DNB June 2020 – Paper 4

PART A

1. a) Rotating anode X-ray tube and its advantages. **(5)**
b) X-ray beam restricting devices and their uses. **(5)**

2. a. Classify water-soluble iodinated contrast media. **(2)**

b. Describe various adverse reactions to iodinated contrast media. **(3)**

c.Describe steps in the diagnosis and management of contrast media–induced anaphylaxis. **(2 + 3)**

3. Enumerate the principles of Computed Radiography (CR) and Digital Radiography (DR). **(3 + 3)** Advantages and disadvantages. **(2 + 2)**

4. a) Principle and technique of phase contrast MR angiography. **(2 + 2)**
b) Advantages, limitations, and clinical applications. **(2 + 2 + 2)**

5. a) Applications of dual energy CT. **(5)**
b) Ultrasound artifacts. **(5)**

PART B

6. Classify the commonly used embolizing agents. **(3)**
Advantages, disadvantages, and clinical applications. **(2 + 2 + 3)**

7. What is plagiarism in scientific writing? **(1)**
Common types of plagiarism. **(4)**
How plagiarism can be detected in scientific writings. **(2)**
Measures to avoid plagiarism. **(3)**

8. a) Predatory journals and publishing. **(5)**
b) Techniques of randomization of samples in a research study. **(5)**

9. a) PC-PNDT Act and its applications. **(5)**
b) TLD Badge: Principles and uses. **(5)**

10. a) Artificial Intelligence in radiology. **(5)**
b) PET MRI. **(5)**

Q1. Discuss the causes and radiological features of Budd Chiari Syndrome. Discuss the role of Interventional Radiology in treatment of Budd Chiari Syndrome.

Answer

Extrahepatic Portal Vein Obstruction (EHPVO) refers to **chronic obstruction of the extrahepatic portal vein**, leading to **portal hypertension and cavernous transformation** of the portal vein. It is a major cause of **non-cirrhotic portal hypertension**, particularly in **children and young adults**.
Imaging plays a crucial role in **diagnosing EHPVO**, assessing the **extent of portal vein obstruction**, detecting **collaterals, biliary involvement (portal biliopathy), and complications like varices and hypersplenism**.

Imaging Modalities for EHPVO

1. Ultrasound (USG) with Doppler – First-Line Modality

- **Most accessible and cost-effective** modality.
- Used for **initial diagnosis** and **follow-up** of EHPVO patients.
- Can differentiate between **acute and chronic portal vein thrombosis**.

2. Contrast-Enhanced CT (CECT) – Gold Standard for Evaluation

- Provides **detailed vascular anatomy**, **collateral pathways**, and **associated portosystemic shunts**.
- Evaluates **portal cavernoma transformation and secondary complications**.
- Helps in **pre-operative planning** for interventions like **shunt surgery**.

3. Magnetic Resonance Imaging (MRI) and MR Venography

- **Non-invasive** and excellent for **vascular mapping**.
- **MR Cholangiopancreatography (MRCP)** helps detect **biliary changes (portal biliopathy)**.
- Useful in **patients allergic to iodinated contrast**.

4. Catheter-Based Digital Subtraction Angiography (DSA) – Gold Standard for Interventions

- Used mainly in **pre-interventional planning** for **shunt procedures**.
- Helps assess **hemodynamics** and **collateral circulation**.

Ultrasound & Doppler Features of EHPVO

1. Absent or Attenuated Portal Vein Flow

- **Portal vein is non-visualized or replaced by fibrotic tissue.**
- **Doppler: No detectable hepatopetal flow in the main portal vein.**
- **Gray-scale USG:** Hypoechoic or echogenic thrombus in the portal vein (if acute).

2. Cavernous Transformation of the Portal Vein

- **Multiple small tortuous periportal collaterals** replace the normal portal vein.
- Appears as a **spongy, irregular, hypoechoic structure at the porta hepatis**.
- **Doppler flow:** Multiple low-velocity hepatopetal (towards the liver) flow signals.
- Often extends to **splenic vein, superior mesenteric vein (SMV)**.

3. Splenomegaly and Collateral Formation

- **Splenic enlargement (>12 cm)** due to portal hypertension.
- **Prominent portosystemic collaterals**, especially:
    - Esophageal and gastric varices
    - Splenorenal collaterals
    - Recanalized paraumbilical vein (caput medusae)

4. Increased Hepatic Arterial Flow (Compensatory Changes)

- Due to **reduced portal flow**, there is **increased hepatic artery velocity**.
- **Doppler:** High resistive index in hepatic artery, with increased peak systolic velocity.

5. Portal Biliopathy (Biliary Changes Due to Chronic Portal Hypertension)

- **Compression of bile ducts by venous collaterals** leads to:
    - Mild intrahepatic biliary dilatation
    - Irregular common bile duct (CBD)
    - No obstructing stone or mass

USG Doppler Patterns in EHPVO

Feature	USG Gray-Scale Findings	Doppler Findings
Normal Portal Vein	Anechoic tubular structure at porta hepatis	Hepatopetal monophasic flow

Acute PVT (Portal Vein Thrombosis)	Hypoechoic echogenic thrombus	No flow or partial flow in PV
Chronic EHPVO	Small or absent PV, replaced by multiple hypoechoic collaterals	Multiple slow-flow periportal collaterals (cavernous transformation)
Portal Biliopathy	Mild bile duct dilatation	No intraductal stones or mass

CT Imaging Features of Extrahepatic Portal Vein Obstruction (EHPVO)

CT Protocol for EHPVO Evaluation

A **multiphasic CT protocol** is essential to assess **vascular flow, collaterals, and complications**.

Phases of Contrast-Enhanced CT (CECT)

CT Phase	Timing	Findings in EHPVO
Non-contrast Phase	Pre-contrast	Detects thrombus (hyperdense in acute PVT, isodense in chronic cases).
Arterial Phase (20-30 sec post-injection)	Early contrast enhancement	Assesses hepatic arterial flow (compensatory increase in chronic EHPVO).
Portal Venous Phase (60-80 sec post-injection)	Peak portal vein enhancement	Detects **non-opacification of the portal vein** and cavernous transformation.
Delayed Phase (2-5 min post-injection)	Late vascular enhancement	Identifies slow-filling collaterals and delayed biliary enhancement in portal biliopathy.

CT Findings in EHPVO

1. Non-Visualization or Attenuation of the Portal Vein

- **Complete absence of normal portal vein opacification**.
- **Acute PVT** → Hyperdense thrombus in the portal vein (pre-contrast).
- **Chronic EHPVO** → Portal vein appears atrophic or completely replaced by **collaterals**.
- No flow-related contrast enhancement in the portal vein.

2. Cavernous Transformation of the Portal Vein

- **Numerous small, tortuous periportal venous collaterals** seen around the expected location of the **portal vein**.
- **Best seen in the portal venous phase** as a network of **low-attenuation serpiginous structures**.
- **Cavernoma replaces the thrombosed portal vein**.

3. Portosystemic Collaterals and Varices

- **Esophageal & Gastric Varices**
  - Dilated, tortuous submucosal veins in the **lower esophagus & gastric fundus**.
  - Can lead to **variceal bleeding**.
- **Splenorenal Shunt**
  - Large venous collateral connecting the **splenic vein** to the **left renal vein**.
  - May lead to **hepatic encephalopathy**.
- **Recanalized Paraumbilical Vein (Caput Medusae)**
  - Dilated **paraumbilical veins tracking along the ligamentum teres**.
  - Seen in **severe portal hypertension**.
- **Retroperitoneal & Mesenteric Collaterals**
  - Prominent **lumbar veins, omental veins, and mesenteric varices**.

4. Splenomegaly and Hypersplenism

- **Massive splenomegaly (>15 cm)** due to chronic portal hypertension.
- **Hypersplenism features:**
  - **Thrombocytopenia**
  - **Leukopenia**
  - **Anemia**
- Splenic infarcts may be seen in **acute PVT**.

5. Portal Biliopathy (Biliary Changes Due to Chronic Portal Hypertension)

- **Compression of the bile ducts by cavernous transformation**.
- **CT findings:**
  - Mild **dilation of intrahepatic bile ducts**.
  - Irregular **narrowing of the common bile duct (CBD)**.
  - No obstructing mass or stones (differentiates from malignancy).

🔹 **Best assessed using MRCP (Magnetic Resonance Cholangiopancreatography).**

MRI Imaging Features of Extrahepatic Portal Vein Obstruction (EHPVO)

MRI Protocol for EHPVO

MRI Sequences Used

Sequence	Purpose
T1-Weighted Imaging (T1WI)	Evaluates liver parenchyma and detects thrombus (hyperintense in subacute cases).
T2-Weighted Imaging (T2WI)	Highlights periportal collaterals (hyperintense).
Dynamic Contrast-Enhanced MRI (DCE-MRI)	Assesses vascular flow in portal phase.
MR Venography (MRV)	Detects absent portal vein and cavernous transformation.
MRCP (Magnetic Resonance Cholangiopancreatography)	Evaluates biliary abnormalities (portal biliopathy).

MRI & MRV Findings in EHPVO

1. Absence of Normal Portal Vein Flow

- **Portal vein not visualized or replaced by collaterals.**
- **MRV shows complete occlusion or near-total attenuation of the main portal vein**.

2. Cavernous Transformation of the Portal Vein

- **Multiple tortuous venous collaterals** replacing the thrombosed portal vein.
- **T2-weighted imaging**: Hyperintense periportal structures (representing small collateral vessels).
- **MRV**: Multiple small periportal collaterals forming a network around the expected portal vein location.

