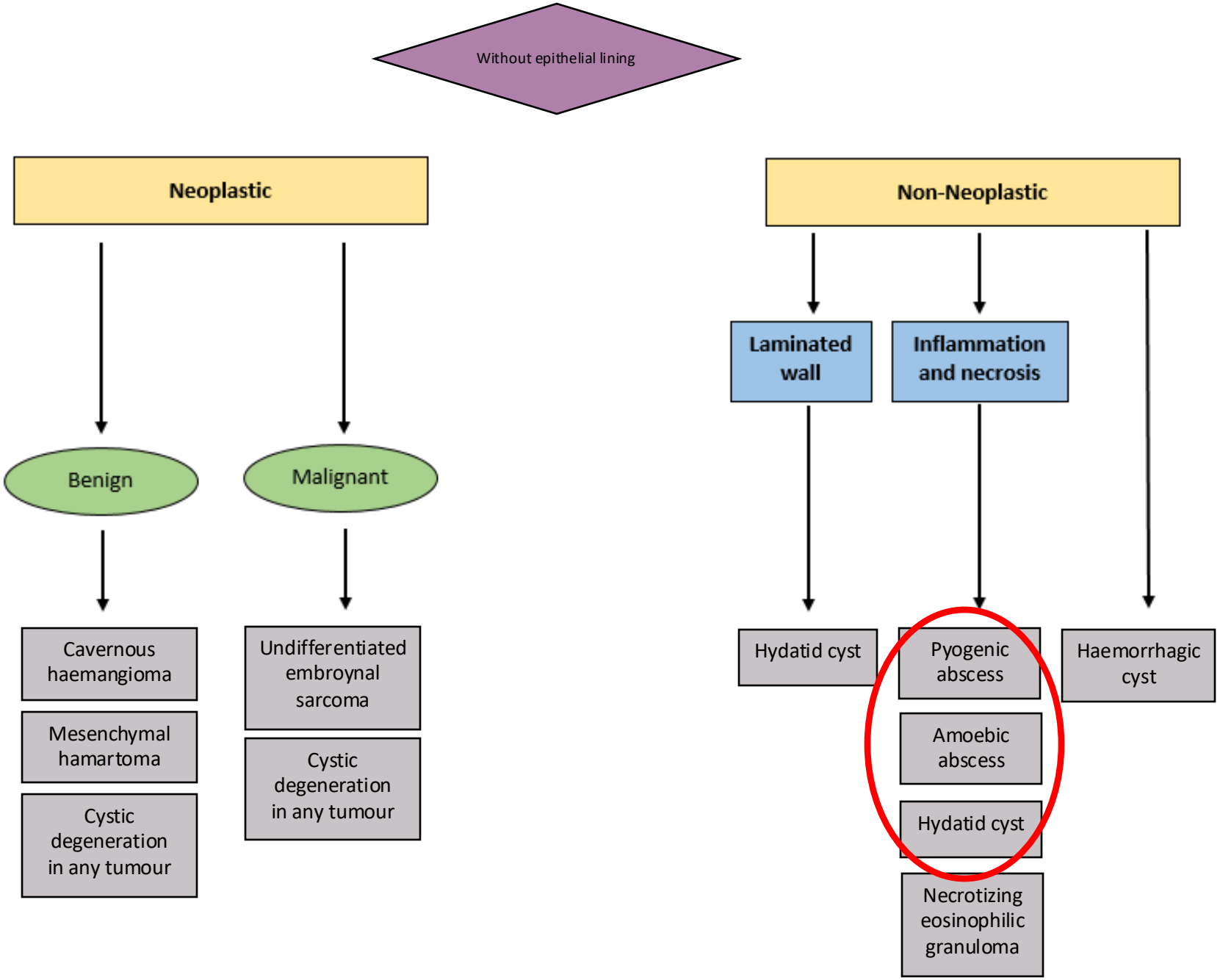
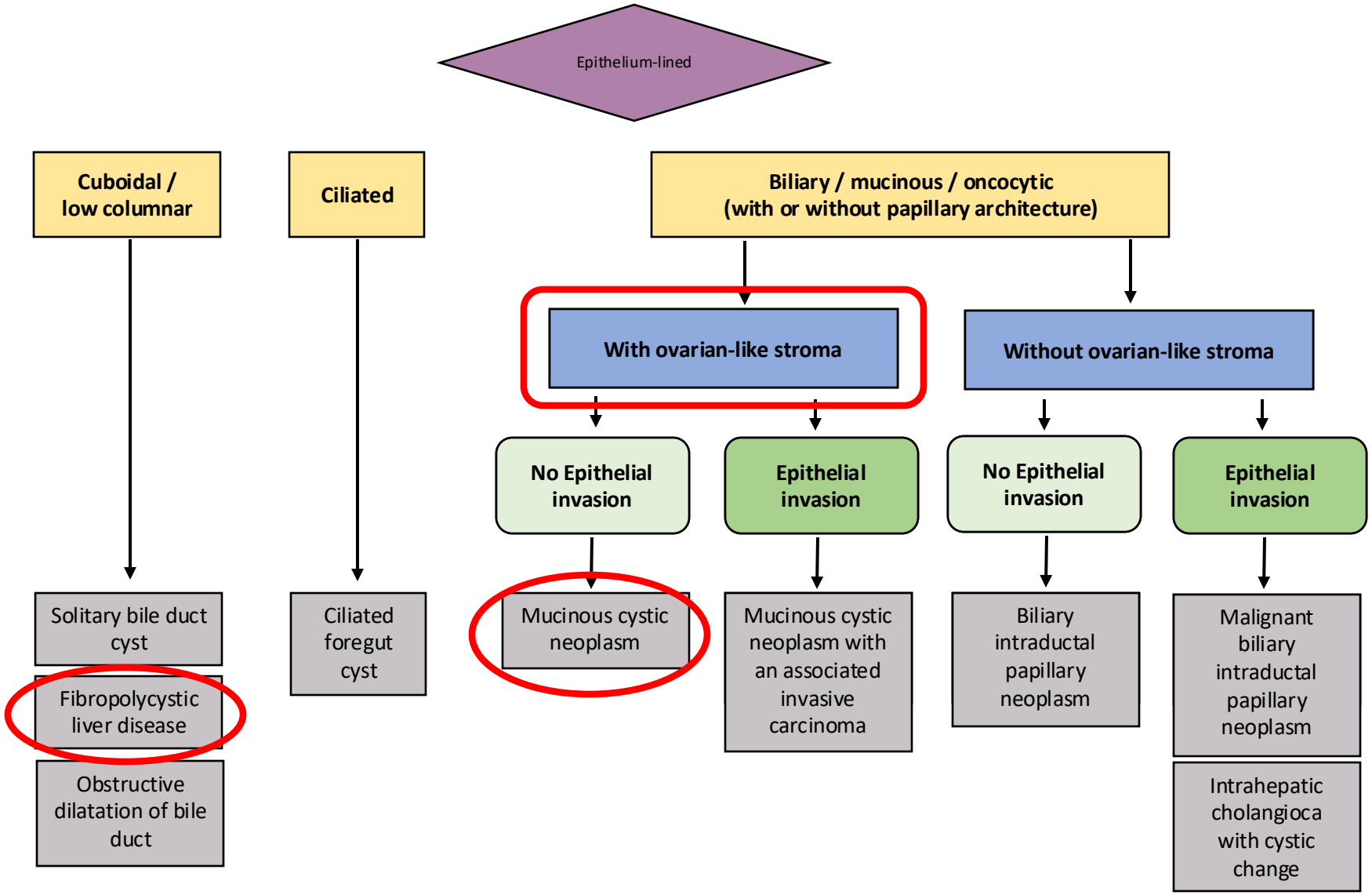
outline

- WHO classification-5Th edition
- Pyogenic liver abscess
- Amoebic liver abscess
- Hydatid cyst
- Simple cysts
- Polycystic liver disease
- Caroli disease
- Biliary harmatomas
- Peribiliary cysts
- Mucinous cystic neoplasms


WHO Classification of Tumours of the Digestive System
Edited by Fred T. Bosman, Fátima Carneiro, Ralph H. Hruban, Neil D. Theise
WHO

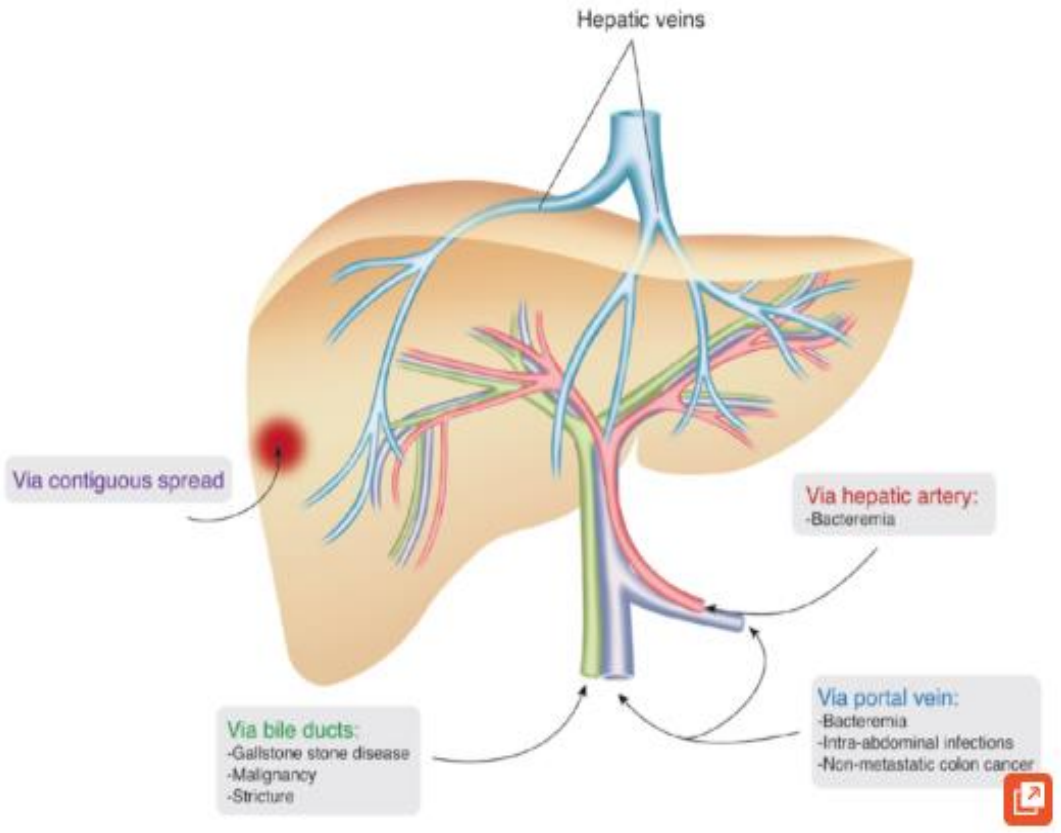

Cystic lesion of the liver
Epithelium-lined
Without epithelial lining


Without epithelial lining
Neoplastic
Benign
Malignant
Cavernous haemangioma
Mesenchymal hamartoma
Cystic degeneration in any tumour
Undifferentiated embroynal sarcoma
Cystic degeneration in any tumour
Non-Neoplastic
Laminated wall
Inflammation and necrosis
Hydatid cyst
Pyogenic abscess
Amoebic abscess
Hydatid cyst
Necrotizing eosinophilic granuloma
Haemorrhagic cyst


