ULTRASOUND SOCIETY OF THE PHILIPPINES

Subspecialty of the Philippine College of Radiology

"We GUT you on this"
CASE REVIEW SERIES

ESSENTIALS and UPDATES on ULTRASONOGRAPHY of the GASTROINTESTINAL TRACT

Online Board Review Series 2025

Sinbad Alfonso A. Apepe MD
July 23, 2025

PORTAL HYPERTENSION

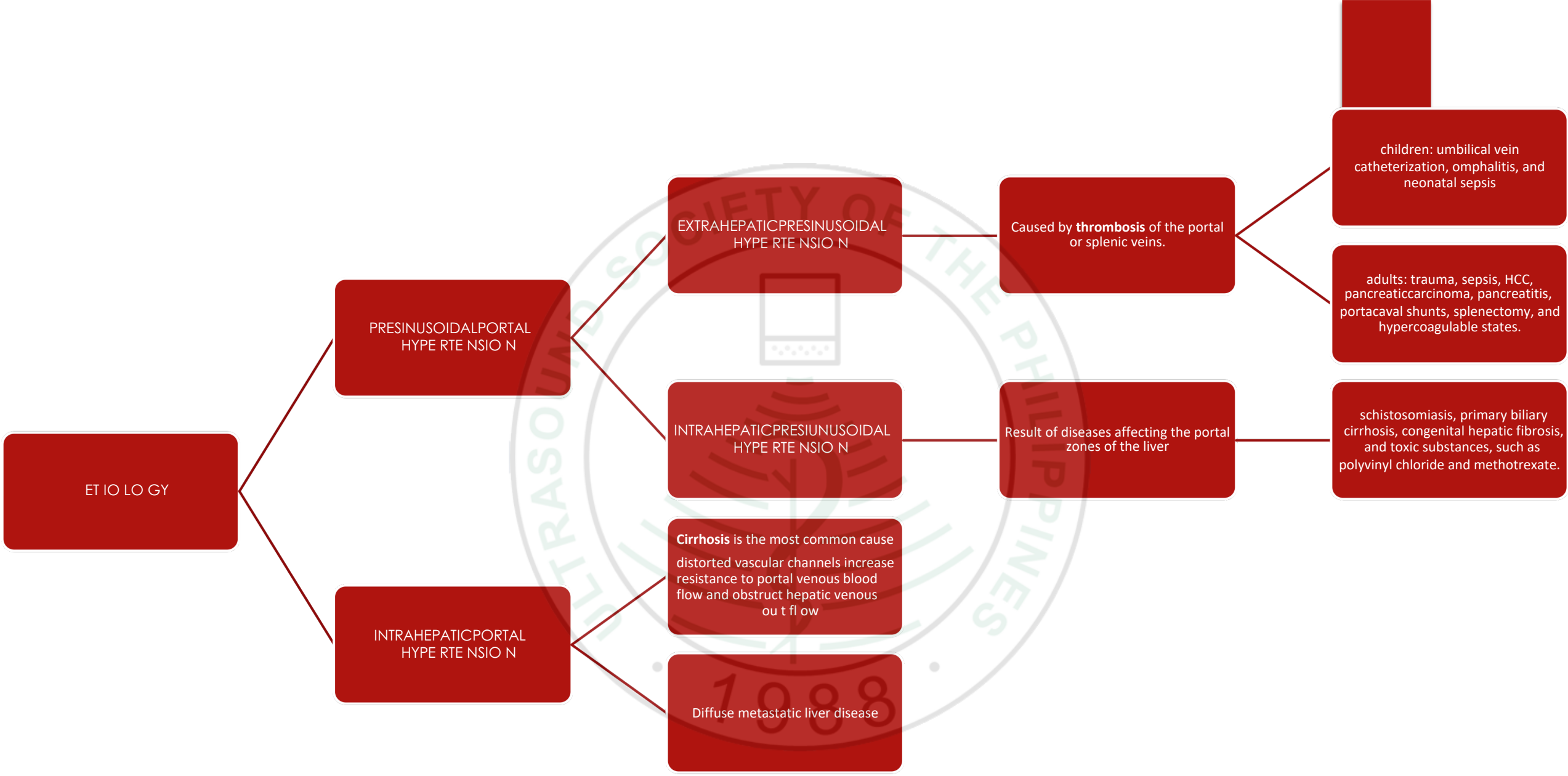
- Normal portal vein pressure = 5 to 10 mm Hg

DEFINITION

- A wedge hepatic vein pressure or diret portal vein pressure **more than 5 mm Hg** greater than IVC pressure
- A splenic vein pressure greater than **15 mm Hg**
- Or a portal vein pressure (measured surgically) **greater than 30 cm H2O**

ETIOLOGY

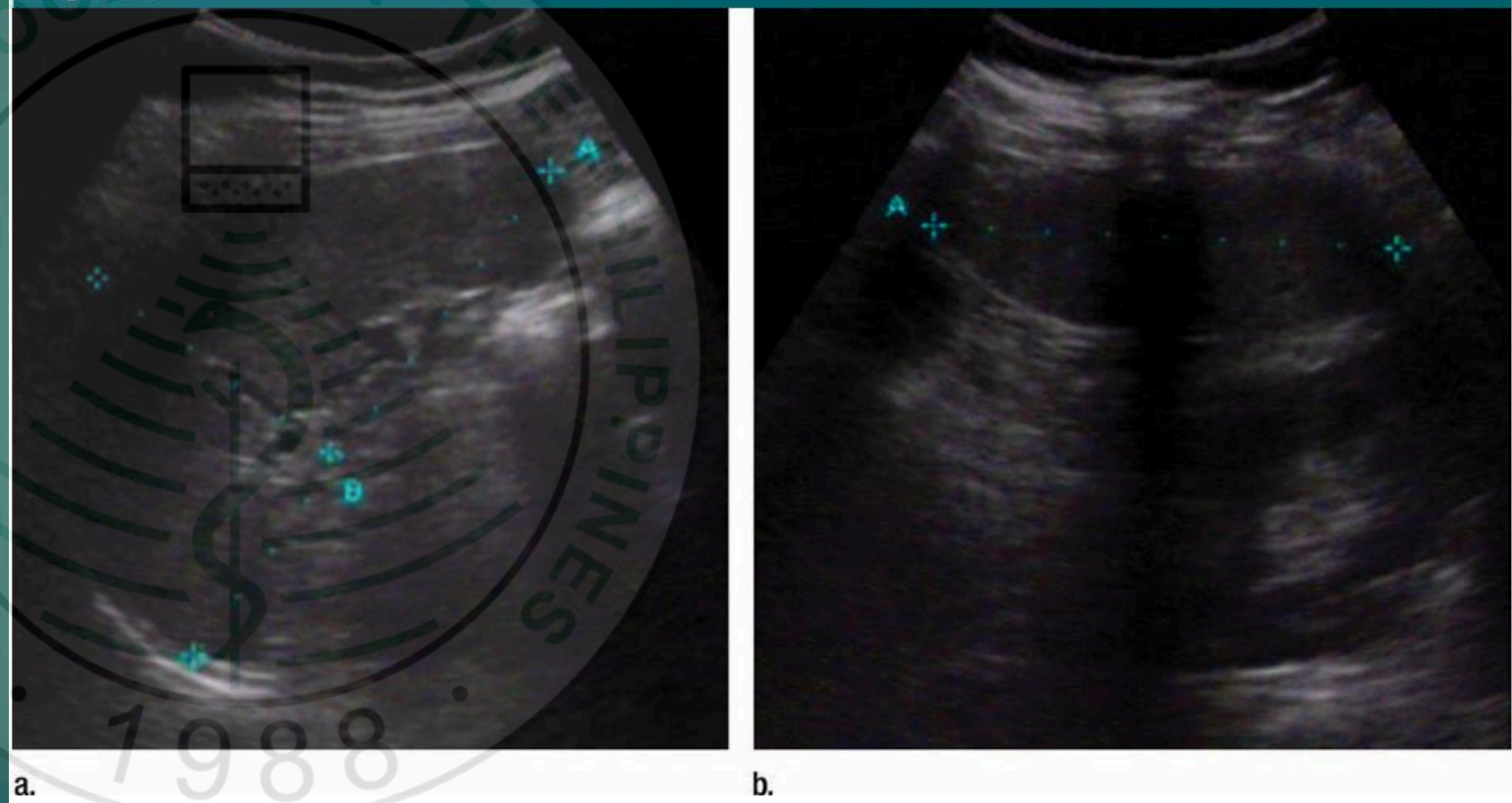
- Presinusoidal or intrahepatic


ET IO LO GY
PRESINUSOIDALPORTAL HYPE RTE NSIO N
INTRAHEPATICPORTAL HYPE RTE NSIO N
EXTRAHEPATICPRESINUSOIDAL HYPE RTE NSIO N
INTRAHEPATICPRESIUNUSOIDAL HYPE RTE NSIO N
Cirrhosis is the most common cause
distorted vascular channels increase resistance to portal venous blood flow and obstruct hepatic venous ou t fl ow
Diffuse metastatic liver disease
Caused by thrombosis of the portal or splenic veins.
Result of diseases affecting the portal zones of the liver
children: umbilical vein catheterization, omphalitis, and neonatal sepsis
adults: trauma, sepsis, HCC, pancreaticcarcinoma, pancreatitis, portacaval shunts, splenectomy, and hypercoagulable states.
schistosomiasis, primary biliary cirrhosis, congenital hepatic fibrosis, and toxic substances, such as polyvinyl chloride and methotrexate.

SONOGRAPHIC FINDINGS

Secondary signs of splenomegaly, ascites and portosystemic venous collaterals

Figure 1



a. b.

Figure 1: Assessment of spleen size with US. Representative US images are shown to illustrate measurement of **(a)** maximum spleen length and width and **(b)** spleen anteroposterior dimension at hilus.

Niknejad M, Cirrhosis in a patient with autosomal dominant polycystic kidney disease. [illegible] 27 Jan 2025] https://doi.org/10.53347/rID-153673

FIVE MAJOR SITES OF PORTOSYSTEMIC VENOUS COLLATERALS

Gastroesophageal junction:

- between the coronary and short gastric veins and esophageal veins
- may lead tolife-threateningorfatalhemorrhage
- >0.7 cm coronaryvein=severeportalhypertension

Paraumbilical vein:

- Runs in the falciform ligament and connects the left portal vein to the systemic epigastricveins near theumbilicus

Splenorenaland gastrorenal:

- Tortuous veins may be seen in the region of the splenic and left renal hilus

Intestinal:

- veins of the ascending and descending colon, duodenum, pancreas, and liver may anastomose with the renal, phrenic, and lumbar veins (systemic tributaries).

Hemorrhoidal:

- The perianal region where the superior rectal veins, which extend from the inferiormesenteric vein, anastomose with the systemic middleand inferior rectal veins.

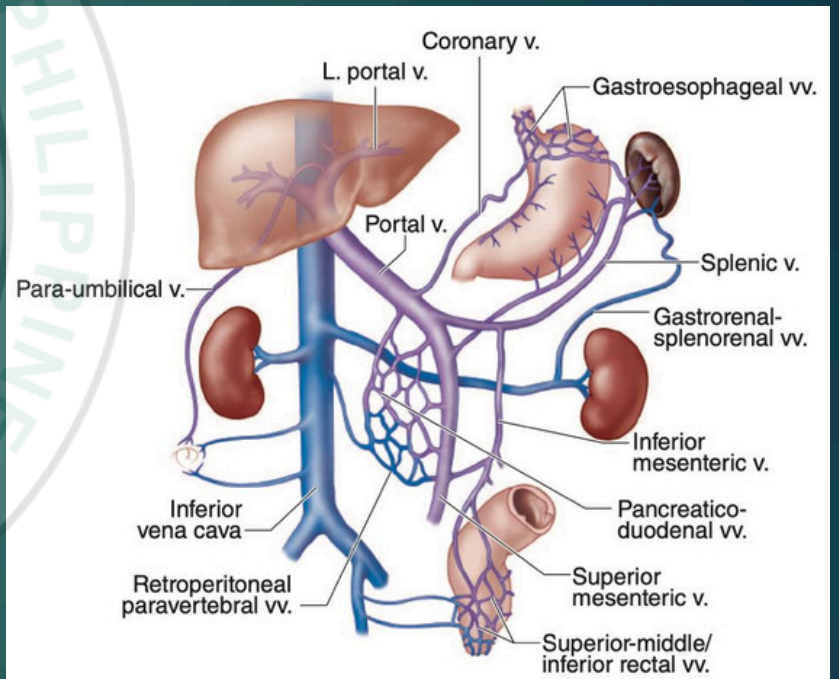
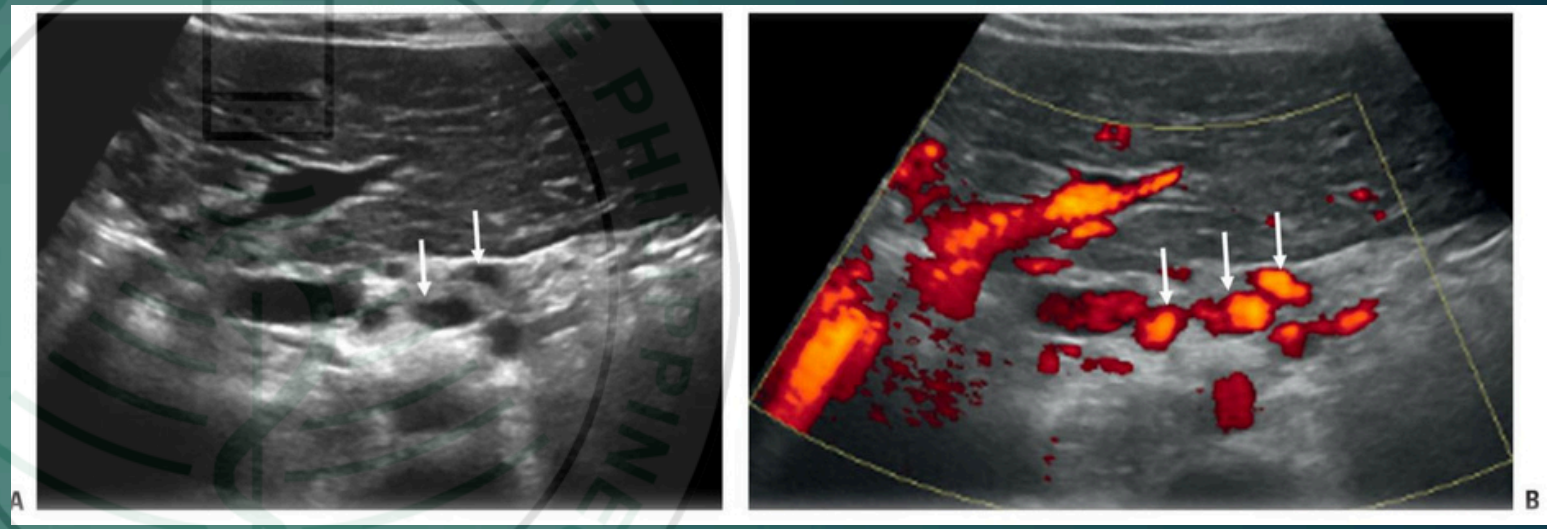


FIG. 4.30 Portal Hypertension. Major sites of portosystemic venous collaterals.

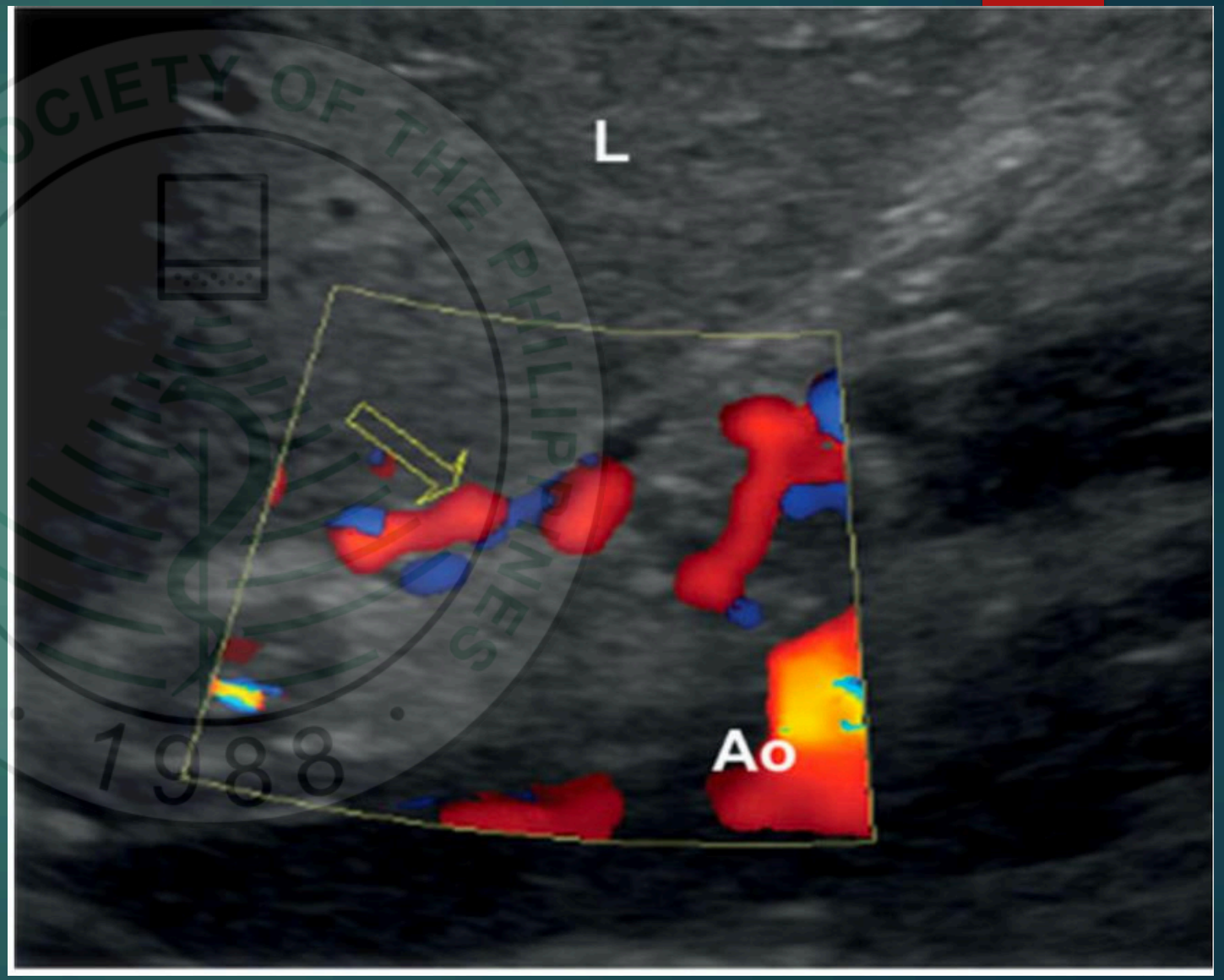
SONOGRAPHIC FINDINGS

Portosystemic venous collaterals - coronary vein



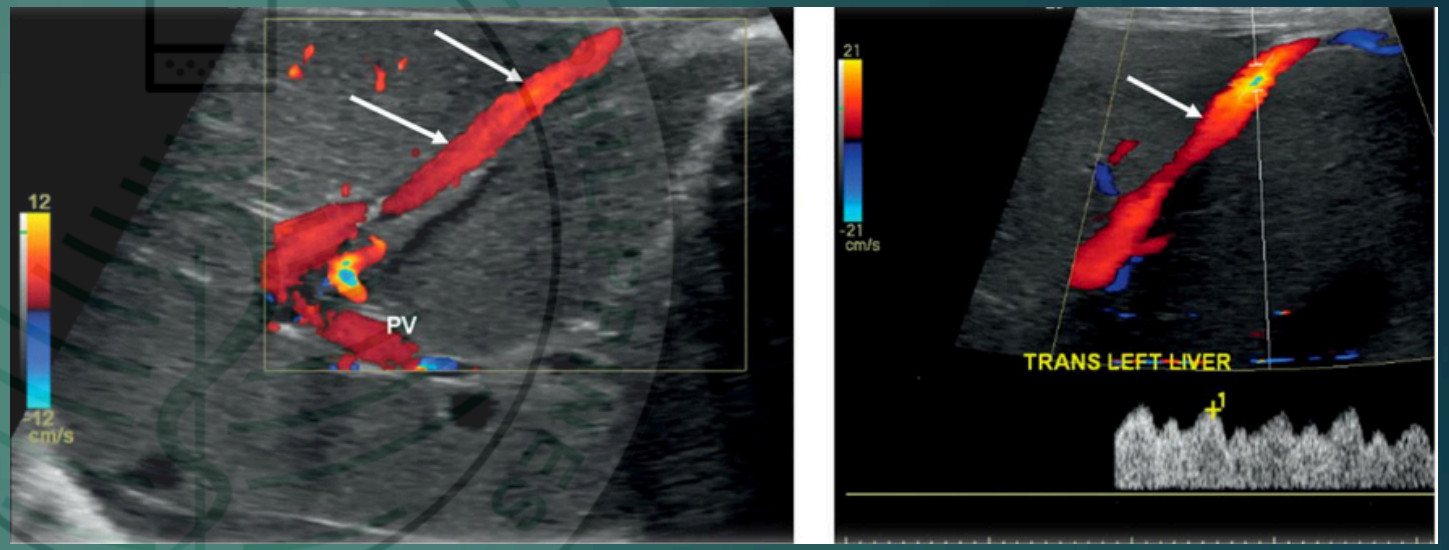
SONOGRAPHIC FINDINGS

Portosystemic venous collaterals - esophageal vein



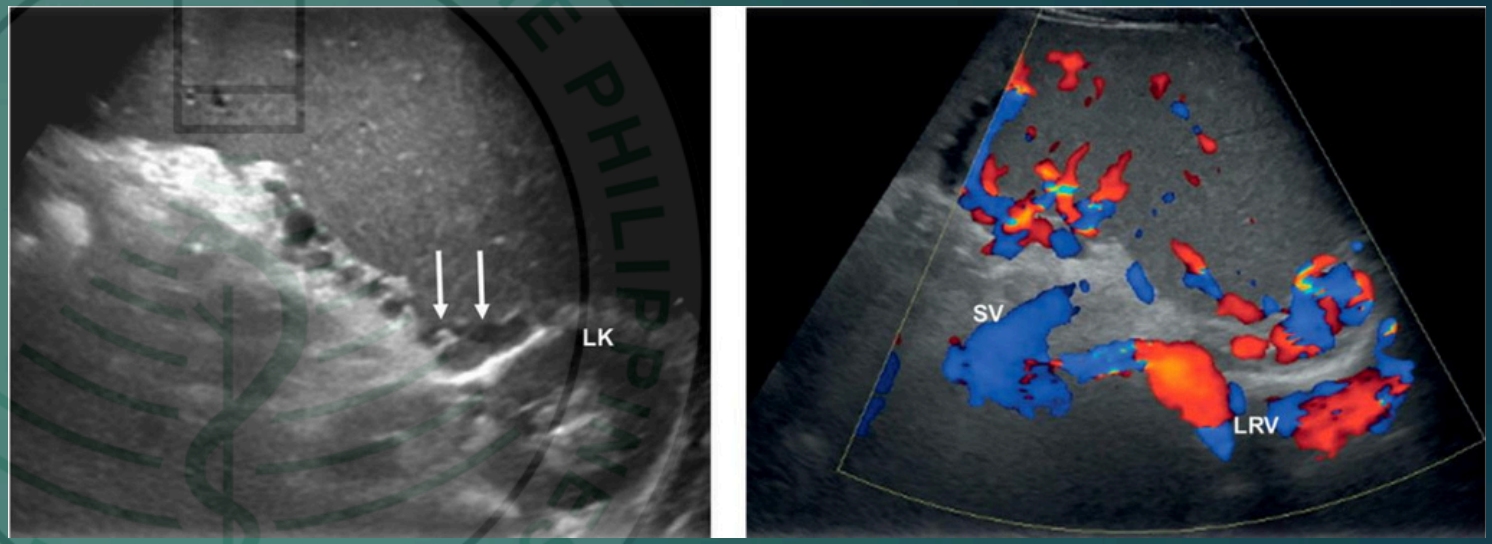
SONOGRAPHIC FINDINGS

Portosystemic venous collaterals - paraumbilical vein



SONOGRAPHIC FINDINGS

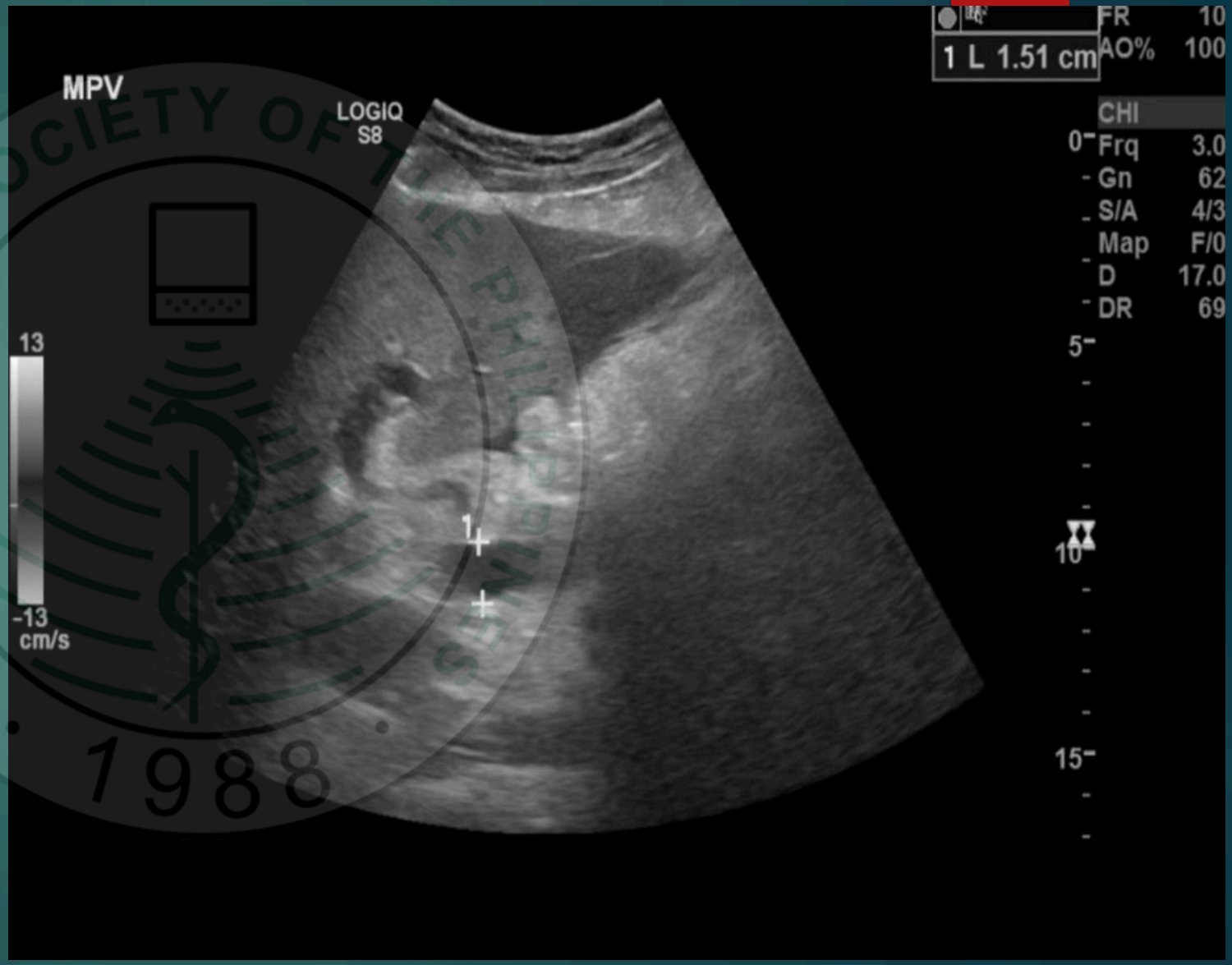
Portosystemic venous collaterals - splenorenal vein



SONOGRAPHIC FINDINGS

> Increased caliber of portal vein >1.3 cm

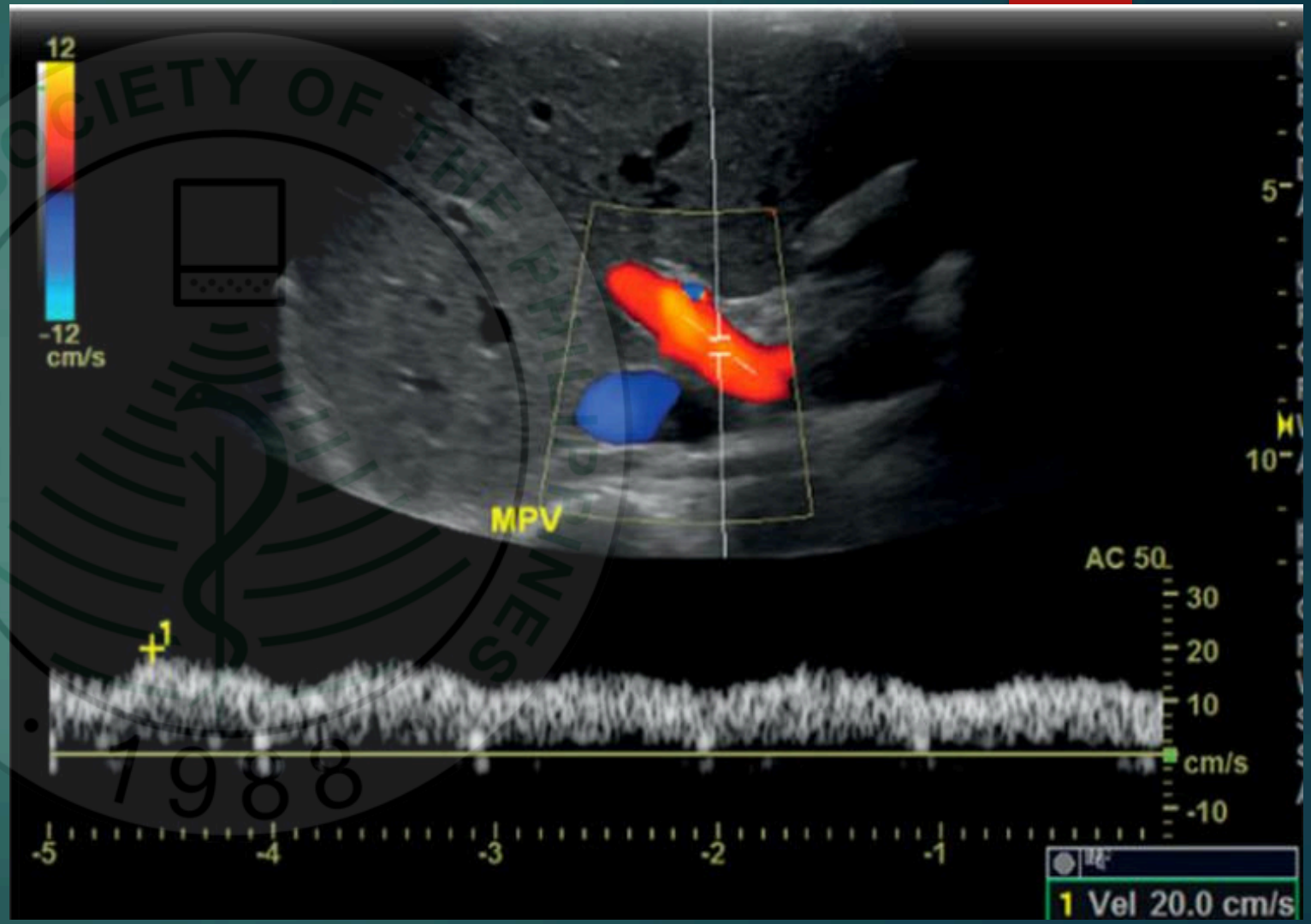
>Increase of less than 20% in the diameter of the portal vein with deep inspiration indicates portal hypertension



DUPLEX DOPPLER SONOGRAPHY

NORMAL FEATURES:

- hepatopetal direction
- velocity 15 to 18 cm/sec
  - increasing postprandially and during in s pi rat io n
  - decreasing after exercise or in the upright position.
  - increase of less than 20% in the diameter of the portal vein wiNORMAth deep inspiration indicates portal hypertension

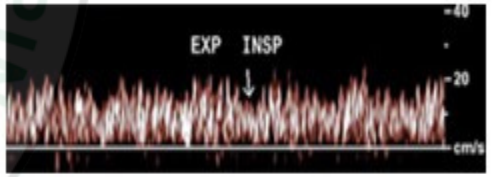


DUPLEX DOPPLER SONOGRAPHY

- IN PORTAL HYPERTENSION:
- monophasic ---> biphasic ---> hepatofugal (away from the liver)

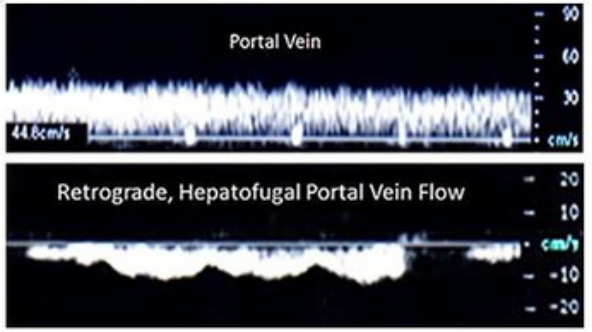
Portal Vein: Hepatopetal Flow

Antegrade flow toward liver is characterized by low velocity mild respiratory and moderate pulsatile variation. This occurs due to hepatic venous flow transmitted back to the portal vein through hepatic sinusoids.[52]



Abnormal portal vein

Portal hypertension causes blunting of the respiratory variation followed by development of bidirectional flow then overt retrograde flow that is away from the liver and termed hepatofugal.