3. Portosystemic Collaterals and Varices

- **Esophageal and gastric varices** seen as dilated serpiginous structures in the **lower esophagus and stomach wall**.
- **Splenorenal shunt**: Large tortuous venous connection between **splenic and left renal veins**.
- **Recanalized paraumbilical vein**: Tortuous veins along the anterior abdominal wall (**Caput Medusae**).
- **Retroperitoneal and mesenteric collaterals** visible on MRV.

4. Splenomegaly and Hypersplenism

- **Enlarged spleen (>15 cm) with multiple dilated splenic veins**.

- **Findings of hypersplenism** (anemia, thrombocytopenia, leukopenia).

MRCP Findings in Portal Biliopathy

Portal biliopathy (biliary changes due to chronic portal hypertension) occurs due to **compression of the bile ducts by dilated periportal collaterals**.

MRCP Features:

- **Irregular strictures of the common bile duct (CBD)**.
- **Mild intrahepatic biliary dilatation**.
- **Tortuous periportal collaterals seen adjacent to bile ducts**.
- **No mass lesion or stones (differentiates from cholangiocarcinoma or primary sclerosing cholangitis).**

Digital Subtraction Angiography (DSA) in Extrahepatic Portal Vein Obstruction (EHPVO)

Digital Subtraction Angiography (DSA) is an **invasive imaging modality** used primarily in **interventional planning** for EHPVO. While **Ultrasound, CT, and MRI** are the main diagnostic tools, **DSA remains the gold standard for vascular interventions**, including **portosystemic shunt procedures and transhepatic recanalization**.

DSA provides **real-time hemodynamic assessment of the portal circulation** and helps in **evaluating collateral pathways, measuring pressure gradients, and guiding therapeutic procedures**.

Role of DSA in EHPVO

1. Diagnostic Role

- Confirms **absence of portal vein flow**.
- Maps **portosystemic collaterals and shunt pathways**.
- Evaluates **portal pressures and pressure gradients**.

2. Interventional Role

- **Balloon angioplasty** and **stenting of portal vein**.
- **Direct intrahepatic portosystemic shunt creation (TIPS alternative in EHPVO).**
- **Embolization of large portosystemic shunts to prevent encephalopathy.**
- **Surgical planning for meso-Rex shunt or splenorenal shunt.**

DSA Findings in EHPVO

1. Non-Filling of the Portal Vein

- **No contrast opacification of the portal vein** in the expected anatomical location.
- **Collateral circulation is seen instead of the normal portal vein.**

2. Cavernous Transformation of the Portal Vein

- Numerous **small tortuous venous collaterals** replace the expected portal vein.
- These collaterals form a **low-flow venous network** at the porta hepatis.

3. Portosystemic Collaterals

- **Esophageal and gastric varices** are visualized during contrast injection.
- **Splenorenal shunt** is identified as an enlarged **venous connection between the splenic and left renal veins**.
- **Recanalized paraumbilical vein (Caput Medusae)** shows contrast draining into the systemic circulation.

4. Increased Hepatic Arterial Flow

- **Compensatory arterialization of the liver** is observed in chronic EHPVO.
- DSA may show **hypertrophied hepatic arteries supplying the liver in the absence of normal portal flow.**

Role of DSA in Interventions

1. Transjugular Intrahepatic Portosystemic Shunt (TIPS)

- **Usually not feasible in EHPVO** due to portal vein thrombosis.
- DSA confirms **lack of portal vein continuity**, making alternative shunts necessary.

2. Percutaneous Transhepatic Portal Vein Recanalization

- **Balloon angioplasty or stent placement** can restore portal venous flow in selected cases.
- Used in **acute or subacute EHPVO** before cavernous transformation occurs.

3. Meso-Rex Bypass Planning

- DSA is crucial for **planning Rex shunt surgery** in pediatric patients.
- Helps determine **feasibility of mesenteric-portal vein bypass**.

4. Embolization of Portosystemic Shunts

- **Large spontaneous portosystemic shunts (e.g., splenorenal shunt)** can cause **hepatic encephalopathy**.

- **DSA-guided embolization** redirects blood flow back to the liver, improving metabolic function.

Q2. a) Imaging workup of a potential renal donor.
b) Imaging evaluation of vascular complications of a transplant kidney.

Answer

Evaluation of a Potential Renal Donor

- **Purpose**: Imaging in living renal donor evaluation is critical to:
    1. Confirm two normally functioning kidneys.
    2. Document detailed vascular anatomy (arterial & venous).
    3. Assess collecting system configuration.
    4. Detect any pathology that may alter surgical decision-making.
- **Gold Standard**: **Multidetector Computed Tomography (MDCT) Angiography** — high spatial resolution, rapid acquisition, multiplanar and 3D reconstructions.

Imaging Goals in Donor Assessment

1. **Anatomy** – Renal size, morphology, cortical thickness.
2. **Vascular Mapping** – Number, origin, and branching pattern of arteries and veins; presence of anomalies.
3. **Collecting System** – Configuration, duplication, strictures, stones.
4. **Parenchymal Pathology** – Masses, cysts, scarring.
5. **Functional Assessment** – Split renal function to determine side for donation.

Imaging Workflow

1. Ultrasound (Screening)

- **Advantages**: Non-invasive, widely available, no radiation.
- **Role**:
  - Detect cysts, masses, hydronephrosis, or stones.
  - Measure kidney size and cortical thickness.
- **Limitations**: Poor for vascular mapping and collecting system evaluation.

2. Nuclear Medicine (Split Renal Function)

- **Common Tracers**: ^99mTc-DTPA, ^99mTc-MAG3.
- **Indication**: Ensures both kidneys have adequate function — usually accept if each kidney has ≥35–40% relative function.
- **Benefit**: Functional confirmation before deciding which kidney to remove.

3. Multidetector CT Angiography (Mainstay)

- **Rationale**:
  - Comprehensive evaluation of vasculature, parenchyma, and collecting system.
  - Fast, high-resolution, multiplanar imaging.
- **Phases**:
  - **Non-contrast phase** – Baseline attenuation, detection of stones, calcifications.
  - **Arterial phase** – Mapping of main and accessory renal arteries, early branching.
  - **Nephrographic phase (70 sec)** – Uniform parenchymal enhancement, mass detection.
  - **Delayed excretory phase (5 min)** – Collecting system anatomy, ureter course.
- **Reconstruction Techniques**:
  - **Axial thin sections** (0.6–0.8 mm) for detailed anatomy.
  - **Multiplanar reformations (MPR)** in coronal/sagittal planes.
  - **Curved planar reformations** for vessel course.
  - **Maximum Intensity Projection (MIP)** for vascular visualization.
  - **3D Volume Rendering** for surgical planning.

4. MR Angiography (Alternative)

- **Indications**:
  - Iodinated contrast allergy.
  - Borderline renal function where contrast risk outweighs benefit.
- **Sequences**:
  - 3D contrast-enhanced MRA.
  - Non-contrast time-of-flight or phase-contrast MRA.

- **Limitations**: Slightly less spatial resolution than CT; poor calcification detection.

MDCT Protocol (Example – Living Donor)

Parameter	Value
Tube Voltage	120 kVp
Effective mA	180
Rotation Time	0.5 s
Detector Collimation	32 × 0.6 mm
Slice Thickness	0.6 mm
Table Feed	23 mm
Kernel	B30f medium smooth
Oral Contrast	1000 mL water
IV Contrast	120 mL + 40 mL saline @ 4 mL/s
Bolus Tracking	100 HU at aorta + 4 s delay
Phases	Basal → Arterial → Nephrographic → Delayed
Reconstructions	Axial (0.8 & 5 mm), Coronal (5 mm), Sagittal length, Curved MPR, Thin MIP, 3D VR

Imaging Checkpoints

A. Vascular Anatomy

Arteries:

- **Normal**: Single artery per kidney.
- **Variants**:
  - Accessory renal arteries (polar arteries).
  - Early branching (<2 cm from origin).
  - Aberrant origin (e.g., iliac artery).
- **Relevance**: Multiple arteries increase surgical complexity, affect anastomosis time.

Veins:

- **Normal**: Single renal vein draining into IVC.
- **Variants**:
  - Multiple veins.
  - Retroaortic left renal vein.
  - Circumaortic left renal vein.
- **Relevance**: Affects surgical dissection and ligation.

B. Parenchyma

- Symmetry in size and enhancement.
- Exclude:
    - Masses (solid or complex cystic lesions).
    - Scarring or cortical thinning.
    - Nephrolithiasis.

C. Collecting System

- Use delayed phase to assess:
    - Duplication anomalies.
    - Narrowing or obstruction.
    - Stones.
- **3D excretory reconstruction** useful for surgical roadmap.

Choosing the Kidney for Donation

- **General Rule**: Prefer **left kidney** due to longer renal vein.
- **Exceptions**:
    - Poorer function in left kidney.
    - Complex arterial anatomy on left side (multiple/early branches).
    - Pathology confined to one side.

Radiology in Renal Donor Assessment & Post-Transplant Vascular Complications

I. Pre-Transplant Renal Assessment

A. Parenchymal Evaluation

Objective: Identify anatomical suitability and exclude pathology that precludes donation.

Imaging Modalities:

- **Ultrasound (US)** – Initial screening, parenchymal echotexture, masses, stones, scars.
- **MDCT Angiography** – Gold standard for full anatomical mapping.
- **MR Angiography** – Alternative if contrast contraindicated.
- **Nuclear Medicine Renogram** – Functional split assessment.

