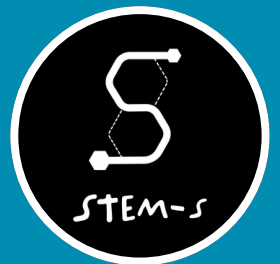


NEET - SS

MEDICAL GROUP

Module Wise Notes



Presented By:
STEM-S

STEM-S

NEET-SS · MedicalGroup·ModuleWise Notes

High Yield Modulewise Notes

(HNM Note)

DETAILED SUBJECT INDEX

12 Modules · 999 Pages · Complete Topic Listing

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HEPATOLOGY

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& METABOLISM

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MODULE
1

NEUROLOGY

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5. Differentiating ALS from Major Mimics

Condition	Key Clinical Differentiator	Diagnostic Finding
MMCB	Pure LMN; no UMN signs; asymmetric	EMG: Conduction Block; high Anti-GM1 titers
Kennedy's	X-linked; pure LMN; mid-adult males	Gynecomastia; reduced fertility; CAG repeat in Androgen Receptor
Hirayama	Unilateral hand/forearm atrophy; stabilizes	MRI (flexion): localized cord atrophy; oblique atrophy pattern
SMA I	Infantile onset ("Floppy baby")	SMN1 deletion; absent reflexes
Tay-Sachs (Adult)	Very slow progression (years)	Cerebellar atrophy on imaging; Hex-A deficiency

Heads up: Don't miss Multifocal Motor Neuropathy (MMCB). It's highly responsive to IVIg, unlike ALS, so getting this diagnosis right actually changes the treatment plan.

6. Visual Anchors -

- MRI (ALS): high T2 signal in the Corticospinal Tracts (Wallerian degeneration) viewed through the internal capsule
- MRI (Hirayama): localized cord atrophy at C6-C7 with an enlarged posterior epidural space on neck flexion
- Pathology (ALS): ubiquitin-positive inclusions (TDP-43) in motor neurons
- MRI (Kennedy's): marked atrophy of the intrinsic hand muscles and proximal legs
- MRI (HSP/SPG4): spinal cord atrophy without signal changes in an adult with scissoring gait
- Clinical (SMA I): "Floppy baby" posture with profound hypotonia and areflexia

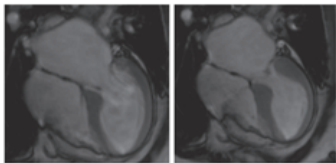
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Heads up: ECG and enzymes will look like an MI, but a clean coronary angiography is what actually differentiates Takotsubo from a real infarct.

2. Orthostatic Dynamics: OH vs POTS

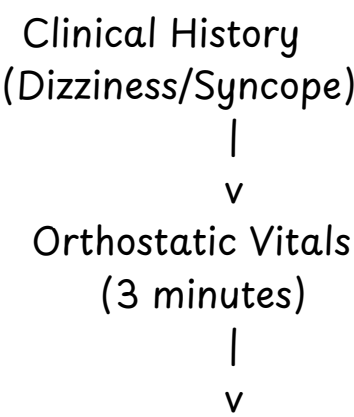
Orthostatic intolerance comes from gravity pulling blood downward (300–800 mL pooling) that the body fails to compensate for.

Orthostatic Hypotension (OH): failure of pressure maintenance. The "20/10 rule": systolic BP drop ≥ 20 mmHg or diastolic drop ≥ 10 mmHg within 3 minutes of standing.

POTS: failure of heart rate stability without hypotension.

Feature	Orthostatic Hypotension (OH)	POTS
Primary Sign	BP Drop ($\geq 20/10$ mmHg)	HR Increase (≥ 30 bpm; ≥ 40 if < 19 yo)
BP Stability	Unstable	Stable (No drop $\geq 20/10$)
Key Patient	Elderly/Autonomic Failure	Young Females (15–45y)
Medication	Fludrocortisone or Droxidopa	Low-dose Propranolol (10–20mg TID)

Diagnostic step ladder (autonomic failure workup)