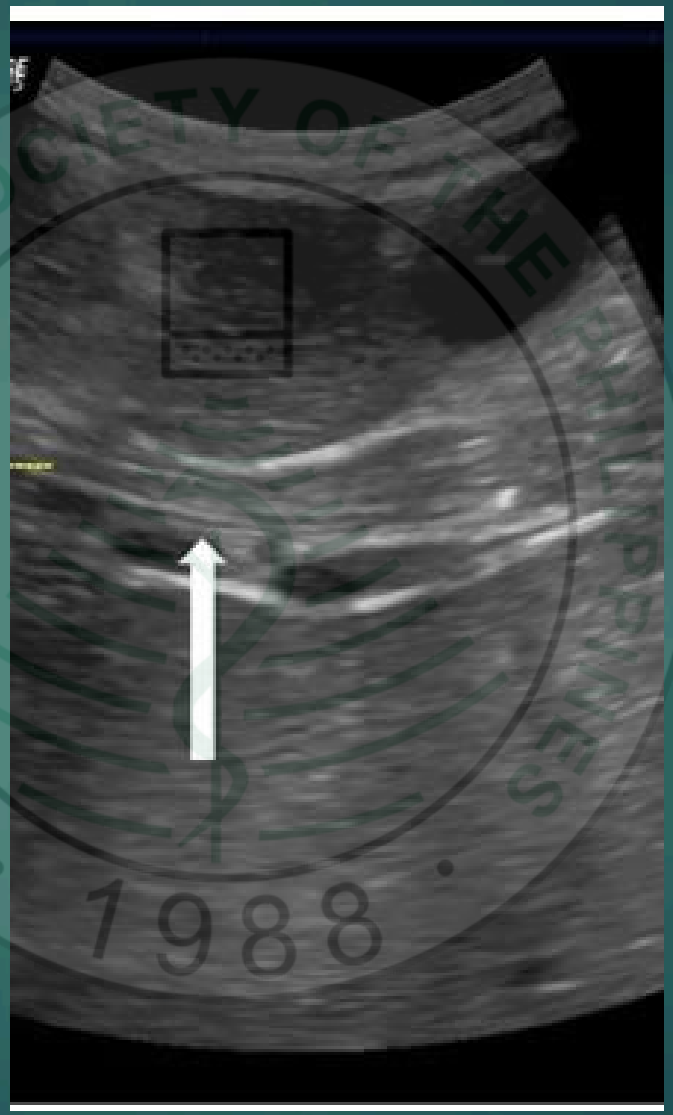


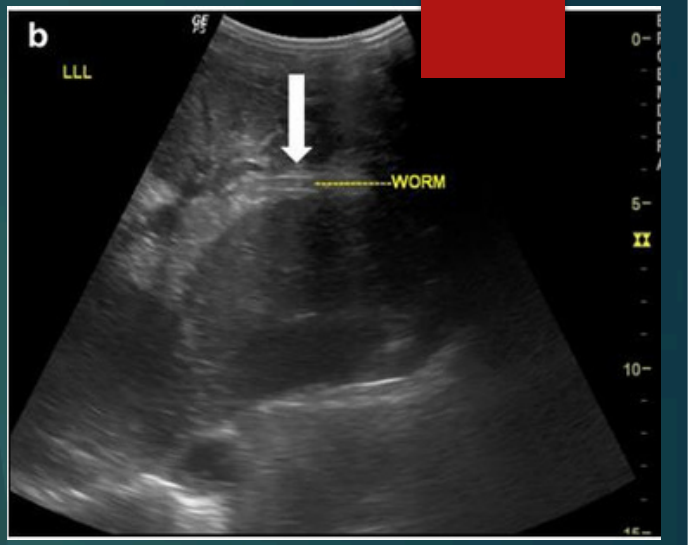
ASCARIASIS

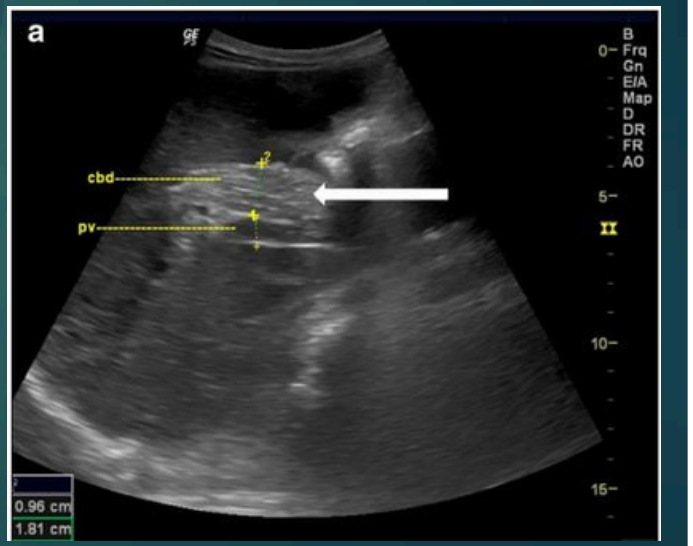
- **Ascariasis lumbricoides**
  - fecal-oral route of transmission
  - most common in child
  - 20 to 30 cm long and up to 6 mm in diameter.
  - It is active within the small bowel and may enter the biliary tree retrogradely through the ampulla of Vater, causing acute biliary obstruction

ASCARIASIS

asingle worm is identified that appears as a tube or as parallel echogenic lines within the bile ducts



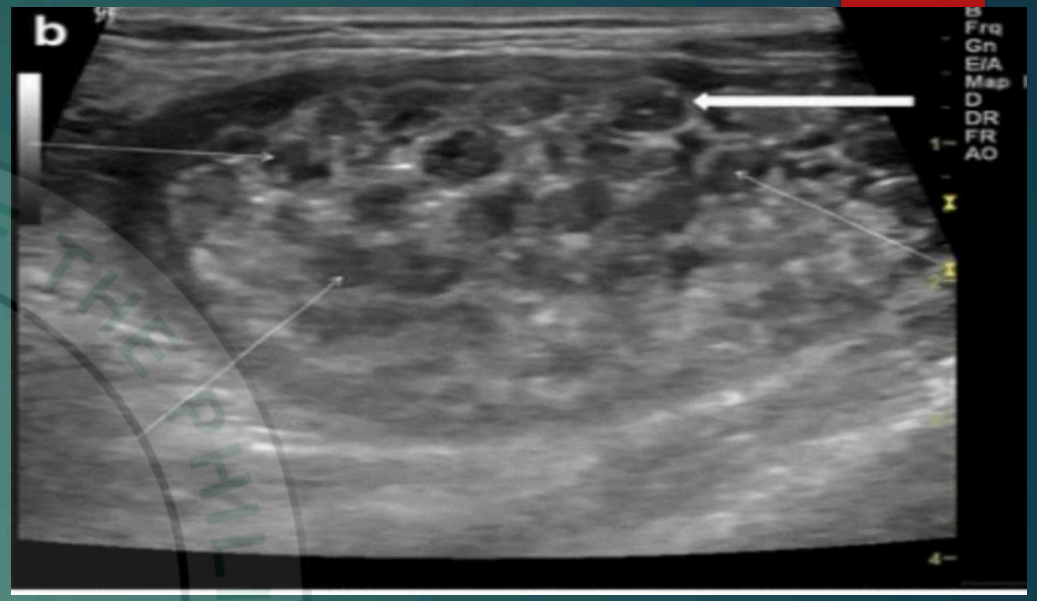




ASCARIASIS

- On the transverse view, the rounded worm surrounded by the duct wall gives a target appearance.

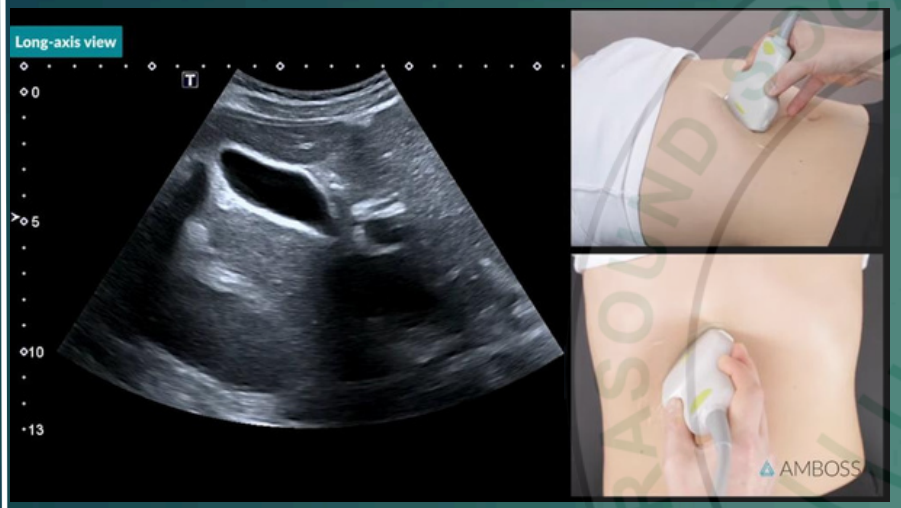
- If the worm is alive and moving during the scan, diagnosis becomes easier.

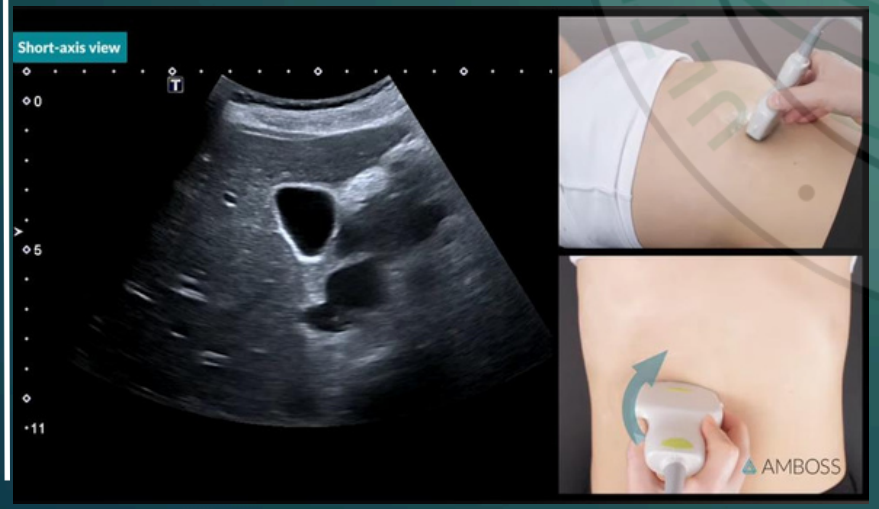


Lyssen, D., Handique, A., Daniala, C. et al. Sonographic images of hepato-pancreatico-biliary and intestinal ascariasis: A pictorial review. Insights Imaging 6, 611–624 (2015). https://doi.org/10.1007/s13244-015-0427-2

GALLBLADDER

THE GALLBLADDER: SONOGRAPHIC TECHNIQUE





-The gallbladder is typically evaluated using routine sagittal and transverse views.

-We can also perform subcostal oblique scans—with the transducer angled so the left edge is more cephalad, pointing toward the right shoulder.

-Importantly, gallbladder evaluation should be done after at least 4 hours of fasting, since eating—especially fatty food—triggers gallbladder contraction.

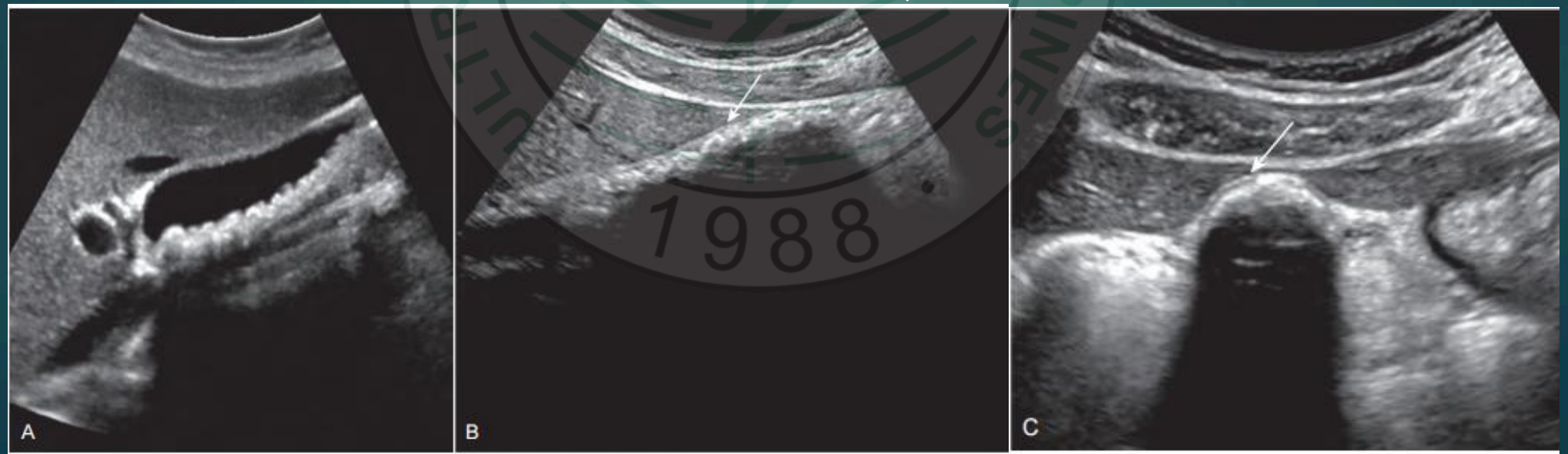
THE GALLBLADDER MAY BE HARD TO VIS UA LIZ E

THIS MAY BE DUE TO:

- PREVIOUS CHOLECYSTECTOMY
- PHYSIOLOGIC CONTRACTION
- CHRONIC CHOLECYSTITIS
- AIR-FILLEDGALLBLADDER/EMPHYSEMATOUS CHOLEYSTITIS
- TUMEFACTIVE SLUDGE
- AGENESIS OF THE GB
- ECTOPIC GB

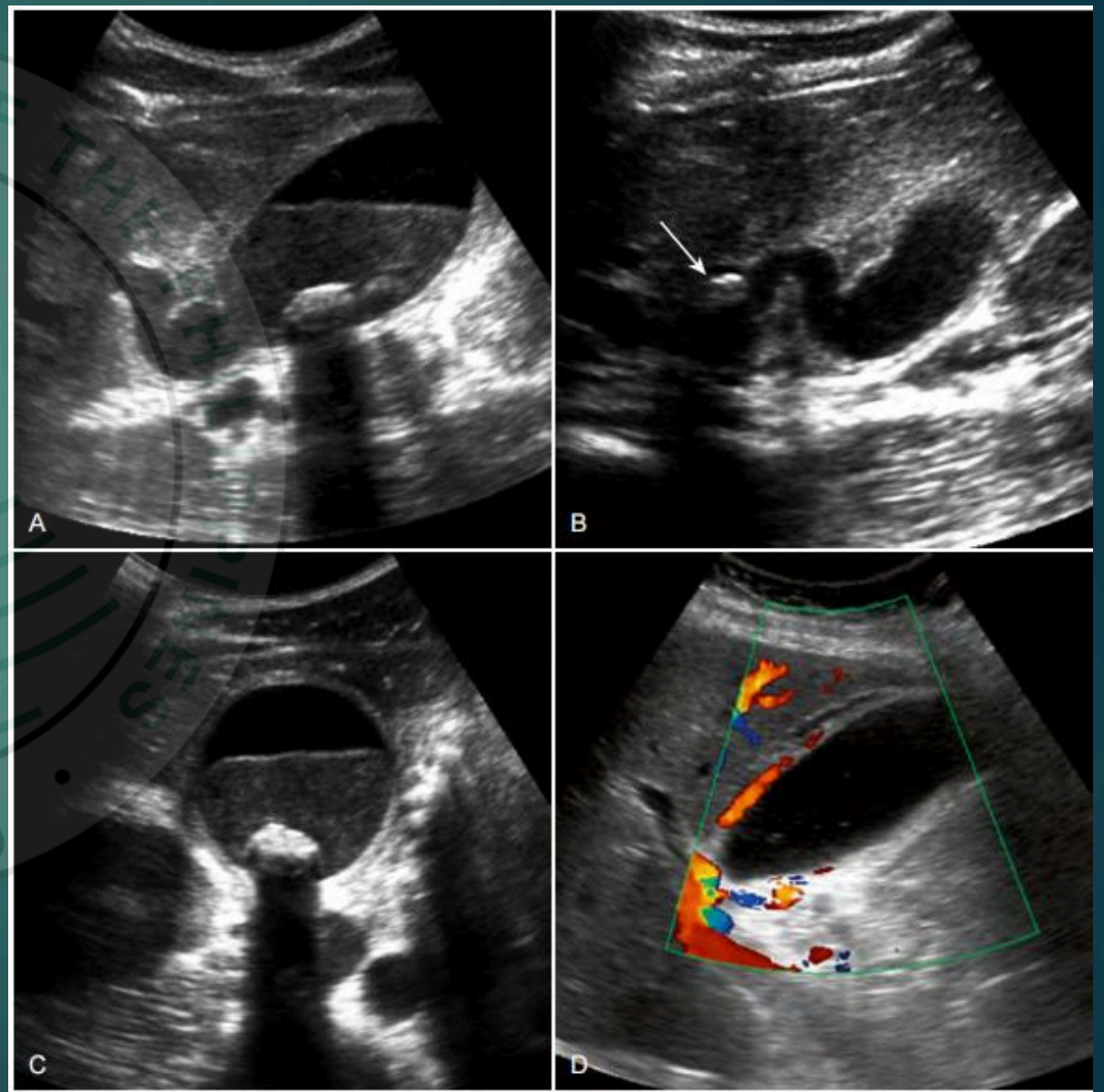
GALLSTONE DISEASE

- MOBILITY IS A KEY FEATURE IN DIFFERENTIATING STONES
- MULTIPLE STONES CAN APPEAR AS A GIANT STONE
- WALL-ECHO-SHADOW COMPLEX/ SIGN
  -GALLBLADDER WALL IS VISUALIZED FIRST IN THE NEAR FIELD
  -THEN THE BRIGHT ECHO OF THE STONE, THEN THE POSTERIOR SHADOWING



ACUTE CHOLECYSTITIS

- SONOLOGRAPHIC FINDINGS INCLUDE
  - -THICKENED GB WALL (>3MM)
  - -DISTENDED GB LUMEN (4 CM OR MO R E)
    - GALLSTONES/ SLUDGE
    - FLUID AROUND THE GB
  - -SONOGRAPHIC MURPHY SIGN
  - -HYPEREMIA OF THE GB WALL ON DOPPLER



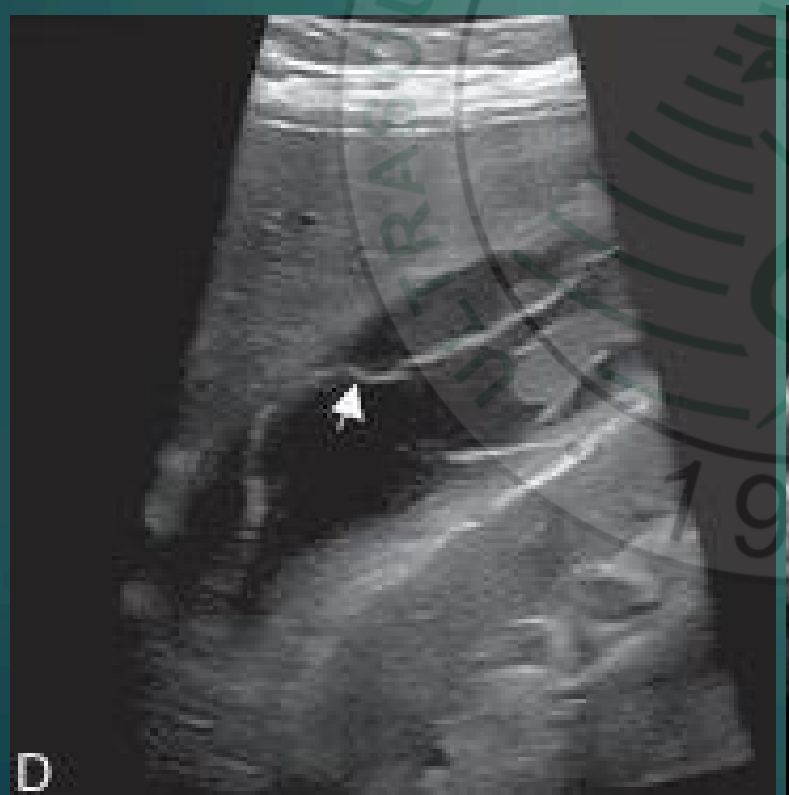
CHOLECYSTITIS COMES IN MULTIPLE FORMS

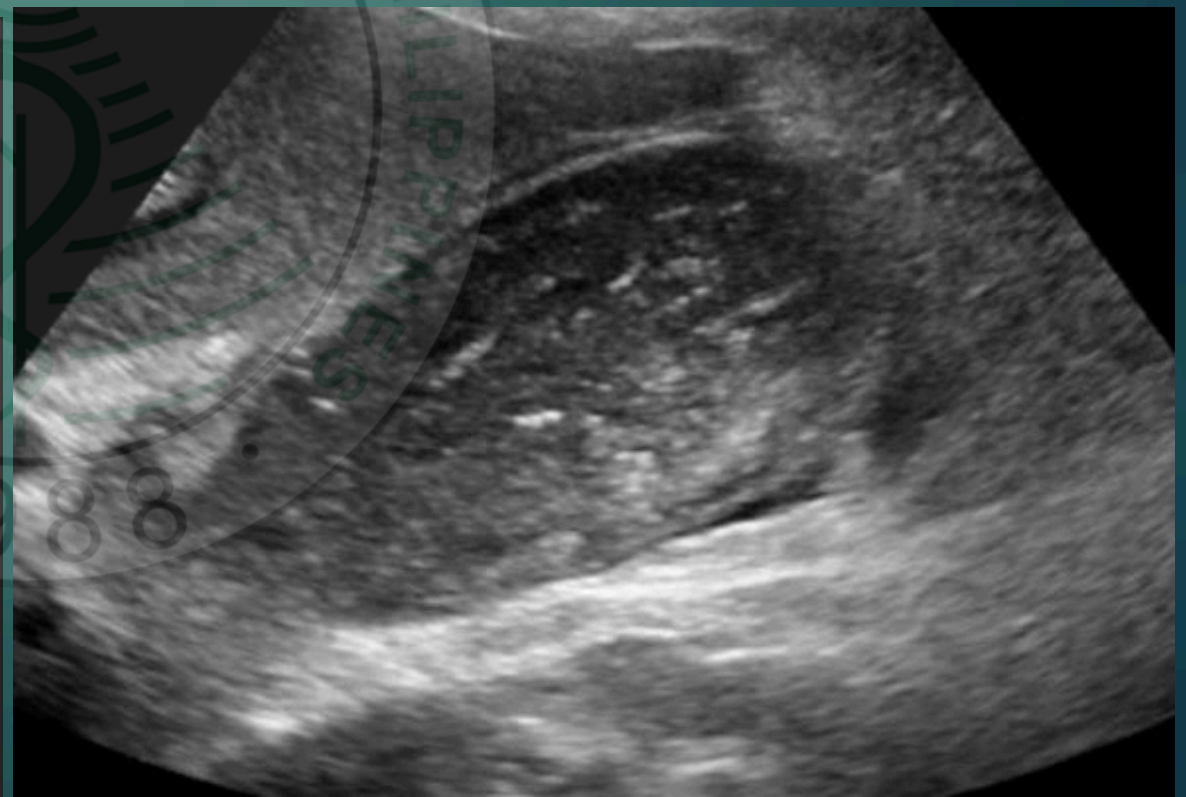
+

- GANGRENOUS CHOLECSYTITIS
- EMPHYSEMATOUS CHOLECYSTITIS
- ACALCULOUS CHOLECYSTITIS
- HEMORRHAGIC CHOLECYSTITIS
- XANTHOGRANULOMATOUS CHOLECYSTITIS