Parameters Assessed:

- **Number** – Confirm two kidneys; rule out unilateral agenesis.
- **Length** – Size symmetry; small kidneys may indicate hypoplasia or scarring.
- **Location** – Identify ectopia (pelvic kidney) or fusion anomalies (horseshoe kidney).
- **Variants** – Duplication, malrotation, abnormal hilum orientation.
- **Parenchymal Diseases** –
    - Exclude: Unilateral agenesis, horseshoe kidney, cortical atrophy, autosomal dominant polycystic kidney disease (ADPKD), medullary sponge kidney, renal papillary necrosis.
    - Conditional:
        - **Renal ectopia** or **UPJ stenosis** — acceptable if surgically correctable and without significant vascular anomaly.
        - **Unilateral small scars** with normal renogram — scarred kidney is preferred for removal.

B. Renal Artery Anatomy & Variants

Normal: Single main renal artery per side, branching ~2 cm from origin.

Variants (seen in up to 25–30% of donors):

- **Accessory arteries** – Polar arteries (superior/inferior).
- **Early branching** – Branching within 2 cm of origin; important for clamping and anastomosis.
- **Aberrant origin** – Arising from iliac or mesenteric arteries.
  Imaging Goal: Detect all variants; multiple arteries increase surgical complexity.

C. Renal Vein Anatomy & Variants

Normal: Single renal vein draining into IVC.

Variants:

- **Multiple renal veins**.

- **Retroaortic left renal vein**.
- **Circumaortic left renal vein**.
  Relevance: Anomalies affect surgical dissection and venous anastomosis.

II. Post-Transplant Vascular Complications

Most occur early in the post-transplant period; imaging, especially **Doppler US**, is first-line due to non-invasiveness and high sensitivity.

1. Renal Artery Stenosis (RAS)

- **Timing**: Usually within first 3 months.
- **Causes**: Atherosclerosis (donor or recipient), technical error at anastomosis, vascular injury.
- **Clinical**: Hypertension ± renal function deterioration; bruit over graft site.
- **Doppler US Findings**:
  - **Focal color aliasing** at stenosis site.
  - **PSV > 200 cm/s** at stenosis.
  - **Velocity ratio > 2:1** stenotic:pre-stenotic segment.
  - **Tardus-parvus waveform** downstream (↓ acceleration, ↓ PSV).
- **Other Imaging**: CTA/MRA for surgical/endovascular planning.

2. Renal Artery Thrombosis

- **Causes**: Vessel kinking, dissection, acute rejection, hypercoagulable states.
- **Clinical**: Oliguria, rapid rise in creatinine.
- **Imaging**:
  - **US**: Absence of arterial flow; echogenic thrombus.
  - **CTA/MRA**: Filling defect with non-opacification of artery ± branches.

3. Renal Vein Thrombosis

- **Incidence**: 0.5–4% of grafts.
- **Causes**: Venous torsion, compression, surgical technical error.
- **Clinical**: Pain, swelling over graft, ipsilateral leg swelling, fever.
- **Imaging**:
  - **US**: Absence of venous flow; enlarged hypoechoic kidney; reversed diastolic arterial flow on Doppler.

4. Pseudoaneurysm

- **Cause**: Usually biopsy-related arterial wall injury.
- **Doppler**: “Yin-yang” swirling pattern within sac.
- **Management**: Small lesions → observe; larger → thrombin injection or endovascular coil.

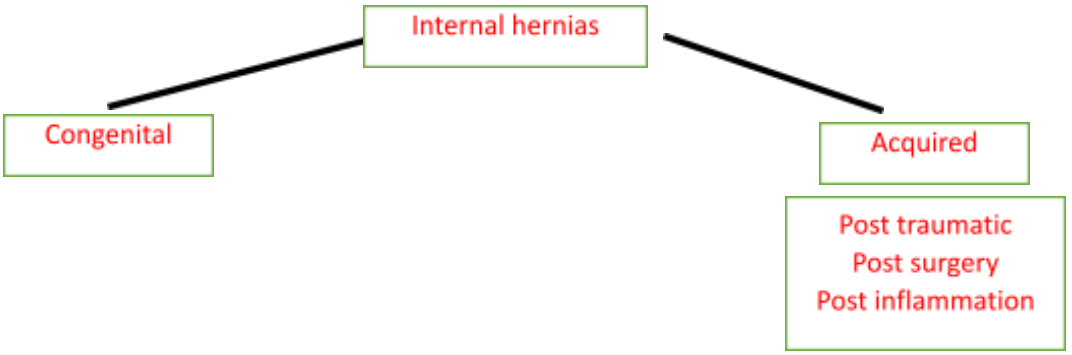
5. Arteriovenous Fistula (AVF)

- **Cause**: Iatrogenic (biopsy needle penetrates artery & vein).
- **Doppler**:
    - Color aliasing at site.
    - Spectral waveform shows high-velocity, low-resistance arterialized venous flow.
- **Course**: Often self-resolving; persistent large fistulas may require embolization.

Q3. Enumerate various internal hernias. Discuss in detail imaging features of any two internal hernias.

Answer

- Internal hernia is the protrusion of a viscus through a normal or abnormal aperture in the peritoneum
  or mesentery, within the confines of the peritoneal cavity.



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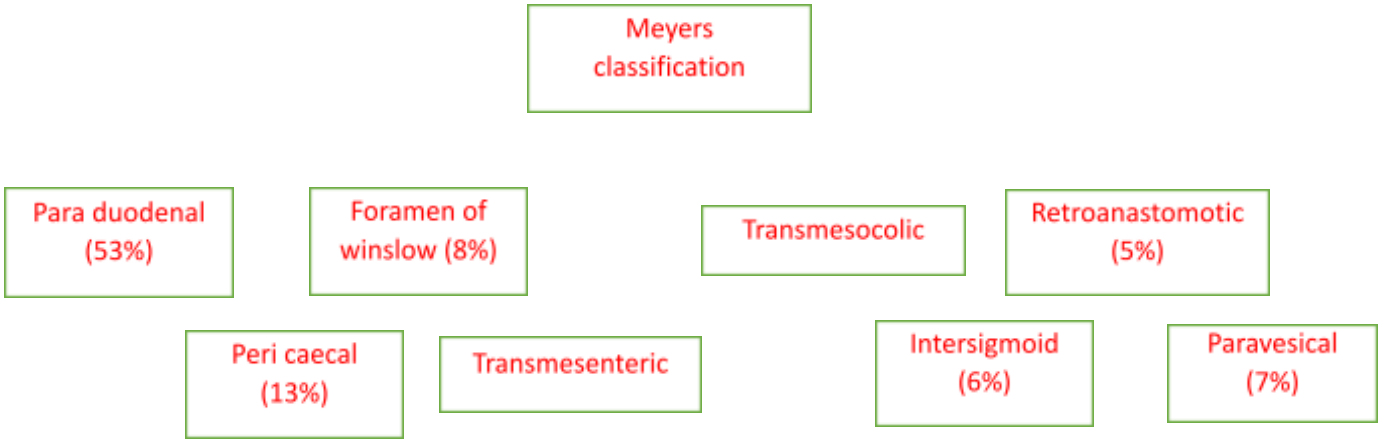

Types of Internal Hernias

Type	Etiology	Site	Findings on Plain Radiographs / Barium Studies	CT Findings
Left Paraduodenal	Congenital	Through Landzert's fossa, behind the 4th part of the duodenum	Cluster of jejunal loops in left hypochondrium, lateral to the 4th part of duodenum; may cause mass effect on posterior stomach wall or displace transverse colon inferiorly	Encapsulated cluster of dilated small bowel between stomach & pancreas, behind pancreas, or between transverse colon & left adrenal gland; inferior mesenteric vein displaced superoanteriorly at neck of hernia sac
Right Paraduodenal	Congenital	Through Waldeyer's fossa (defect in first part of jejunal mesentery)	Clustered loops lateral & inferior to 2nd part of duodenum; associated with small-bowel nonrotation	Encapsulated cluster lateral & inferior to 2nd part of duodenum; small-bowel nonrotation; superior mesenteric artery displaced anteriorly
Pericecal	Congenital / Acquired	Through defect in cecal mesentery into right paracolic gutter	Clustered distal ileal loops posterior & lateral to cecum in right paracolic gutter	Same as plain film: clustered distal ileal loops posterior & lateral to cecum in right paracolic gutter
Foramen of Winslow	Congenital	Through foramen of Winslow	Circumscribed loops medial & posterior to stomach	Loops in lesser sac between liver & inferior vena cava
Intersigmoid	Congenital / Acquired	Fossae or defects in/around sigmoid mesocolon	U- or C-shaped cluster of small bowel posterior & lateral to sigmoid colon	Same as plain film: U- or C-shaped cluster posterior & lateral to sigmoid colon

Trans - mesenteric	- Congenital in children (defect in small bowel mesentery) - Acquired in adults (post-abdominal surgery)	Defect in small bowel mesentery	Variable; in postoperative cases, air within gastric remnant; may mimic left paraduodenal hernia	Small bowel lateral to colon; displaced omental fat with small bowel abutting abdominal wall
Retro - anastomotic	Acquired	Through surgical anastomosis defect	Variable	Variable