Epithelium-lined
Cuboidal / low columnar
Ciliated
Biliary / mucinous / oncocytic (with or without papillary architecture)
With ovarian-like stroma
Without ovarian-like stroma
No Epithelial invasion
Epithelial invasion
No Epithelial invasion
Epithelial invasion
Solitary bile duct cyst
Fibropolycystic liver disease
Obstructive dilatation of bile duct
Ciliated foregut cyst
Mucinous cystic neoplasm
Mucinous cystic neoplasm with an associated invasive carcinoma
Biliary intraductal papillary neoplasm
Malignant biliary intraductal papillary neoplasm
Intrahepatic cholangioca with cystic change

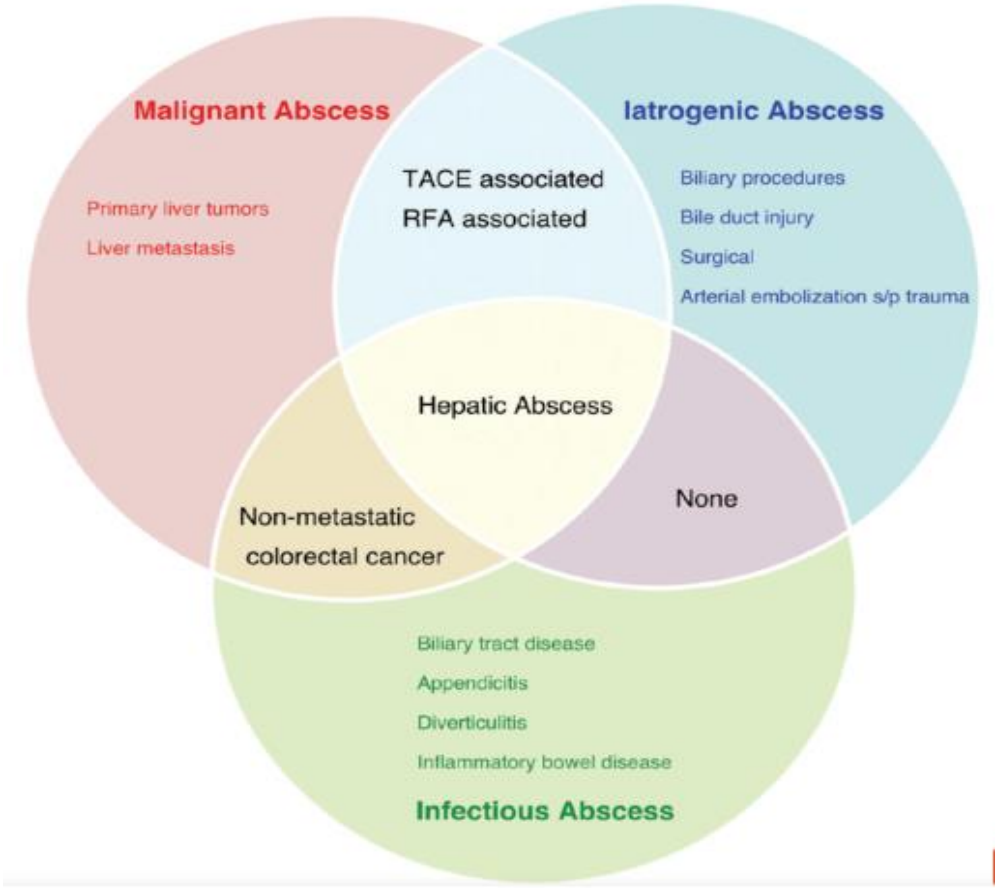
Pyogenic liver abscess(PLA)

- Localized collection of pus within the liver tissue
- Rare 2.3 /100,000 in the USA, higher in East Asia countries upto 17.6/100,000
- Higher in males 3.3 vs 1.3/100,00
- Develop secondary to biliary disease, portal pyemia of various causes, arterial hematogenous seeding, or via direct spread.



Taken from: Mavilia M, Molina M, Wu G. The Evolving Nature of Hepatic Abscess: A Review. J Clin Transl Hepatol. 2016

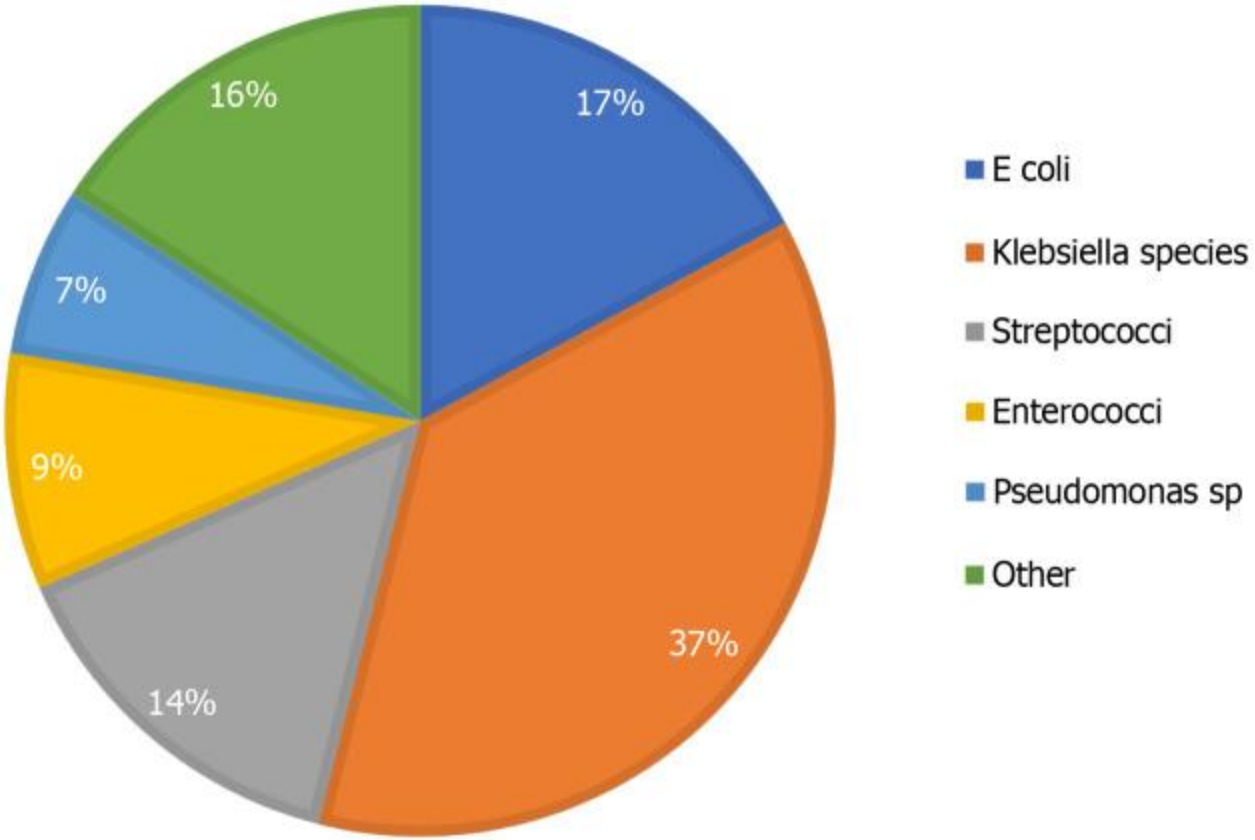
- Risk factors
  - Diabetes mellitus
  - Hepato-biliary dx
  - Pancreatic dx
  - Liver transplant
  - PPI use
  - Geographic location(E.Asia)
  - Colorectal cancer



▸ World J Hepatol. 2024 Dec 27;16(12):1417–1428. doi: 10.4254/wjh.v16.i12.1417

Clinicopathological presentation of liver abscesses and hydatid liver disease from two South African tertiary hospitals

Krevosha Pillay [1], Zafar Ahmed Khan [2,3], Ekene Emmanuel Nweke [4], Jones Omoshoro-Jones [5,6]



- Most abscesses are polymicrobial
- E.coli and Klebsiella sp commonly isolated organisms W.W (Similar findings in GSH)
- 48% ass. With biliary pathology
- (74.77%) pyogenic, amoebic (16.22%)and hydatid(9.01%)

Clinical features

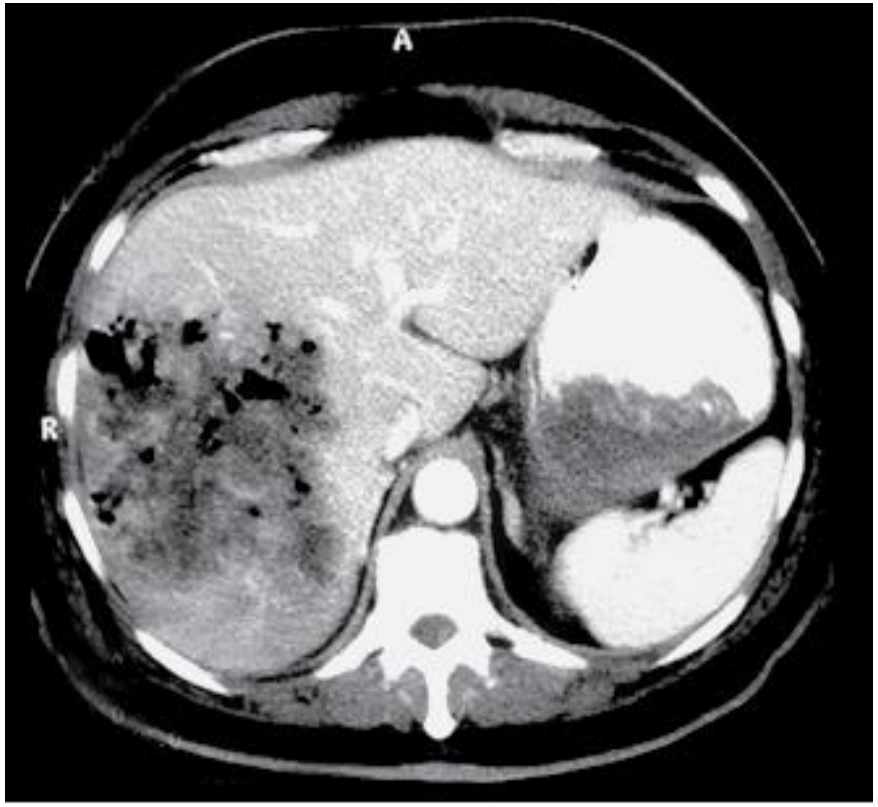
- Typical-Fever and abdominal pain. Others nausea, vomiting, anorexia, weight loss, and malaise.
- Approx. ½ pxs have RUQ tenderness, hepatomegaly or jaundice
- Less frequent: right pleural effusion, ascites, Murphy's sign, hypotension