CHOLECYSTITIS COMES IN MULTIPLE FORMS

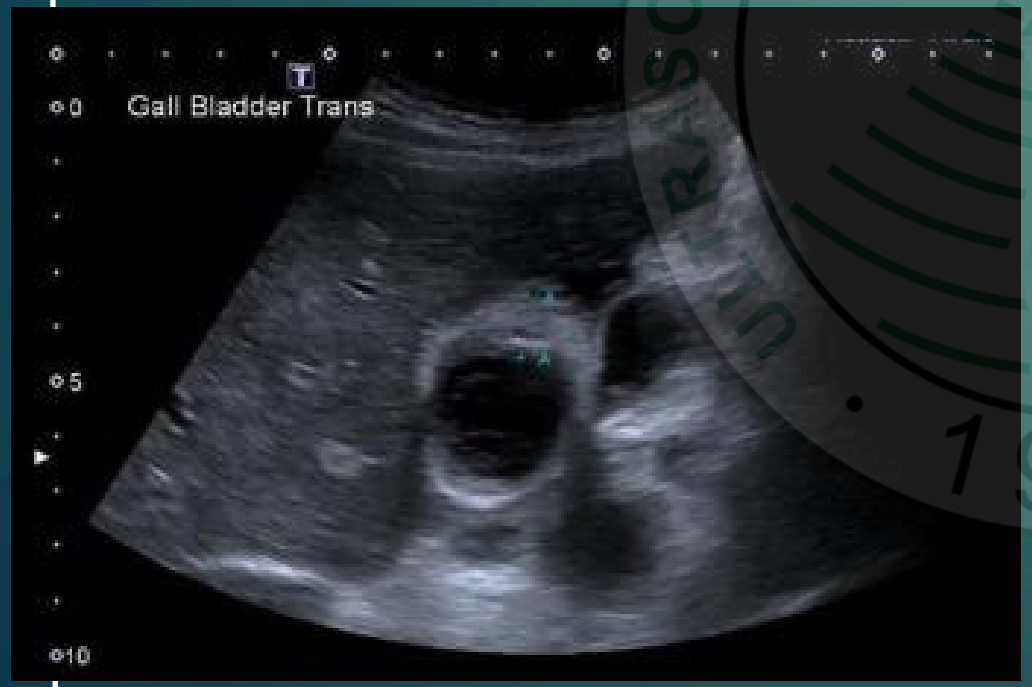
- GANGRENOUS CHOLECYSTITIS
  -NONLAYERINGBANDSOFECHOGENIC TISSUE ARE SEEN

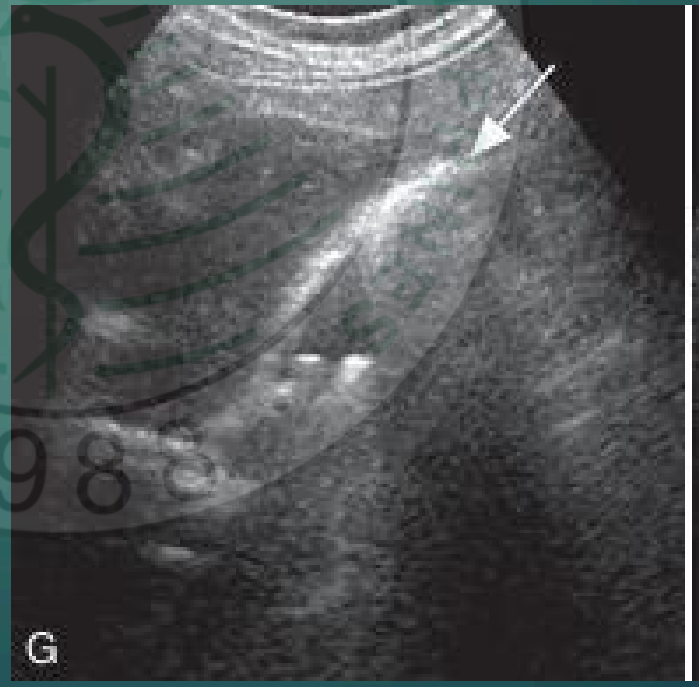


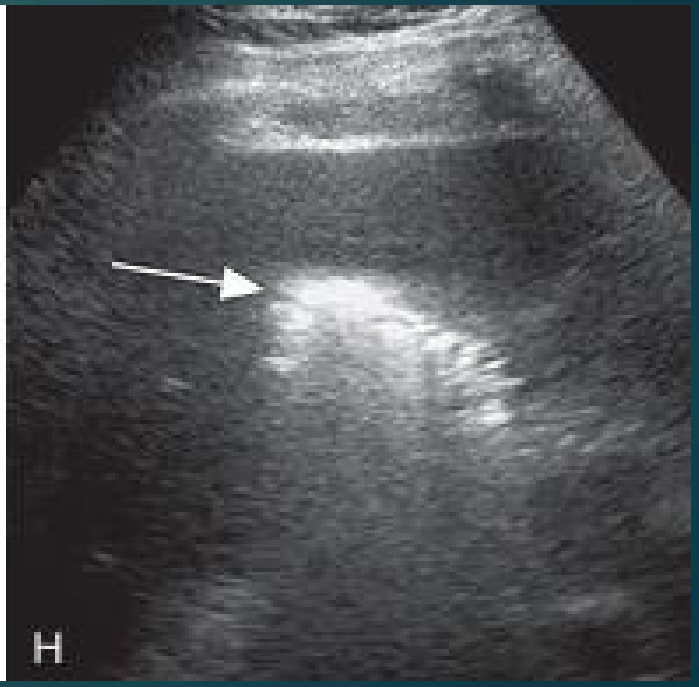


CHOLECYSTITIS COMES IN MULTIPLE FORMS

- EMPHYSEMATOUS CHOLECYSTITIS
  - More common in diabetics, higher incidence of perforation

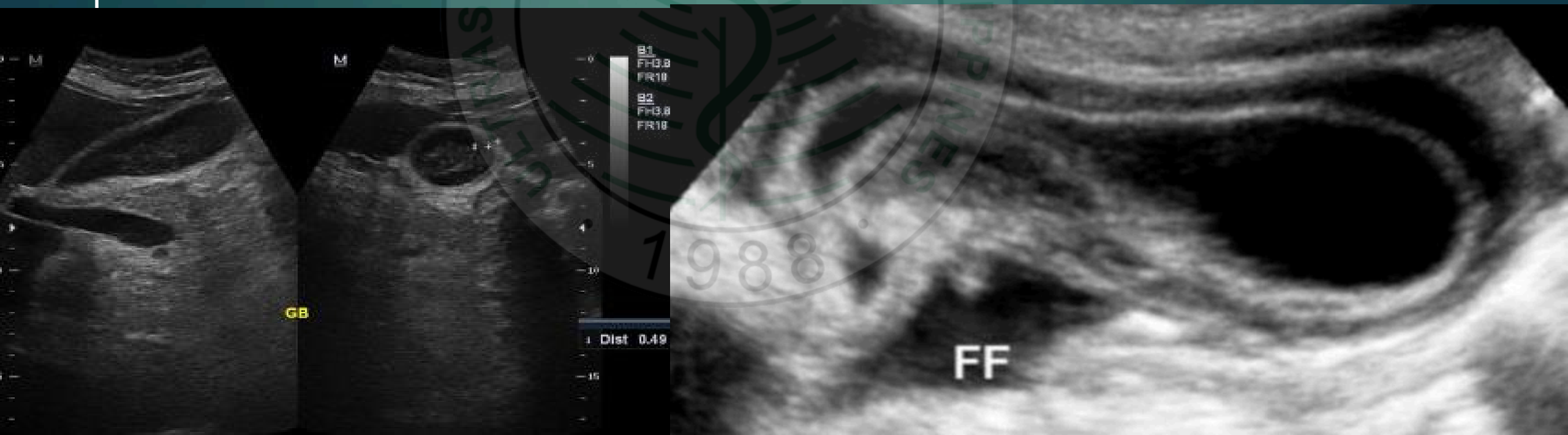






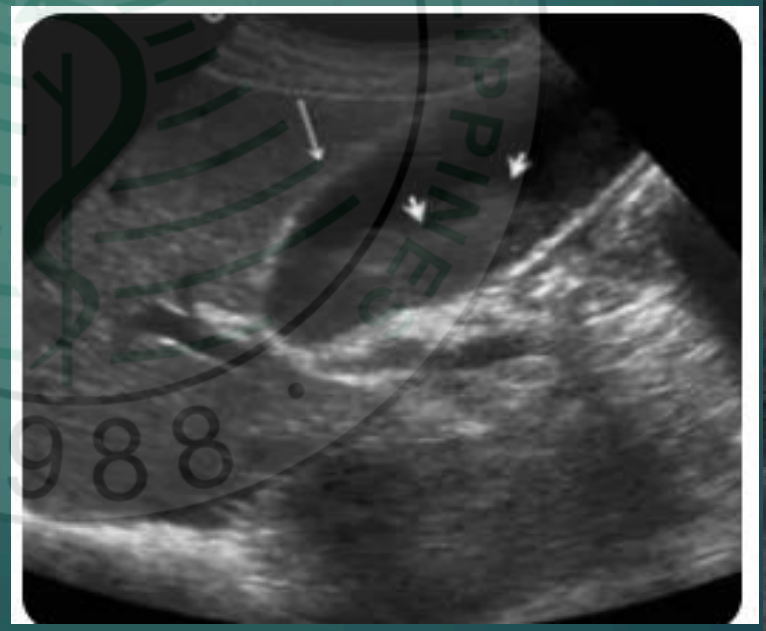
CHOLECYSTITIS COMES IN MULTIPLE FORMS

- ACALCULOUS CHOLECYSTITIS
  - More common in critically ill patients
  - SIGNS OF CHOLECYSTITIS WITHOUT A STONE



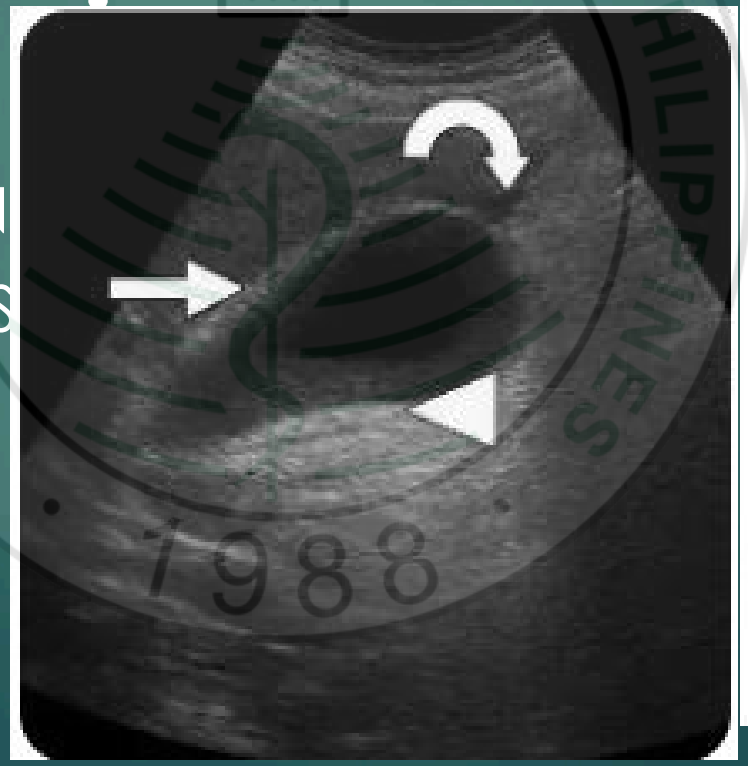
CHOLECYSTITIS COMES IN MULTIPLE FORMS

- HEMORRHAGIC CHOLECYSTITIS
  - Has a variety of causes (anticoagulation, chronic renal or cardiopulmonary disease)
  - RELIES ON THE FAMILIARITYOF THE



CHOLECYSTITIS COMES IN MULTIPLE FORMS

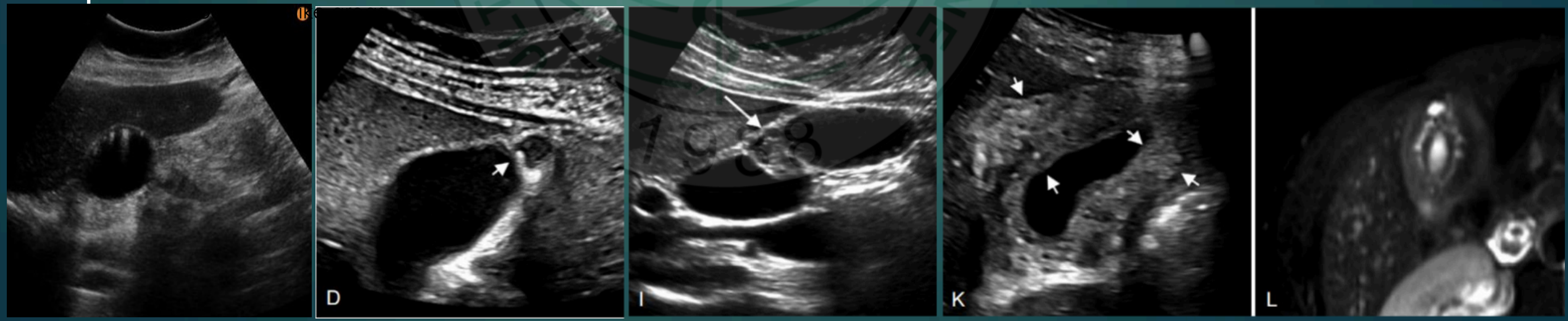
- XANTHOGRANULOMATOUS CHOLECYSTITIS
  - LIPID LADEN MACROPHAGES WITHIN
  - HYPOECHOIC NODULES THE GB WALLS OR BANDS WITHIN THE THICKENED WALL
    -



ADENOMYOMATOSIS

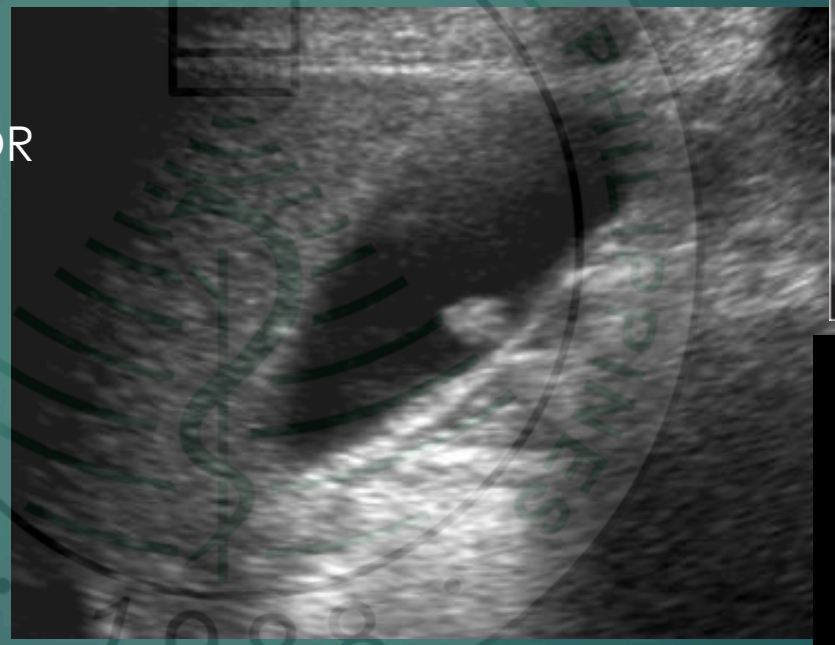
- Benign condition due to exaggerationofthe normal invaginations of the luminal epithelium with proliferation of the smooth muscle
- Thickening of the walls with cystic spaces
- Focal or diffuse
- Comet tail artifacts – most common appearance
- Adenomyomas – 2nd most common

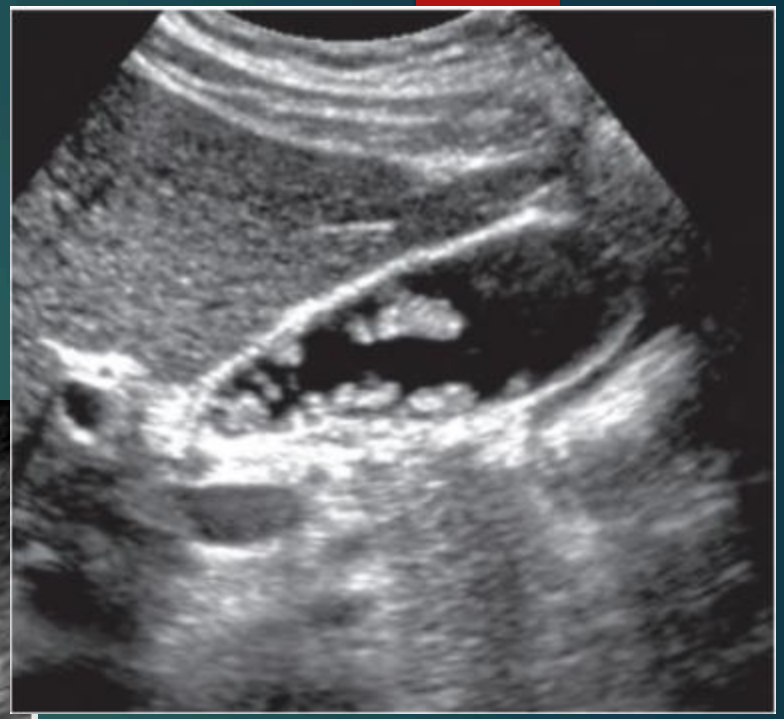
- MOSTDIAGNOSTICFINDING = PRESENCE OF CYSTIC SPACES



GALLBLADDER POLYPS

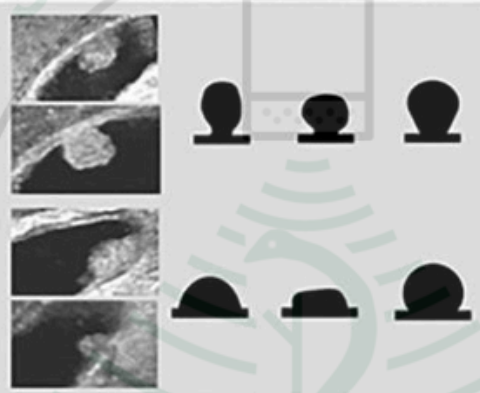
- SIZE: <6-7MM ARE NEGLIGIBLE, 6-9MM MAY BE FOLLOWED UP, >10MM ARE FOR RESECTION
- MULTIPLE POLYPS ARE MORE LIKELY TO BE BENIGN
- SESSILE SHAPE + THICKENED GB WALL = MORE LIKELY MALIGNANT
- HYPOECHOIC FOCI/ APPEARANCE WITHIN THE POLYP + CENTRAL VESSEL = MORE LIKELY MALIGNANT
- APPROXIMATELY HALF OF POLYPOID LESIONS ARE CHOLESTEROL POLYPS
- POLYPS ARE ATTACHED TO THE WALL





CLE | **Figure 1.** Summary of the 2022 SRU gallbladder polyp consensus conference recommendations.

SRU Gallbladder Polyp Consensus Conference Guidelines

Extremely Low Risk[e]	Pedunculated ball-on-the-wall Pedunculated with thin stalk		• ≤ 9 mm[a]: No follow-up • 10-14 mm: Follow-up US at 6, 12, 24 months[b,c] • ≥ 15 mm: Surgical consult
Low Risk[d,e]	Pedunculated with thick or wide stalk Sessile		• ≤ 6 mm: No follow-up • 7-9 mm: Follow-up US at 12 months[b] • 10-14 mm: Follow-up US at 6, 12, 24, 36 months[b] vs surgical consult • ≥ 15 mm: Surgical consult
Indeterminate Risk[e]	Focal wall thickening ≥ 4 mm adjacent to polyp		• ≤ 6 mm: Follow-up US at 6, 12, 24, 36 months[b] vs surgical consult • ≥ 7 mm: Surgical consult

Footnotes:

[a] Polyp size should be rounded to nearest millimeter

[b] On follow-up: Increase of ≥ 4 mm in ≤ 12 months OR reaches threshold size within category - recommend surgical consult
Decrease of ≥ 4 mm - stop following

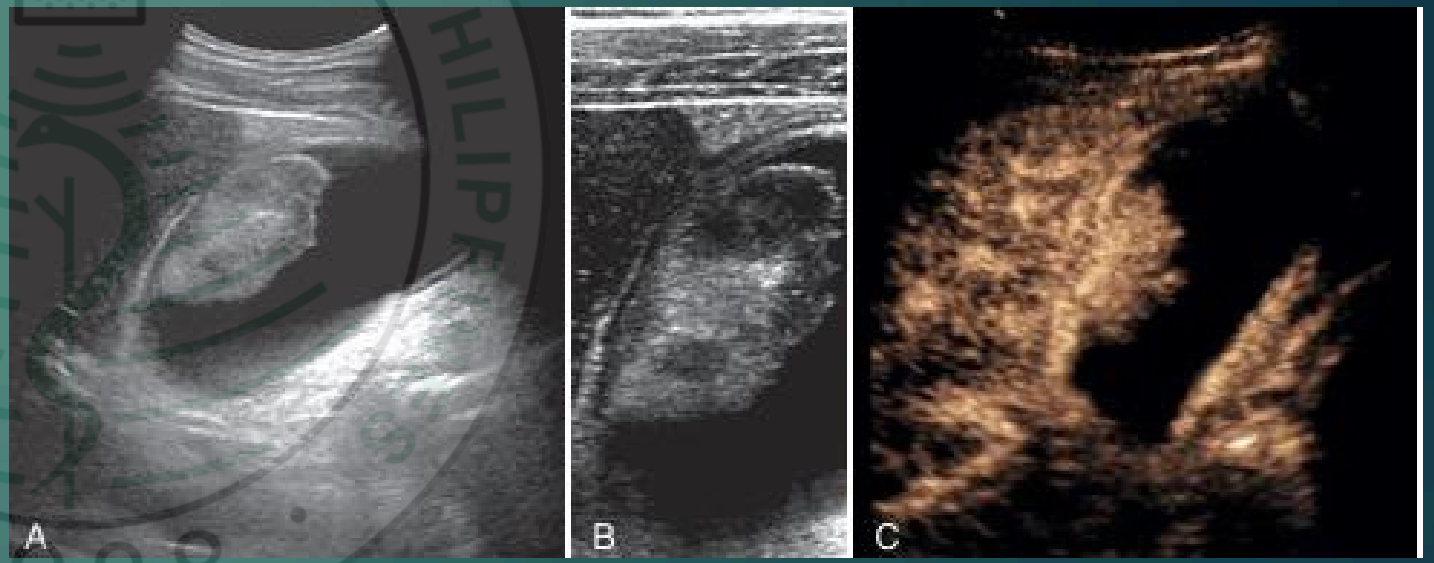
[c] Surgical consult may be an acceptable alternative for polyps 10-14 mm in Extremely Low Risk category

[d] It is optional to consider polyps Low Risk instead of Extremely Low Risk if certain ethnicities are known (North Indian, North/South American Indigenous, local incidence)

[e] If unsure between categories, choose Low Risk category

GALLBLADDER MALIGNANCY

- CARCINOMA IS ASSOCIATED WITH CHRONIC GALLSTONE DISEASE AND RESULTANT + D YSPLA SI A
- 98% ARE ADENOCARCINOMAS
  - Squamous cell CA and metastasis account for the rest
- PATTERNS OF DISEASE
  - MASS ARISING WITHIN THE GB FOSSA
  - OBLITERATING THE GB
  - FOCAL OR DIFFUSE IRREGULAR WALL THICKENING
  - INTRALUMINAL POLYPOID MASS
- CONTIGUOUS LIVER INVASION IS COMMON
  - LYMPHATIC SPREAD IS ALSO COMMON



GALLBLADDER METASTASIS

- MELANOMA CAUSES 50 TO 60% OF METASTASES TO THE GB
- HYPERECHOIC, BROAD-BASED POLYPOID LESIONS, POTENTIALLY MULTIPLE AND MORE THAN 10MM IN DIAMETER
- DIFFUSELY INFILTRATIVE AND CAUSES CONTRACTION OF THE GB

CHOLANGIOCARCINOMA

- UNCOMMON NEOPLASM ARISING FROM ANY PORTION OF THE BILIARY TREE
- FREQUENCY RISES WITH AGE
- PRIMARYSCLEROSING CHOLANGITISISTHEMOSTCOMMONRISK FACTOR IN THE WEST
- RECURRENT BILIARY INFECTION AND GB STONE DISEASE ARE ALSO RISKFACTORS
  -

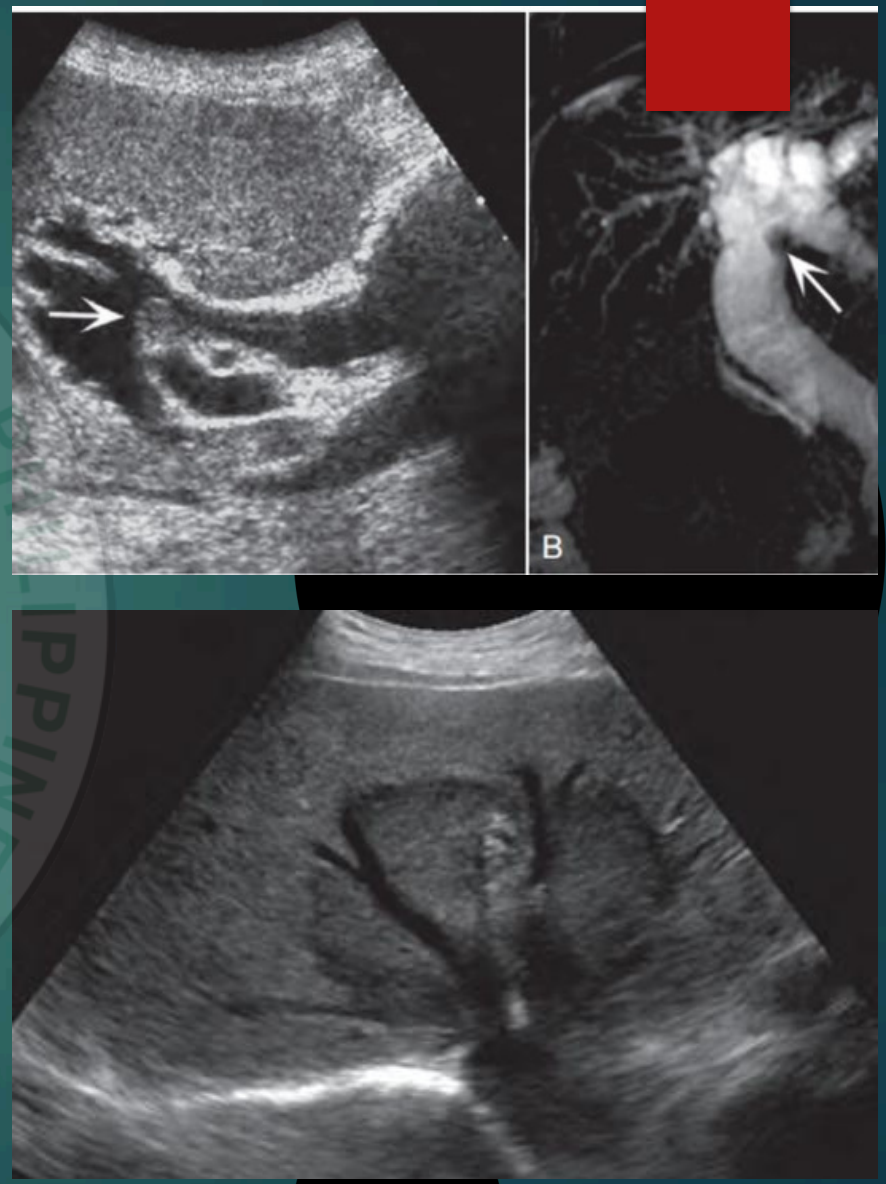
CHOLANGIOCARCINOMA

- CLASSIFIED BASED ON LOCATION
  - INTRAHEPATIC
  - HILAR
  - DISTAL

- 90% ARE ADENOCARCINOMAS
  - SQUAMOUSCARCINOMA–NEXT MOST COMMON
    -

CHOLANGIOCARCINOMA

- INTRAHEPATIC – ALSO CALLED PERIPHERAL CHOLANGIOCARCINOMA
- ARISE FROM SECOND ORDER OR HIGHER ORDER BRANCHES OF THE BILIARY TREE
- MOST COMMON MANIFESTATION IS A LARGE HEPATIC MASS
  - MUCH HIGHER INCIDENCE OF DUCTAL OB STR UCT ION
- MAY ALSO APPEAR AS PURELY INTRADUCTAL MASS
  - INTRADUCTALINTRAHEPATIC CH OL AN G IO CA RCI N OMA
  - DISTEND THE DUCTS WITH MUCIN
- ONLY MAKE UP ~10% OF CASES

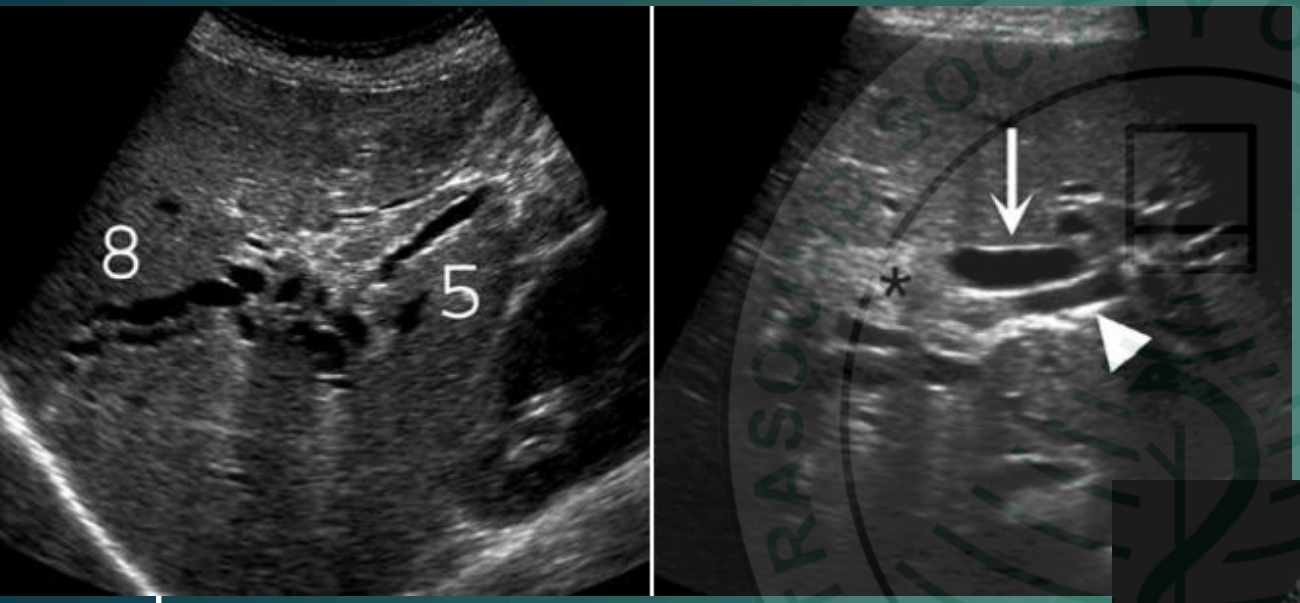


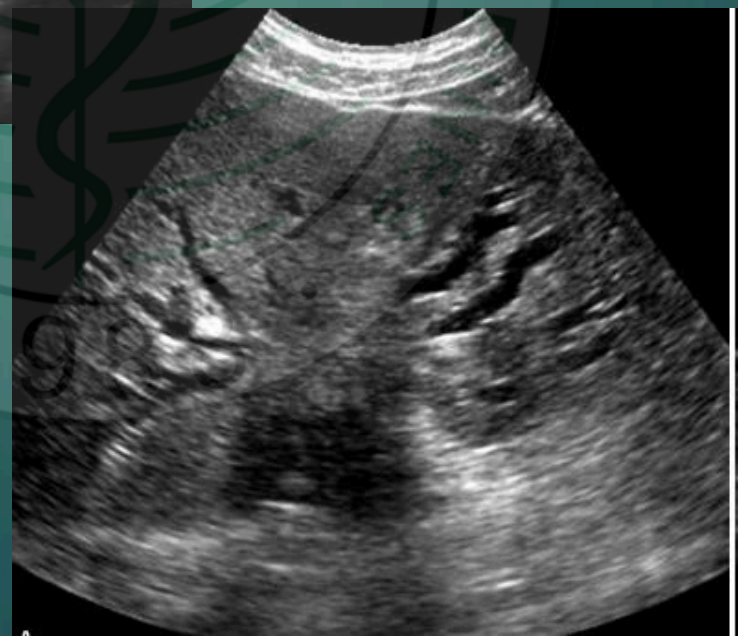
CHOLANGIOCARCINOMA

- HILAR – ALSO CALLED KLATSKIN CHOLANGIOCARCINOMA
- MAKES UP ~60% OF CASES
- PRESENT WITH JAUNDICE, PRURITIUS, AND ELEVATED LIVER ENZYMES
- BEGINS IN THE RIGHT OR LEFT BILE DUCT
  - EXTENDING PROXIMALLY TO THE HIGHER ORDER BRANCHES
  - EXTENDINGDISTALLYINTOTHE COMMON HEPATICDUCT AND CONTRALATERALDUCTS(MAY INVOLVE THEPORTALVEIN AND ARTERIES)
- CHRONIC DUCTAL OBSTRUCTION LEADS TO ATROPHY OF THE LOBE
- CLASSIC APPEARANCE
  - DILATATION OF THE HIGHER ORDER INTRAHEPATIC BILE DUCTS NONUNION OF THE RIGHT AND LEFT HEPATIC BILE DUCTS
  -