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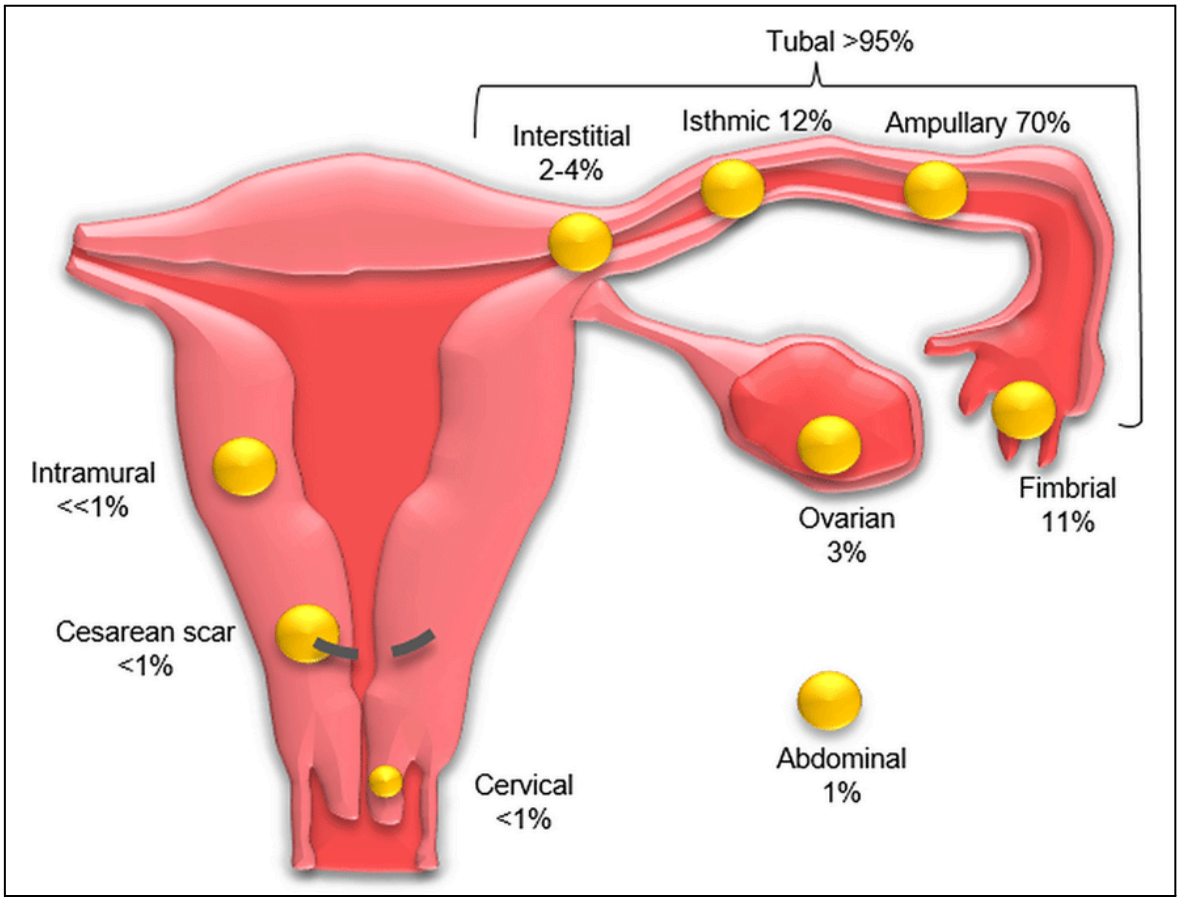
Q4. Describe imaging algorithm in a suspected case ectopic pregnancy. Describe role of imaging in differentiating three common clinical mimics of ectopic pregnancy.

Answer



Ectopic pregnancy refers to **implantation of a fertilized ovum outside the uterine endometrial cavity**, most commonly within the **fallopian tube (95–98%)**. It accounts for **2% of all pregnancies** and remains the **leading cause of maternal death in the first trimester**, primarily due to hemorrhage from rupture.
Early radiologic diagnosis is essential to reduce morbidity and mortality, guide treatment (medical vs. surgical vs. expectant), and preserve future fertility.

Clinical and Diagnostic Approach

Clinical Red Flags

- **First trimester bleeding and/or lower abdominal pain**
- **Positive pregnancy test (β-hCG)**
- **No intrauterine pregnancy (IUP) on ultrasound**
  In ART (Assisted Reproductive Technology) populations, the risk of heterotopic pregnancy rises up to **1 in 100**, compared to **1 in 30,000** in spontaneous pregnancies.

Role of β-hCG in Imaging Evaluation

Serum **β-hCG levels** are crucial in determining whether an intrauterine gestational sac should be visible and guiding interpretation of sonographic findings.

β-hCG (IRP)	Ultrasound Correlation
<1500 mIU/mL	IUP may not yet be visible – indeterminate
1500–2000 mIU/mL	*Discriminatory zone*: IUP may start appearing via TVS
>2000 mIU/mL	**Gestational sac should be visible** on transvaginal US

Absence of IUP when β-hCG > 2000 mIU/mL → **strongly suggestive of ectopic pregnancy or recent spontaneous abortion**.

Imaging Modalities

Modality	Indication/Strengths
Transvaginal US (TVS)	Modality of choice: best resolution for endometrial and adnexal structures
Transabdominal US	Supplemental; better for hemoperitoneum, obese patients, large field of view
MRI	Problem-solving tool in equivocal cases (e.g., interstitial/cervical/abdominal ectopics)
CT (rarely used)	May reveal hemoperitoneum in undiagnosed ruptured ectopics in emergency settings

Normal First Trimester Sonographic Landmarks

Gestational Age (LMP)	Finding	Modality
4.5–5 weeks	Intradecidual sign	TVS
~5 weeks	Gestational sac, possibly double sac sign	TVS
5.5 weeks	Yolk sac visible	TVS
6 weeks	Embryonic pole + cardiac activity	TVS, M-mode

Double sac sign = Two concentric echogenic rings: **decidua capsularis** and **decidua parietalis**

Imaging Features of Ectopic Pregnancy

1. Extrauterine Findings (most to least specific)

Sign	Description
Live embryo in adnexa	Extrauterine cardiac activity (100% specific)
Tubal ring sign	Echogenic ring with or without yolk sac, often medial to ovary
Complex adnexal mass	Heterogeneous mass separate from ovary
Ring of fire sign (Doppler)	Peripheral vascularity – seen in both corpus luteum and ectopic
Free fluid (echogenic)	Suggestive of hemoperitoneum, may imply rupture

2. Intrauterine Mimics

Entity	Distinguishing Feature
Pseudogestational sac	Central location, single layer, no yolk sac, no double decidual sign
Decidual cysts	Peripheral, multiple, thin-walled, subendometrial location

Special Ectopic Pregnancy Locations

Tubal Ectopic Pregnancy

- **Most common** (95–98%) – ampullary > isthmic > fimbrial
- **Classic appearance**: Adnexal mass with yolk sac ± fetal pole
- **Tubal ring** often noted; use sliding organ sign to assess mobility

Interstitial (Cornual) Pregnancy

- **Located in intramural part of tube**
- **Risk of catastrophic rupture** due to late presentation (8–16 weeks)
- **Signs**:
    - Gestational sac >1 cm from uterine cavity
    - **Interstitial line sign** (echogenic line from cavity to sac)
    - <5 mm myometrial mantle surrounding sac

Cervical Ectopic Pregnancy

- **Gestational sac in cervical canal**
- Risks severe bleeding if curettage attempted
- **Sliding organ sign** helps differentiate from abortion in progress
- Treatment: Methotrexate or KCl injection

Cesarean Scar Pregnancy

- **Implantation in lower uterine segment scar**
- Myometrial thinning (<2 mm) anteriorly
- High risk of **placenta accreta spectrum (PAS)** if pregnancy continues
- MR imaging can aid in diagnosis

Ovarian Ectopic

- Rare (0.5–3% of ectopics)
- Mass within ovary that doesn't move separately
- Often mistaken for hemorrhagic corpus luteum

Abdominal Ectopic

- Very rare; often implanted on bowel/omentum
- Diagnosis is difficult; often requires MRI or surgical confirmation

Heterotopic Pregnancy

- Coexisting IUP + ectopic
- More common with **ART**
- Presence of IUP should **not exclude** ectopic – thorough adnexal evaluation essential

Pregnancy of Unknown Location (PUL)

Occurs when:

- β-hCG is positive
- No intrauterine gestation
- No ectopic mass on US

Likely Diagnosis	Based on hCG level
Early viable IUP	<2000 mIU/mL
Ectopic pregnancy	>2000–3000 mIU/mL with no IUP
Spontaneous abortion	Falling hCG + empty uterus

Expectant management may be considered if patient is stable, hCG <200, and no adnexal mass or fluid.

Management Based on Imaging & Clinical Findings

Finding	Management
Live ectopic, >3.5 cm, unstable, cardiac activity	Surgery (laparoscopy/laparotomy)

Mass <3.5 cm, hCG <4000, no cardiac activity	Methotrexate (single or multi-dose)
Pregnancy of unknown location, low hCG	Expectant with β-hCG and US follow-up
Scar, cervical, or interstitial ectopics	Local MTX/KCl injection or preop embolization

Methotrexate Contraindications

- Active pulmonary disease, PUD, liver/kidney dysfunction, immunosuppression, cardiac activity (relative)

Q5. Discuss the classification and imaging features of cystic neoplasms of pancreas.

Answer

Cystic pancreatic tumors represent a **heterogeneous group** of lesions with variable **histology, malignant potential, and prognosis**.
Many are incidentally detected on **cross-sectional imaging**, particularly due to increased use of high-resolution **MDCT and MRI**.