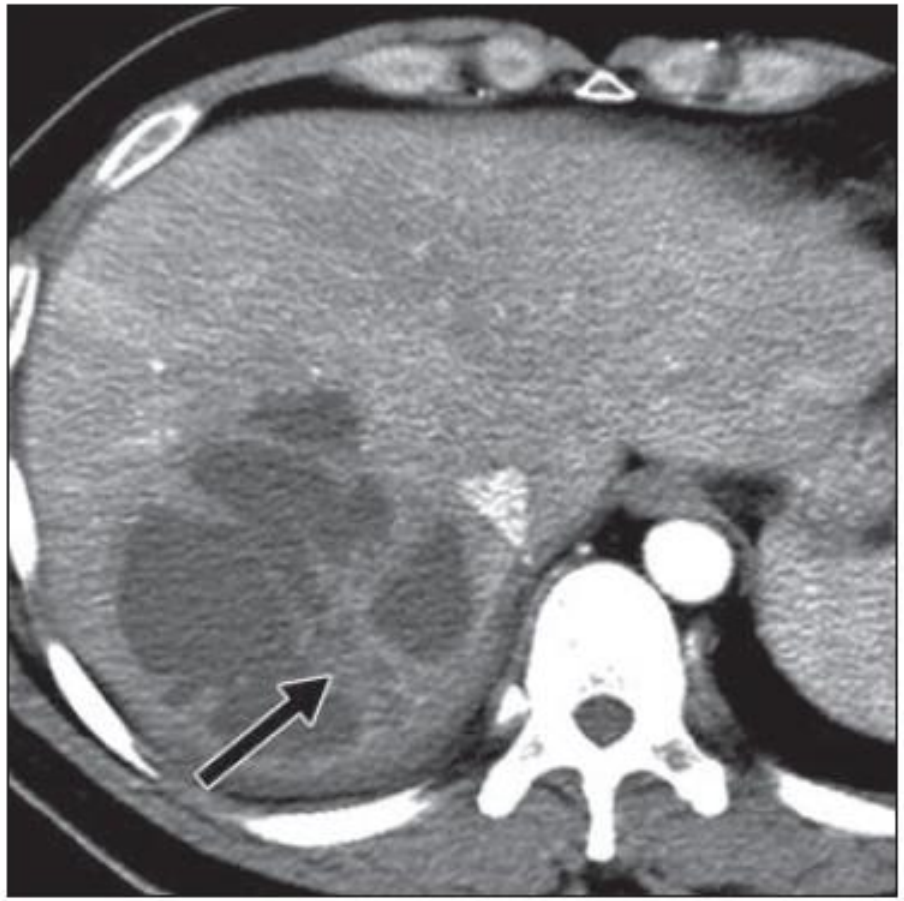
Investigations: Elevated bilirubin, AST, ALP and low albumin-non specific

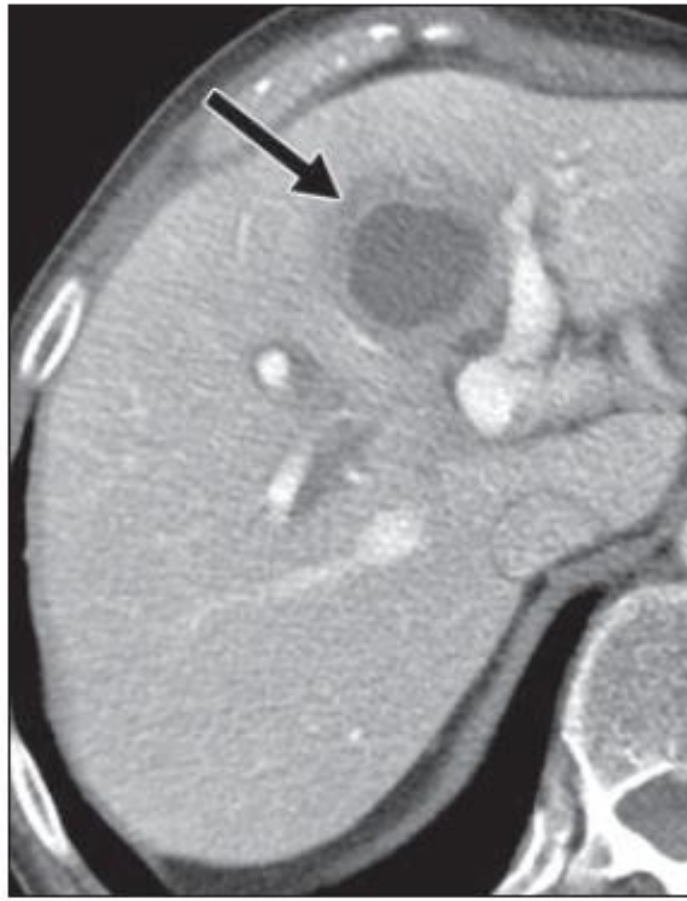
- CBC-Leukocytosis and normocytic normochromic anaemia
- Obtain blood for cultures(aerobic and anaerobic)
- *E.histolytica* serology

Imaging

- CT and U/S first choice
- CT higher sensitivity(95 vs 85%)
- **U/S -** can range from hypoechoic to hyperechoic lesions with internal echoes reflecting debris or septation.
- **CT**- well-defined, round lesion with central hypoattenuation, can also be more complex with loculated sub collections or an irregular border
- Pyogenic and amoebic abscesses can not be differentiated on imaging accurately
- **Aspiration**- CT or U/S guided for obtaining sample for gram stain and culture, and drainage of large abscesses







A contrast-enhanced CT scan of the upper abdomen demonstrates a large gas-containing abscess in the right lobe of the liver. This location is easily amenable to percutaneous CT-guided drainage

Medical management

- **The empiric regimen** should cover streptococci, enteric gram-negative bacilli, and anaerobes. The empiric regimen should also cover *E. histolytica* until the causative pathogen(s) is found or amebic abscess is excluded.
- Should be initiated before aspiration.
- Preferred regimens
- A 3rd or later generation cephalosporin plus metronidazole
- Piperacillin-tazobactam with or without metronidazole
- Ampicillin + gentamicin + metronidazole(up to 72 hours)
- **Duration of treatment**:
- Good response to drainage-IV 2-4 weeks IV, incomplete drainage 4-6 weeks IV
- Total duration: 4-6 weeks

Management

- **Single, unilocular abscesses with a diameter ≤5 cm** – Percutaneous drainage with either catheter placement or needle aspiration only is acceptable.
- **Single, unilocular abscesses with diameter >5 cm** –percutaneous drainage with placement of a catheter
- **Multiple or multiloculated abscesses** – The decision on drainage approach on an individual basis by a MDT taking into account the number, size, and accessibility of the abscess(es), the experience of the surgeons and radiologists, and the underlying condition and comorbidities of the patient.
- **Surgical drainage**(laparascopic or open)inadequate response to percutaneous drainage after 7 days or who have abscesses with viscous contents obstructing the drainage catheter.
- May also be considered for accompanying biliary disease or intra-abdominal tumor.
- **Outcome**: 2-12% mortality R/F:Female, open surgery, malignancy,liver failure and anaerobic and/or enterococcal infection.

Amoebic liver abscess

- Extraintestinal manifestation of amebiasis
- Almost all cases are caused by *E. histolytica,* but cases by *Entamoeba dispar* reported.
- 7-10 fold more in males in the 4^{th} and 5^{th} decade compared to females.
- In the west seen in immigrants and travelers from India, **Africa**, Mexico, and parts of Central and S.America.
- Defective cell mediated immunity appears to increase chances of invasive amoebic abscess in patients with dysentery but this is compounded by oral-anal sexual contact in this group of patients.

Clinical presentation

- Sub-acute(1-2 weeks) or chronic presentation (months)
- RUQ pain
- Fever
- Cough
- Malaise
- weight loss
- Anorexia
- Hiccup
- Concurrent diarrhea in less than 1/3 with others reporting it in the previous months
- **P/E**-Hepatomegaly and RUQ tenderness(50%), Jaundice (10%)
- Rupture into the pleural cavity or peritoneal cavity may occur with related symptoms
- Similar presentation HIV positive and negative patients.

Diagnosis

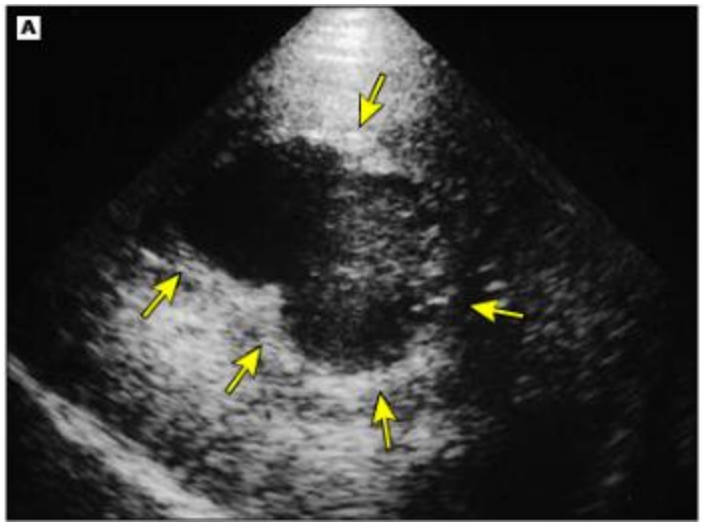
Lab:

- Leukocytosis without eosinophilia
- Elevated ALP
- Elevated transaminases
- Stool microscopy, antigen and serological testing(co-existence uncommon)

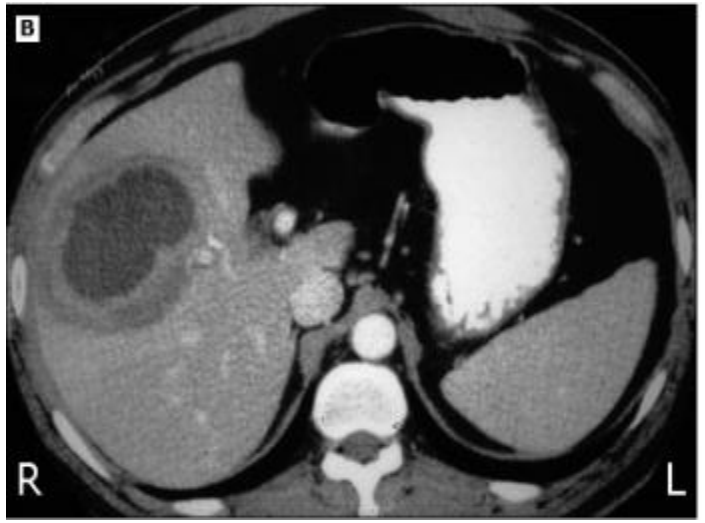
Imaging:

- **U/S**- appears as a round, well-defined hypoechoic mass
- **CT**- low-density mass with a peripheral enhancing rim.
- **MRI**-low-signal intensity on T1-weighted images and high-signal intensity on T2-weighted image
- **CXR**-elevation of the right hemidiaphragm

37 year old man with amebic liver abscess



(A) Sonogram shows a round mass consisting of a band of peripheral solid part (arrows), and central liquefied part showing low-level internal echoes.



(B) Contrast-enhanced computed tomography scan shows a peripheral solid part and central liquefied part. At surgery, the central portion was liquefied and contained "anchovy paste."