CHOLANGIOCARCINOMA

- HILAR – ALSOCALLEDKLATSKINCHOLANGIOCARCINOMA





CHOLANGIOCARCINOMA

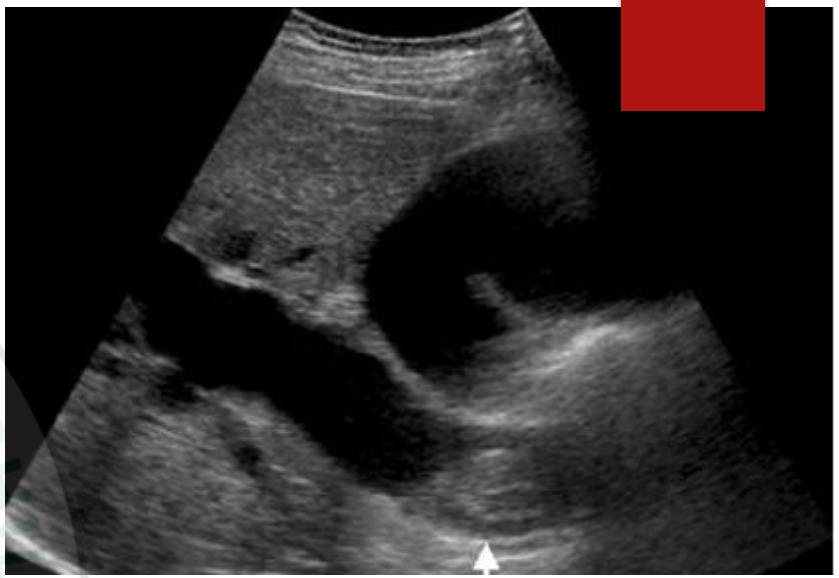
- DI ST AL–CLINICALLYINDISTINGUISHABLEFROM HILAR FORMS
  - PROGRESSIVE JAUNDICEIN 75TO 90%OFPATIENTS
- MAKES UP ~30% OF CASES
- MAY EXTEND PROXIMALLY INTO THE CYSTIC AND RIGHT AND LEFT HEPATIC DUCTS
- MAY ALSO EXTEND BEYOND THE DUCT WALL
- SPREAD TO THE ADJACENT LYMPH NODES IS COMMON
- ITSAPPEARANCEIS VARIABLE
  - POLYPOID TUMORS CAN APPEAR AS A WELL-DEFINED, DUCT EXPANDING MASS
  - NODULAR-SCLEROSINGTUMORAPPEARS ASA FOCAL, IRREGULAR DUCTAL CONSTRICTION AND WALL THICKENING

CHOLANGIOCARCINOMA

- DISTAL – CLINICALLY INDISTINGUISHABLE FROM HILAR F ORM S
- MAY EXTEND PROXIMALLY INTO THE CYSTIC AND RIGHT AND LEFT HEPATIC DUCTS
- MAY ALSO EXTEND BEYOND THE DUCT WALL
- SPREAD TO THE ADJACENT LYMPH NODES IS COMMON

ITS APPEARANCE IS VARIABLE

- POLYPOID TUMORS CAN APPEAR AS A WELL-DEFINED, DUCT EXPANDING MA
- NODULAR-SCLEROSING TUMOR APPEARS AS A FOCAL, IRREGULAR DUCTAL CONSTRICTION AND WALL THICKENING



SPECTRUM OF FINDINGS IN PANCREATITIS

ACUTE PANCREATITIS

- International Symposium on Acute Pancreatitis (Atlanta, 1992)
  - An acute inflammatory processof thepancreas with variable involvement of other regional tissues or remote organ systems associated with raised pancreatic enzyme levelsin blood and/or urine.”
- Mild acute pancreatitis: resolves spontaneously
- Acute interstitial/edematous pancreatitis: enlarged and congested gland
- Necrotizing pancreatitis

- PREDOMINANT CAUSES OF ACUTE PANCREATITIS
  - G alls tones
  - Alcohol abuse

- All patients whopresent with their first episode of acute pancreatitis should have sonography to evaluate the biliary tree for g alls ton es

- Other causes of pancreatitis
  - Biliary sludge (microlithiasis)
  - Neoplasm, infection, pancreas divisum, toxins, drugs, and genetic, traumatic, and iatrogenic (endoscopy, postoperative) factors

APPROACH TO IMAGING

2 most useful imaging modalities in patients with acute pancreatitis:

-
  - Abdominal sonography
  - CECT
- Secondary diagnostic and therapeutic studies:
  - MRI, MRCP, ERCP, and endoscopic ultrasound
- Ultrasound: detect gallstones and bile duct obstruction
- CECT
  - Indicated early in the clinical course of patients with severe pancreatitis, mainly to diagnose pancreatic necrosis
  - when seeking delayed complications of acute pancreatitis (most accurate ex ami nati on)

Imaging in Acute Pancreatitis

ROLE OF ULTRASOUND

Detect gallstones as a cause of acute pancreatitis
Detect bile duct dilation and obstruction
Diagnose unsuspected acute pancreatitis or confirm diagnosis of acute pancreatitis
Guide aspiration and drainage

ROLE OF COMPUTED TOMOGRAPHY

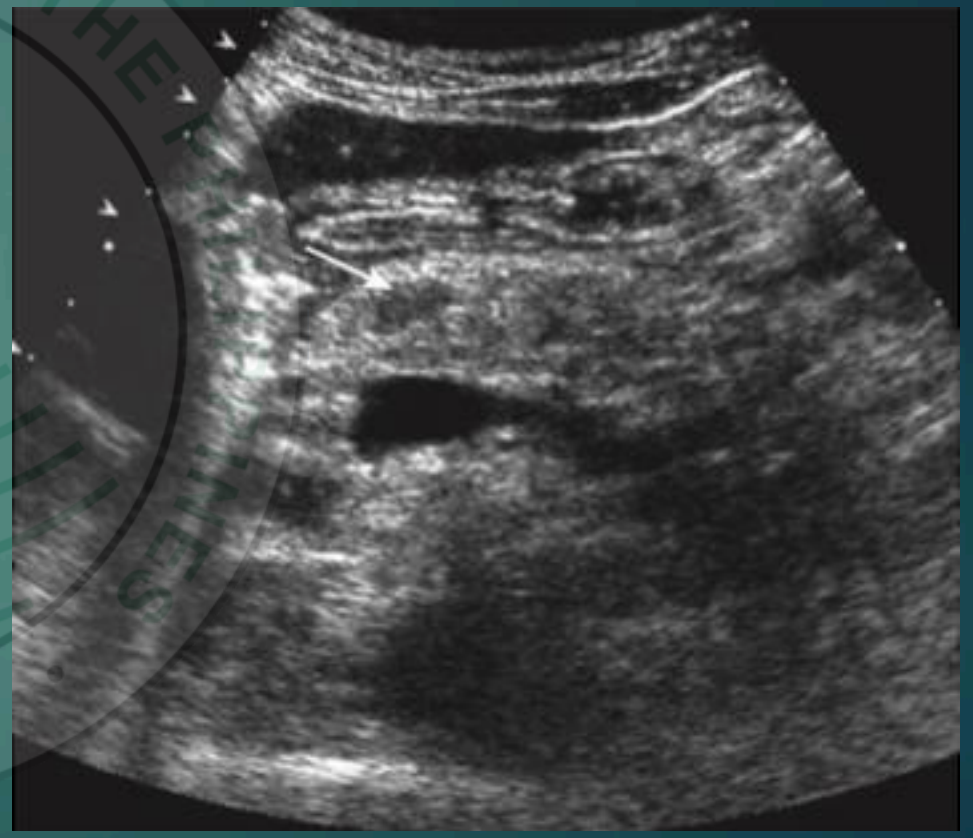
Detect pancreatic necrosis (patients with suspected severe pancreatitis)
Detect complications of acute pancreatitis
Diagnose unsuspected acute pancreatitis or confirm diagnosis of acute pancreatitis
Diagnose conditions mimicking acute pancreatitis, including gastrointestinal ischemia, ulceration, or perforation and ruptured abdominal aortic aneurysm
Guide aspiration and drainage

- MRCP and MRI: often reserved for patients in whom CT or ultrasound does not provide adequate information to guide management and for those who have a contraindication to CECT
- Endoscopic ultrasound: more sensitive than abdominal ultrasound for the detection of common bile duct stones and can be useful in suspected gallstone pancreatitis
- ERCP
  - oFormerly botha diagnosticand a therapeutic modality, is now usually reserved for therapy becauseof its complications and expense

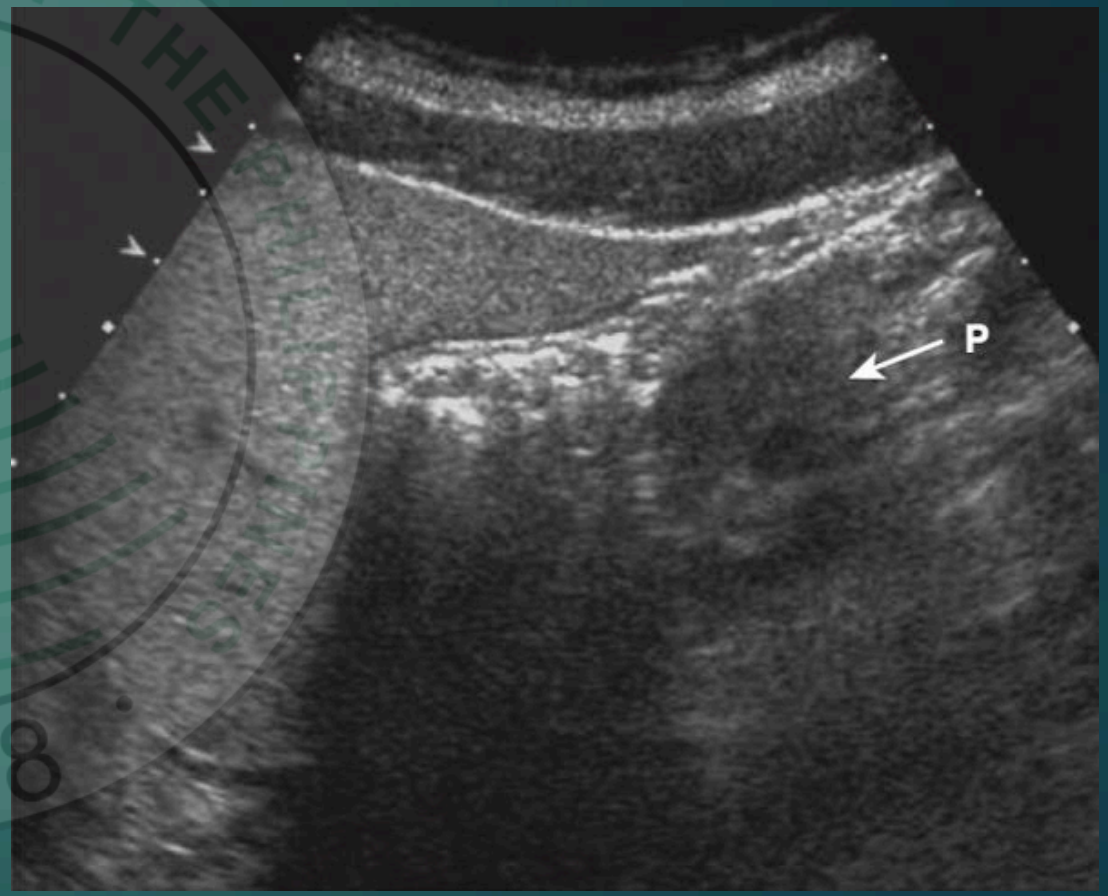
ULTRASOUND FINDINGS

- Focus of ultrasound: evaluation of the gallbladder and bile ducts in patients with acute pancreatitis
- Serum amylase and serum lipase: sensitivity and specificity of 90-95% in diagnosing acute p anc re a titi s

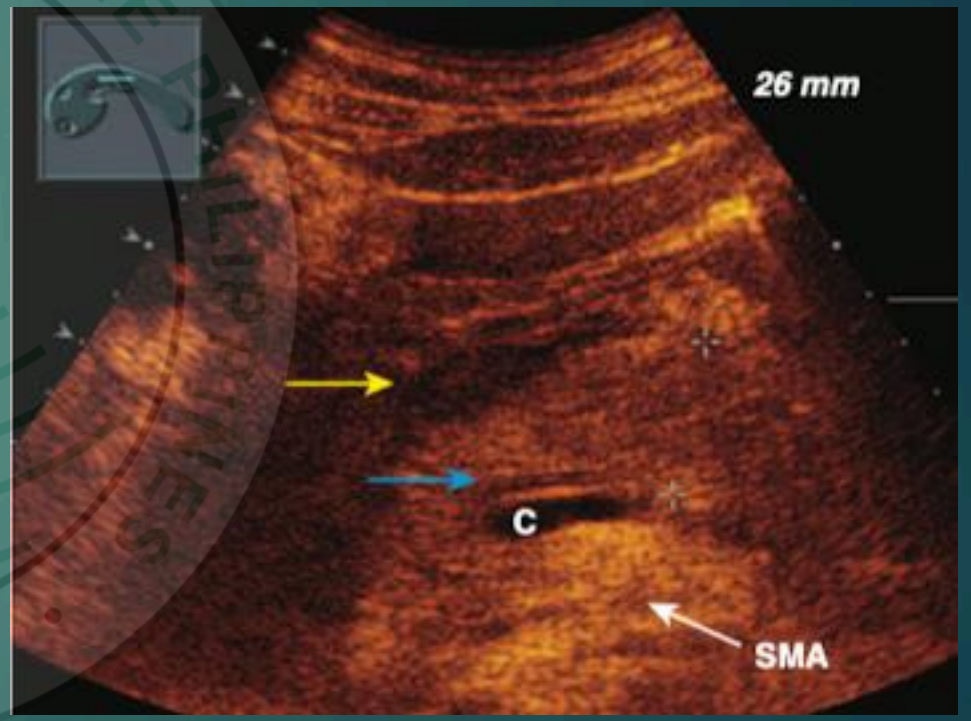
  - Pancreatic echogenicity typically decreases
  - because of interstitial edema (44%) – focal or diffuse In some patients, echogenicity is
  - normal In rare cases, echogenicity may
  - increase
  because of hemorrhage, necrosis, or fat sa poni fi c ati on



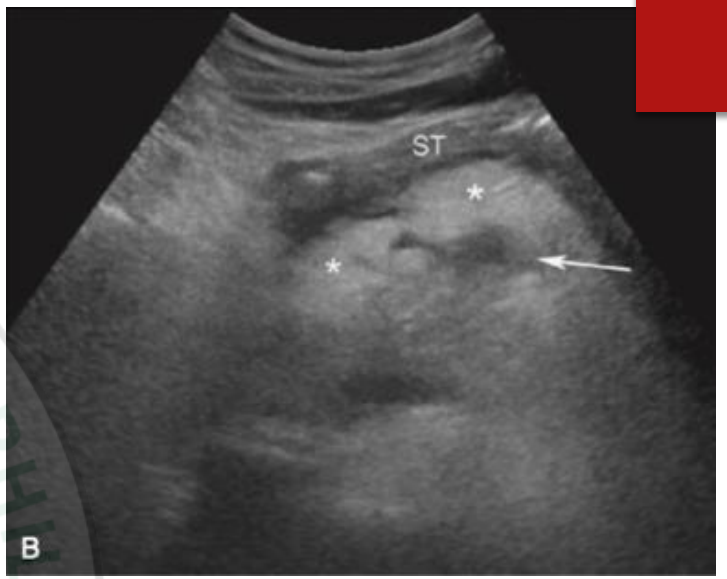
- Pseudopancreatitis: when there is fatty infiltration of the liver, the normal pancreas may appear hypoechoic

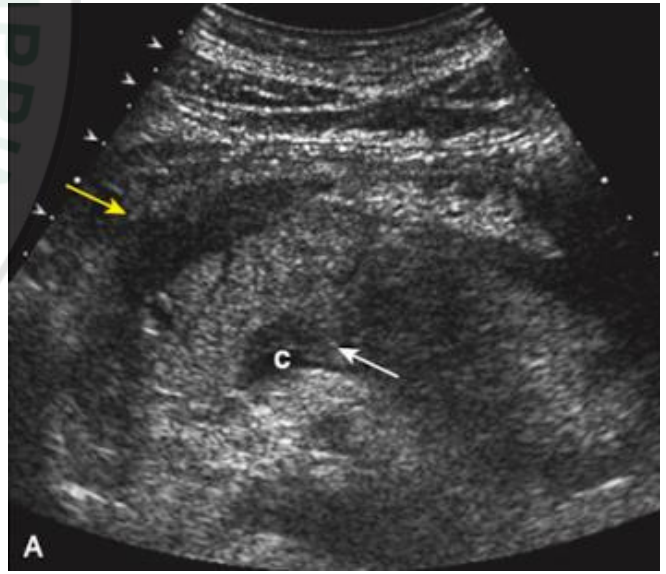


- Enlargement of the pancreas is almost universal
- In 1995 Guerra et al.
  - Thickness ofthe bodyof the normal pancreas in 261 adults was 10.1 mm (±3.8 mm; range, 4-23 mm)
- In2005, Finstad et al.
  - Seriesof patients with acute pancreatitis,the mean AP measurement of the pancreatic body at the SMA level was 21.1 mm (±6.4 mm; range, 12-45 mm)
- 22mm(meanplus3standarddeviations)astheupperlimitofnormal pancreatic thickness

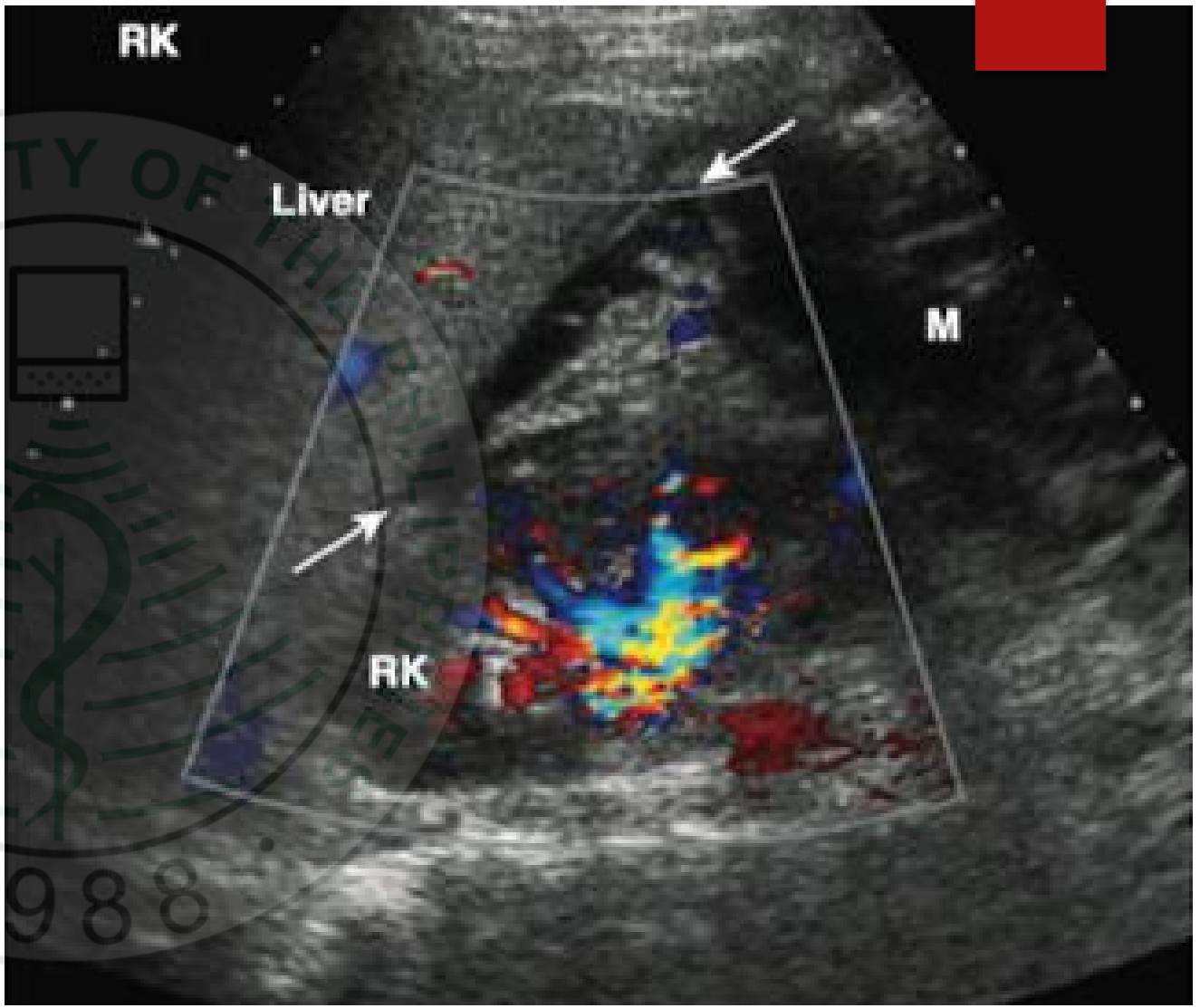


- Pancreatitis-associated inflammation
  - Least subjective,mostcommon,andthus most useful finding
- Extrapancreatic inflammatory changes may be detected even when the pancreatic contour is normal and the pancreas is not obviously enlarged

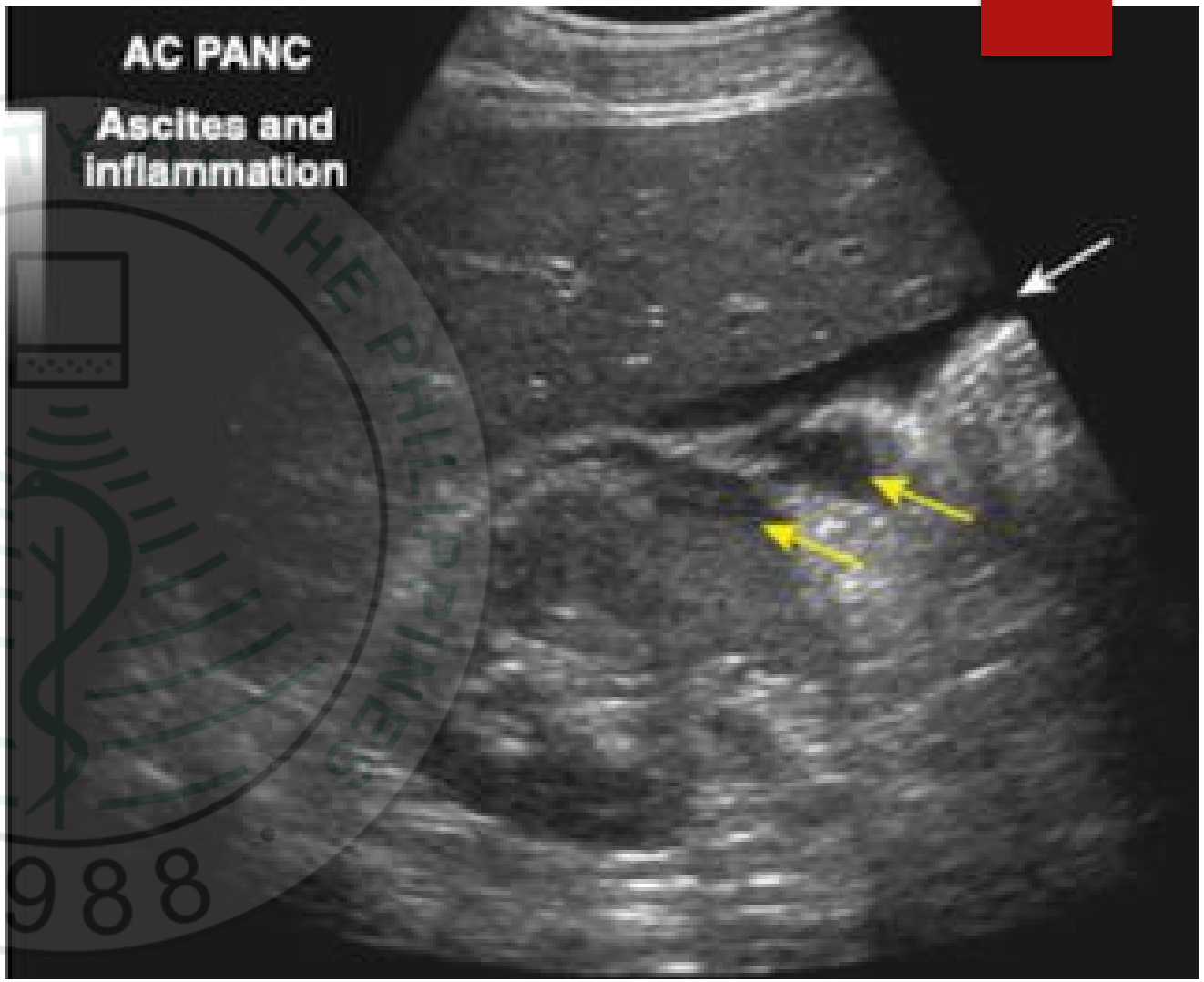




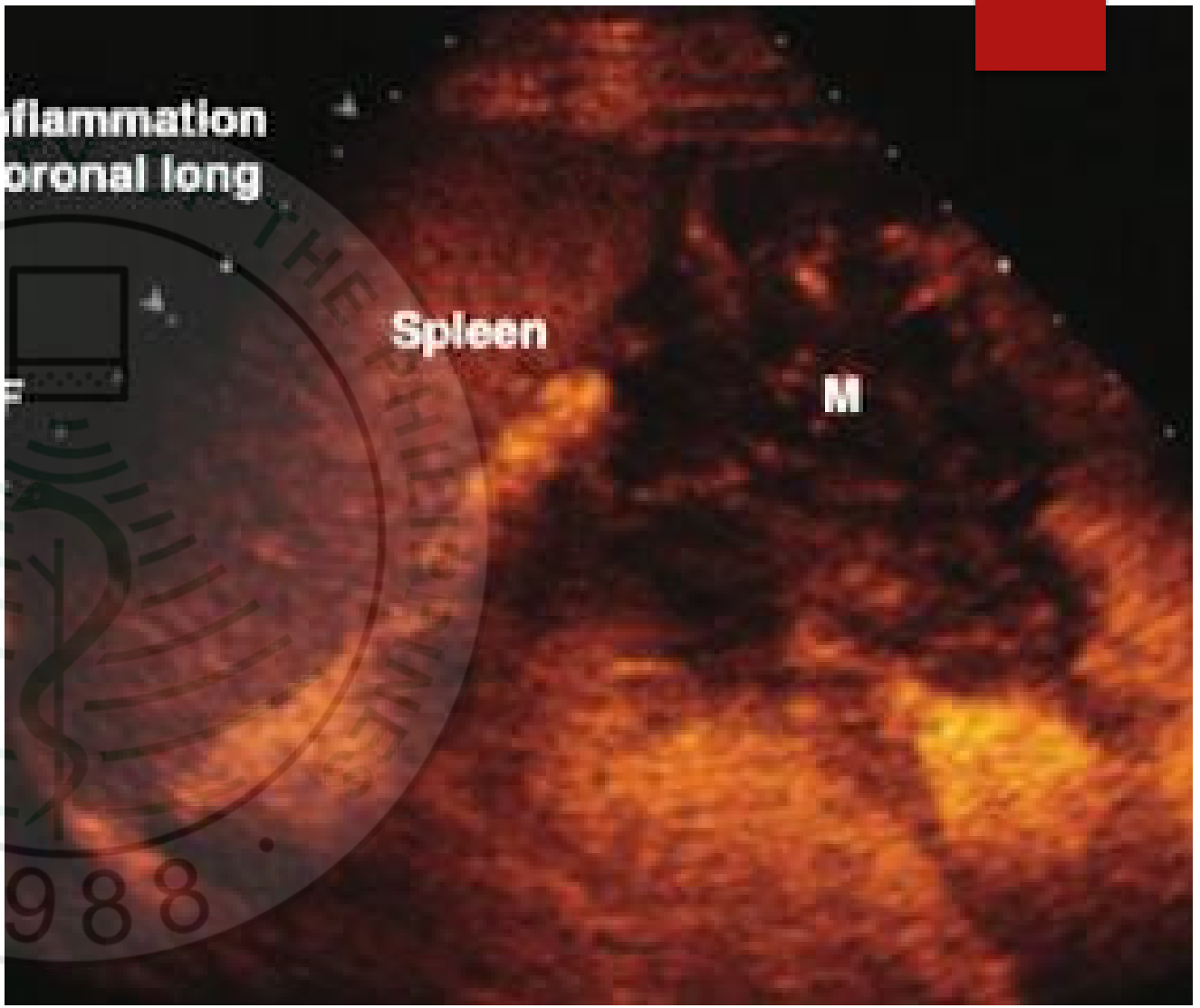
- It may be difficult or impossible to distinguish inflammation from fluid
  - Fluid collections: often have convex margins, thicker and more localized, may cause a mass effect, and sometimes have through transmission of sound
- Inflammation is most often seen ventral and adjacent to the pancreas in the:
  - Prepancreatic
    r etr op er it oneu m
  - Right and let anterior pararenal spaces
    Perirenal spaces
  - Transverse mesocolon