CLASSIFICATION

1. By Incidence:

Common Tumors	**Rare Tumors**
- Serous cystadenoma (SCA)	- Cystic lymphangioma
- Mucinous cystic neoplasm (MCN)	- Cystic teratoma
- Intraductal papillary mucinous neoplasm (IPMN)	- Paraganglioma
- Solid pseudopapillary neoplasm (SPN)	- Cystic degeneration in NETs, adenocarcinoma, metastases

2. Morphologic Classification (Imaging-based):

Type	**Imaging Features**	**Examples / DDx**
Unilocular Cysts	- Single, thin-walled cyst - No internal septations, mural nodules, or solid components	- **Pancreatic pseudocyst** (post-pancreatitis) - **Early IPMN** (side-branch type)
Microcystic Lesions	- >6 **tiny cysts (<2 cm)** - **Central stellate scar** ± coarse calcification - Honeycomb appearance on MRI/CT	- **Serous cystadenoma (SCA)**
Macrocystic Lesions	- <6 **larger cysts (>2 cm)** - Smooth external contour - May have peripheral septations	- **Mucinous cystic neoplasm (MCN)** - **Side-branch IPMN**

Cysts with Solid Component	- **Mural nodule** or **enhancing solid part** within cyst - May show diffusion restriction or vascularity	- **Solid pseudopapillary neoplasm (SPN)** - **Cystic neuroendocrine tumor (NET)** - **Malignant MCN/IPMN**

MODALITY-SPECIFIC IMAGING

Modality	Utility
USG	Initial detection; can assess wall, septae, and Doppler vascularity; operator dependent
MDCT	Workhorse modality; assesses cyst morphology, enhancement, septae, nodules, calcification
MRI / MRCP	Superior soft-tissue contrast, internal architecture, and ductal communication; no radiation
EUS ± FNA	For indeterminate lesions or fluid analysis; detects mural nodules, ductal communication

Common cystic tumors

1. **Serous Cystic Neoplasm (SCN) of Pancreas**
    - **A benign exocrine pancreatic neoplasm.**
    - **Accounts for ~1–2% of all pancreatic tumors.**
    - **No malignant potential (exceptionally rare serous cystadenocarcinoma).**

Epidemiology

- Strong female predilection.
- Peak incidence: ~6th–7th decade.
- Associated with **von Hippel–Lindau (VHL)** syndrome (multifocal/multicentric in this setting).

Clinical Presentation

- Usually **asymptomatic**, detected incidentally.
- May cause mass effect if large (>4 cm): pain, gastric outlet obstruction, or bile duct compression.

Gross Pathology

- Typically **microcystic**: composed of numerous small cysts (<2 cm) giving **"honeycomb"** appearance.
- Well-defined, lobulated lesion.
- May have **central stellate scar**, sometimes with calcification (seen in ~30% cases; pathognomonic).

- No mucin production.

Imaging Features

- **Ultrasound (USG)**: Hypoechoic, lobulated lesion. Appears solid if cysts are very small.
- **CT**:
  - Lobulated, **multicystic lesion** with enhancing **thin septae**.
  - **Central stellate scar with/without calcification**.
  - **No communication with pancreatic duct**.
  - Displacement (not invasion) of adjacent structures.
- **MRI**:
  - Cysts: Hyperintense on T2W; low signal fibrous septae.
  - Septae/scar: Early intense enhancement with persistent delayed enhancement.
  - No mural nodules or soft tissue component.
- **EUS-FNA**:
  - Thin, clear fluid.
  - Low **CEA** and **amylase**.
  - Presence of **glycogen-rich cuboidal epithelium** (PAS+).

Management

- Conservative for asymptomatic lesions <4 cm.
- Surgical resection if:
  - Symptomatic.
  - Size >4 cm or rapid growth (>2 cm/year).
  - Indeterminate imaging.

2. **<u>Mucinous Cystic Neoplasm (MCN) of Pancreas</u>**
   - **Mucin-producing epithelial neoplasm with ovarian-type stroma (defining histologic criterion).**
   - **Considered pre-malignant to malignant (adenoma → borderline → carcinoma in situ → invasive carcinoma).**
   - **~2.5% of pancreatic tumors.**

Epidemiology

- Almost **exclusive to women** (99%).
- Mean age ~40–50 years.
- No syndromic associations (unlike SCN or IPMN).

Location

- **Exclusively in pancreatic body and tail**.
- Never communicates with pancreatic duct (helps differentiate from side-branch IPMN).

Clinical Presentation

- Usually asymptomatic.
- Larger lesions may present with:
    - Abdominal discomfort or pain.
    - Palpable mass.
    - Rarely jaundice (if large enough to compress ducts).

Gross Pathology

- **Macrocystic**, encapsulated lesion with **thick fibrous capsule**.
- Typically **unilocular or multilocular** (few locules >2 cm).
- Mucin-filled locules.
- May show **curvilinear calcification**, **septations**, **mural nodules**.
- Does **not** communicate with the pancreatic duct.

Imaging Features

- **Ultrasound (USG)**: Anechoic or complex cystic mass with septations or debris.
- **CT**:
    - **Macrocystic lesion** with **thick wall**, internal septae.
    - **Mural nodules** or enhancing soft tissue components suggest malignancy.
    - **Curvilinear wall calcification** (specific).
- **MRI**:
    - Cystic fluid: T2 hyperintense, variable T1 signal (proteinaceous mucin).
    - Thick enhancing wall/septae.
    - **No ductal communication**.
- **EUS-FNA**:
    - Viscous, mucin-rich fluid.
    - **High CEA** (>192 ng/mL), **low amylase**.
    - Ovarian-type stroma on histology is diagnostic.

Malignant Potential

- Malignant transformation risk increases with:
    - Size >4 cm.
    - Mural nodules.
    - Thickened septae.
    - Calcifications.
    - Elevated tumor markers.

Management

- **Surgical resection is recommended** in most cases due to potential for malignancy.
- Conservative follow-up may be considered for:

- Small (<3 cm), asymptomatic lesions without worrisome features, in non-surgical candidates.

3. **<u>Intraductal Papillary Mucinous Neoplasm (IPMN)</u>**
    - **Mucin-producing neoplasm arising from ductal epithelium of the pancreas.**
    - **Exhibits intraductal papillary proliferation, mucin secretion, and ductal dilatation.**
    - **Premalignant with potential for invasive carcinoma.**

Classification

- Based on ductal involvement:
    - **Main duct IPMN (MD-IPMN)** – high malignant potential (~60–70%)
    - **Branch duct IPMN (BD-IPMN)** – lower malignant risk (~20–30%)
    - **Mixed-type IPMN** – both involved

Epidemiology

- **Elderly males** (mean age ~65 years).
- Increasingly detected due to cross-sectional imaging.

Clinical Presentation

- Often **asymptomatic**.
- May present with:
    - Abdominal pain
    - Recurrent pancreatitis (due to mucin plugging)
    - Jaundice (if main duct involved)
    - Diabetes (longstanding duct obstruction)

Imaging Findings

1. Ultrasound

- Nonspecific; may show:
    - Dilated duct
    - Cystic lesion in uncinate/ head region
    - Internal echoes/mucin

2. CT

- **Main Duct IPMN**: Diffuse or segmental **pancreatic duct dilatation >5 mm**, no obvious obstruction.
- **Branch Duct IPMN**:
    - Cystic lesions in head/uncinate
    - **Communication with main duct** (key feature)
- **Mural nodules**, wall enhancement = red flags for malignancy.
- Calcifications are uncommon.

3. MRI + MRCP *(Modality of Choice)*

- **T2-weighted**: High signal cysts; main duct or side-branch dilatation.
- **MRCP**: Best to demonstrate ductal communication.
- **T1 post-contrast**: Enhancing mural nodules suggest malignancy.
- Ancillary signs:
    - **"Grape-like" cystic clusters** in uncinate (BD-IPMN)
    - **"Fish-mouth papilla"** with mucin extrusion on endoscopy

Malignancy Risk – High-Risk Features

- Main duct diameter >10 mm
- Enhancing mural nodule
- Abrupt ductal caliber change with atrophy
- Cyst size >3 cm (for BD-IPMN)
- Elevated CEA (>192 ng/mL) on EUS-FNA

Management

- **Main duct and mixed IPMN**: Surgery
- **Side branch IPMN**:
    - Observe if <3 cm, no mural nodules, no symptoms.
    - Surgery if >3 cm, mural nodules, positive cytology, or high-risk stigmata.

4. <u>Solid Pseudopapillary Neoplasm (SPN)</u>

- **Low-grade malignant epithelial tumor of pancreas with solid and cystic components.**
- **Previously called Frantz tumor.**
- **Very distinct demographic and behavior.**

Epidemiology

- **Young women** (~90%) in their **second to third decade**.
- Rare (<2% of all pancreatic neoplasms).

Clinical Presentation

- Often **asymptomatic** or presents as:
    - Abdominal lump or discomfort
    - Rarely jaundice or rupture/hemorrhage
- May be large at diagnosis (mean size ~8–10 cm)

Pathology

- Encapsulated tumor with **solid periphery** and **cystic/hemorrhagic center**.
- Microscopy: sheets of cells with pseudopapillary architecture.
- β-catenin nuclear staining is typical.

Imaging Features

1. Ultrasound

- Well-defined heterogeneous mass.
- Mixed echogenicity due to solid and hemorrhagic cystic areas.

- Often confused with pseudocyst.

2. CT

- **Encapsulated mass** with **solid and cystic areas**.
- Peripheral **solid enhancing component**.
- **Capsule enhancement** with delayed fill-in of solid part.
- No significant ductal obstruction.