Adopted from UpToDate

Drainage

- Diagnostic and therapeutic
- Needle aspiration under ultrasound or CT guidance or insertion of a pigtail catheter
- Acellular, proteinaceous debris, and a brown fluid likened to "anchovy paste
- Trophozoites are seen on microscopy of the aspirate in <20%
- Antigen and PCR testing increases diagnostic yield
- Indications
  - cyst >10 cm in diameter
  - Imminent risk of rupture(left lobe lesions)
  - Clinical deterioration or lack of response to empiric therapy
  - Exclusion of alternative diagnoses
- When to remove drain
  - Size of the residual abscess (eg, <3 cm)
  - Improvement in abdominal pain
  - No fever for 48 hours
  - Drainage output <10 mL per 24 hours for two consecutive days,
  - Improvement in leukocyte count

Medical management

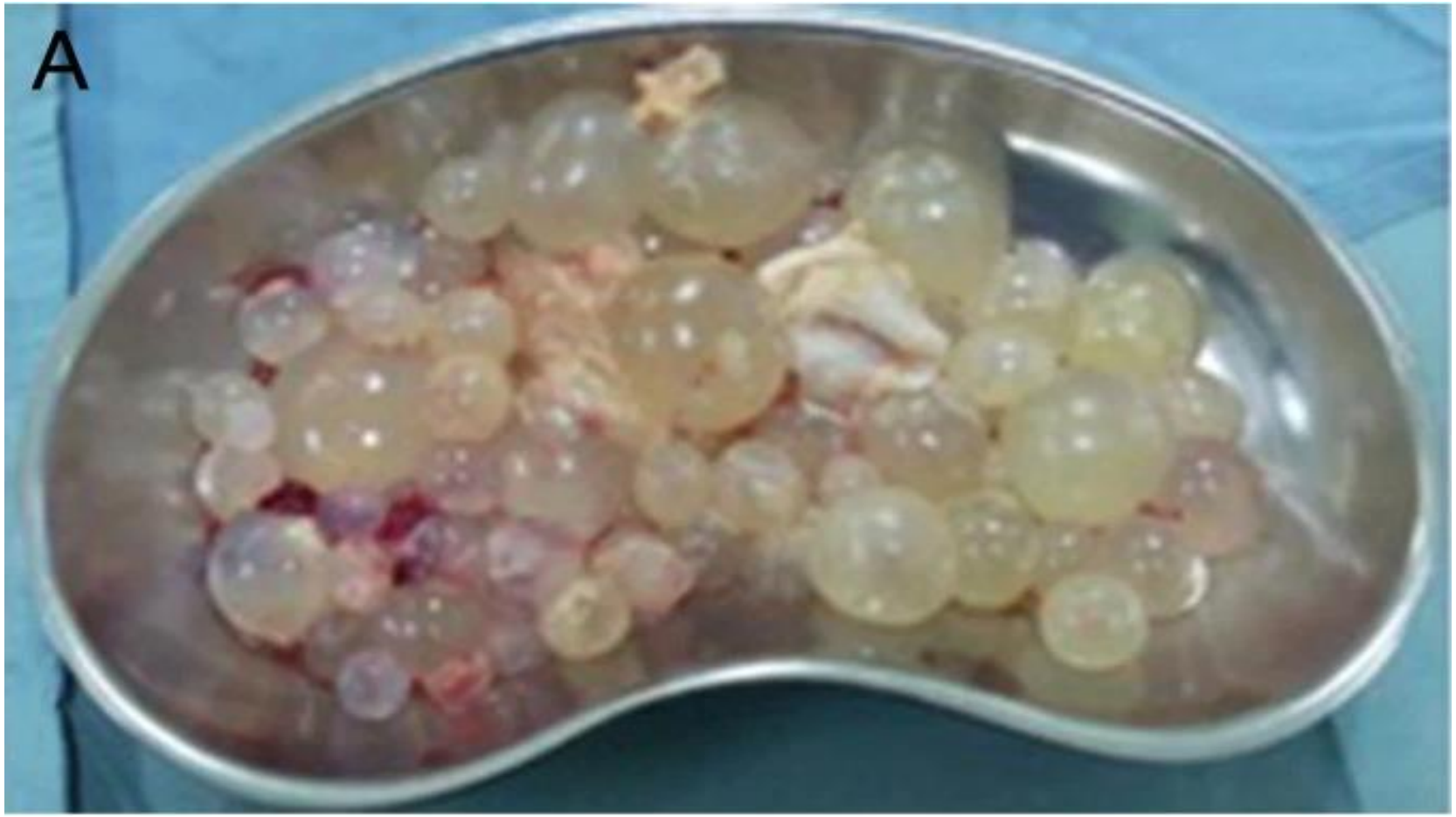
- Empiric treatment should be started pending stool, Ag and serology and aspirate tests
- **Tissue agents** — metronidazole 500 to 750 mg PO TID for 7 to 10 days, tinidazole 2 g once daily for 5 days ->90% cure rate
- Alt. Nitazoxanide 500 mg twice daily for 10 days
- **Luminal agents:** paromomycin 25 to 30 mg/kg per day orally in three divided doses for 7 days)
- Diiodohydroxyquin (650 mg orally three times daily for 20 days for adults and 30 to 40 mg/kg per day in 3 divided doses for 20 days for children), or diloxanide furoate (500 mg orally three times daily for 10 days for adults and 20 mg/kg per day in three divided doses for 10 days for children

- **In pregnancy** :Metronidazole is preferred in severe disease though safety studies are lacking.
- Alt.Chloroquine (600 mg base daily for two days, followed by 300 mg base daily for three weeks) followed by paromomycin

Outcome:

- <1% mortality in uncomplicated disease

Liver cysts

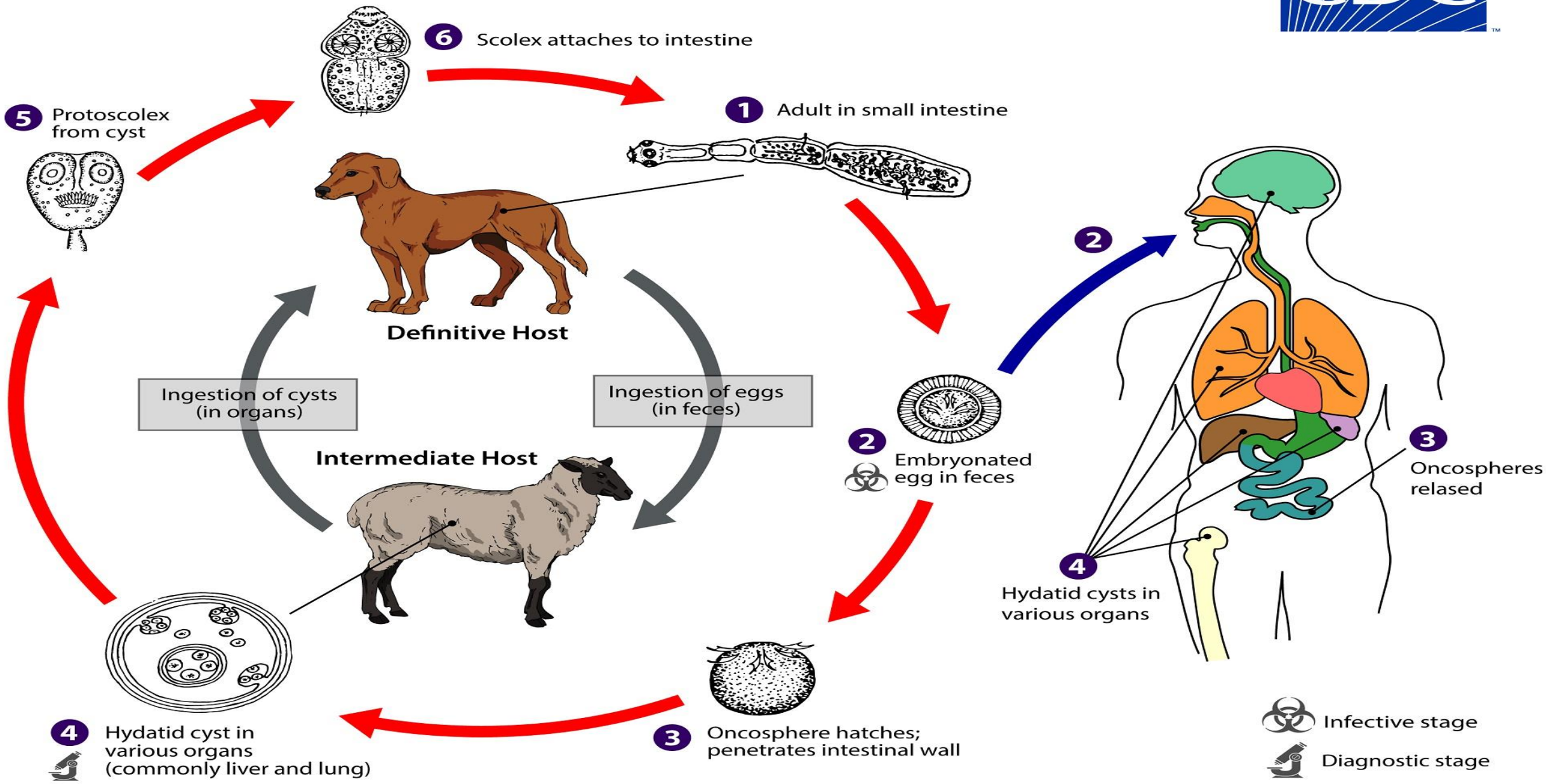


Hydatid cyst/Cystic echinococcus(CE)

- Caused by the larval stages of cestodes (tapeworms) of the genus *Echinococcus*.
- Humans are accidental intermediate hosts and become infected by ingesting parasite eggs, often through contaminated food, water, or soil, or through contact with infected animals.
- Prevalent in rural, underdeveloped areas, especially where people raise livestock and have close contact with dogs.

Cystic Echinococcosis

Echinococcus granulosus sensu lato



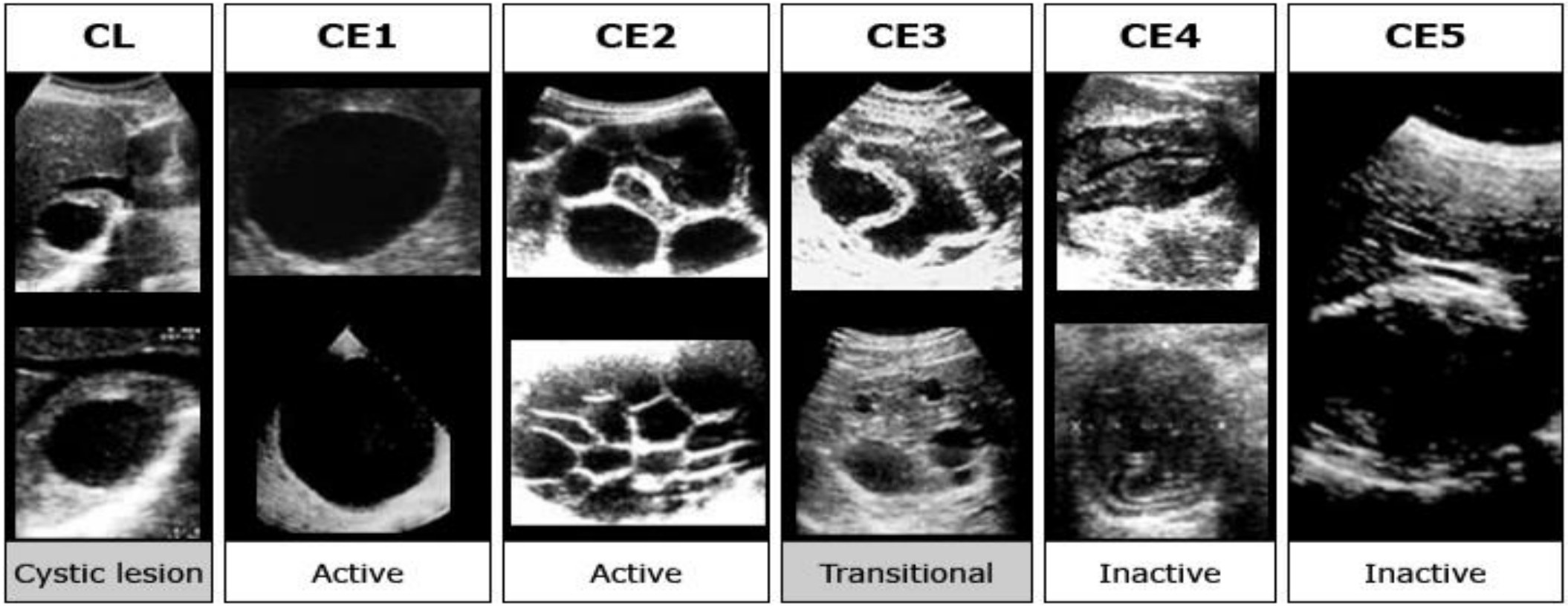
Clinical manifestations

- Most asymptomatic in- 50%
- Symptoms depend on size and site
- Liver commonest site(2/3) others lungs (25%), brain, muscle, kidneys, bone, heart, and pancreas.
- In the Rt liver lobe in 60-85% with frequent symptoms being RUQ pain, nausea and vomiting, rare in cysts<10cm.
- Pressure or mass effect on the bile ducts, portal and hepatic veins, or the IVC> cholestasis, PHT, venous obstruction, or Budd-Chiari syndrome.
- Rupture-peritonitis, allergic reaction(anaphylaxis),spread
- Secondary bacterial infection leading to pyogenic liver abscesses can occur.