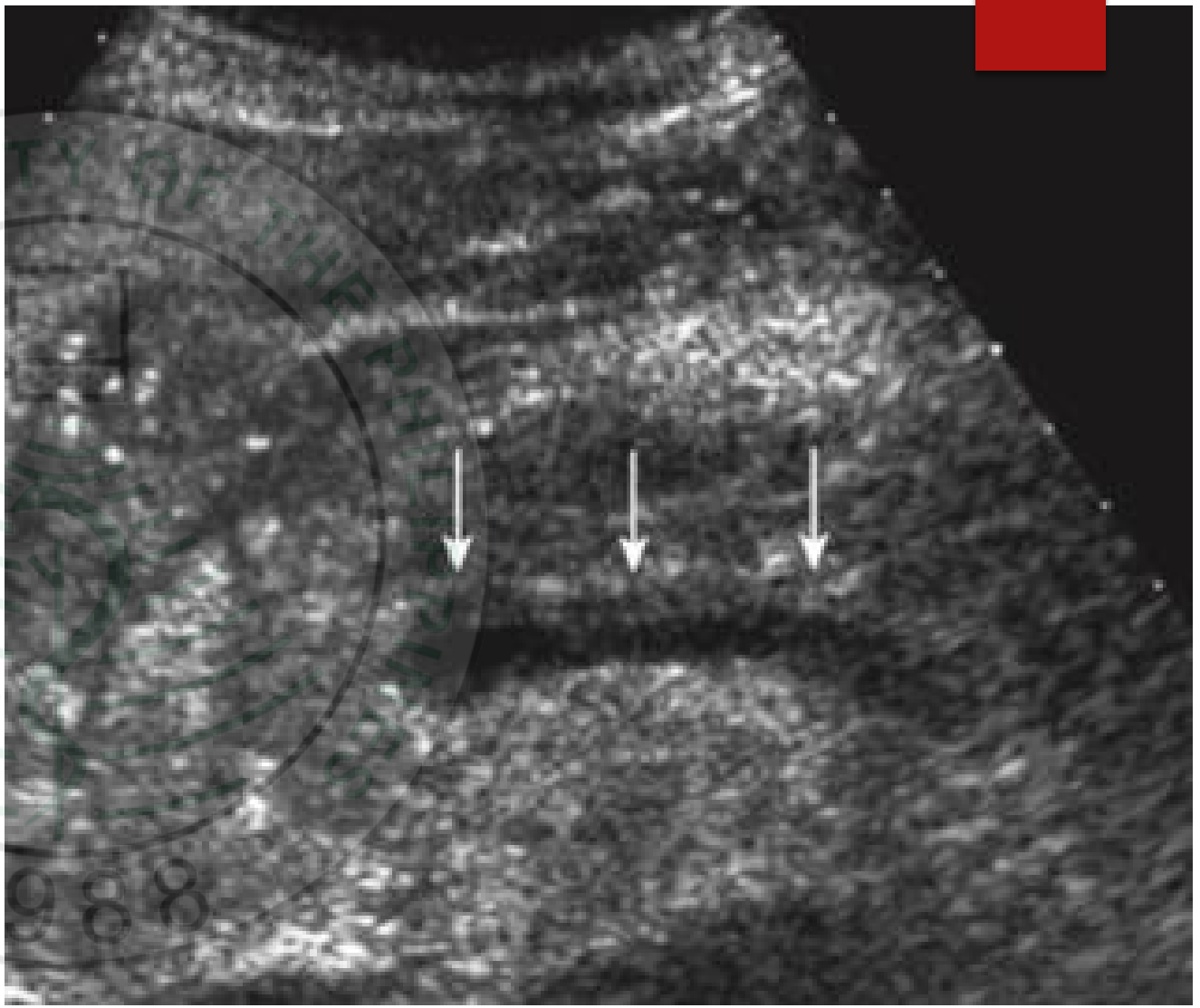
- Pancreatic inflammation is typically hypoechoic or anechoic and conforms to a known retroperitoneal or peritoneal space

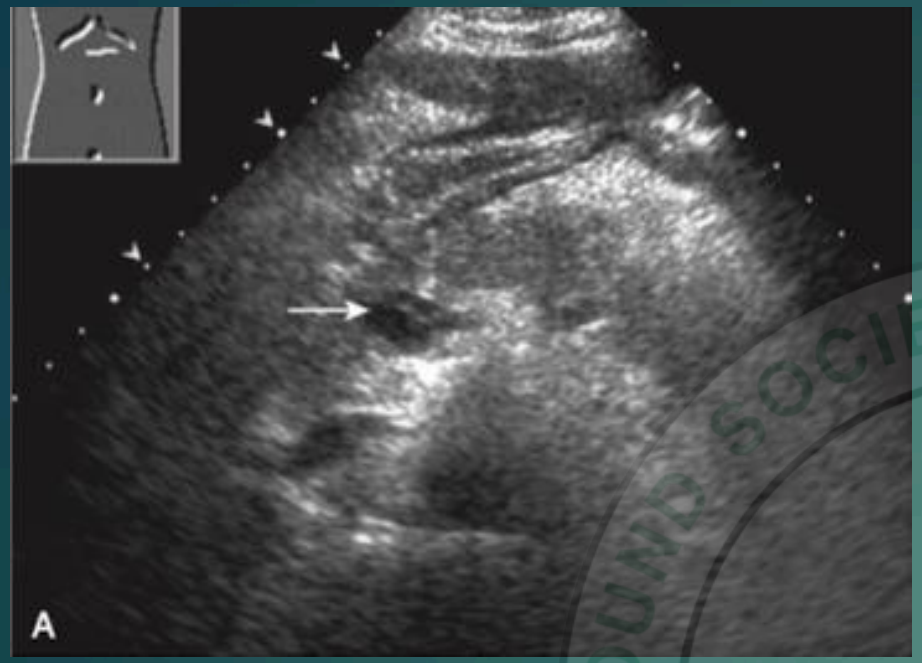


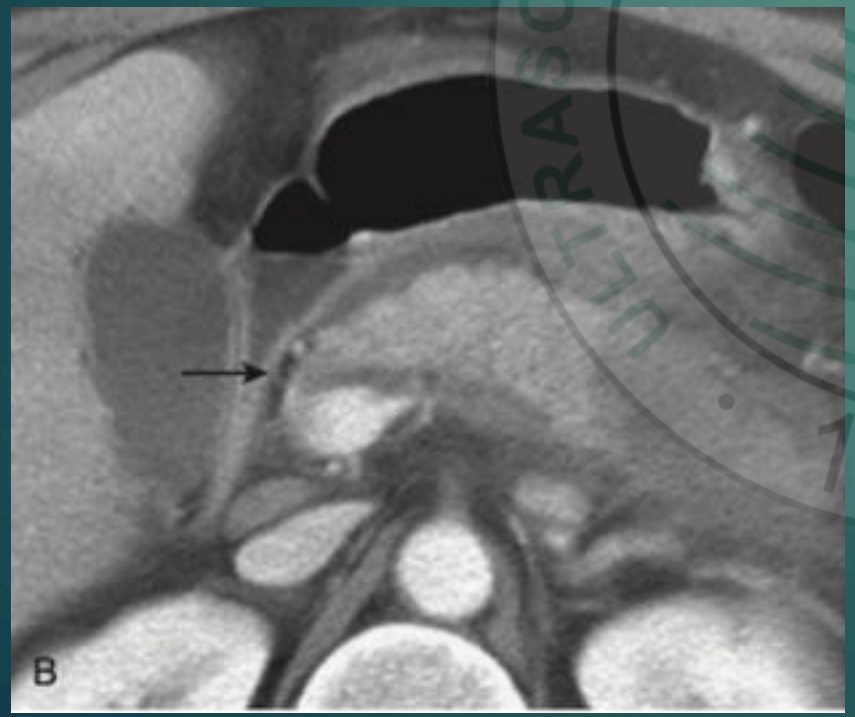
- Inflammatory mass (formerly called a phlegmon) may be pr es en t

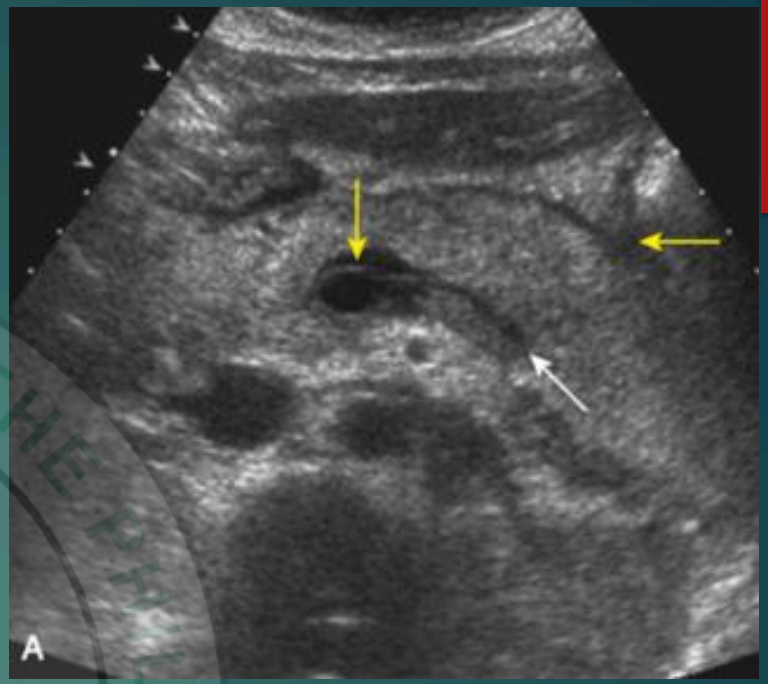


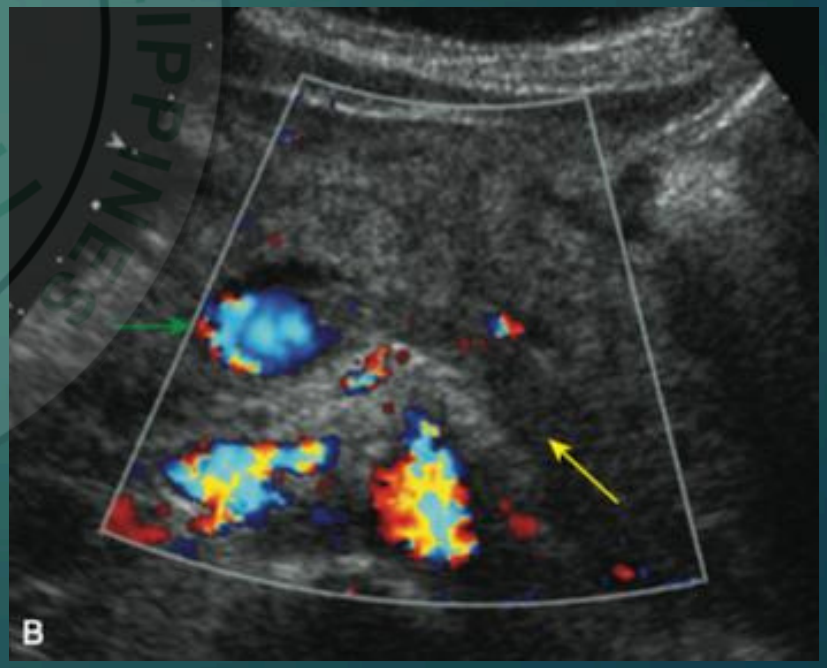
- CHARACTERISTIC OF ACUTE PANCREATITIS
  - Spread of inflammation along perivascular spaces, especially the splenic vein and splenoportal confluence




A


B


A


B

LOCAL COMPLICATIONS OF ACUTE P AN CRE AT IT IS

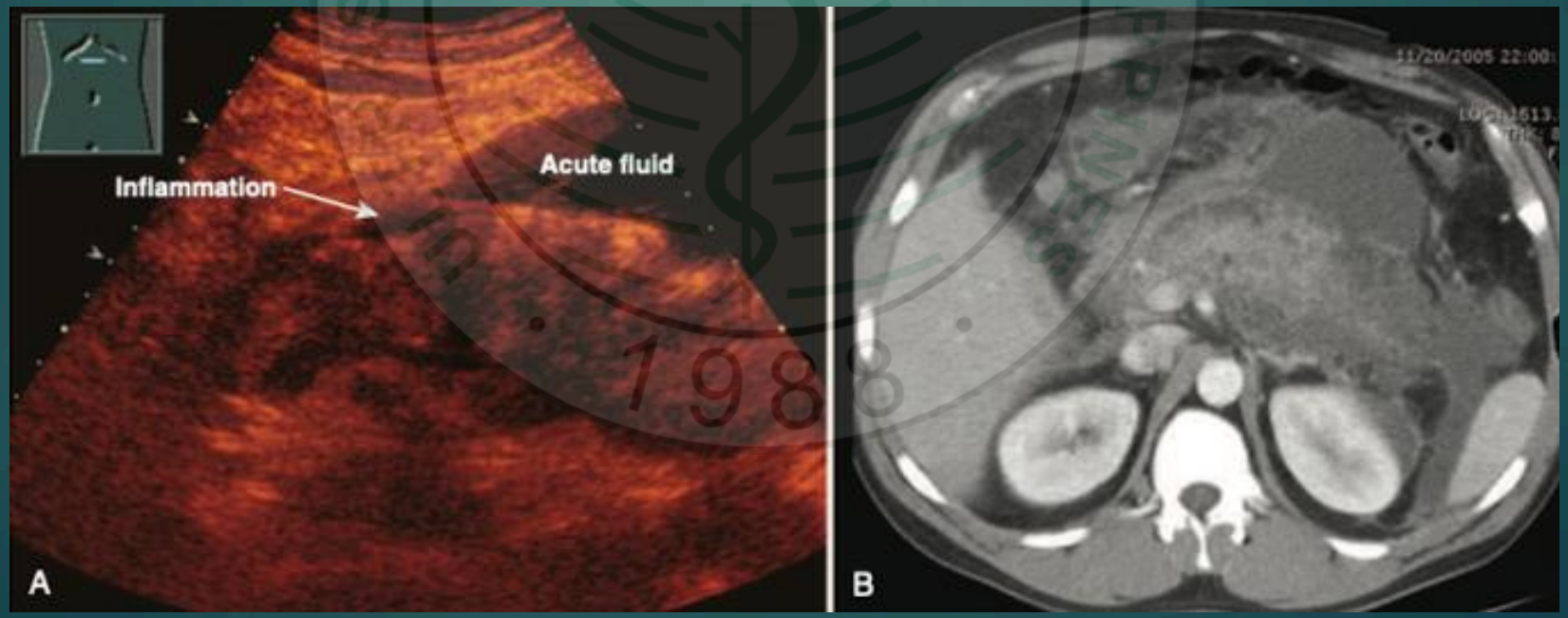
- Acute fluid collections
- Pancreatic pseudocysts
- Pancreatic abscess
- Necrosis
- Infected necrosis
- He m orr ha ge
- Venous thrombosis
- Ps eu do an e ur ys ms

ACUTE FLUID COLLECTIONS

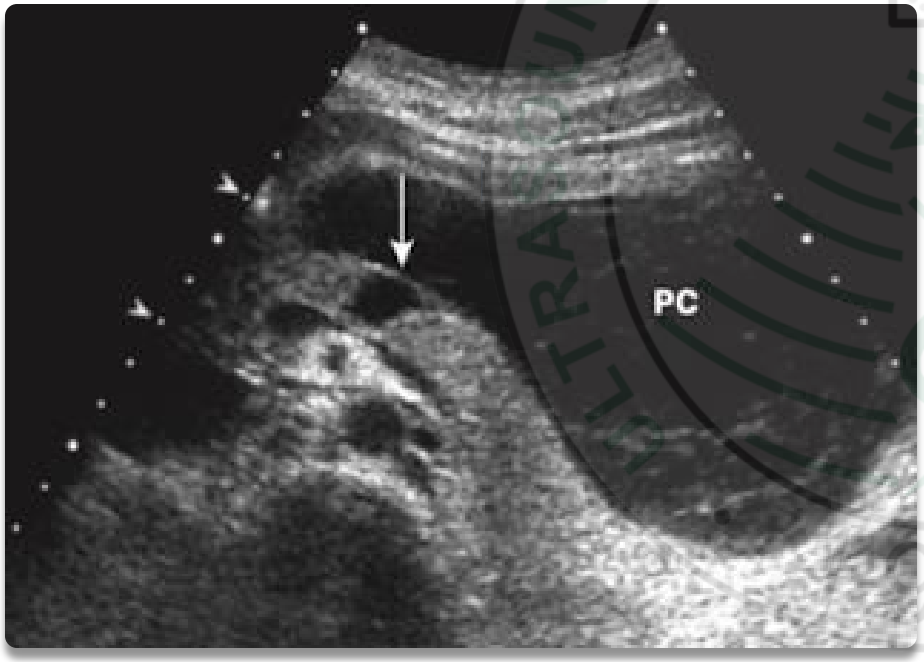
- 40% of patients with acute pancreatitis develop acute fluid collections
  - Half of these appear to resolve spontaneously
- Drainage or other intervention in acute collections is inappropriate
  - Atlanta Classification: differentiation between acute fluid collection and pseudocyst should be made after 4 weeks from the onset of disease
  - Others suggest: fluid collection that persists for 6 weeks can be considered a pseudocyst

ACUTE FLUID COLLECTIONS

- Pancreatitis-associated fluid collections represent a spectrum of disease and thus can be problematic to classify



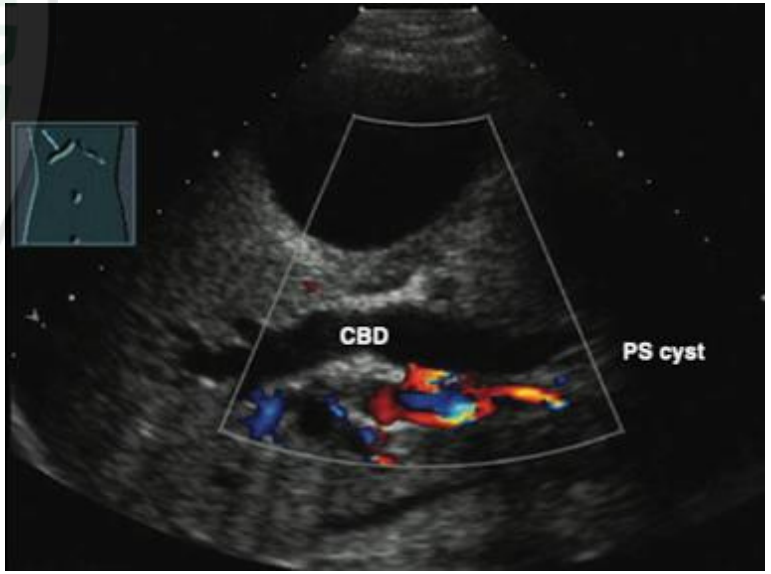
PANCREATIC PSEUDOCYST



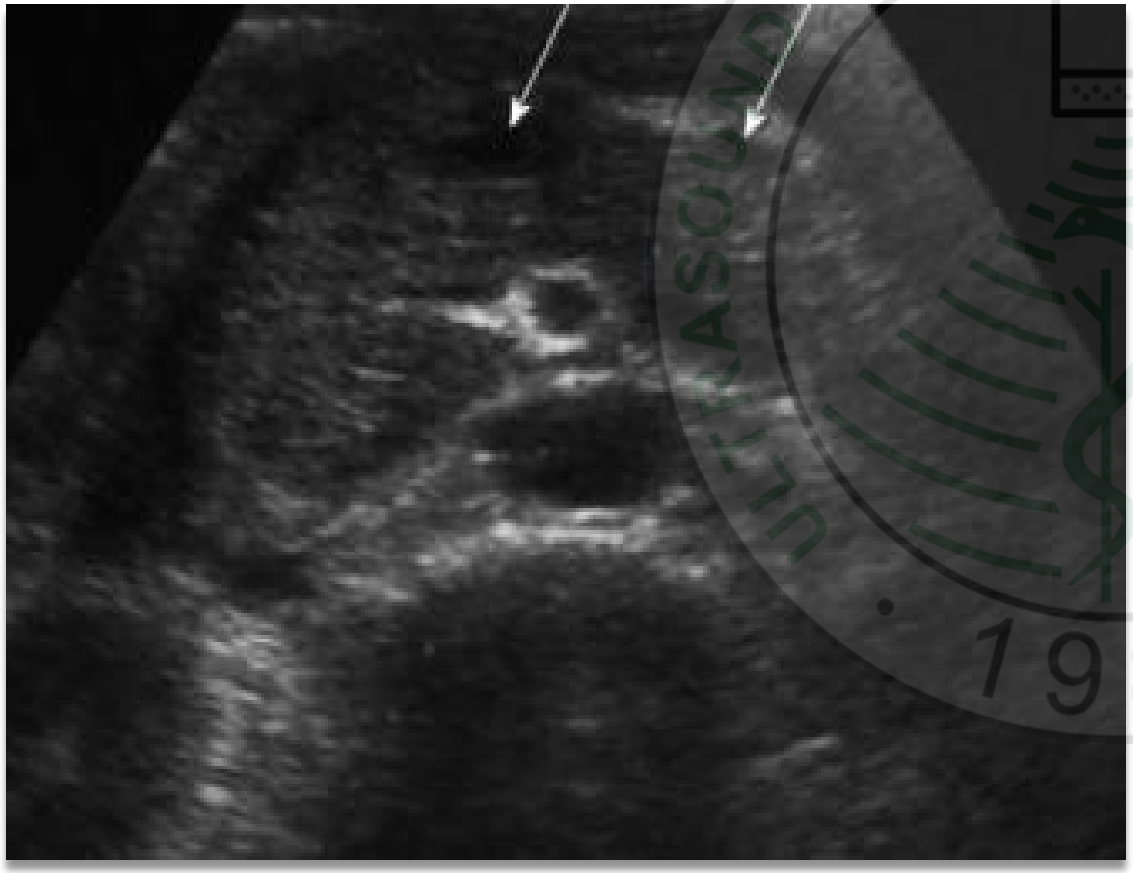
- Well-known complication of acute and chronic pancreatitis
- More common in patients with chronic than acute pancreatitis
- Its wall consist of fibrous and granulation tissue
- It does not have an epithelial lining, unlike true cysts or cystic neoplasms. This distinction may be difficulton imaging

- The major criterion for diagnosing a pseudocyst:
- Clinical history
- Imaging evidence of acute or chronic pancreatitis

- Variable appearance
- Range in appearance from almost purely cystic to collections with considerable mural irregularity, septations, and internal echogenicity
- Resolve spontaneously – conservative management
- Indications for drainage of a pseudocyst:
  - Abdominal pain
  - Biliary obstruction
  - Gastrointestinal (usually duodenal) obstruction

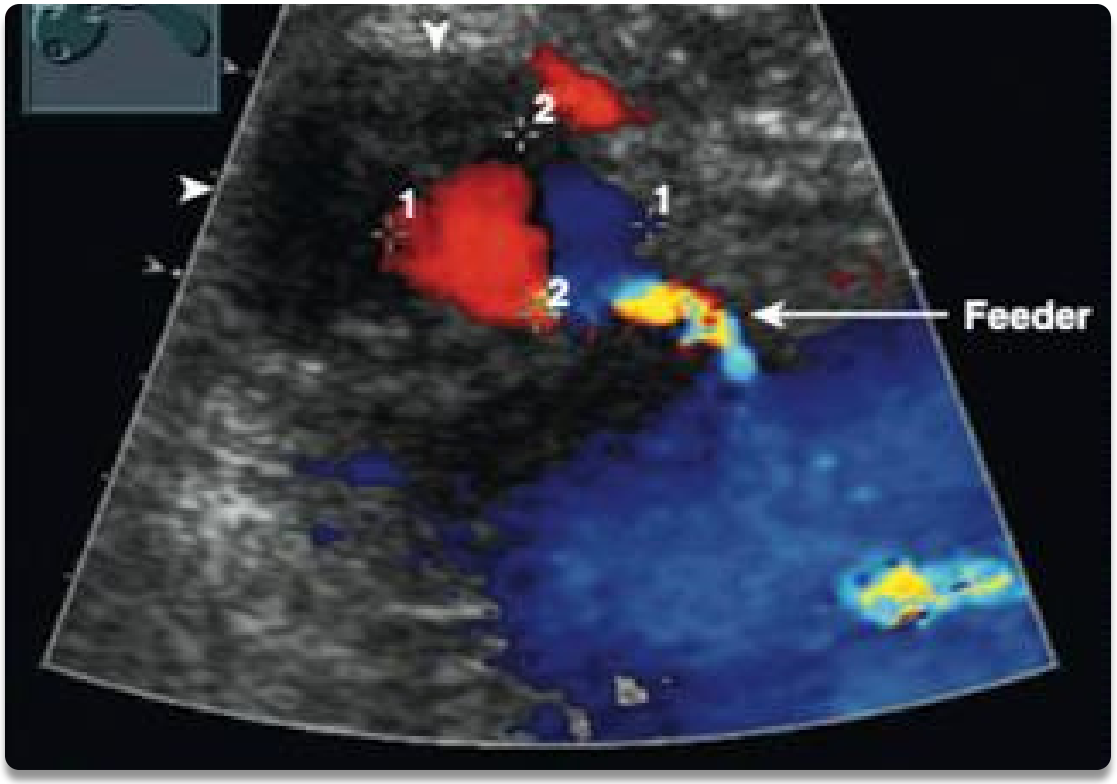


NECROSIS AND ABSCESS



- Atlanta Classification system: nonenhanced pancreatic parenchyma >3 cm or involving >30% of the area of the pancreas on CECT
- Necrosis cannot be definitively diagnosed by ul traso und
- 2 distinct types of acute pancreatitis-associated abscess:
  - (1) Aninfectedfluid collection/pseudocyst,which has minimal necrosis (The original Atlanta Classification pancreatic abscess)
  - (2) Infected necrosis with a fluid collection, which arises from infection of necrotic pancreatic tissue.