3. MRI

- **T1**: High signal (hemorrhagic/cystic areas)
- **T2**: Heterogeneous hyperintensity
- **Capsule**: Hypointense rim, delayed enhancement
- **Solid components enhance progressively**
- May show **fluid–fluid levels** (hemorrhage)

Differentials

- Cystic neuroendocrine tumor
- Mucinous cystic neoplasm
- Pancreatic pseudocyst (esp. in young women)

Management

- **Surgical resection** is curative in most.
- Malignant behavior seen in ~15% (capsular invasion, vascular invasion, metastasis).
- Excellent prognosis: >95% 5-year survival.

5. **<u>Cystic Neuroendocrine Tumor (Cystic pNET) of the Pancreas</u>**
    - **A rare cystic variant of pancreatic neuroendocrine tumors (pNETs), arising from ductal endocrine differentiation, not true islet cells.**
    - **Most nonfunctioning pNETs (NF-pNETs) can undergo cystic or necrotic degeneration, especially when large.**
    - **Important to differentiate from other cystic neoplasms (e.g., MCN, SPN) due to different biological behavior and management.**

Epidemiology

- Occurs in **middle-aged adults** (40–60 years).
- No gender predilection for cystic variants.
- May be **sporadic** or syndromic (e.g., MEN1, VHL).
- **Cystic morphology** is more common in **nonfunctioning** and **larger** tumors.

Clinical Presentation

- Frequently **asymptomatic**; may present with:
    - Palpable mass
    - Abdominal discomfort
    - Symptoms of mass effect (biliary obstruction, pain)

- Hormonal syndromes **absent** in nonfunctioning types.

Pathology

- Well-differentiated neuroendocrine tumor with **central necrosis or cystic degeneration**.
- Peripheral viable solid rim containing neuroendocrine cells.
- Cystic spaces may be hemorrhagic or necrotic.
- Immunohistochemistry: Chromogranin A, synaptophysin positive.

<u>WHO Grading</u>

- Based on **Ki-67 index** and **mitotic count**:
  - **Grade 1 (G1)**: Ki-67 <3%
  - **Grade 2 (G2)**: Ki-67 3–20%
  - **Grade 3 (G3)**: Ki-67 >20%

<u>Imaging Features</u>

1. Ultrasound

- Anechoic or complex cystic lesion
- **Peripheral solid rim** with or without Doppler flow
- May mimic pseudocyst or SPN

2. CT

- Well-defined, **hypodense lesion** with a **hypervascular enhancing rim** (best appreciated in arterial phase)
- **Delayed progressive enhancement** of solid rim
- Central non-enhancing cystic or necrotic component
- May show **calcifications** in rim (more common in NF-pNETs)

3. MRI

- **T1W**: Variable signal; hemorrhagic areas appear hyperintense
- **T2W**: Hyperintense central cystic component
- **Post-contrast**: Early **arterial hyperenhancement of solid peripheral rim**, followed by washout or persistent enhancement
- May show **fluid–fluid levels**
- Useful to distinguish from MCN or SPN based on enhancement pattern

4. Nuclear Imaging

- **68Ga-DOTATATE PET-CT**: High sensitivity for somatostatin receptor-positive pNETs
- Distinguishes low-grade pNETs from other cystic lesions

Differentiation from Other Cystic Lesions

Feature	Cystic pNET	MCN	SPN

Rim enhancement	**Yes (arterial)**	No	Delayed progressive
Functionality	Often nonfunctional	Nonfunctional	Nonfunctional
Location	Body > Tail	Tail	Tail
Age group	40–60 yrs	Middle-aged women	Young women
Internal contents	Necrotic/cystic	Mucin	Hemorrhagic/necrotic
PET-DOTATATE uptake	**Positive**	Negative	Negative

EUS-FNA

- Shows **thin, yellow serous fluid**
- High **chromogranin A**, **synaptophysin**
- Low amylase and CEA
- **Cytology** confirms neuroendocrine cells with granular cytoplasm

Management

- **Surgical resection** for:
    - Size >2 cm
    - Suspicious imaging features
    - Confirmed pNET on biopsy
- **Surveillance** may be considered for small, asymptomatic lesions without mural nodules.
- Excellent prognosis if well-differentiated.

Parameter	**SCN**	**MCN**	**IPMN**
Ductal Communication	**No**	No	**Yes**
Fluid Viscosity	Low	High	High
CEA Level	**<5 ng/mL**	>192 ng/mL	>192 ng/mL
Amylase Level	**Low**	Low	**High**
Mucin Presence	No	Yes	Yes

MANAGEMENT STRATEGY

Lesion	**Surgery**	**Surveillance**
MCN	Always indicated	–

Main duct IPMN	Always indicated	–
Side branch IPMN	If ≥3 cm or worrisome features	Yes, if small and no high-risk signs
SPN	Always indicated	–
SCA	Only if >4 cm or symptomatic	Yes

Q6. Describe TNM staging of bladder cancer. Draw a schematic diagram of T staging of bladder tumour. Enumerate the role of ultrasound, computed tomography and MRI in the diagnosis and staging of the bladder carcinoma

Answer

TNM staging for bladder cancer categorizes tumors based on Tumor size and invasion (T), lymph Node involvement (N), and distant Metastasis (M).

T-Primary tumour	
TX	Primary tumour cannot be assessed
T0	No evidence of primary tumour
Ta	Non-invasive papillary carcinoma
Tis	Carcinoma in situ
T1	Tumour invades subepithelial connective tissue layer
T2	Tumour invades muscle
	T2a Tumour invades superficial muscle
	T2b Tumour invades deep muscle
T3	T3 tumours -invade perivesical tissue
	T3a -microscopically
	T3b-macroscopically (extravesical mass)
T4	Tumour invades any of the following: prostatic stroma, seminal vesicles, uterus, vagina, pelvic wall, abdominal wall
	T4a Tumour invades prostatic stroma, seminal vesicles, uterus or vagina
	T4b Tumour invades pelvic wall or abdominal wall
N-Regional lymph nodes	
NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in a single lymph node in the true pelvis
N2	Metastasis in multiple regional lymph nodes in true pelvis
N3	Metastasis in common iliac lymph node(s)
M-Distant metastasis	
M0	No distant metastasis
	M1a Non-regional lymph nodes
	M1b Other distant metastasis

Role of Imaging Modalities in Bladder Carcinoma

1. Ultrasound (USG)

- **Role**: Visualizes the intravesical position of many tumors.
- **Advantages**:
  - Can detect many intravesical lesions.
  - Transrectal US provides better visualization of tumors involving the posterior wall, bladder neck, prostate, and seminal vesicles.
  - Widely available and non-invasive.
- **Limitations**:

    - Cannot accurately evaluate the depth of penetration into the bladder wall.
    - Perivesical invasion is infrequently determined.
    - Extravesical spread may be recognized indirectly by asymmetry or infiltration signs.
    - Does not optimally assess regional lymph node status.
- **Accuracy**: Transabdominal US for depth of infiltration ranges from 55% to 95%.

2. Computed Tomography (CT)

- **Role**: Primarily used for staging rather than detection of the primary tumor.
- **Advantages**:
    - More reliable for advanced disease.
    - Detects **extravesical extension** — seen as increased density or stranding of perivesical fat and poorly defined outer bladder wall.
    - Identifies pelvic side wall involvement — soft tissue mass into obturator internus or strands extending to pelvic wall.
    - Suggests seminal vesicle invasion — obliteration of vesicle angle by soft tissue mass.
    - Detects lymph nodal metastases — nodes >10 mm short axis considered malignant; obturator and external iliac nodes are first to involve.
- **Limitations**:
    - Cannot distinguish early stages — unable to separate lamina propria (T1) from superficial muscle (T2) or deep muscle invasion (T3a).
    - Less accurate than MRI for perivesical infiltration and nodal metastasis.

3. Magnetic Resonance Imaging (MRI)

- **Role**: Most accurate technique for staging invasive bladder carcinoma.
- **Technique**:
    - **T2-weighted** or **postcontrast T1-weighted** sequences are used to assess tumor infiltration.
    - A **low-intensity line on T2W** represents uninvolved bladder wall — helps differentiate stage T3a from T3b.
- **Advantages**:
    - Detects muscularis propria invasion — interruption of low signal muscle layer, irregular bladder wall, or perivesical stranding.
    - Multiplanar imaging allows assessment of invasion into adjacent organs:
        - **Sagittal imaging** for uterine/vaginal invasion.

        - Seminal vesicle invasion indicated by vesicle enlargement, T2 hypointensity, and loss of normal angle with bladder.
        - Prostate/rectal infiltration appears as direct tumor spread with T2 signal changes.
    - Particularly useful for dome or base tumors due to multiplanar capability.
    - T1W better for lymph node evaluation due to contrast with adjacent fat.
    - With ultra-small superparamagnetic iron oxide (USPIO) agents, MRI can detect metastases in normal-sized nodes and differentiate reactive from malignant nodes.
- **Limitations**:
    - More expensive and less available than CT.
    - Requires expertise in interpretation.

Q7. Describe various indications, protocol, advantages and disadvantages of fetal MRI.

Answer

Fetal MRI provides detailed in-utero imaging of the developing fetus. Because fetal movement is common, fast sequences are essential. The modality is particularly valuable when ultrasound findings are inconclusive. MRI offers high-resolution assessment of fetal anatomy, including the brain, upper aerodigestive tract, thorax, abdomen, pelvis, and musculoskeletal system.
MRI can be performed from the second trimester onward. Its use in the first trimester is controversial due to concerns about biosafety, small fetal size, and uncertain biological effects at that stage.