Diagnosis

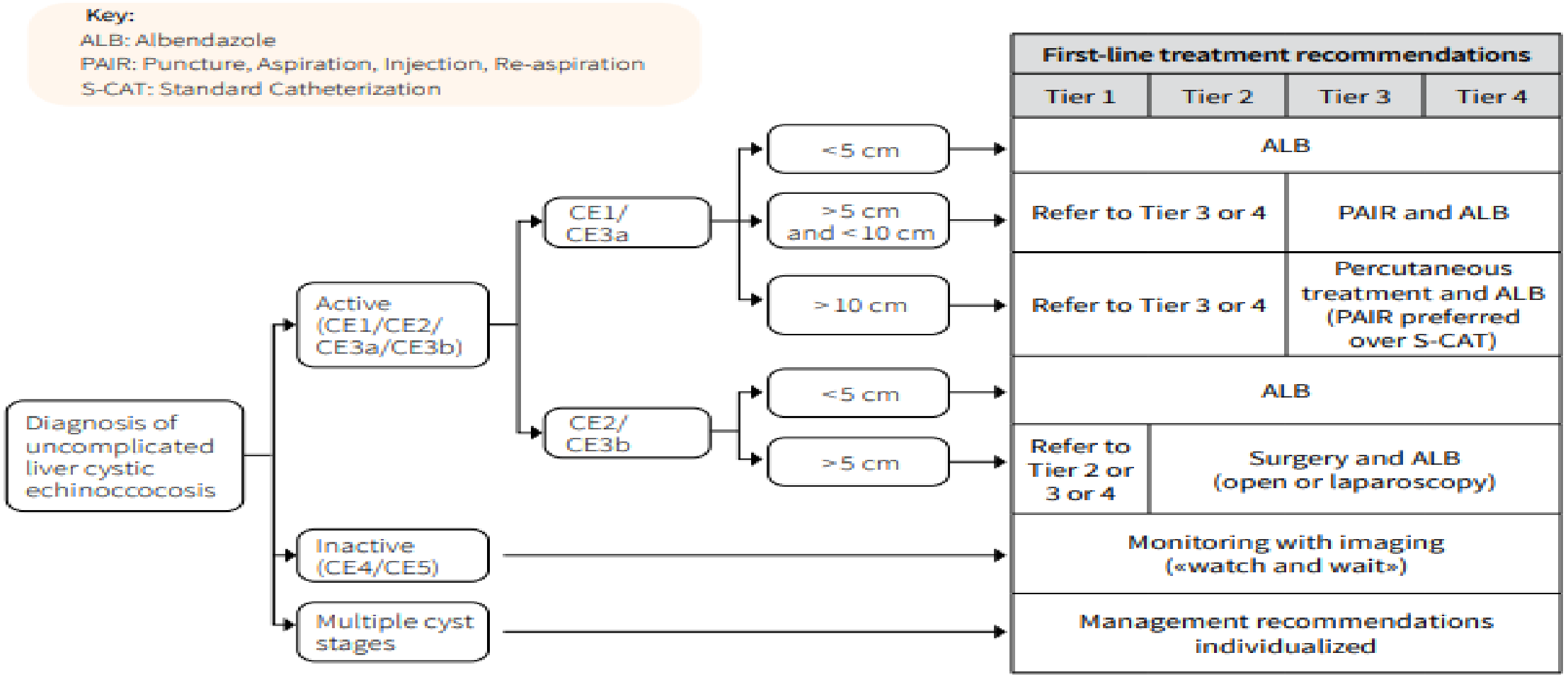
- Lab- Nonspecific leukopenia or thrombocytopenia, mild eosinophilia, and nonspecific liver function abnormalities.
- Dx-imaging and serology-antibody higher sensitivity
- Aspiration of cysts- controversial ,risk of anaphylaxis
- AUS –sensitivity 90-95%,an anechoic, smooth, round cyst similar to simple cysts, daughter cysts give characteristic septation.
- Shifting the patient's position during U/S may demonstrate "hydatid sand," which consists predominantly of hooklets and scolexes from the protoscolices.
- CT helps define features especially if wall calcified
- MRI can define but insensitive to calcification

Ultrasonographic classification of cysts due to cystic echinococcosis



snow flake sign Honeycomb sign Water lily sign Ball of wool sign Calcified shell

WHO guidelines for the treatment of cystic echinococcosis



Footnotes: Tiers (for full details see Table 2, p.7):
Tier 1: Medical doctor, basic laboratory capacity, ultrasound referral availability.
Tier 2: Tier 1 plus general surgeon, anaesthetic and operating theatre capacity, on-site ultrasonography.
Tier 3: Tier 2 plus laparoscopic surgeon, physician trained in PAIR, S-CAT, CT and fluoroscopy capacity.
Tier 4: Tier 3 plus thoracic surgery and interventional radiology capacity, MRI and MRCP capacity, advanced laboratory capacity.

Fig. 1. Algorithm for first-line treatment of uncomplicated liver CE cysts according to different health tiers based on health provider resources and capabilities

Management

- Asymptomatic, inactive, calcified cysts: watch and wait
- Active single compartment cyst <5cm(CE1,CE3a)- albendazole (ALB) monotherapy, 15mg/kg/day max.400mg BD, with fatty food for up to 6 months.
- **AEs**: Reversible hepatotoxitity1-5%,cytopenia 1%,alopecia 1%(monitor LFTS,CBC 2 weekly in the 1st 3 month of RX. Alt: praziquantel, mebendazole
- Active >5cm cysts or cysts with multiple compartments(CE2,CE3b)- Albendazole and PAIR/Surgery/Catheterisation (adjunctive treatment)
- ALB started 1 day pre-OP to up to 6 months

- **PAIR** (Puncture, Aspiration, Injection and Re-aspiration with scolicidal agent (ethanol) under U/S or CT guidance
- Cysts >5cm with no daughter cysts who aren't surgical candidates or decline surgery, or relapse post-op.
- Not recommended if biliary fistulas present or communications with the biliary tree (biliary sclerosis)
- **Modified catheterization techniques** — used to remove the entire endocyst and daughter cysts from the cyst cavity using large-bore catheters and cutting devices together with an aspiration apparatus.
- Used in combination with albendazole

- **Surgery**: Treatment of choice for ruptured cyst, cysts with biliary fistulae, cysts compressing vital structures, cysts with secondary infection or hemorrhage
- Intact cyst removal preferred.
- Albendazole +/- praziquantel should be started 1 week pre op and continued for 4 weeks post op(longer with spillage)
- **ERCP**
  - cholestatic jaundice and cholangitis-sphincterotomy with removal of membranes and cysts
  - External biliary fistula-stenting
  - Sphincter of Oddi stenosis-sphincterotomy
  - Bile duct stricture-Balloon dilatation and temporaly stent
- Follow up: CE recurs years after RX
- Follow up for up to for 5 years is reasonable.

Table 3- Uncommon causes of hepatic abscesses

Rare pathogens that can cause hepatic abscesses	Important considerations
Mycobacterium	• Rare • Generally manifests as multiple abscesses • Consider this in patients at risk or with previous exposure who do not have typical pyogenic organisms on liver aspirate culture
Candida	• Think about this in patients with hematologic malignancies or other immunosuppressive conditions • Can also be a co-pathogen with other bacterial organisms
Fasciola	• Endemic in central and South America, Europe, Asia, Africa and Middle East, with sheep and cattle as the main hosts • Morbidity is proportional to fluke burden • Chronic phase can be asymptomatic or present with RUQ pain, nausea, vomiting, jaundice or pancreatitis • Diagnosis can be made with duodenal aspirates, eggs in stool or bile specimens • Diagnostic clues include eosinophilia, abnormal liver chemistries and anemia

Adapted from: Davis J, McDonald M. Pyogenic liver abscess. UpToDate. Sep 2020. and Leder K, Weller P. Liver flukes:fascioliasis. UpToDate. Sept 2020

Hepatic cysts

- Fluid-filled lesions lined by a single cell layer
- Congenital and rare diseases that result from abnormal development of the embryonic ductal plate.
- Incidental finding on imaging, symptomatic in 15%

Simple cysts

- Congenital, no communication with intra hepatic biliary tree
- F>M
- **Symptoms**- abdominal pain, early satiety, nausea, and vomiting from mass effect.
- **Complications**: Infection, haemorrhage, rupture – RARE >10cm (trauma)
- Compression of adjacent organs – BILE DUCTS (SEGMENT IV)(ALP, Jaundice

Diagnosis

- **U/S** is the modality of choice with a sensitivity and specificity of 90% ,anechoic content with posterior enhancement.
- solitary *vs.* multiple and architecture (simple *vs.* complicated *vs.* complex cyst).
- **Complicated and complex cysts**-presence of calcifications, septations, mural thickening or nodularity, debris-containing fluid, haemorrhagic or proteinaceous contents, fluid levels, wall enhancement, and associated bile duct dilatation.
- Further imaging with MRI/CT required to guide intervention and surveillance

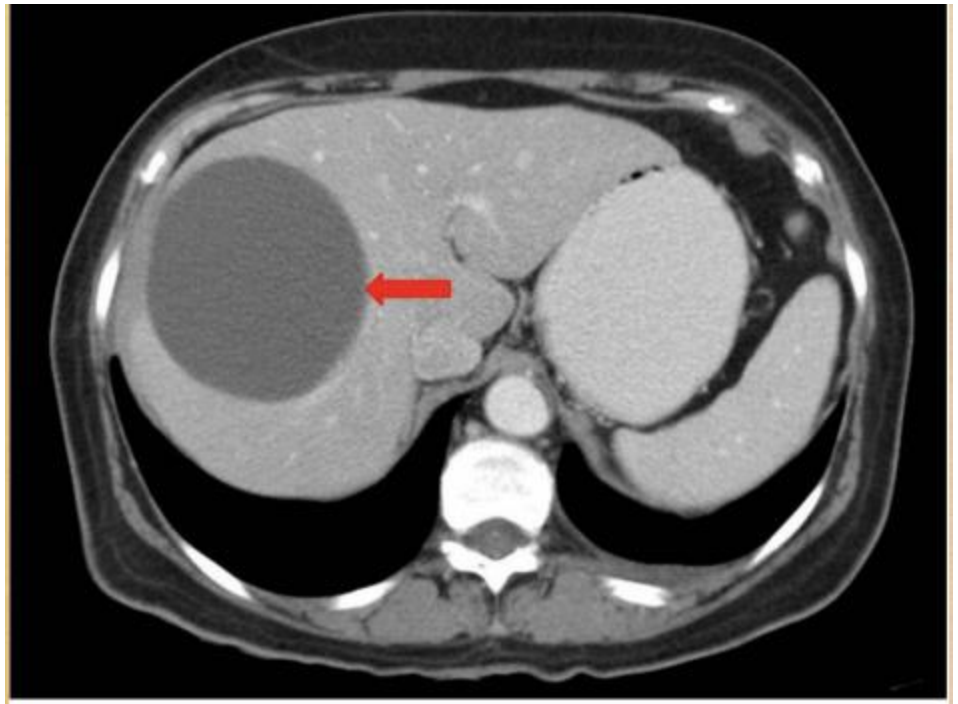


Fig. 1: Contrast enhanced abdominal CT showing a simple hepatic cyst (arrow).