ORGANISM(S)	DRUG (DOSE, DURATION)	COMMENTS
Staphylococci (<i>S. aureus</i> and coagulase-negative) — MSSA infecting native valves (no noninfected devices) and complicated right-sided and left-sided endocarditis	Nafcillin, oxacillin, or flucloxacillin (2 g IV q4h) for 4–6 weeks	Addition of gentamicin is not recommended. For uncomplicated right-sided endocarditis, a 2-week course may be effective.
	Cefazolin (2 g IV q8h) for 4–6 weeks	Can use cefazolin in patients with mild penicillin allergy; see text regarding cefazolin vs antistaphylococcal penicillin as primary therapy. Addition of gentamicin not recommended.
	Vancomycin (15 mg/kg IV q12h) for 4–6 weeks	Only use vancomycin for patients with immediate (urticarial) or severe penicillin allergy until allergy consultation can be obtained for β -lactam desensitization evaluation; addition of gentamicin not recommended.
MRSA infecting native valves (no noninfected devices)	Vancomycin (15 mg/kg IV q8–12h) or daptomycin (8–10 mg/kg IV q24h) for 4–6 weeks	No role for routine use of rifampin (see text). For daptomycin treatment, see text.
MSSA infecting prosthetic valves	Nafcillin, oxacillin, or flucloxacillin (2 g IV q4h) for 6–8 weeks plus Gentamicin (1 mg/kg IM or IV q8h) for 2 weeks plus Rifampin (300 mg PO q8h) for 6–8 weeks	Use gentamicin during initial 2 weeks; determine gentamicin susceptibility and await blood culture clearance before initiating rifampin; if patient is highly allergic to penicillin, use regimen for MRSA and obtain allergy consultation; if β -lactam allergy is of the minor nonimmediate type, cefazolin can be substituted for oxacillin, nafcillin, or flucloxacillin.
MRSA infecting prosthetic valves	Vancomycin (15 mg/kg IV q12h) for 6–8 weeks plus Gentamicin (1 mg/kg IM or IV q8h) for 2 weeks plus Rifampin (300 mg PO q8h for 6–8 weeks)	Use gentamicin during initial 2 weeks; determine gentamicin susceptibility and await blood culture clearance before initiating rifampin. Daptomycin (8–10 mg/kg daily) is an alternative to vancomycin.

Pathology / Classification
Angina Phenotypes

Feature / Parameter	Stable Angina (CCS)	Unstable Angina (UA)	Vasospastic (Prinzmetal)	Microvascular (Syndrome X)
Pathophysiology	Fixed atherosclerotic stenosis (>70% lumen); supply-demand mismatch on exertion.	Subtotally occlusive thrombus or dynamic plaque destabilization.	Episodic coronary artery spasm / smooth muscle hyperreactivity.	Microvascular endothelial dysfunction & abnormal cardiac nociception.
Pain Trigger & Timing	Predictable exertion or emotional stress; resolves in 2–5 min with rest/NTG.	Rest pain (>20 min), recent onset (<2 wks), or crescendo pattern.	Recurrent rest pain, classically in early morning / midnight.	Exertional chest pain, often persisting long after exercise stops.
ECG Findings	Normal at rest; transient ST depression / T-inversion during exercise.	Transient ST depression or T-wave inversion during pain.	Transient ST-segment ELEVATION during pain; resolves with NTG.	Normal resting ECG; inducible ST depression during stress testing.
Biomarkers & Angio	Troponin Negative; Stable plaque on Angiography.	Troponin NEGATIVE; Subtotal thrombus or ulcerated plaque on Angio.	Troponin Negative; Normal coronaries or non-obstructive spasm on Angio.	Troponin Negative; Normal epicardial coronaries on Angiography.

ACC/AHA Blood Pressure Categories

BP Category	Systolic BP (mmHg)	Diastolic BP (mmHg)	Initial Management Recommendation
Normal Blood Pressure	<120 mmHg	and <80 mmHg	Reassess annually; promote healthy lifestyle habits.
Elevated Blood Pressure	120–129 mmHg	and <80 mmHg	Nonpharmacologic lifestyle therapy; reassess in 3–6 months.
Stage 1 Hypertension	130–139 mmHg	or 80–89 mmHg	Lifestyle alone if ASCVD <10%; Add 1 drug if ASCVD ≥10%, DM, or CKD.
Stage 2 Hypertension	≥140 mmHg	or ≥90 mmHg	Lifestyle + Initial Antihypertensive Drug Therapy (2 drugs if BP >20/10 over goal).