General Indications
Fetal MRI is often requested when an abnormality is suspected on ultrasound but visualization is limited by fetal position, maternal habitus, oligohydramnios, ossification, or a restricted field of view. It is also used when ultrasound detects a poorly defined abnormality requiring further clarification for management or prognostication. MRI is indicated in high-risk pregnancies where ultrasound cannot adequately assess certain pathologies.

Specific Indications

1. **Central Nervous System (Brain and Spine)**

MRI helps evaluate suspected malformations such as familial disorders (e.g., tuberous sclerosis, lissencephaly, callosal dysgenesis), cephaloceles, intracranial masses, cortical malformations, posterior fossa anomalies, holoprosencephaly, corpus callosum abnormalities, ventriculomegaly, vascular malformations, hydranencephaly, infarcts, and hemorrhage. It is also used in monochorionic twin complications and suspected spinal anomalies, including neural tube defects, sacrococcygeal teratoma, caudal regression, and vertebral anomalies.

2. **Head and Neck**

MRI evaluates facial and neck masses, vascular and lymphatic malformations, teratomas, clefts, congenital cysts, and airway compromise.

3. **Thoracic**

Indications include congenital lung and airway malformations, diaphragmatic hernias, pleural effusions, mediastinal masses, esophageal atresia, and lung volumetry in suspected pulmonary hypoplasia.

4. **Abdominal and Pelvic**

MRI is used for suspected abdominal masses or cysts, genitourinary anomalies (especially when ultrasound is limited by severe oligohydramnios), anorectal malformations, complex bowel obstructions, and abdominal wall defects.

5. **Musculoskeletal**

It aids in evaluating masses, malformations, and skeletal dysplasias.

6. **Multiple Gestations**

In monochorionic twins, MRI can assess vascular anatomy before laser therapy. In conjoined twins, it helps define shared anatomy to guide counseling and delivery planning.

7. **Placental Evaluation**

MRI is valuable when ultrasound cannot adequately assess placental anomalies, gestational trophoblastic disease, or abnormal placentation (e.g., accreta spectrum).

Limitations

Challenges include reduced signal-to-noise ratio, partial volume effects (especially before 18 weeks), maternal size limitations, claustrophobia, contraindicated implants, and difficulty assessing cardiac structures due to rapid fetal heart rate and movement.

Standard MRI Sequences

- **Single-shot fast spin-echo (SSFSE) T2-weighted** for high-contrast anatomic detail
- **T1-weighted** imaging for fat, calcification, and hemorrhage
- **Steady-state free-precession (SSFP)** for cardiac and vascular imaging

Biosafety Considerations

Fetal MRI does not use ionizing radiation. However, the effects of static magnetic fields, radiofrequency pulses, and gradient switching on early fetal development are not fully understood. The ACR recommends performing MRI after 18 weeks' gestation. Use of 1.5 T MRI in the second and third trimesters has not been linked to hearing loss or developmental issues in multiple studies.

Practical Imaging Approach

Maternal and Pregnancy Structures

Assess placenta (location, morphology, invasion), cord insertion and vessel number, amniotic cavity (bands, fluid), and cervix length.

Fetal Signs of Life
Look for cardiac signal void and normal motion of amniotic fluid.

Central Nervous System

- Measure biometry (BPD, OFD, HC) in correct planes.
- Evaluate for anencephaly, macro/microcephaly, hydrocephalus, posterior fossa malformations, vermian hypoplasia, and midline defects (e.g., holoprosencephaly, callosal agenesis).
- Assess sulcation and gyration milestones to estimate gestational age.
- Examine lamination patterns and basal ganglia signal evolution over gestation.

Face and Calvarium
Check facial profile, lips, alveolar margins, ears, external meati, pinnae, and cochlea development.

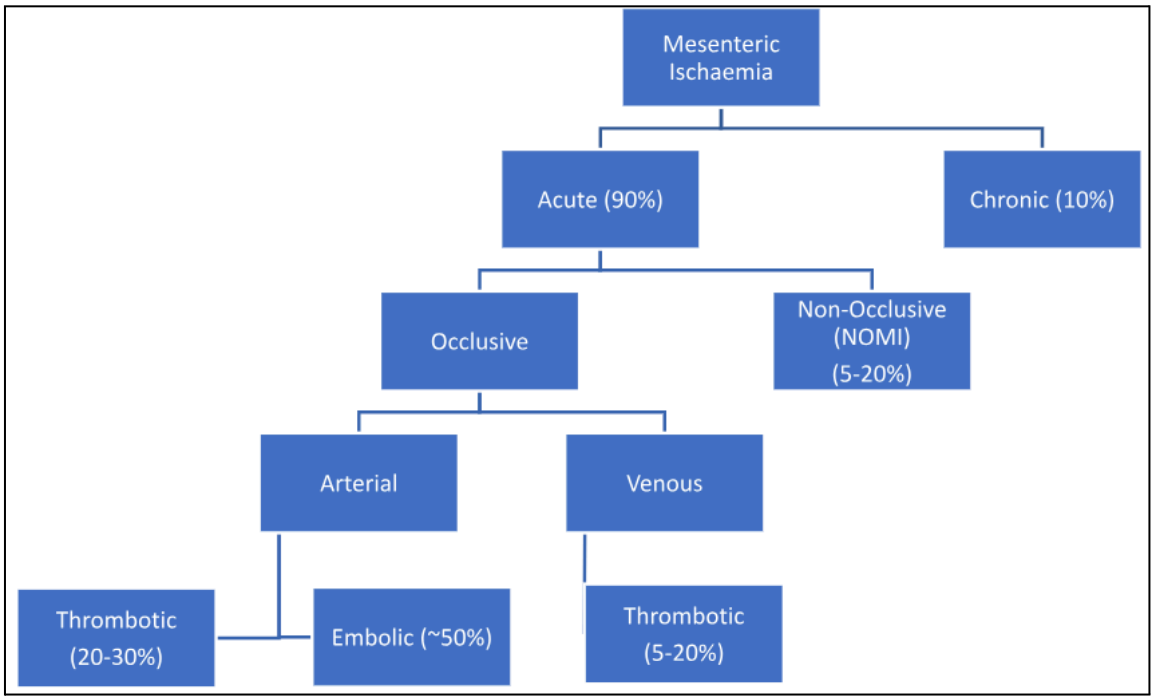
Spine
Evaluate for spina bifida, neurenteric cyst, and teratomas.

Thorax and Abdomen
Confirm situs, diaphragmatic integrity, stomach position and filling, liver, gallbladder, kidneys (size, pelvis diameter), and bladder shape.

Q8. What are the various causes of acute mesenteric ischemia? Discuss the role of radiology in its diagnosis and management.

Answer

Mesenteric ischemia refers to compromised blood supply (arterial or venous) to the small intestine and proximal colon, leading to **intestinal hypoxia**, **metabolic derangement**, and potential **bowel necrosis**.



Type	Subtype	Etiology

Acute Mesenteric Ischemia	**➤ Occlusive Arterial**	SMA embolism (AF), thrombosis (atherosclerosis, dissection)
	➤ Occlusive Venous	Mesenteric vein thrombosis (hypercoagulable states, OCPs, IBD, SLE)
	➤ Non-Occlusive (NOMI)	Low-flow states: shock, sepsis, vasoconstrictors (e.g., digoxin, ergotamine)
Chronic Mesenteric Ischemia	—	Atherosclerosis (usually ≥2 major vessels), fibromuscular dysplasia

Clinical Features

Acute	**Chronic**
Sudden severe abdominal pain (pain > signs)	Postprandial abdominal pain ("intestinal angina")
Vomiting, diarrhea, occult bleeding	Food fear, significant weight loss
Peritonitis (late), shock, metabolic acidosis	Abdominal bruit may be heard

Parameter	**Mesenteric Arterial Embolism (MAE)**	**Mesenteric Arterial Thrombosis (MAT)**	**Mesenteric Venous Thrombosis (MVT)**	**Non-Occlusive Mesenteric Ischemia (NOMI)**
Frequency	~50%	20–30%	5–20%	5–20%
Presentation	Acute	Acute on chronic	Subacute	Acute or subacute
Age Group	Middle-aged, elderly	Elderly	Younger	Critically ill (any age)
Risk Factors	AF, embolic heart disease, recent MI, endocarditis	Atherosclerosis, proximal vessel disease	Hypercoagulable states, pancreatitis, PHTN	Shock, cardiac surgery, vasopressors, multiorgan failure
Mortality Rate	Intermediate (~65.8%)	Intermediate (~65.8%)	Lowest (~42.5%)	Highest (~68.5%)

Bowel Involvement	Short segment, distal SMA territory	Long segment, proximal SMA/IMA	Venous territory, segmental, possibly discontinuous	Patchy, long segments, does **not** follow vascular territories
Bowel Wall Thickness	Normal or thinned; thickened if reperfused	Same as MAE	**Thickened** (due to edema / hemorrhage)	Same as MAE
Wall Attenuation (NCCT)	May show hyperdensity with hemorrhagic infarction	Same as MAE	Low (edema) or high (hemorrhagic infarct)	Same as MAE
Enhancement (CECT)	↓ or absent; target sign if reperfusion occurs	Same as MAE	Target sign; mucosal hyperenhancement	Patchy ↓/↑; heterogeneous; target appearance possible
Bowel Dilatation	Minimal or hypotonic	Same as MAE	**Moderate to prominent** (due to intraluminal exudation)	Same as MAE
Mesenteric Vessels	SMA embolus (mid/distal); "polo mint sign"	Proximal SMA/IMA occlusion ± wall calcification	SMV/IMV filling defect; possible rim enhancement	**No occlusion**; diffusely reduced caliber of mesenteric arteries
Mesenteric Fat	Late stranding (if infarcted)	Same as MAE	**Early and prominent stranding** (fluid exudation)	Same as MAE
Additional Imaging Features	Emboli / infarcts in kidneys, spleen, etc	Atherosclerotic changes in other arteries	Portal/splenic vein thrombosis extension	CT hypoperfusion complex (flat IVC, hyperenhancing adrenals, ↓aortic caliber)