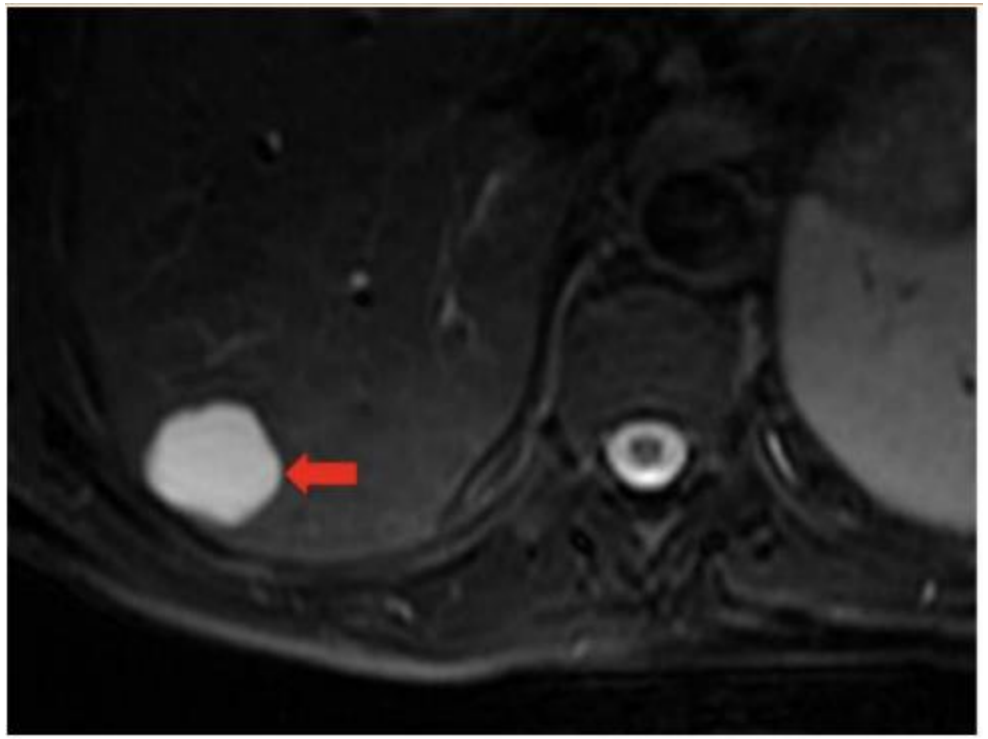


Fig. 2: Axial T2-weighted MR image showing a simple hepatic cyst (arrow).

Management

- **Asymptomatic:** Left alone
- **Symptomatic**: Volume reducing therapies
- **Percutaneous aspiration sclerotherapy**(Recurrence-higher)
- **Cyst fenestration-** Laparoscopy + drainage +cyst wall excision
  - Recurrence <8%
- **Partial hepatectomy and fenestration**- Multiple/Large
  - Perioperative mortality (14%)
  - Liver failure- mean 6-8 years with need for liver transplant (2-3%)
- **Success**: symptom relief not volume reduction.

complications

- Hemorrhage:Conservative Management– Avoid interventions during active hemorrhage
- Cyst infection: Fluoroquinolones or 3rd generation cephalosporins

Polycystic Liver disease

- Polycystic liver disease is a genetic disorder responsible for the progressive development of multiple cysts in the liver,>10.

  - Autosomal dominant polycystic kidney disease
  - Autosomal dominant polycystic liver disease

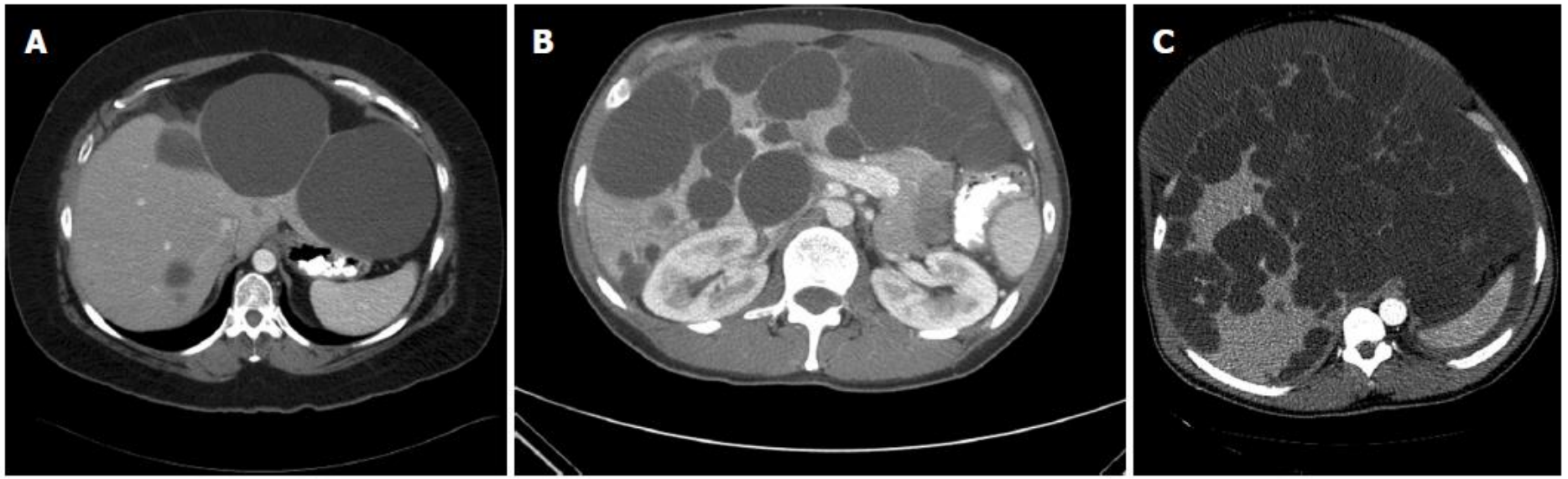
- These two forms of PCLD (with or without ADPKD) are linked to distinct gene mutations.
- Both, however, have an autosomal dominant transmission and almost identical clinical courses.
- One third of patients with isolated PCLD may have a few kidney cysts (usually one or two) as does the general population of adult patients

Clinical features

- Usually asymptomatic
- Symptoms – female/larger & more numerous cysts (10-15%)
- Abdominal pain/discomfort, postprandial fullness, hepatomegaly, dyspnoea
- Severe pain: Infection, intracystic hemorrhage, rupture, pendunculated cyst torsion, jaundice (5%)
- Complications- Ascites/Hepatic hydrothorax – PHT (congenital hepatic fibrosis) – rarely varices

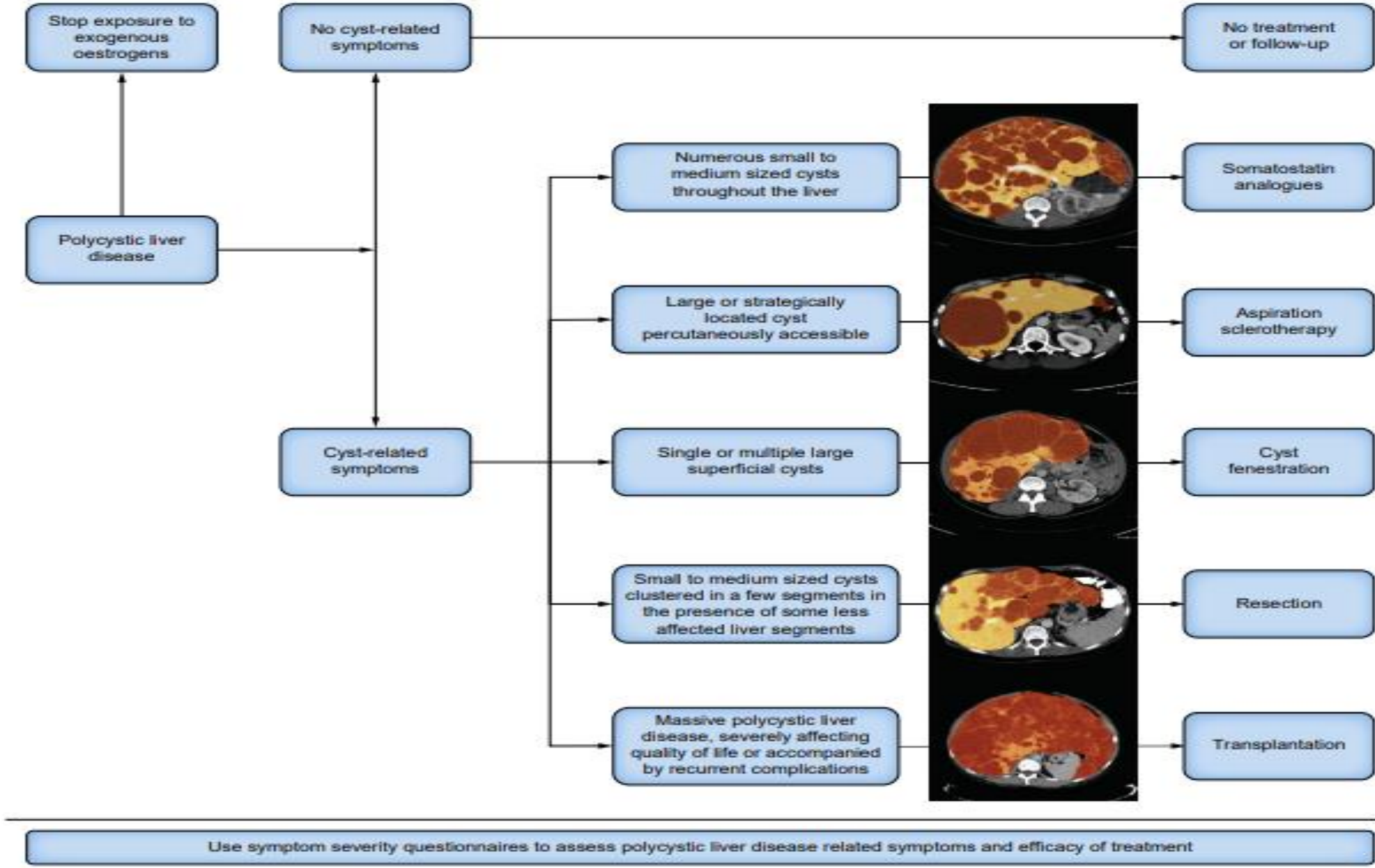
Diagnosis

- LFTs normal- Mildly elevated ALP/GGT
- CA19.9 – elevated-not diagnostic
- U/S; CT & MRI-PLD will appear as multiple hepatic cysts with the same features as simple solitary cysts
- KDIGO – AUS for all ADPKD
- Screening of family members of PLD is not recommended unless symptoms develop
- Liver volume - 1.8% increase annually (4.8% females < 48 YRS)
- Symptomatic patients management in expert centres
- Little room for genetic testing



Gigot classification

- Type I patients have a limited number (< 10) of large cysts with large areas of non-cystic parenchyma (Figure 1A)
- Type Ⅱ PCLD have diffuse involvement of liver parenchyma by medium sized cysts with remaining large areas of non-cystic parenchyma (Figure 1B).
- Type Ⅲ patients are characterized by massive, diffuse involvement of liver parenchyma by small and medium sized liver cysts and only a few areas of normal liver parenchyma between cysts (Figure 1C).