VASCULAR COMPLICATIONS



- Both acute and chronic pancreatitis
- Most important vascular complications:
  - Pseudoaneurysms
  - Venous thrombosis
- Venous and small vessel disease: most cases of clinically insignificant hemorrhagic pancreatitis
- Pseudoaneurysm of major vessels: cases of potentially fatal hemorrhage

Hemosuccus pancreaticus

- Gastrointestinal bleeding caused by bleeding into the pancreatic duct

Splenic vein thrombosis is most common

"Sinistral" (let-sided) portal hypertension

- Condition wherein splenic vein thrombosis results in upper GI bleeding from gastric varices

CHRONIC PA NCR EA TIT IS

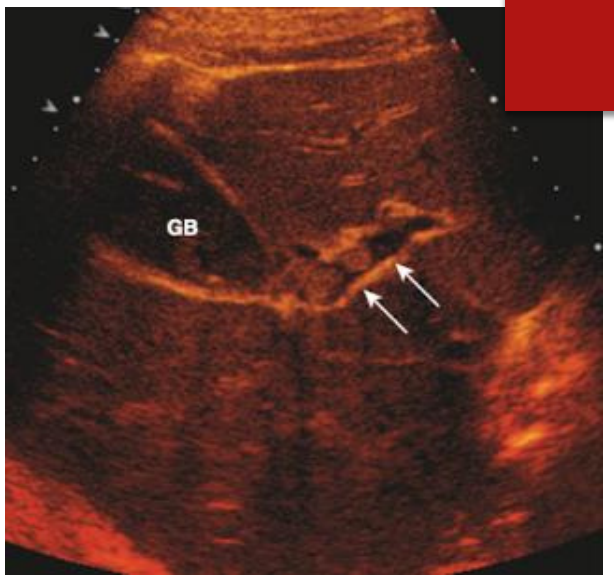
- Characterized by intermittent pancreatic inflammation with progressive, irreversible damage to the gland
- Key histologic features:
  - Fi br o sis
  - Acinar atrophy
  - Chronic inflammation
  - Distorted and blocked ducts
- Leads to permanent structural change and deficient endocrine and exocrine function

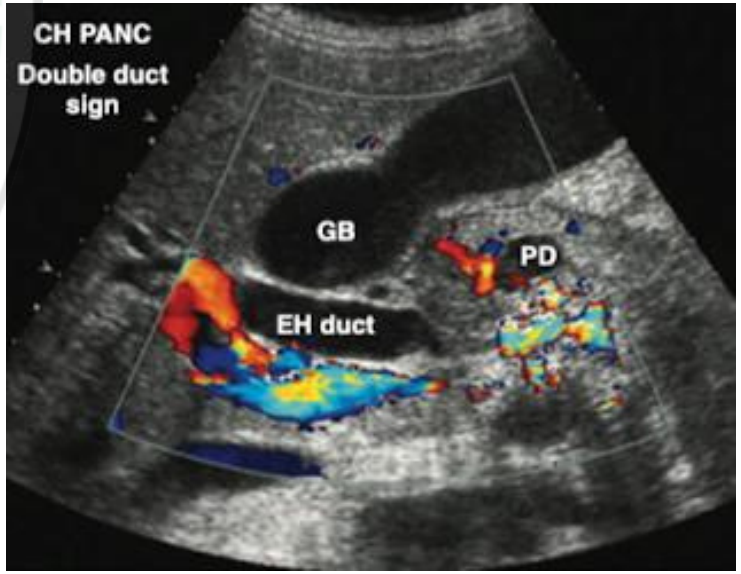
CHRONIC PANCREATITIS

- Some lasting morphologic changes include:
  - Alterations inparenchymal texture
  - Glandular atrophy
  - Glandular enlargement
  - Focal masses
  - Dilation and beading of the pancreatic duct (often with intraductal calcifications)
  - Pseudocysts
- Alcoholism: predominant cause
- Other causes of chronic pancreatitis:
  - Pancreatic duct obstruction caused by strictures
  - Hypertriglyceridemia
  - Hypercalcemia
  - Autoimmune pancreatitis
  - Tropical pancreatitis
  - Other genetic mutations
- Characterized clinically by:
  - Pain
  - M al ab sorp tion
  - Diabetes

- Uncomplicated chronic pancreatitis is usually treated conservatively
- Invasive interventions: reserved for complications

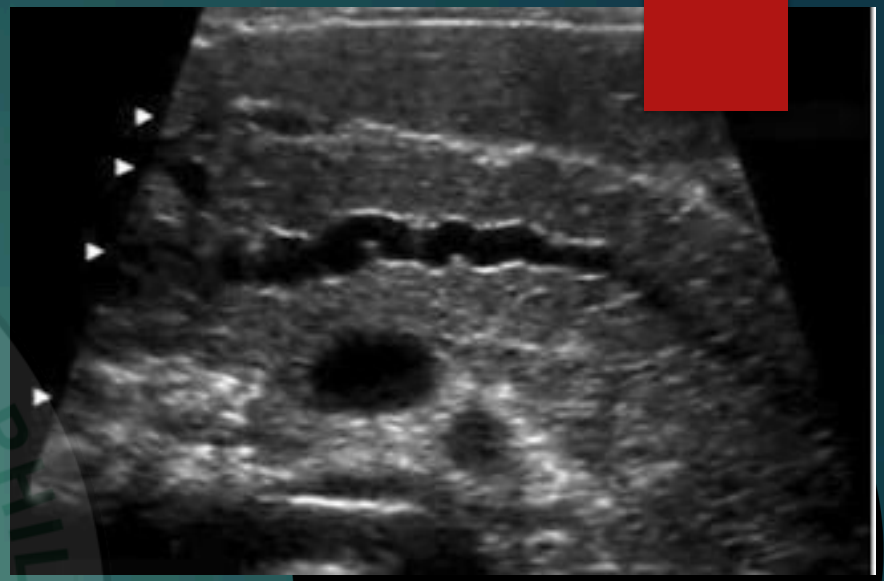
- Obstruction and thrombosis of the portal veins may o cc ur
- Double-duct sign: obstruction of the pancreatic and bile ducts

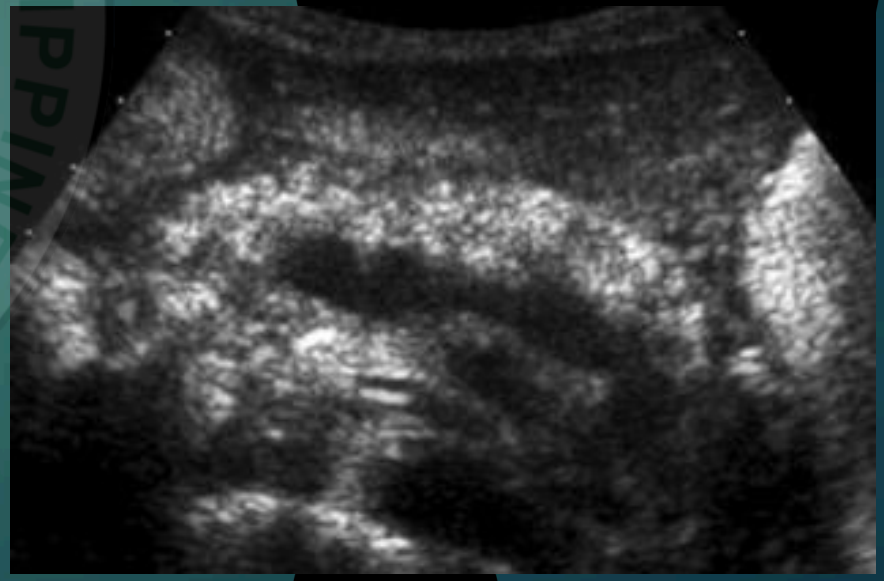




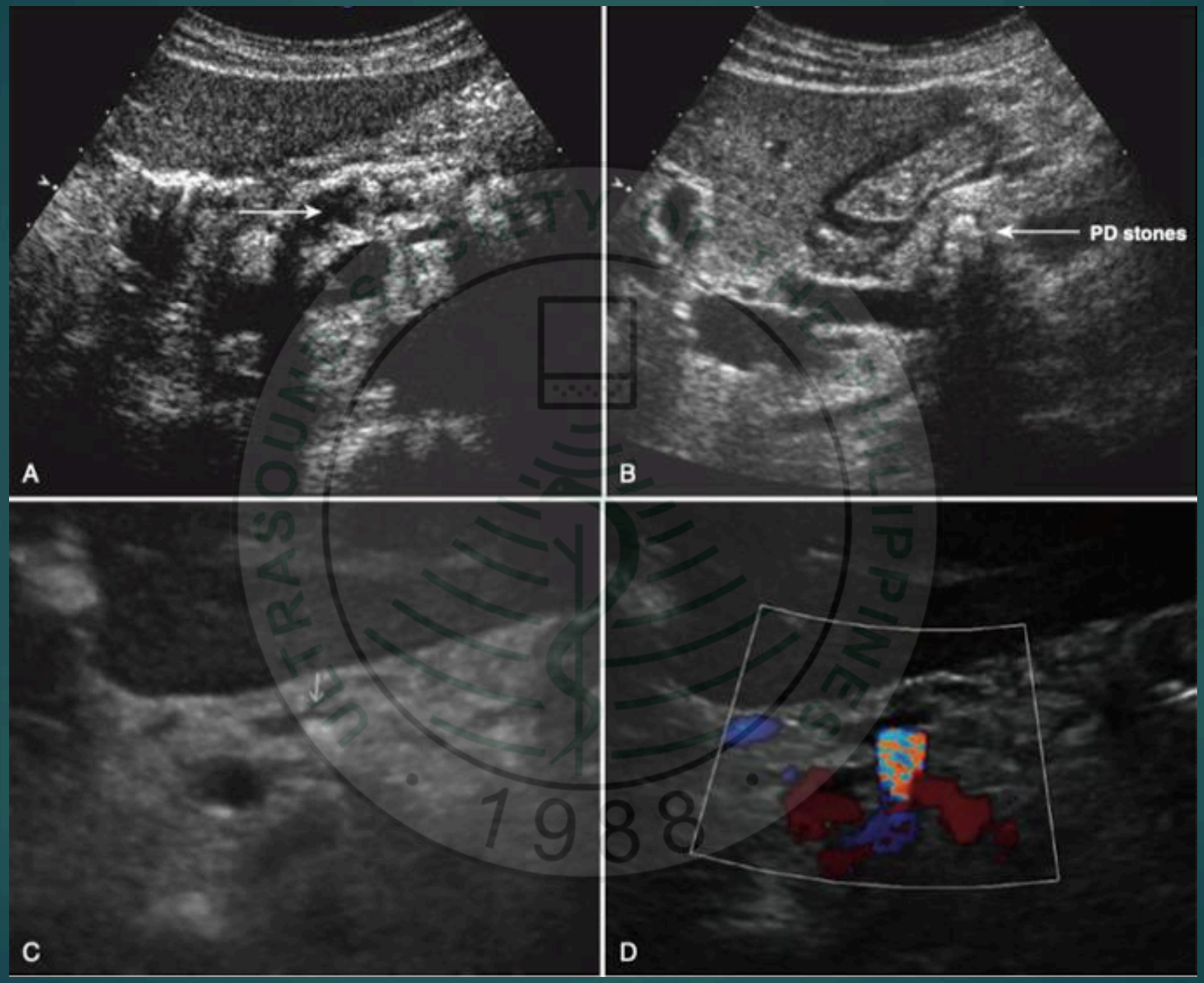
ULTRASOUND FINDINGS

- Hallmark of chronic pancreatitis:
  - Ductal dilation
  - Ca lc if ic a tio ns
- When these findings are present in a patient with pain and a history of alcoholism, the diagnosis of chronic pancreatitis is secure



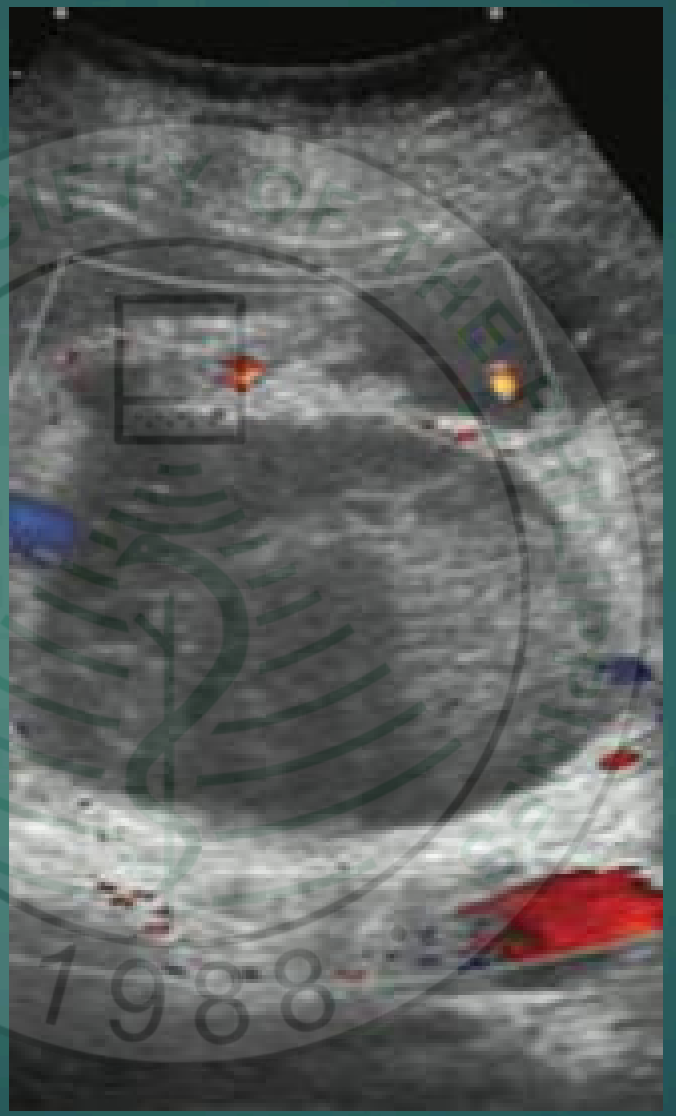


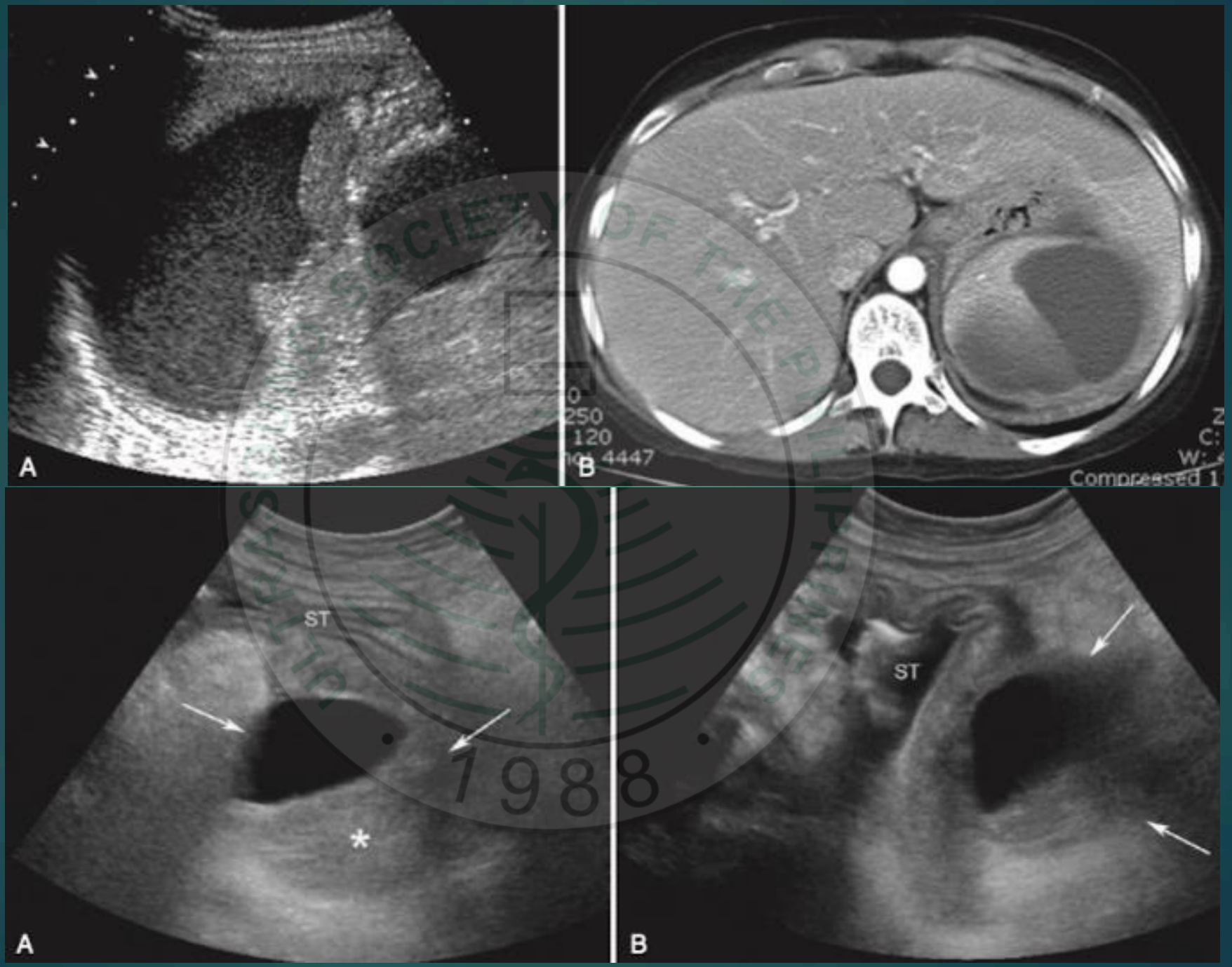
- CT > US: in detecting calcifications and ductal dilation Calcifications are often made much more conspicuous on ultrasound
- images by looking for the color comet-tail artifact, also known as the "twinkling artifact"


PD stones
A
B
C
D

PANCREATIC PSEUDOCYST

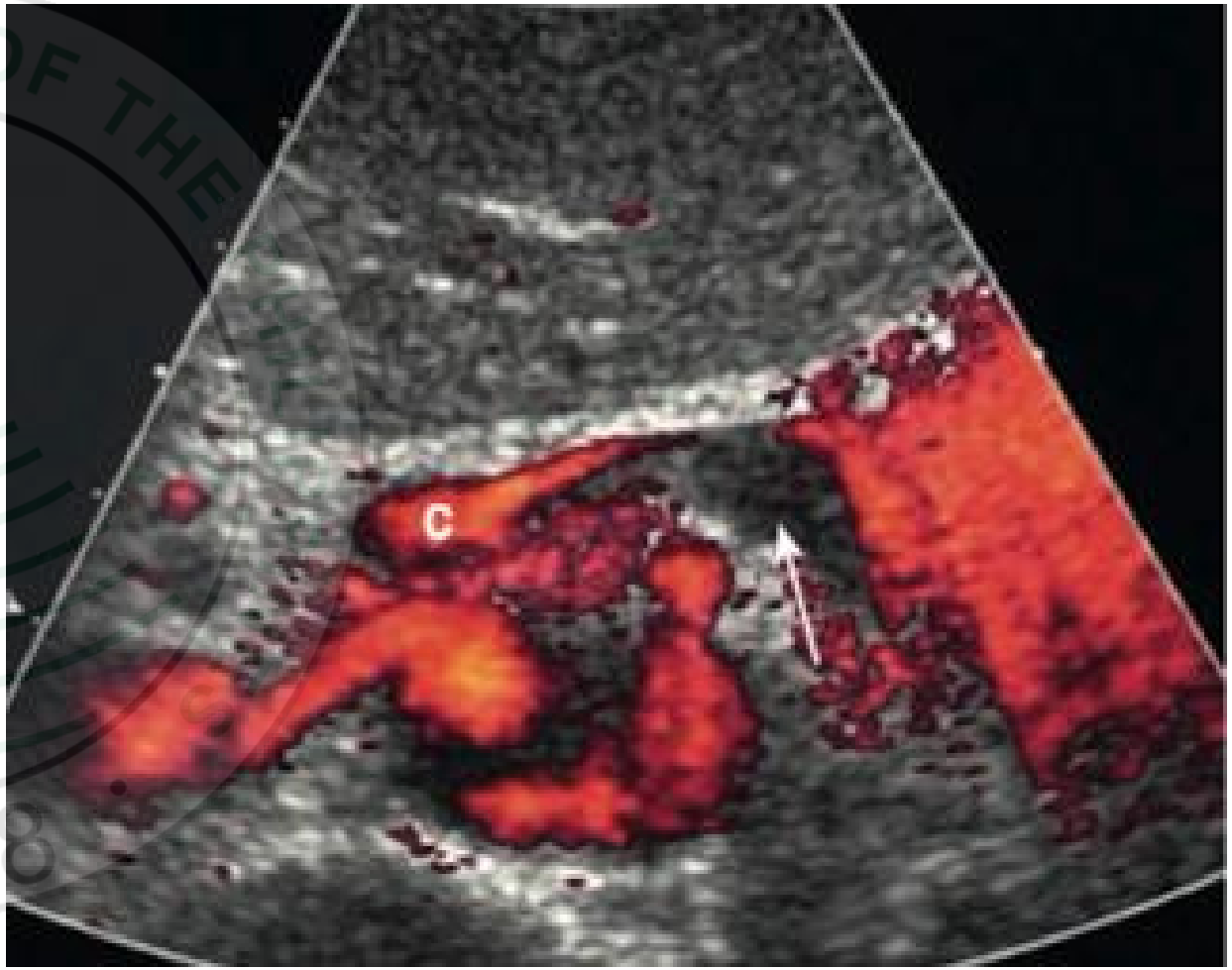
- Morecommonin chronic pancreatitis (20-40%)
  - vs acutepancreatitis (5-16%)
- Various shapes, contain necrotic debris, hemorrhage, or even have a completely solid pa tte rn

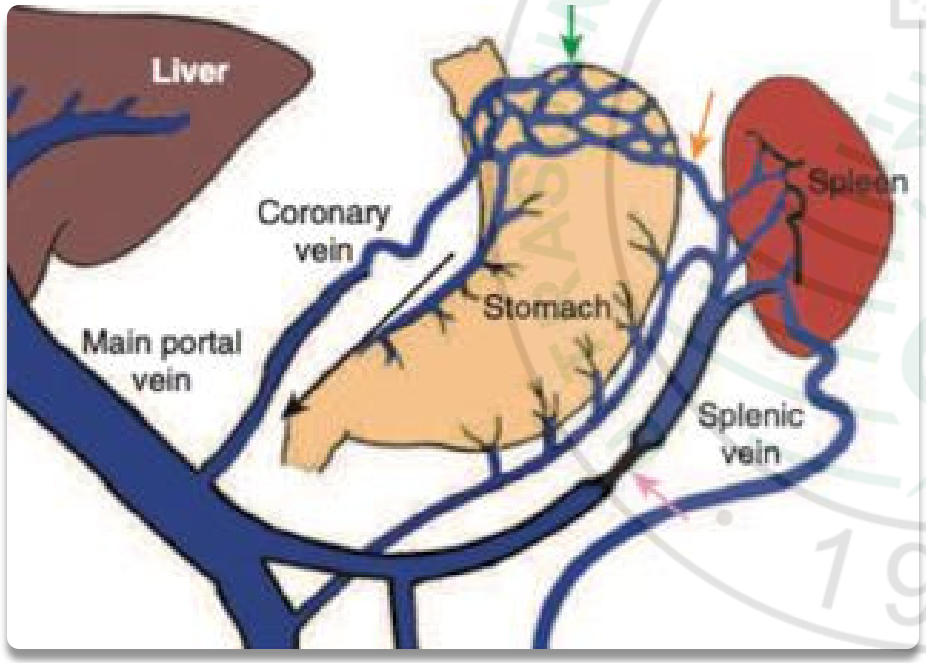



A
B
ST
*
A
ST
B

PORTAL AND SPLENIC VEIN THROMBOSIS

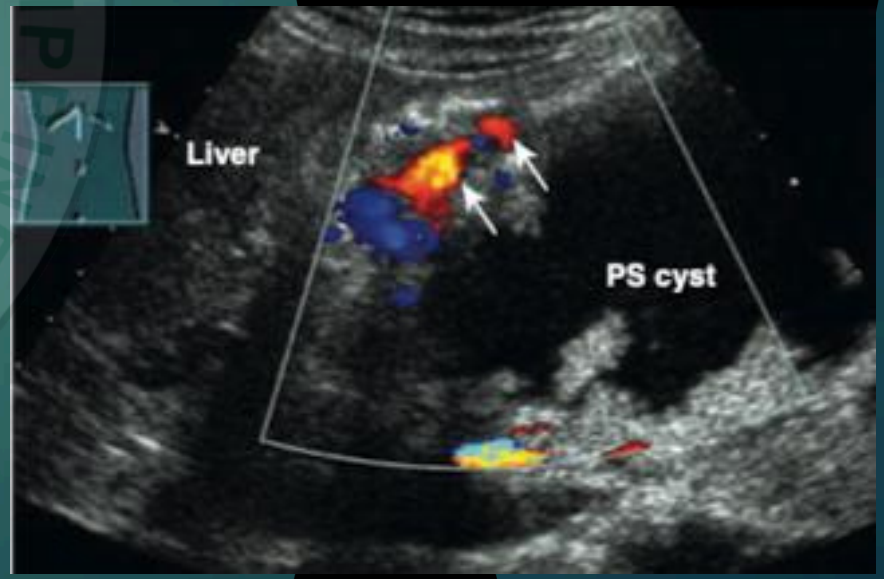
- Thrombosisofthe portalvenous system can occur in chronic pancreatitis because of:
  - (1) Intimalinjury from recurrentacute inflammation
  - (2) Chronic fibrosis and inflammation
  - (3) Compression by either a pseudocyst or an enlarged pancreas
- Splenic vein thrombosis: common in patients with chronic pancreatitis
- Portal vein thrombosis: occurs less frequently



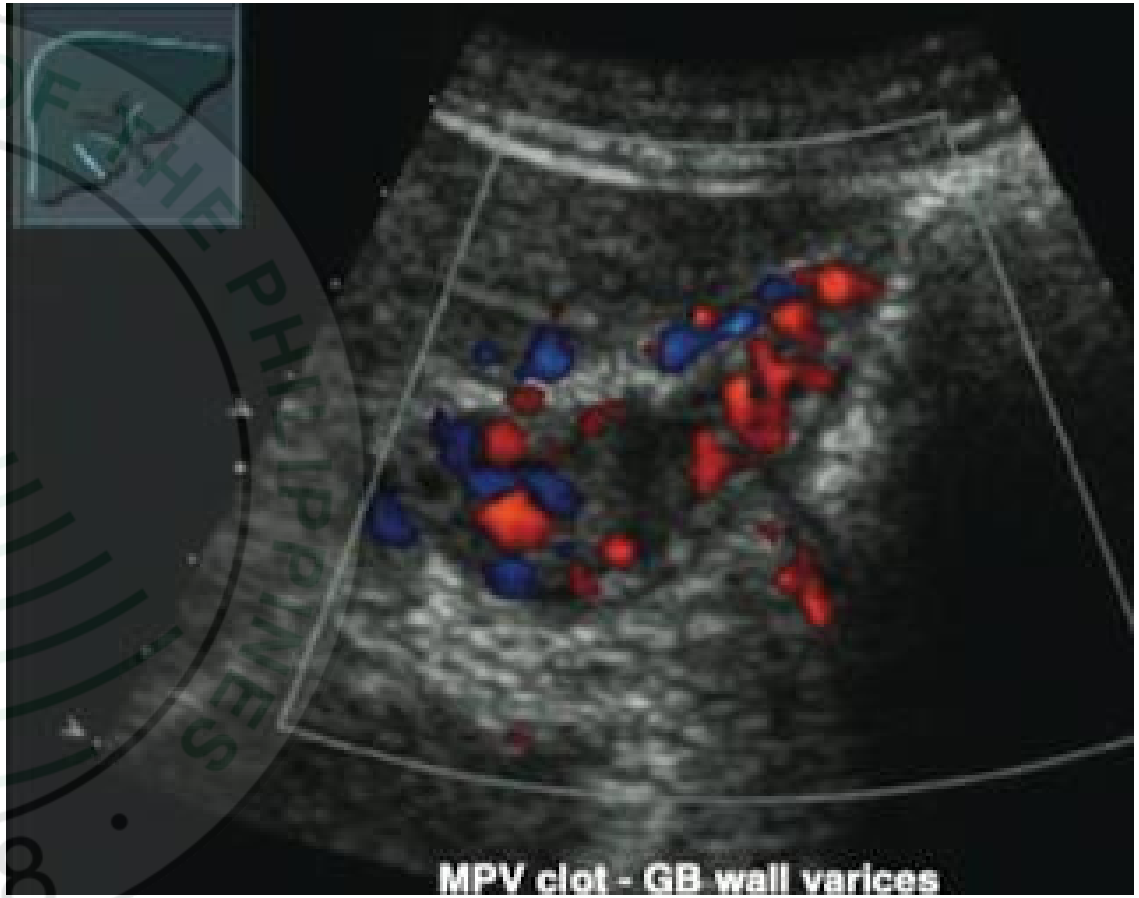


- Pancreatitis-associated thrombus in the splenic or portal vein often results in collaterals
- These collaterals conduct blood ~~toward the liver~~, bypassing the clot
- Splenic vein thrombosis will often result in left-sided ("sinistral") portal hypertension
- This can result in ~~isolated gastric varices~~, which can cause life-threatening ~~GI bleeding~~

- The hepatopetal pathway to bypass the splenic vein clot includes short gastric collaterals that lead to the gastric mural varices, then flow toward the liver in the coronary vein
- Diagnosis of splenic vein clot may depend on detection of collaterals, such as short gastric varices or an enlarged coronary vein



- Gallbladder wall varices
  - Presentin30%ofpatients with portal vein thrombosis
  - These can be successfully diagnosed with color Doppler or gray-scale im a gi ng



MASSES ASSCOCIATED WITH CHRONIC P AN CRE AT IT IS

Focal pancreatic masses occur in 30% of patients who have chronic pancreatitis

(+) calcification: makes the diagnosis of chronic pancreatitis likely

4-6% of ductal adenocarcinomas have calcifications

Pattern of calcification is different from that of patients with the usual chronic pancreatitis

CHRONIC PANCREATITIS VS CARCINOMA

CHRONIC PANCREATITIS FINDINGS	CARCINOMA FINDINGS	NONSPECIFIC FINDINGS
Calcifications are multiple and ductal	Only one or few coarse ca lci fic atio ns	Uncalcified isoechoic or hypoechoic mass occurring in a patient without clinical or imaging evidence of chronic pancreatitis
Hyperechoic masses, even without discrete calcifications		Double-duct sign
Multiple dilated branch ducts in the pancreatic head		
Pseudocysts	Obstructive pseudocyst usuallyareperipheraltobody or tail lesions	

FEAT URE	ACUTE PANCREATITIS	CHRONIC PANCREATITIS
On set	Su dde n	Progre ssi ve
Cause	Gallstones and alcohol	Alcohol (predominant cause)
Pain	Severe, acute epigastric pain radiating to the back	Recurrent or persistent upper abdominal pain
Enzymes (Amylase/Lipase)	Elevated (often >3× upper normal limit)	May be normal or mildly elevated
So no graphy	Enlarged, hypoechoic pancreas; Pancreatitis-associated inflammation	Ductaldilation and calcifications (Hallmark)
C ompl ica tion	Fluid collections, abscess and necrosis	Pancreatic insufficiency (endocrine and exocrine), diabetes, chronic pain
P se udo cys t	Less common (5-16%)	More common (20-40%)
Vascular complications	Occurs in both acute and chronic	
Re ve rsib ili ty	Potentially reversibleif treated early	I rrevers ible
M anage ment	Supportive (fluids, pain control, treat underlying cause)	Symptomatic (enzyme replacement, analgesics), lifestyle changes, surgery if needed

REFERENCES

- Rumack CM et al.Diagnostic Ultrasound, 5th ed.
- Banks PA, et al."Classification of acute pancreatitis—2012: revision of the Atlanta classification."
- h ttps :/ /ww w. re se arc h gate . ne t/ fi gu re /U l tras ou n d-ap pe ara nc e-o f-i n fe cte d - pa nc re ati c-n ec ro si s-be f ore -an d-af te r-th e-tre at me nt -o f_f i g4_26 697 430 0
- R adi o pa ed i a. org

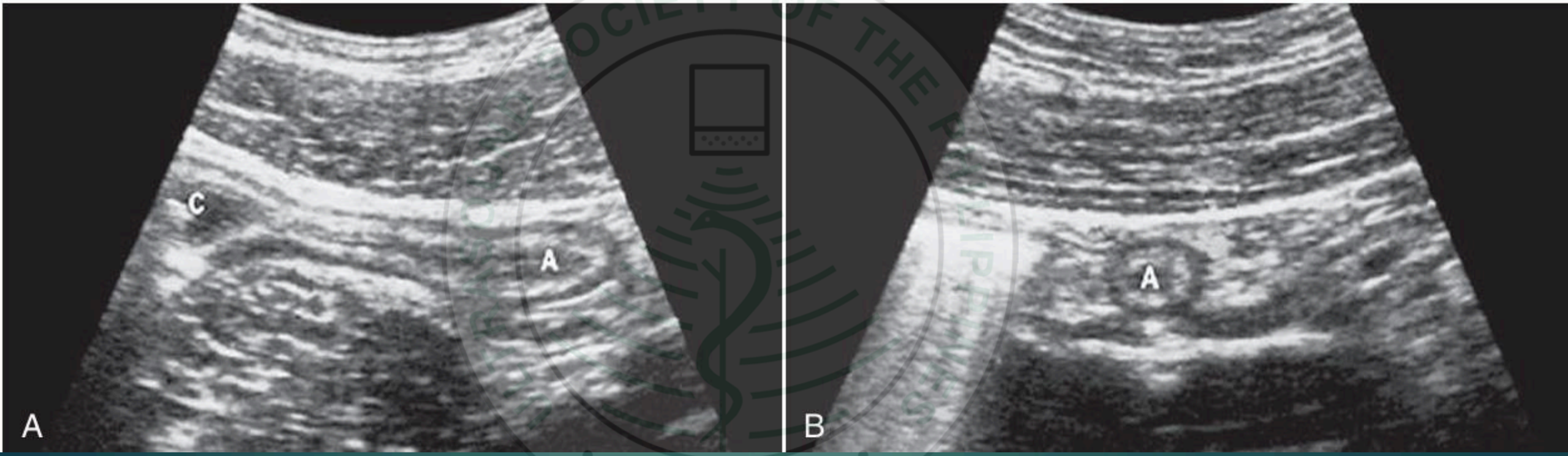
Acute appendicitis

- Most common explanation for acute abd ome n
- RLQ pain, tenderness, and le uko cyto si s
- Symptoms may overlap with other GI co nd itio ns
- CT and ultrasound
- Pathophysiology of acute app end ici tis:
  - Obstruction of appendiceal lu men
  - Increased intraluminal pressure
  - Hypoxia of mucosa