Treatment

Condition	Management
Acute Arterial Occlusion	Endovascular thrombolysis ± stenting; laparotomy if peritonitis
Venous Thrombosis	Anticoagulation, consider thrombolysis if bowel viability threatened
NOMI	Supportive care, intra-arterial vasodilators (papaverine, nitroglycerin)
Chronic Ischemia	Endovascular angioplasty ± stenting (SMA), surgical bypass for refractory cases

- **Second-look laparotomy** may be needed to assess delayed necrosis
- Avoid vasoconstrictors in NOMI

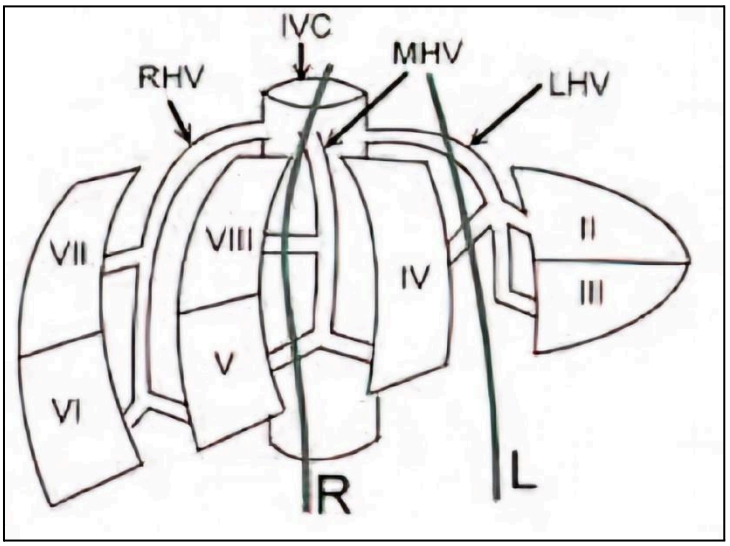
Radiology Signs

Sign	Meaning
"Target sign"	Mural stratification in venous ischemia
Pneumatosis intestinalis	Late stage, transmural necrosis
Arc of Riolan visualized	Chronic occlusion with collaterals
SMA cutoff with retracted loops	Acute arterial embolism

Q9. Describe briefly segmental anatomy of liver with line diagram. Describe imaging (CT & MRI) features in hepatocellular carcinoma.

Answer

1. Liver Anatomy – Couinaud Classification



- **Functional Segmentation:**
  - **8 segments — each with:**
    - **Its own portal vein, hepatic artery, and bile duct in the center.**
    - **Hepatic vein drainage at the periphery.**
- **Planes:**
  - **Right hepatic vein** → divides right lobe into **anterior (V, VIII)** and **posterior (VI, VII)** segments.
  - **Middle hepatic vein** (Cantlie's line) → divides **functional right & left liver** (right & left hemiliver). Runs from gallbladder fossa to IVC.
  - **Umbilical plane** → divides left lobe into:
    - **Medial: segment IV**
    - **Lateral: segments II & III**

      (Only vertical plane not defined by a hepatic vein)
- **Portal vein division:**
  - **Horizontal plane → upper & lower segments.**
  - **Right & left portal veins give superior & inferior branches to each segment.**

Anatomical Landmarks

- **Cantlie's line: Middle of gallbladder fossa → IVC.**
- **Right lobe: Segments V–VIII.**
- **Left lobe: Segments II–IV (Segment I = caudate lobe, independent drainage).**

Imaging Features of Hepatocellular Carcinoma (HCC)

CT

1. **Arterial phase hyperenhancement (APHE)**
    - **Hallmark: brighter than liver in arterial phase.**
2. **Washout** in portal venous/delayed phase
    - **Hypoattenuation vs. background liver.**
3. **Capsule appearance**
    - **Peripheral enhancement in PV or delayed phase.**
4. **Growth patterns**:
    - **Solitary, multifocal, infiltrative.**
    - **May have necrosis or hemorrhage.**
5. **Vascular invasion**:
    - **Tumor thrombus in portal/hepatic veins.**

MRI

1. **T1**: Hypo- or isointense.
2. **T2**: Slightly hyperintense.
3. **DWI**: Restricted diffusion (bright on high b-value, dark on ADC map).
4. **Dynamic phases**:
    - **Arterial phase: APHE.**
    - **Portal venous/delayed: Washout.**
    - **Hepatobiliary phase (HBP) with Gd-EOB-DTPA:**
        - **HCC appears hypointense (no hepatocyte uptake).**
5. **Capsule**: Delayed enhancement.
6. **Fat content**: Drop in opposed-phase vs. in-phase images.
7. **Iron content**: Signal loss on T1 & T2.

Diagnostic Triad (LI-RADS major features):

- **APHE**
- **Washout (PV or delayed)**
- **Capsule (delayed enhancement)**

→ In at-risk patients, presence of all = *definite HCC*.

Q10. Role of plain radiograph in a case of acute abdomen.
b) Role of MRI in acute pancreatitis.

Answer

Role of Plain Radiographs in Acute Abdomen

Although **ultrasound** and **CT** dominate modern acute abdomen workup, **plain abdominal radiographs (AXR)** still have value in certain emergency settings because they are **fast, accessible, inexpensive**, and can be done in unstable patients.

1. Detection of Pneumoperitoneum (Free Air)

- **Cause**: Perforated hollow viscus (e.g., gastric ulcer, bowel perforation).
- **Signs**:
    - **Erect CXR/AXR**: Crescent of air under diaphragm (best seen under right dome).
    - **Rigler's sign**: Visualization of both sides of bowel wall.
    - **Football sign**: Large pneumoperitoneum outlining peritoneal cavity.
- **Clinical importance**: Surgical emergency.

2. Bowel Gas Pattern Assessment

- **Small Bowel Obstruction (SBO)**:
    - Dilated loops (>3 cm diameter).
    - Central location.
    - **Valvulae conniventes** across full width.
    - Multiple air-fluid levels (upright view).
- **Large Bowel Obstruction (LBO)**:
    - Peripheral location.
    - Haustral pattern (does not cross full width).
    - Caecal dilatation >9 cm = risk of perforation.
- **Ileus**:
    - Diffuse dilatation of both small and large bowel.
    - Fewer or absent air-fluid levels.

3. Calcifications

- **Gallstones**: ~10–15% radiopaque.
- **Renal/Ureteric calculi**: Commonly visible.
- **Pancreatic calcifications**: Chronic pancreatitis.
- **Vascular calcifications**: Aneurysm suspicion.

4. Soft Tissue and Organ Shadows

- **Organomegaly**:
  - Enlarged liver or spleen displacing bowel gas.
- **Masses**:
  - Abnormal displacement of bowel loops.

5. Foreign Bodies

- **Radiopaque**: Metal, some plastics, glass.
- **Drug packets** in body packers.

6. Baseline and Serial Imaging

- Useful in:
  - **Obstruction monitoring** (non-operative cases).
  - **Post-op ileus resolution tracking**.

Role of MRI in Acute Pancreatitis

MRI (often with **MRCP**) plays an important role in the **evaluation, complication assessment, and follow-up** of acute pancreatitis, especially when CT is inconclusive or contraindicated.

1. Detailed Pancreatic Anatomy Visualization

- **High soft-tissue contrast** → clear depiction of:
  - Pancreatic parenchyma
  - Main and accessory pancreatic ducts
  - Peripancreatic tissues
- Detects **subtle edema, hemorrhage, or ductal changes**.
- Can show **small stones, strictures, and ductal irregularities** not visible on CT.

2. Complication Assessment

- **Necrosis**:
  - Differentiates **viable vs. necrotic** tissue (T1 signal drop, lack of enhancement).
- **Fluid collections**:
  - T2-weighted sequences show extent and content.
  - Differentiates **acute necrotic collections (ANCs)** from **pseudocysts**.
- **Biliary complications**:
  - With MRCP, detects obstruction from **choledocholithiasis** or inflammatory strictures.

3. MRCP for Non-Invasive Ductal Imaging

- No contrast injection required.
- Visualizes:
    - Common bile duct (CBD)
    - Pancreatic duct
    - Secondary ducts
- Detects **stones, strictures, pancreas divisum** (important in recurrent pancreatitis).

4. Tissue Characterization & Chemical Analysis

- T1/T2 signal differences help differentiate:
    - Necrosis vs. fluid
    - Hemorrhagic vs. serous content
- **Fat-suppressed imaging** highlights fatty infiltration or peripancreatic fat necrosis.

5. No Radiation

- Ideal for:
    - Pregnant patients
    - Young patients
    - Recurrent imaging follow-up

When to Use MRI in Acute Pancreatitis

1. **Inconclusive CT** findings
2. **Suspected ductal cause** → MRCP for stones or strictures
3. **Differentiating necrosis from fluid collections**
4. **Follow-up** for evolving or resolving complications
5. **Radiation or iodinated contrast contraindications** (e.g., allergy, renal impairment)
6. **Evaluation of chronic changes** in recurrent acute pancreatitis

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