Stop exposure to exogenous oestrogens
Polycystic liver disease
No cyst-related symptoms
Cyst-related symptoms
No treatment or follow-up
Numerous small to medium sized cysts throughout the liver
Somatostatin analogues
Large or strategically located cyst percutaneously accessible
Aspiration sclerotherapy
Single or multiple large superficial cysts
Cyst fenestration
Small to medium sized cysts clustered in a few segments in the presence of some less affected liver segments
Resection
Massive polycystic liver disease, severely affecting quality of life or accompanied by recurrent complications
Transplantation
Use symptom severity questionnaires to assess polycystic liver disease related symptoms and efficacy of treatment

Criteria to refer patients with polycystic liver disease for liver transplantation

1. Clinically apparent liver disease due to massive polycystic liver severely affecting quality of life
2. Massive polycystic liver disease and complication(s), that can exclusively be treated by liver transplantation

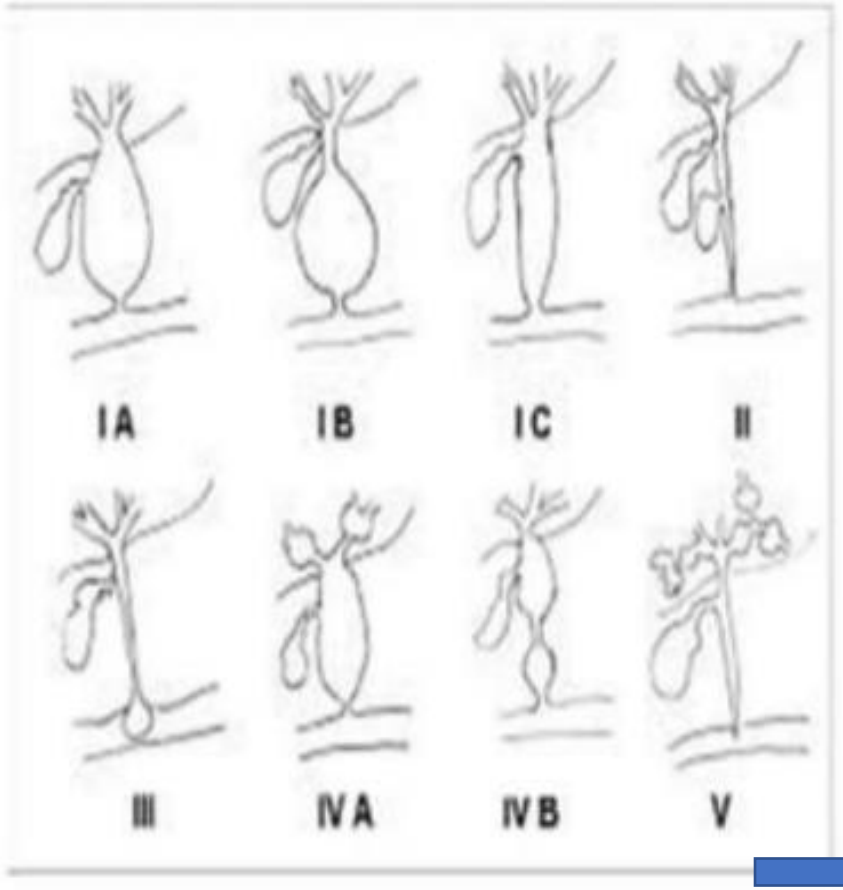
Complications include: severe malnutrition, hepatic venous outflow obstruction, ascites, portal hypertension, variceal haemorrhage, recurrent hepatic cyst infections

3. Failure of non-transplant related interventions and contraindications for non-transplant related interventions

Criteria to consider referral for combined liver-kidney transplantation

1. Creatinine clearance <30 ml/min

Todani classification of choledochal cysts



Type-IA	cystic dilation of the extrahepatic duct.
Type-IB	Focal segmental dilation of the extrahepatic duct
Type-IC	Fusiform dilation of the entire extrahepatic bile duct.
Type-II	simple diverticula of the common bile duct.
Type-III	cyst/choledochocele distal intramural dilation of the common bile duct within the duodenal wall.
Type-IVA	combined intrahepatic and extrahepatic duct dilation
Type-IVB	multiple extrahepatic bile duct dilations.
Type-V	Caroli disease/ multiple intrahepatic bile duct dilation

Caroli disease and syndrome

- Caroli disease (type V) dilatation of intrahepatic bile ducts from failure of proper remodelling and resorption of the ductal plate during foetal development.
- Caroli syndrome-congenital liver fibrosis and kidney cysts(collecting tubes and distal tubules) in addition to type V biliary dilatations.
- Likely a phenotype of ARPKD
- Prevalence of cholangiocarcinoma in Caroli disease and Caroli syndrome 7% compared to 0.05% in general population.
- Thought to be as a result of carcinogenic effect of recurrent cholangitis, cholestasis and irritation from gallstone.
- Diagnostic accuracy for cholangiocarcinome highest with MRI.
- Annual screening recommended.

Clinical features and diagnosis

- Cholestasis leading to stone formation, cholangitis, abscess formation
- Liver fibrosis (can be assessed by non-invasive methods)
- Portal hypertension and its complications
- Cholangiocarcinoma
- Renal: fusiform dilatations of renal collecting ducts and distal tubuli- renal impairment

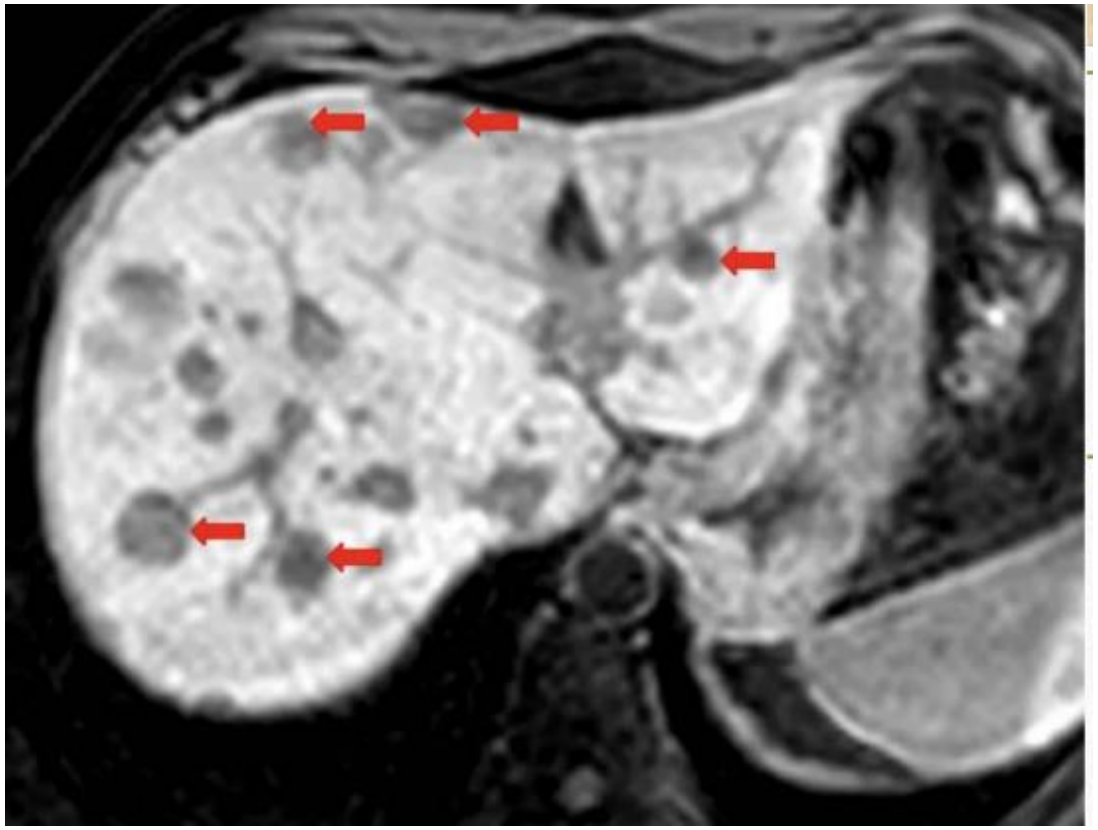


Fig. 5: Axial unenhanced T1-weighted MR image of a patient with Caroli disease, showing multiple hypointense cystic structures (arrows), corresponding to saccular dilatations of the biliary tree.

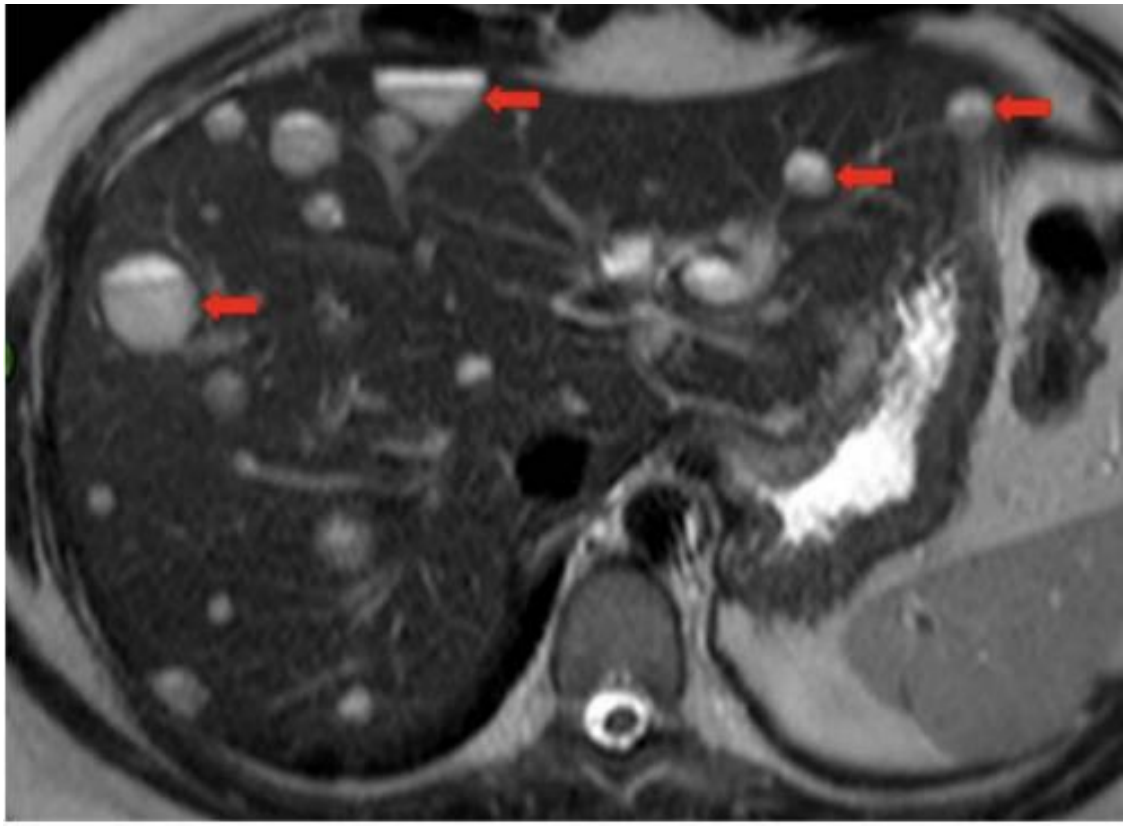


Fig. 6: Axial T2-weighted MR image showing multiple hyperintense cystic lesions (arrows), corresponding to intrahepatic bile duct dilatations in a patient with Caroli disease.