Normal appendix

- Compressible
- wall thickness of 3 mm or less
- Diameter of 6mm or 7mm
- Ovoid morphology - normal

Normal appendix



Acute appendicitis

Acute Appendicitis: Sonographic Diagnosis

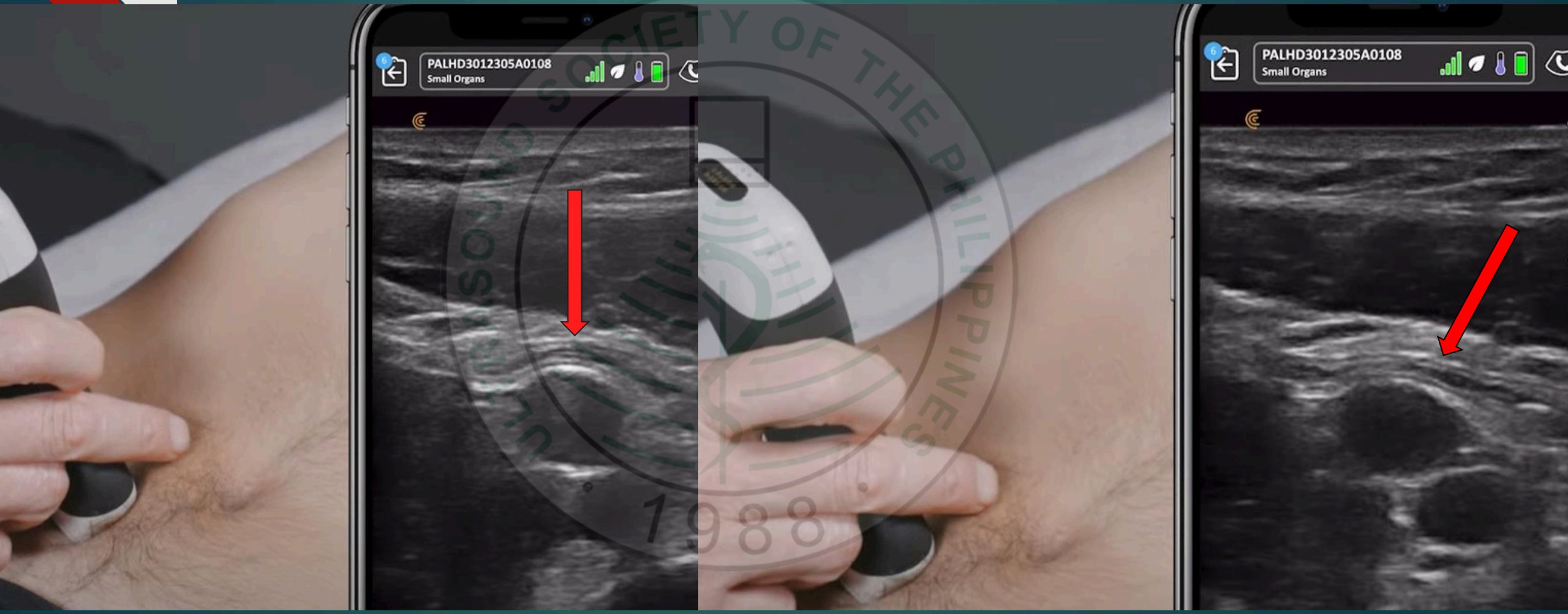
IDENTIFY APPENDIX

Blind ended
Noncompressible
Aperistaltic tube
Gut signature
Arising from base of cecum (typically appendix is caudal to the base of the cecum but it may also be retrocecal and retroileal)
Diameter greater than 6 mm (some use 7 mm for greater specificity)

SUPPORTIVE FEATURES

Inflamed perienteric fat
Pericecal collections
Appendicolith

Acute appendicitis



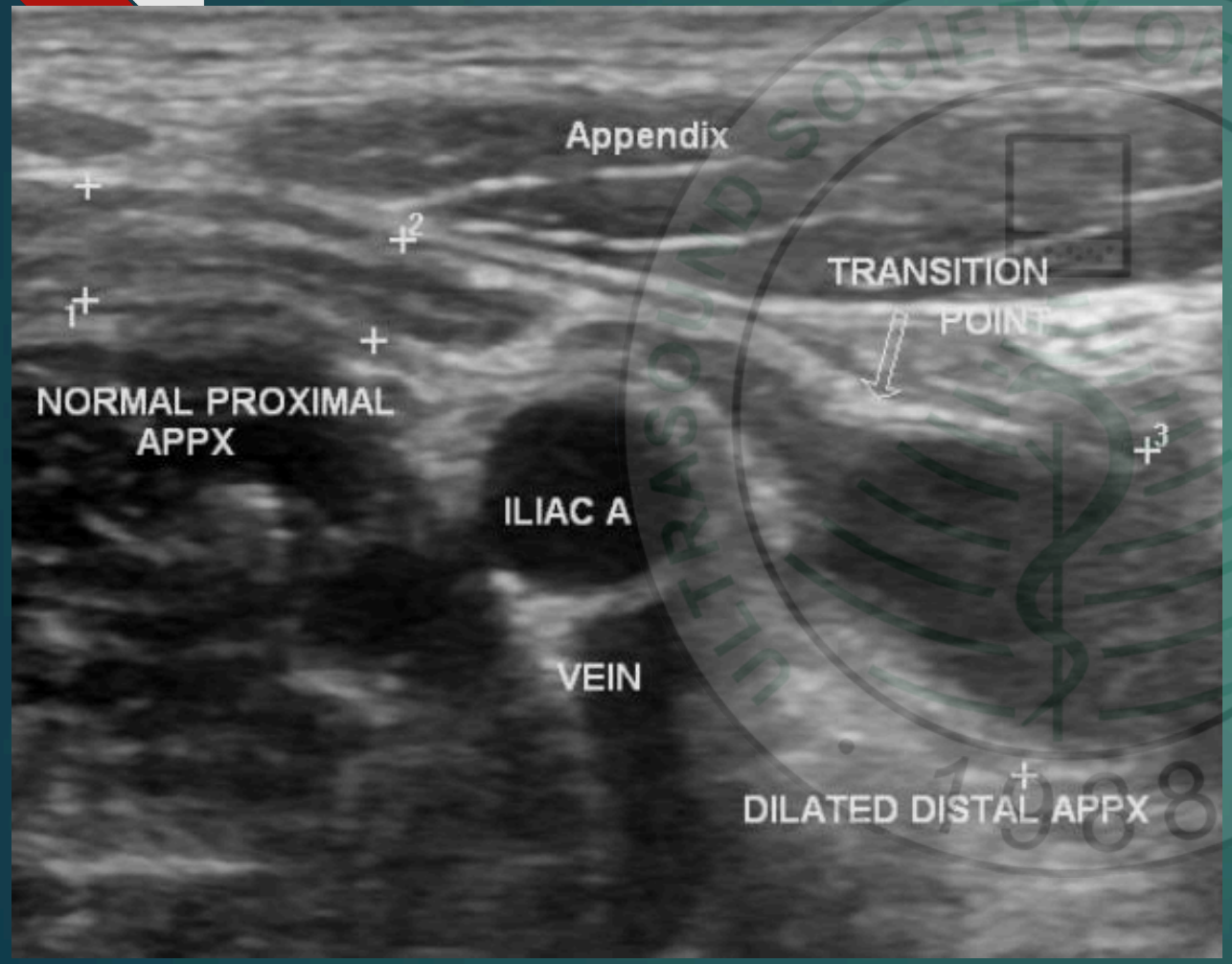
SIGNS OF ACUTE APPENDICITIS:

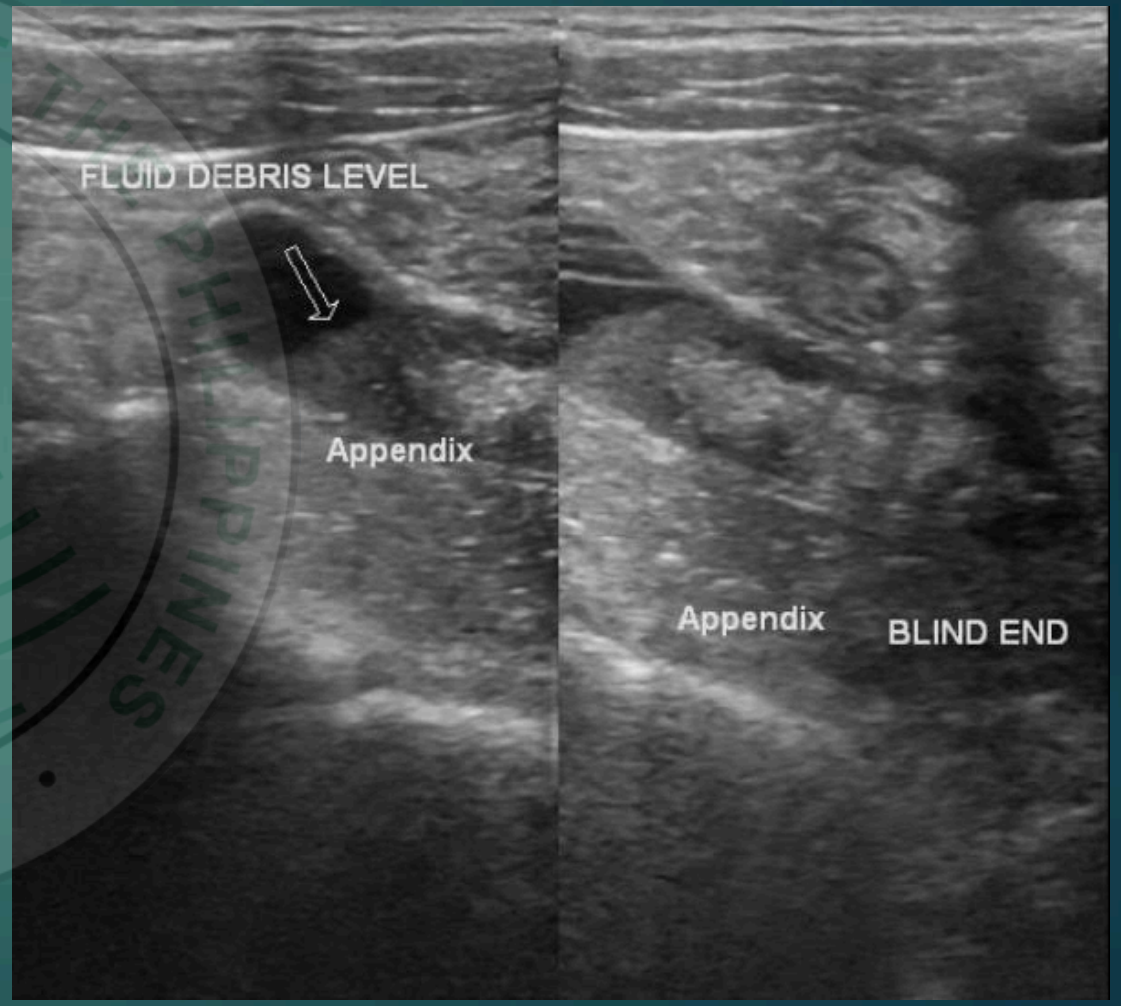
Visualization of an appendix that had a non-compressible, tubular, blind-ended, nonperistaltic app earan ce

A measured diameter of 6 mm or more

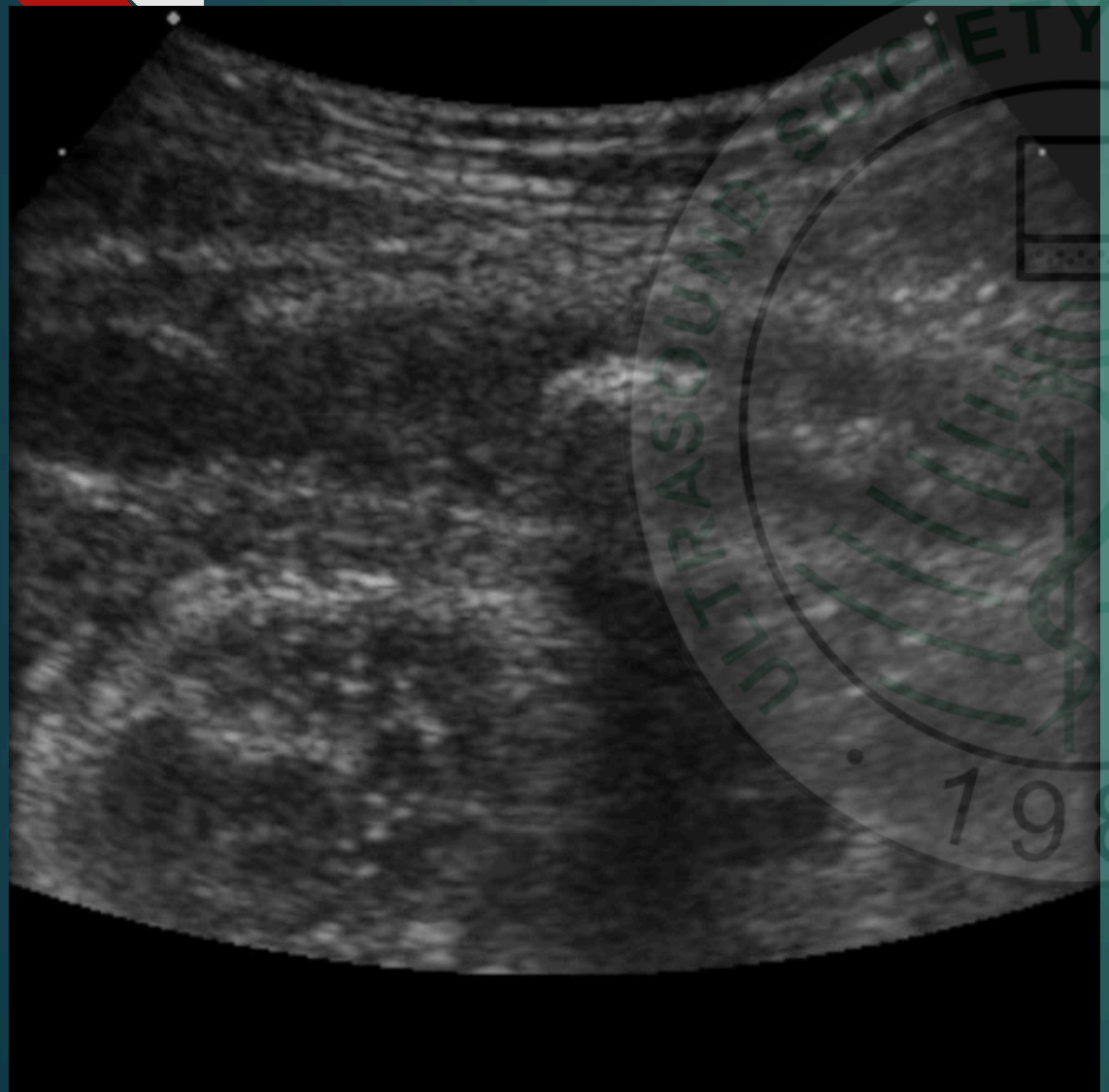
This inflamed appendix with a target app earan ce