Management

- Cholangitis: antibiotics/ERCP
- Gallstones: UDCA(13-15 mg/kg/day) for all just as is in PBC
- Abscesses: drainage
- Monitor for complications(osteoporosis) as a result of cholestasis
- **Liver resection**: Relieves symptoms, particularly in patients with Caroli disease with restricted bile duct involvement
- **Liver transplant**: If Recurrent cholangitis and 1). Bi-lobar involvement or 2). Mono-lobar involvement in combo with liver fibrosis or portal hypertension and liver resection is not an option

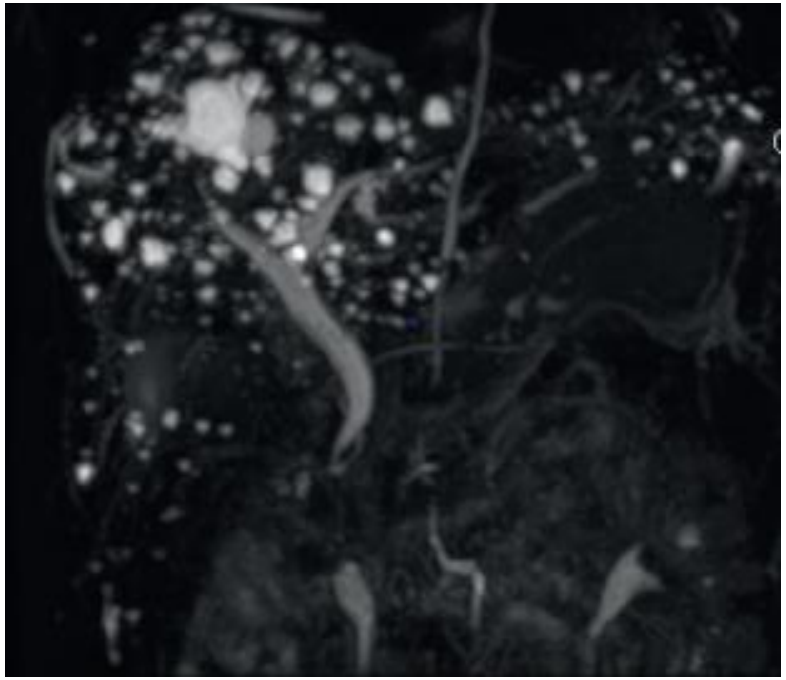
Biliary harmatomas and peribiliary cysts

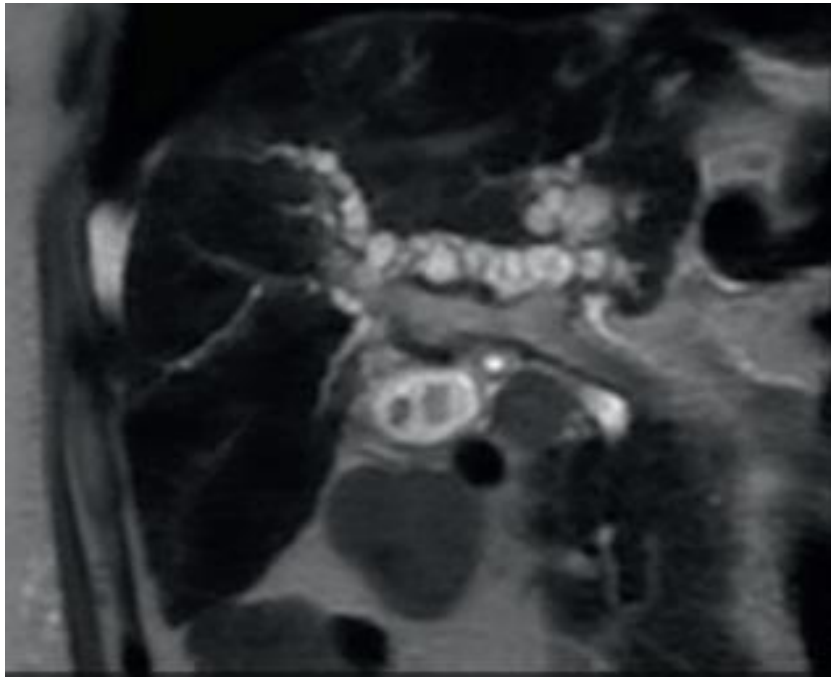
Biliary harmatomas AKA Von Meyenburg complexes

- Part of the spectrum of ductal plate malformations.
- Typically tiny e typically recognised as tiny <1 cm hypodense lesions throughout the liver with normal extra- and intrahepatic bile ducts.
- May mimic liver metastasis at first glance.

Peribiliary cysts- (predominantly perihilar and on both sides of the bile ducts) and small size

- Seen in patients with PHT and cirrhosis and may appear as discrete cysts, tubular structures paralleling the portal structures, or a string of cysts that simulate abnormal bile ducts.
- No further follow up required unless associated with hepatic fibrosis or caroli disease





(L)Biliary hamartomas, MRI cholangiography showing small innumerable T2 hyperintense cystic lesions scattered throughout the hepatic parenchyma without any communication with bile ducts, which resembles a “starry sky”; (R)peribiliary cysts, coronal T2-weighted MRI showing well-demarcated hyperintense cysts in a hilar distribution

Mucinous cystic Neoplasms

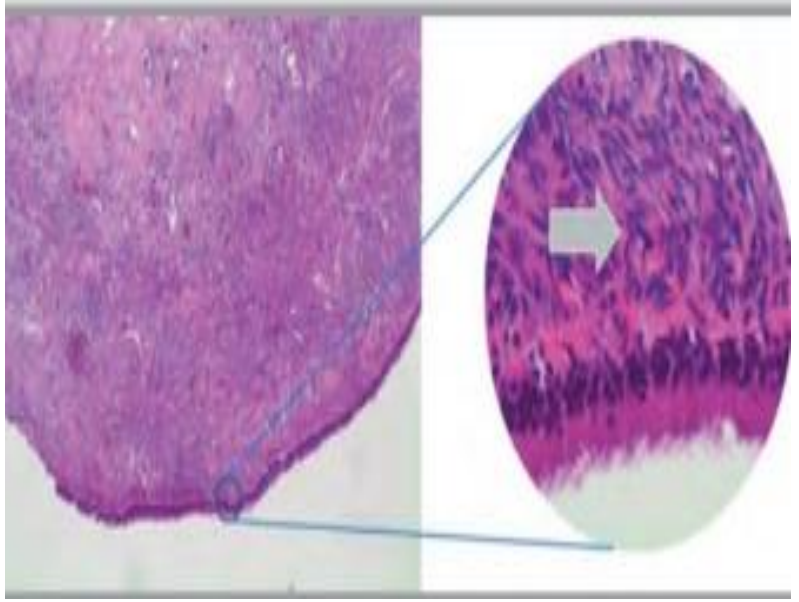
- MCNs are extremely rare.
- Also ‘biliary cystadenoma’ or ‘biliary cystadenocarcinoma’ in the literature.
- Cystic epithelial neoplasm lined by cuboidal, columnar, or flattened mucin producing epithelium overlying ovarian-like hypercellular stroma.
- Malignant transformation rates of up to 30% described.
- Presence of ovarian-like hypercellular stroma and absence of bile duct communication are the 2 hallmarks that differentiate MCNs from intraductal papillary neoplasms of the bile duct

Table 1: Comparison World Health Classification of Mucinous Cystic Liver Neoplasms

WHO 2000 Prior Nomenclature	WHO 2010 Current Nomenclature	Key Features
Biliary cystadenoma	Non-invasive Biliary Mucinous Cystic Neoplasm (ni-BMCN)	Needs ovarian-like stroma No typical bile duct communication
Biliary cystadenocarcinoma	Invasive Biliary Mucinous Cystic Neoplasm (i-BMCN)	Needs ovarian-like stroma No typical bile duct communication
Not previously classified	Intraductal papillary mucinous neoplasm – biliary type (IPMN-B)	No ovarian-like stroma Needs bile duct communication

Kloppers et al, South African Gastroenterology Review, 2016

Figure 1: Low and high power histology slides of a ni-BMCN wall with the arrow indicating the ovarian-like stroma



Clinical features

- Middle aged females
- 86% symptomatic- abdominal pain, fullness, or early satiety due to large size and mass effect.
- May present with low-grade dysplasia, high-grade dysplasia, or invasive carcinoma(3-6%).
- Serum CEA and CA19-9 elevated but lacks diagnostic accuracy.

Investigations

- MCNs are typically solitary, large, well circumscribed cystic lesions, either multiloculated (90%) or unilocular, that predominantly form in the left liver lobe.

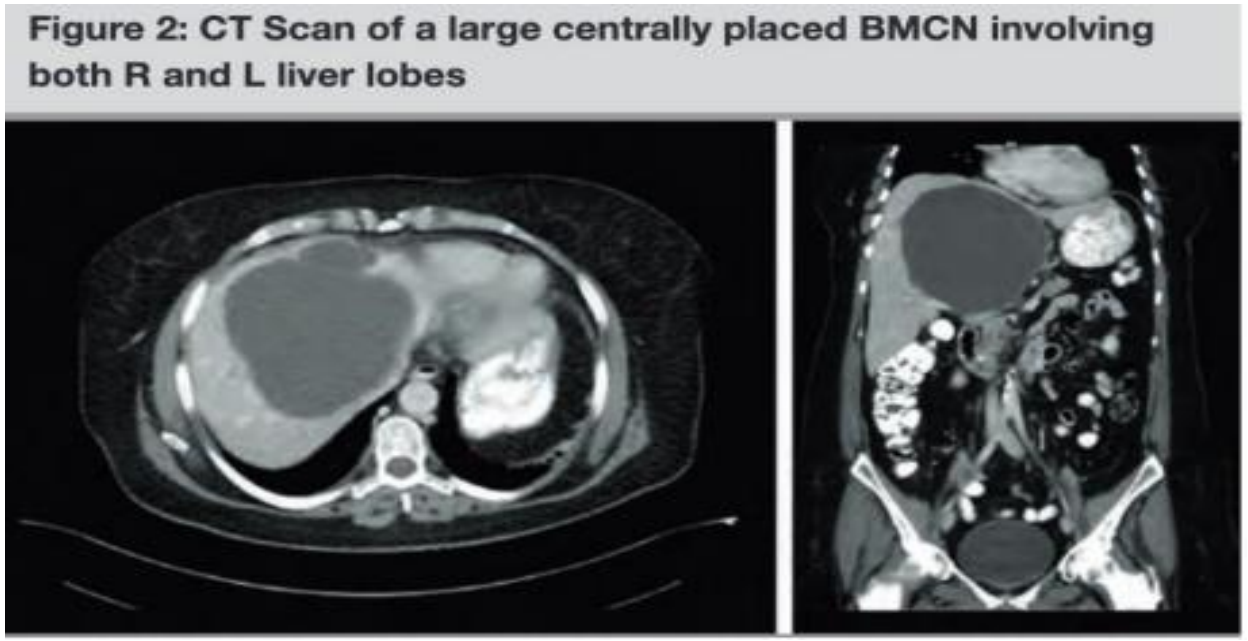


Figure 2: CT Scan of a large centrally placed BMCN involving both R and L liver lobes

Table 4. Worrisome features in mucinous cystic neoplasms of the liver.

Major worrisome features	Minor worrisome features
Thick septation	Upstream biliary dilatation
Nodularity	Thin septations
	Internal haemorrhage
	Perfusional change
	<3 coexistent hepatic cysts

- A combination of 1 or more major and 1 or more minor feature listed in Table 4 may be considered as worrisome features for MCNs.recommendation
- MRI should be used to characterise hepatic cysts with worrisome feature
- (TAG-72) in cyst fluid may help to distinguish between simple hepatic cysts and MCNs of the liver; (>25U/ml (Sen:79% & Spec:97%)

Management

- Mainstay→ **Complete resection with negative** margins (peripheral or one liver lobe)
- Enucleation if central and involving major vascular or biliary structures
- Aspiration, internal drainage, intra-tumoral sclerosant injection, incomplete resection→ 90-100% recurrence rate
- Irresectable disease: Fenestration and complete fulguration of internal cystic lining.(high recurrence)
- Cases of major liver resection,hemihepatectomy and liver transplant have been reported after unsuccessful resections.

Take home messages

- Pyogenic abscess, cover for amebic abscess until excluded and causative agent identified.
- Screen for colorectal cancer in patients with pyogenic abscess
- Liver cysts are common and not all need further imaging and treatment.
- MCNs carry a significant malignant potential and surgical excision with clear margins is treatment of choice

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Thank you
Asante