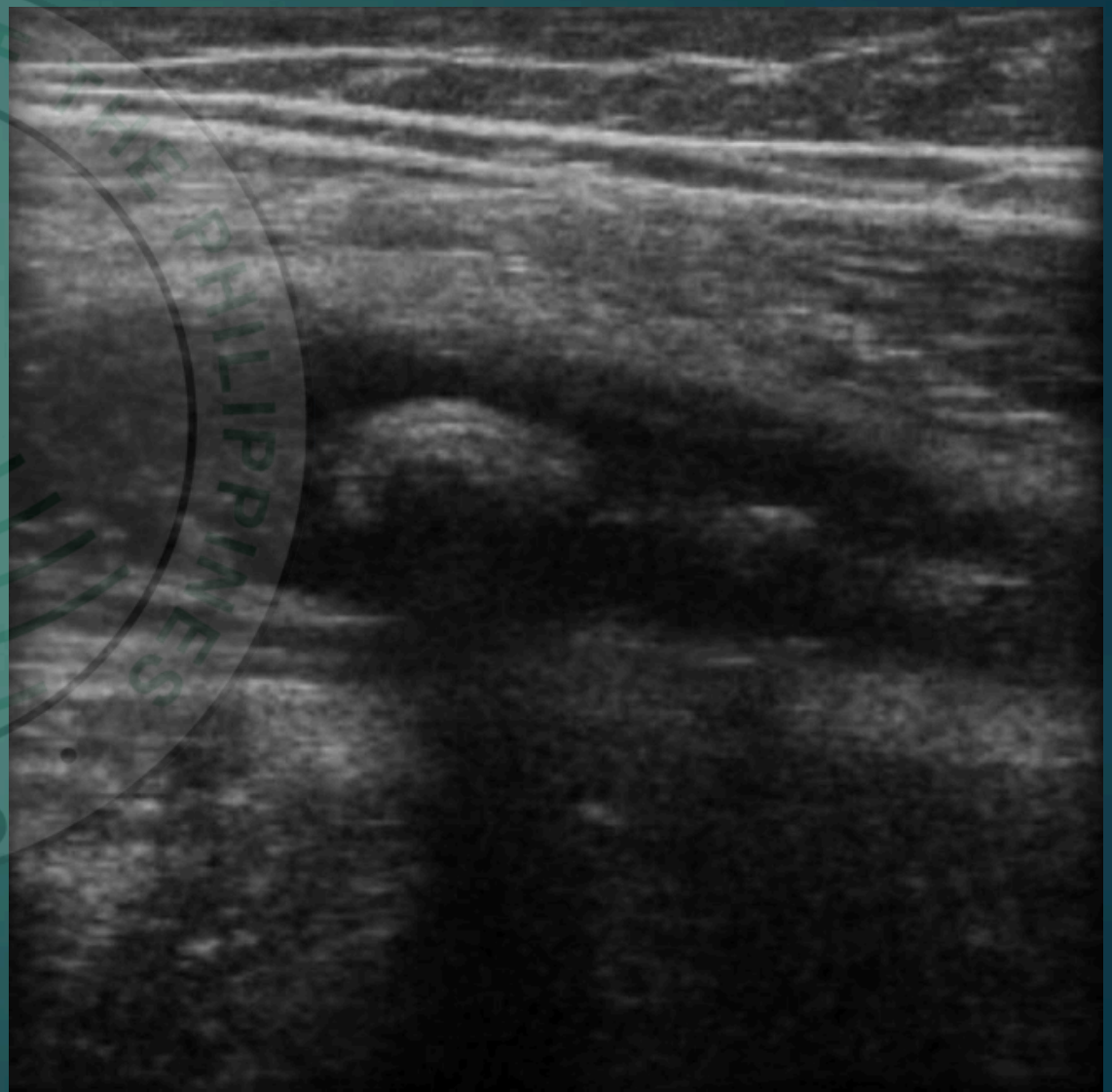
Acute appendicitis



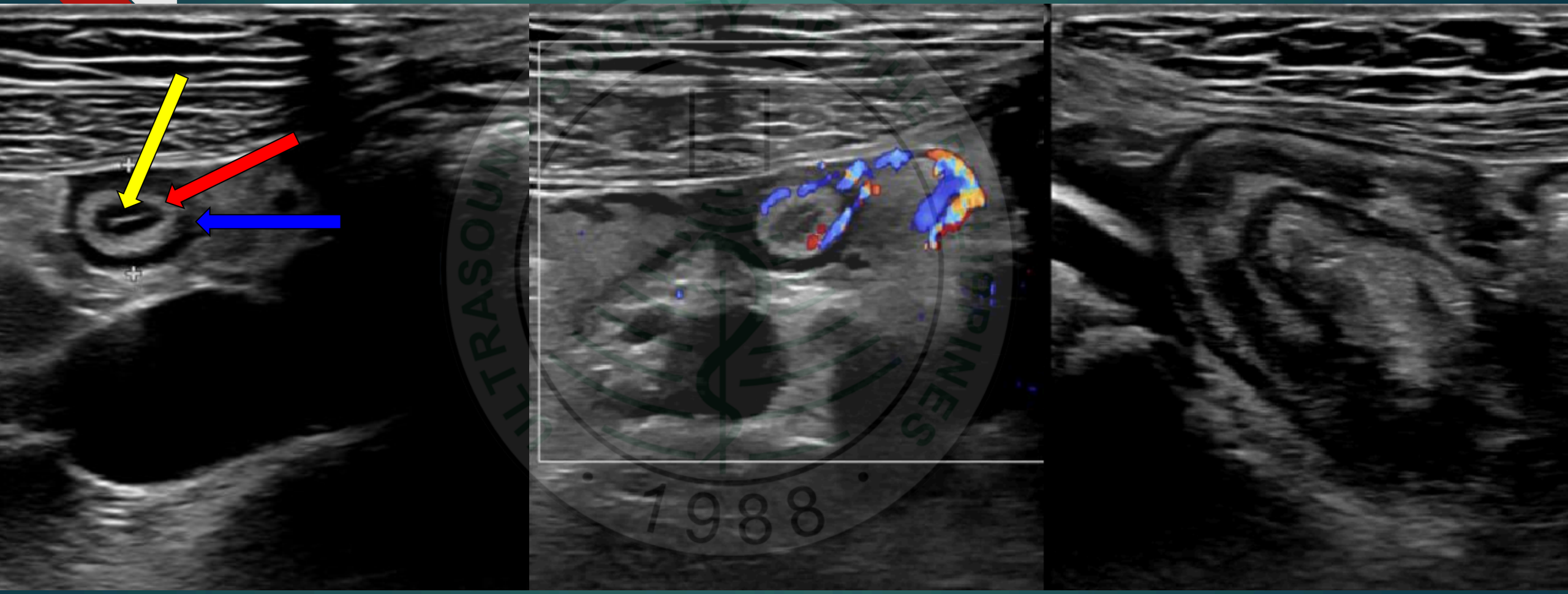


Acute appendicitis

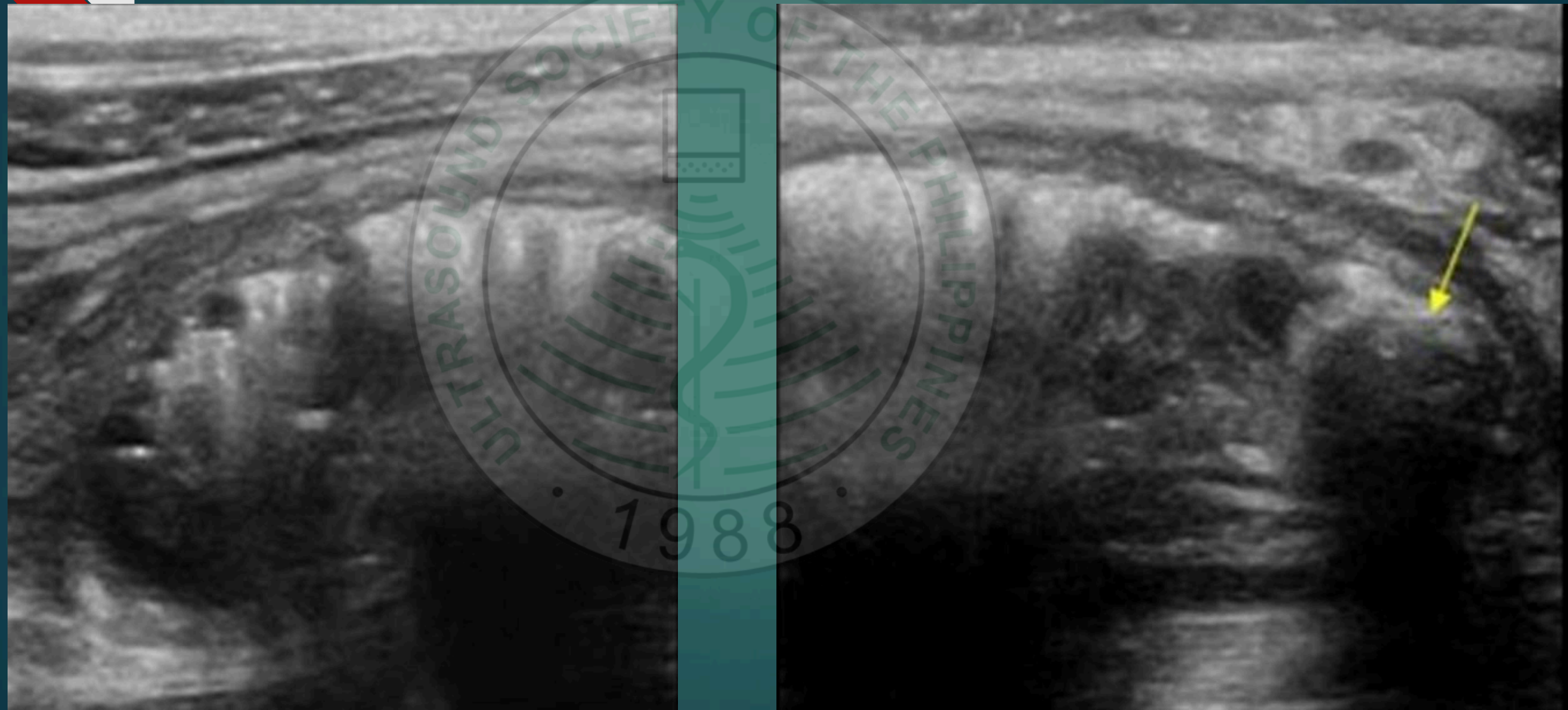




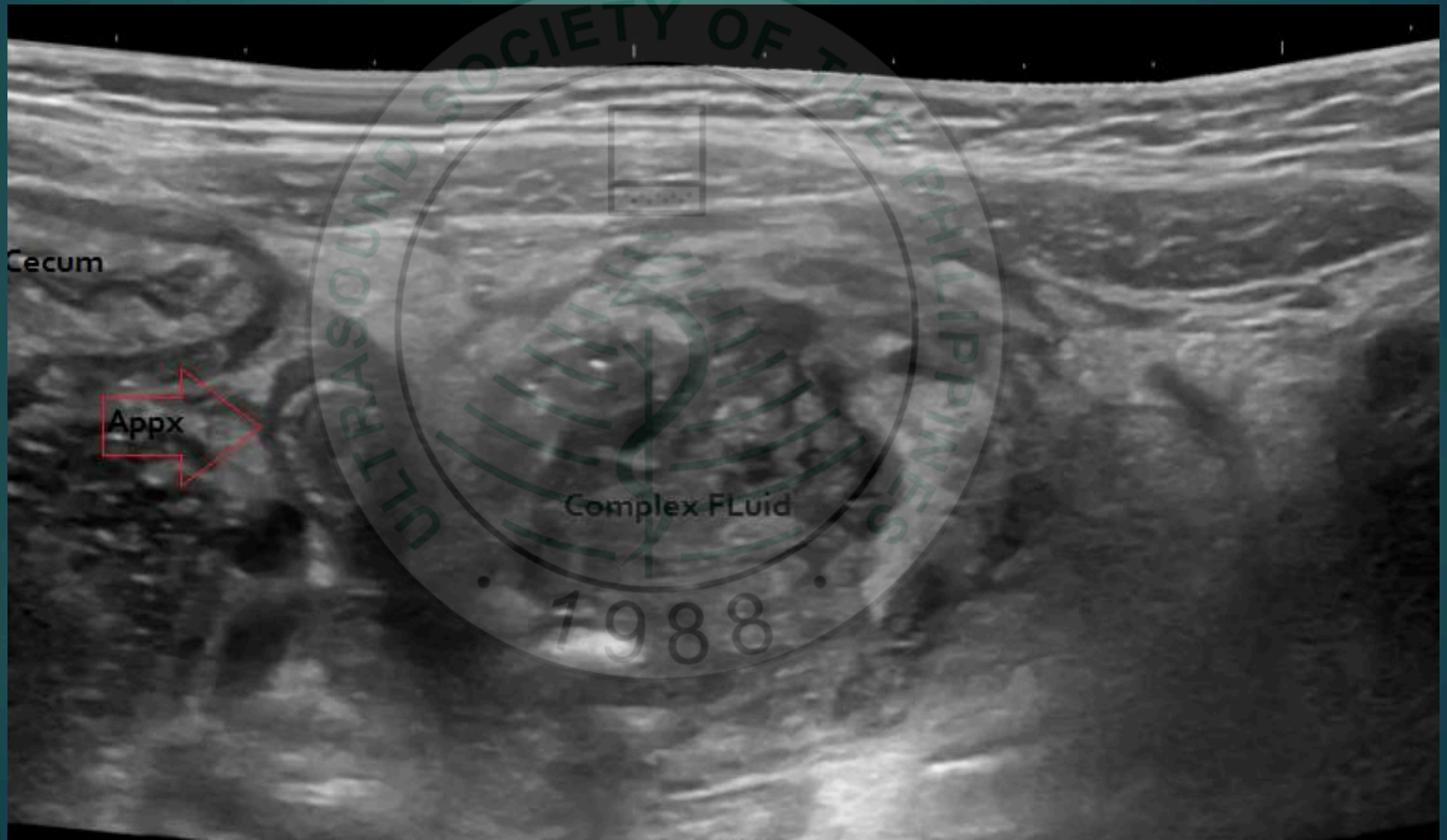
Acute appendicitis



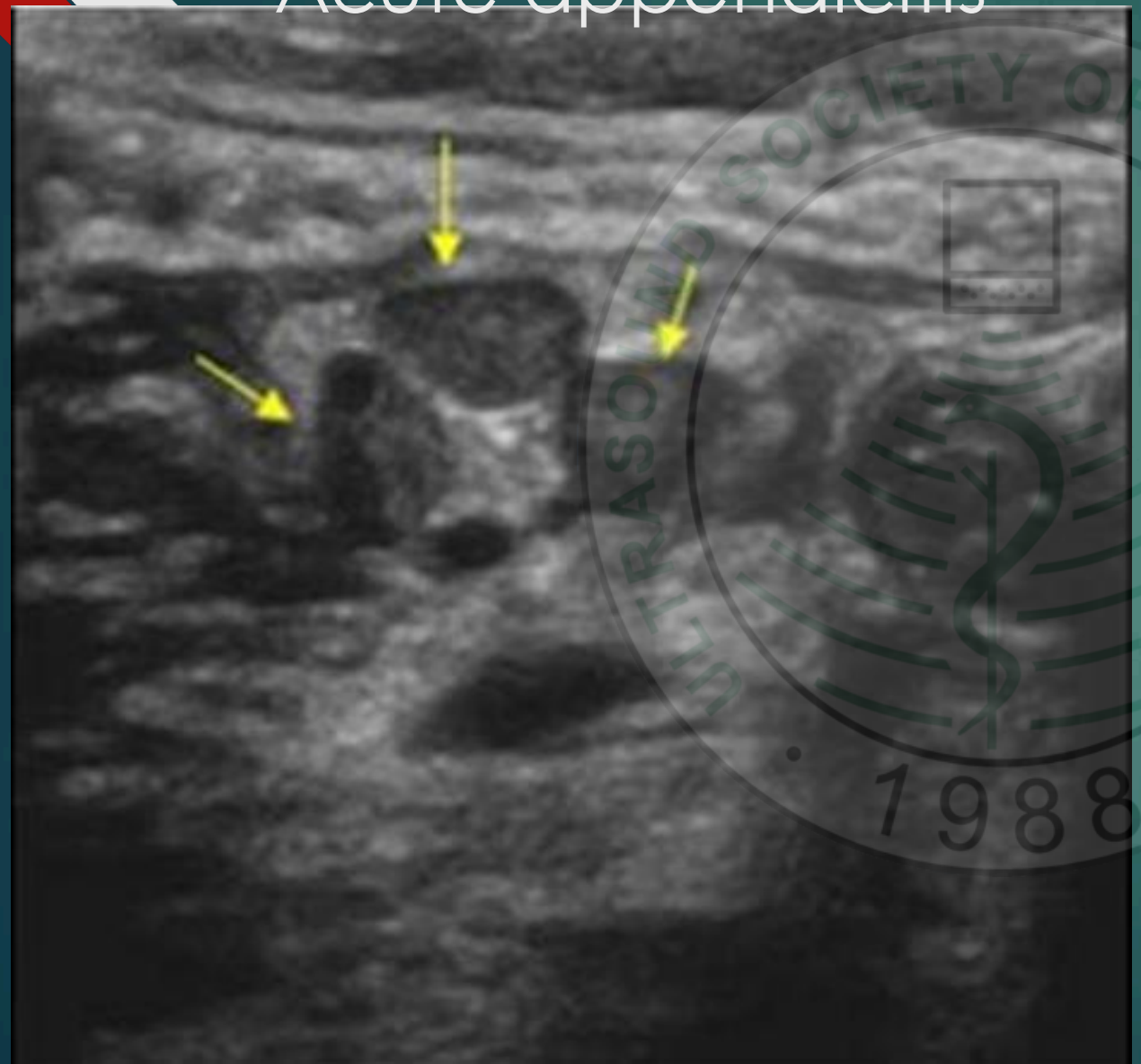
Acute appendicitis

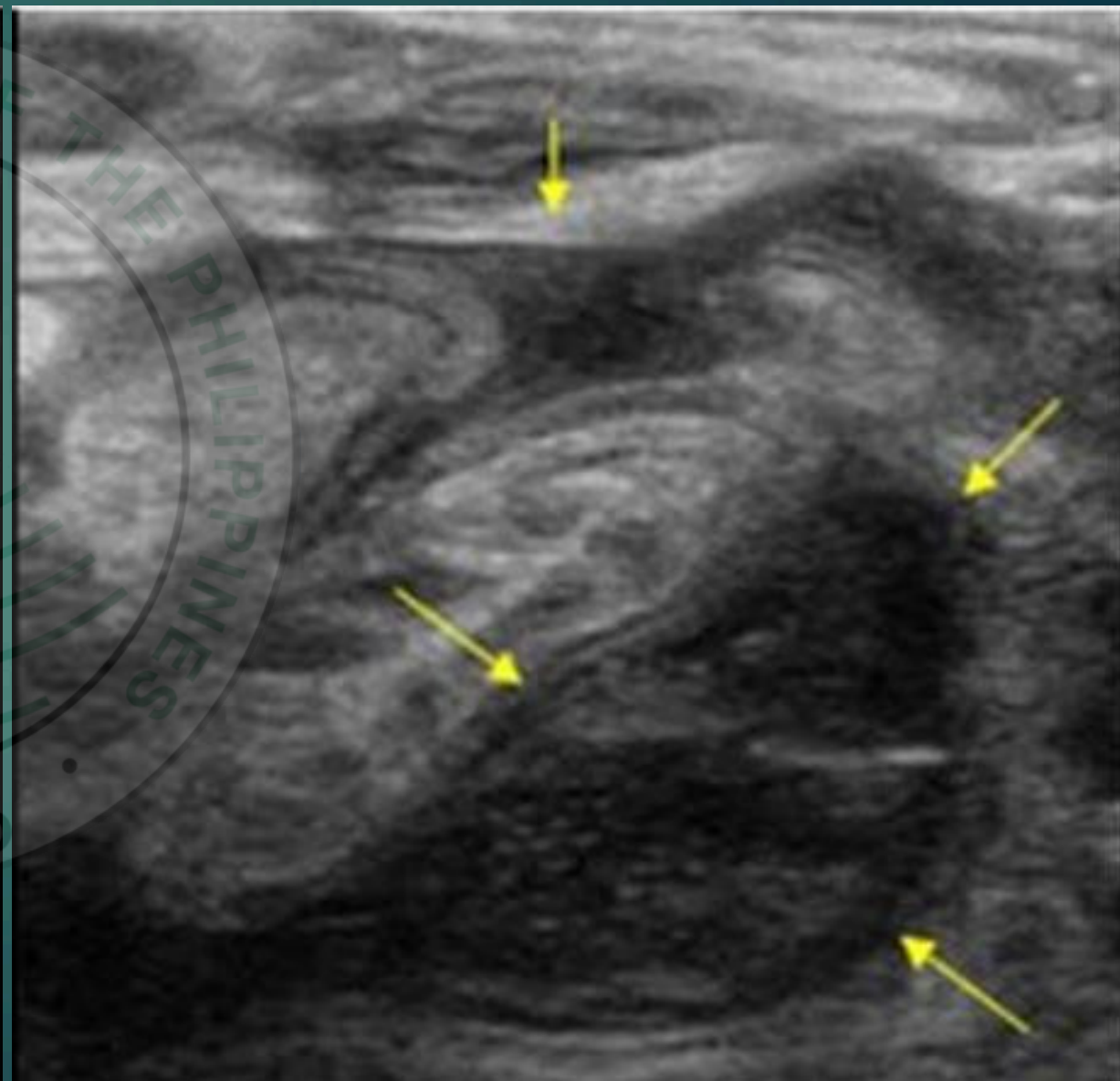


Acute appendicitis



Acute appendicitis





References

- Rumack CM et al. Diagnostic Ultrasound, 5th ed.
- Davamatira, D. (n.d.). Acute appendicitis. In Radiopaedia. Retrieved June 29, 2025, from https://radiopaedia.org/articles/acute-appendicitis-2?lang=us
- SonographicTendencies. (2016, December 7). Appendix ultrasound. Sonographic Tendencies. Retrieved June 29, 2025, from https://sonographictendencies.com/2016/12/07/appendix-ultrasound/
- Al-Marzooq, T. J. M., Hassan, Q. A., Majeed, G. H., Manea, A. N., & Abboud, A. H. (2018). The diagnostic accuracy of sonography, with graded compression, to image acute appendicitis compared to histopathologic results. Journal of Diagnostic Medical Sonography, 34(2), 85–89. https://doi.org/10.1177/8756479317712196

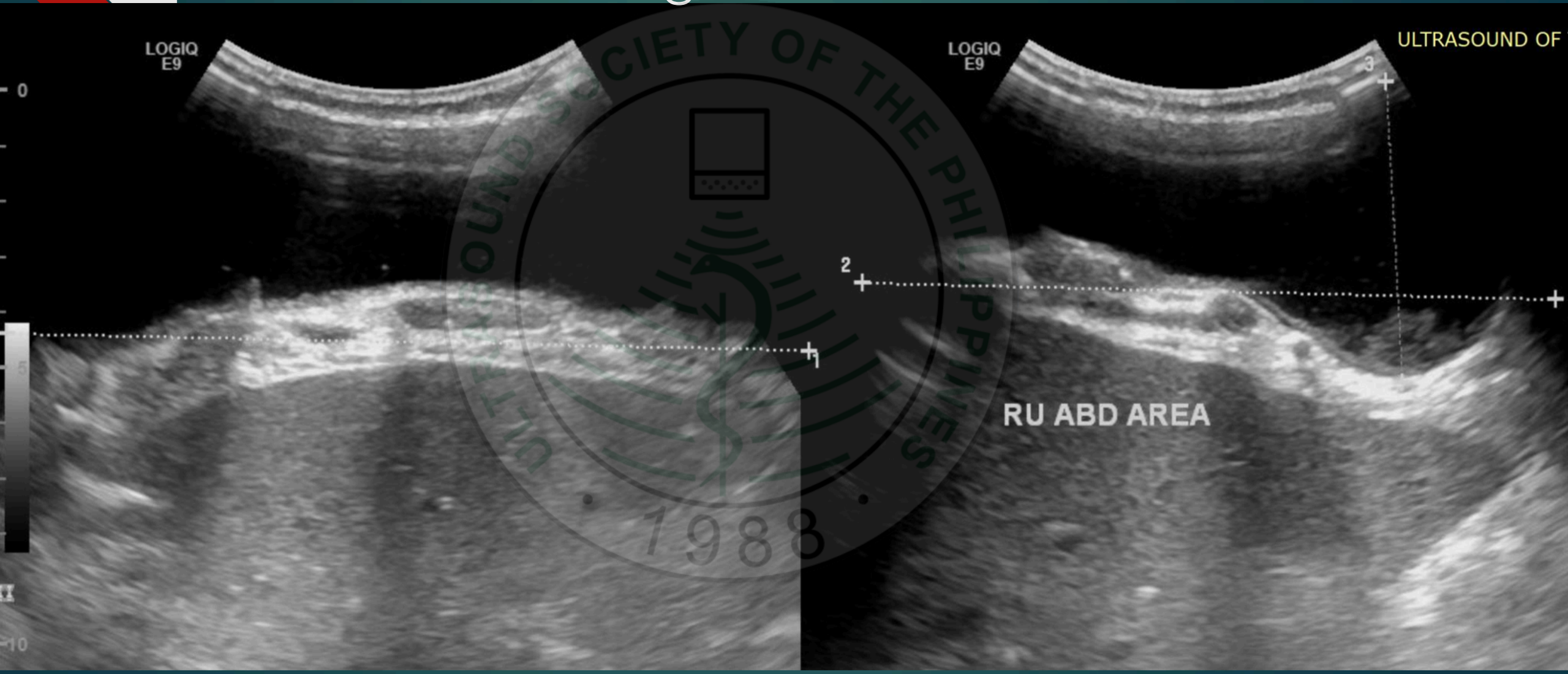
Mesenteric Cysts

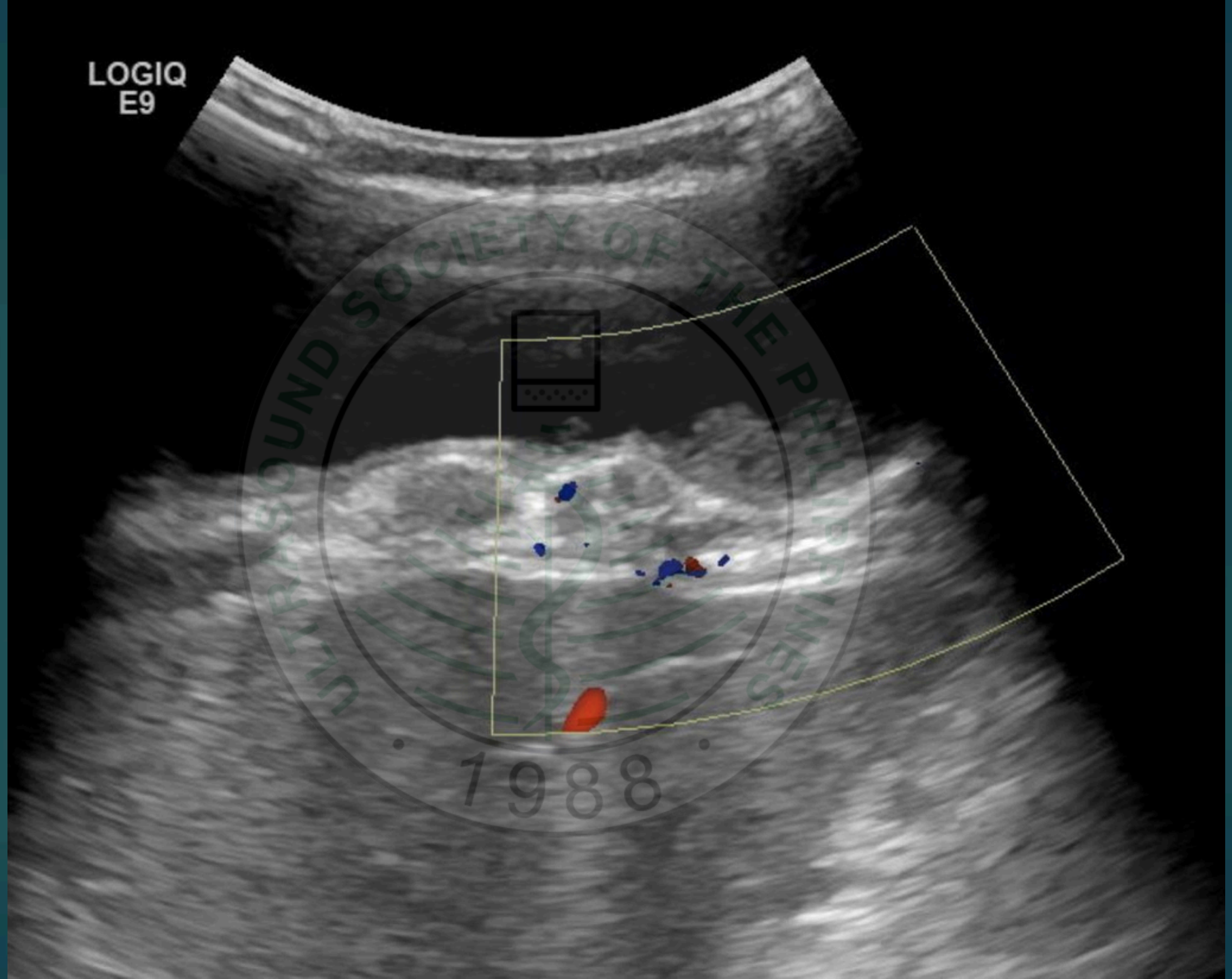
Interesting case

CASE

- 31/M comes in with a chief complaint of an enlarging palpable mass in the upper abdominal region. No other associated symptoms were noted. Past medical history was unremarkable. Initial sonographic assessment was ordered and an ultrasound of the upper abdomen was done.

Ultrasound findings




LOGIQ
E9
ULTRASOUND SOCIETY OF THE PHILIPPINES
1988

Mesenteric cysts

- Rare intraabdominal masses
- Reported incidence of 0.5-1 per 100,000-250,000 admissions
- Slight female predilection.
- May present as abdominal distention
- Vary in size
- Vary in composition
- Imaging via CT or US

Mesenteric cysts

Lymphatic: simple lymphatic cyst and lymphangioma

Mesothelial: simple mesothelial cyst, benign cystic mesothelioma, and malignant cystic mesothelioma

Enteric: enteric cyst and enteric duplication cyst

Urogenital

Non-pancreatic pseudocysts(infectious and traumatic cysts)

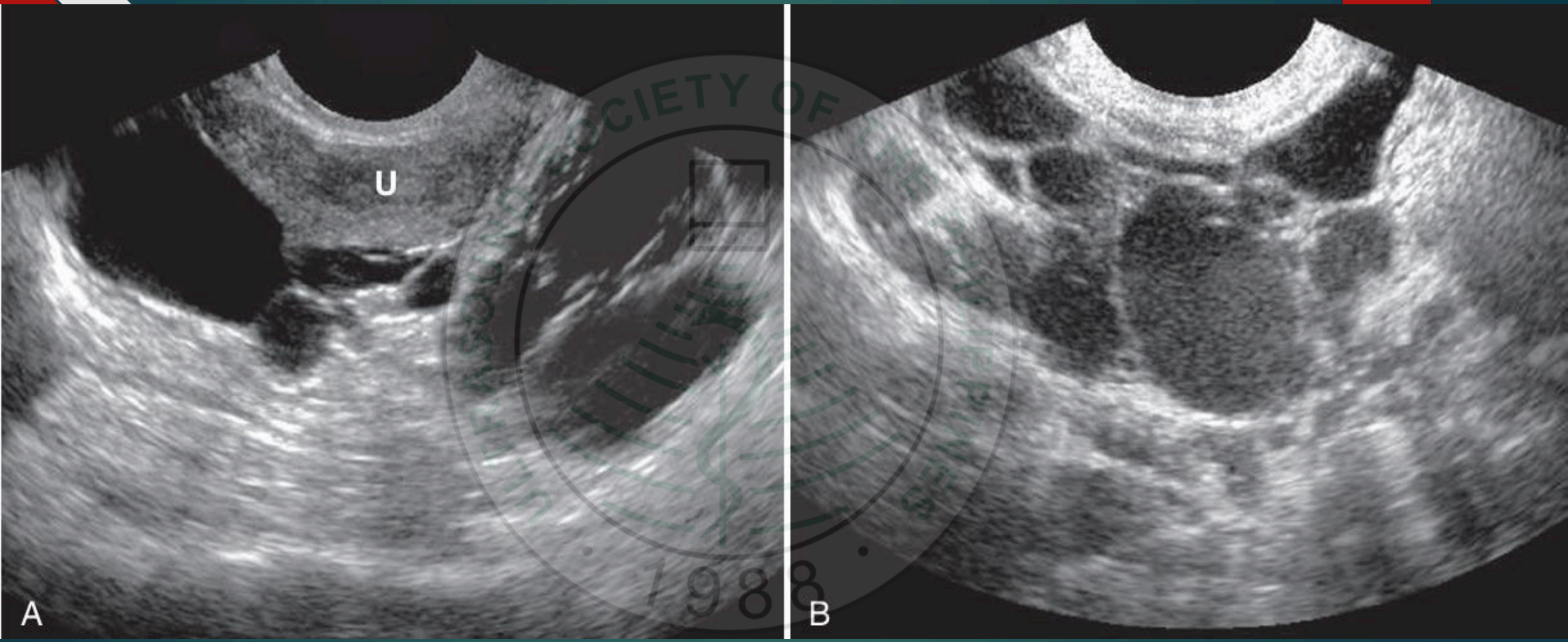
Lymphatic malformations (lymphangiomas)

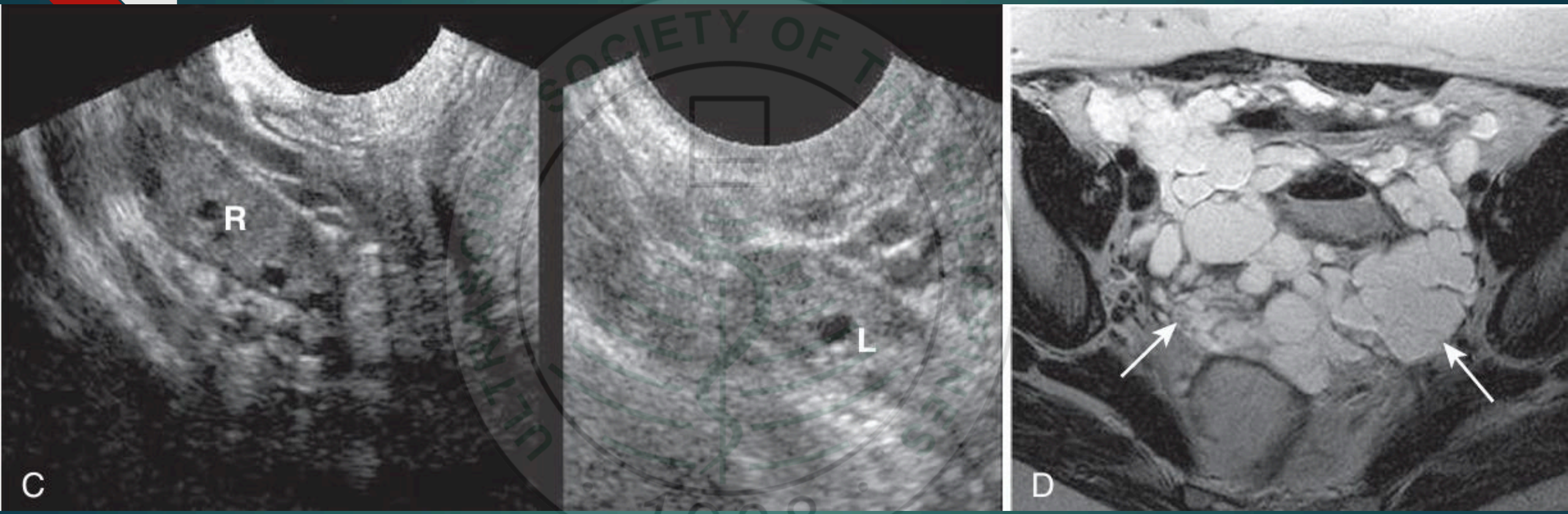
- One of the more common types of mesenteric cysts
- Benign lesions of vascular origin
- Vascular malformations rather than tumors
- They can present at any age but most often occur in the pediatric population

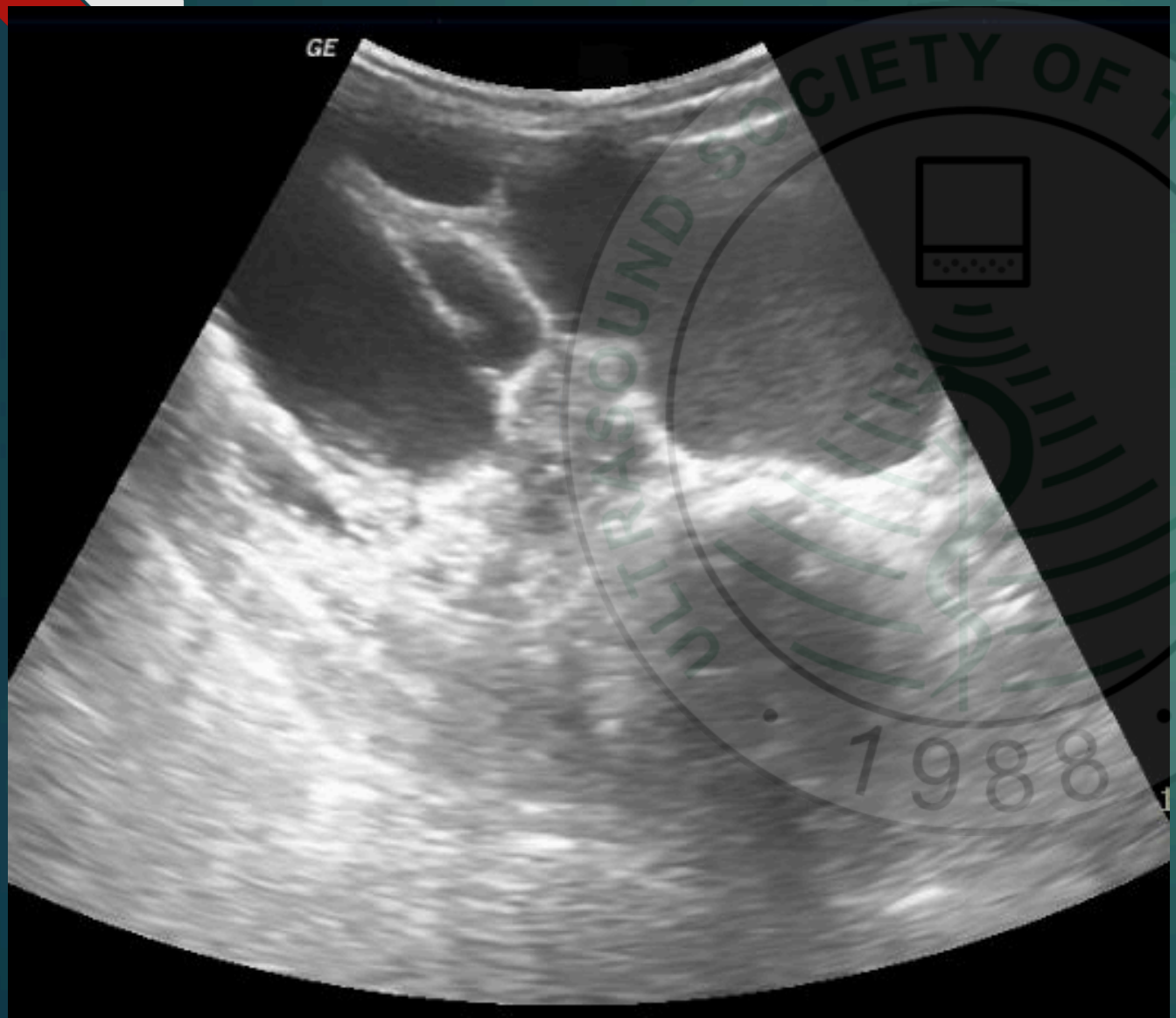
Lymphatic malformations (lymphangiomas)

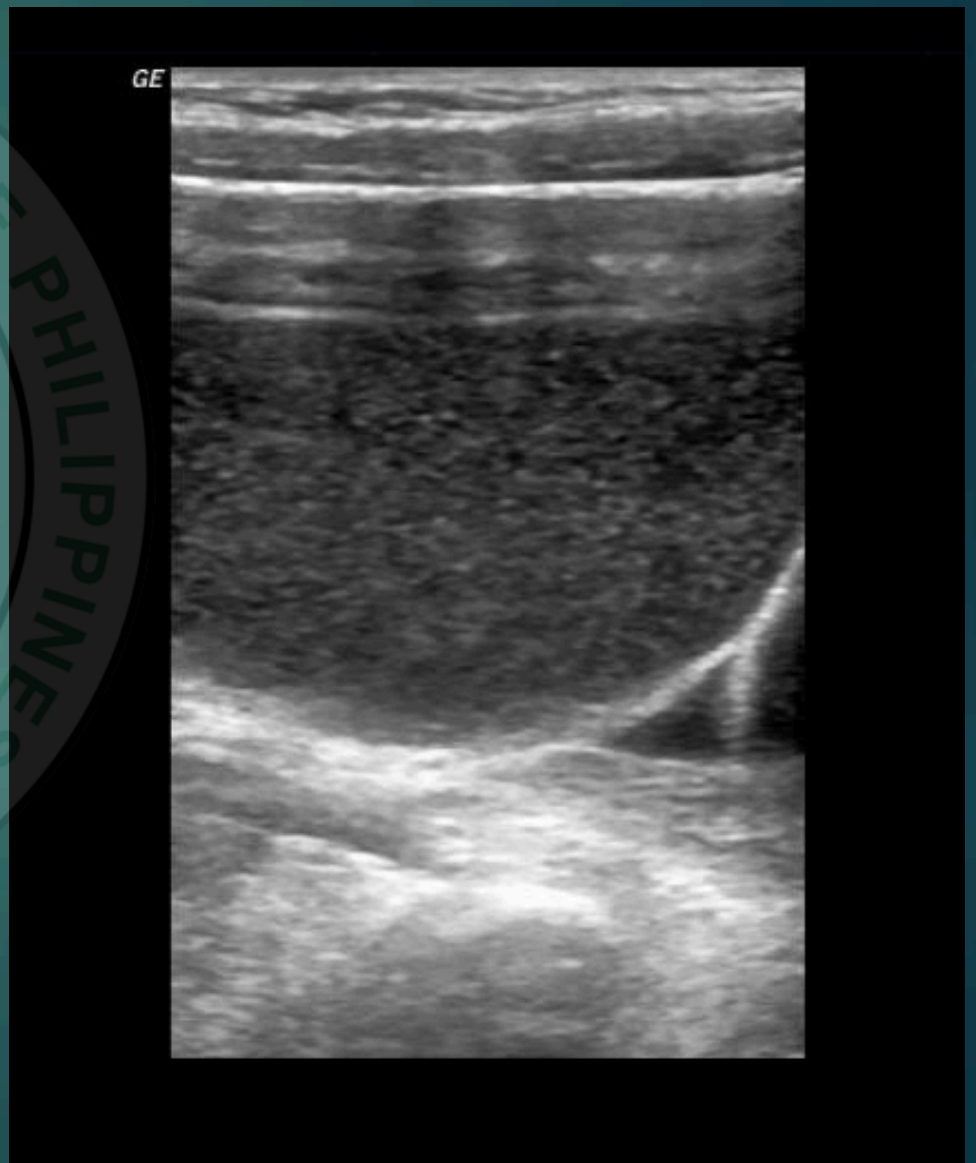
- Sonographic findings
  - multilocular cystic masses internal septa of varying thickness
  - cystic contents: usually anechoic; hyperechoic if contain debris, high lipid concentration, infection or hemorrhage
  - wide variations exist: solid areas, or mostly solid with cystic foci
  - color Doppler: +/- arterial or venous flow in the septa
  -
  -

Example images


U
A
B


R
L
C
D


GE


GE

Treatment and prognosis

- Conservative treatment
- Surgical intervention
- Malignant transformation
- Rare chance of recurrence once removed

References

- Rumack CM et al. Diagnostic Ultrasound, 5th ed.
- Pithawa, A.K., Bansal, A.S., & Kochar, S.P.S. (2014). Mesenteric cyst: A rare intra-abdominal tumour. Medical Journal Armed Forces India, 70(1), 79–82. https://doi.org/10.1016/j.mjafi.2012.06.010
- Radiopaedia. (n.d.). Lymphatic malformations. Retrieved June 29, 2025, from https://radiopaedia.org/articles/lymphatic-malformations-1
- Radiopaedia. (n.d.). Mesenteric cyst. Retrieved June 29, 2025, from https://radiopaedia.org/articles/mesenteric-cyst

THANK YOU!