Antihypertensive Drug Classes

Drug Class & Examples	Primary Indications & Benefits	Absolute Contraindications	High-Yield Exam Traps / Adverse Effects
Thiazides (Chlorthalidone, Indapamide)	First-line step 1; Chlorthalidone preferred due to long t _{1/2} .	Gout (hyperuricemia); severe hyponatremia.	Causes hypokalemia, hyponatremia, hypercalcemia, hyperuricemia, hyperglycemia.
ACEIs / ARBs (Lisinopril, Losartan)	First-line in CKD, Proteinuria, Diabetes, HFrEF, Post-MI.	PREGNANCY (teratogenic); bilateral RAS; Angioedema.	Dry cough (ACEI only); Hyperkalemia; AKI in bilateral renal artery stenosis.
DHP CCBs (Amlodipine, Nifedipine LA)	First-line step 1; excellent in Black adults & isolated systolic HTN.	Severe aortic stenosis; avoid in HFrEF.	Dose-dependent pedal edema (arteriolar vasodilation); headache; flushing.
Non-DHP CCBs (Verapamil, Diltiazem)	Angina, Atrial Fibrillation rate control, vasospastic HTN.	HFrEF (EF <40%); 2nd/3rd degree AV block; Beta-blocker co-use.	Strong negative inotropes/chronotropes. Verapamil causes severe constipation.
MRAs (Spironolactone, Eplerenone)	Step 4 in Resistant HTN; HFrEF; Primary Aldosteronism.	Hyperkalemia (K ⁺ >5.0 mEq/L); Severe CKD (eGFR <30).	Spironolactone causes gynecomastia, breast tenderness, erectile dysfunction.

Nephrotic syndrome spectrum

Defined by the triad: edema, hypoalbuminemia (<3.5 g/dL), and proteinuria >3.5 g/day.

Hypercatabolic state with negative nitrogen balance — not just from urinary protein loss, but from lysosomal breakdown and catabolism of filtered albumin inside PCT epithelial cells, which outpaces hepatic albumin synthesis.

Hypoalbuminemia triggers a hepatic surge in lipid synthesis and a hypercoagulable state (urinary loss of antithrombin-3 and plasminogen, plus hyperfibrinogenemia). Risk of renal vein thrombosis (RVT) is highest with Membranous Nephropathy (MN) — presents as sudden oliguria, back pain, hematuria, or pulmonary embolism.

In pediatric patients with serum albumin <2 g/dL, severe tissue ischemia can trigger a "nephrotic crisis" — severe abdominal pain mimicking a surgical acute abdomen.

Uremic syndrome and azotemia

Impairs filtration, leading to accumulation of nitrogenous wastes.

Prerenal azotemia: BUN:Cr ratio >20 . Both are filtered, but urea is actively reabsorbed in the medullary collecting duct along with water under ADH during concentrated, low-flow states — creatinine is not reabsorbed.

Intrinsic renal azotemia: GFR impairment with tubular dysfunction. Damaged tubules can't concentrate urine or reabsorb sodium/urea, so urine is isosmotic (UOsm ~ 280 – 300 mOsm/kg), BUN/Cr ratio <15 , and FENa 1–3%.

The diagnostic step-ladder

Step 1: Clinical History & Drug Review

(Rule out NSAIDs, Lithium, Cisplatin)



Step 2: Urinalysis & "Liquid Biopsy"

(Spun sediment: acanthocytes, casts, crystals)



Step 3: GFR Assessment

(Inulin clearance [gold standard] vs CKD-EPI estimation)



Pathology

Differential Diagnosis of Acute Post-Transplant Allograft Dysfunction

Condition	Timing Post-Transplant	Pathophysiology / Mechanism	Key Histologic & Diagnostic Findings	First-Line Management
Hyperacute Rejection	Minutes to hours (<24h)	Preformed anti-donor HLA Class I antibodies (Type II hypersensitivity)	Intravascular thrombosis, neutrophilic vasculitis, graft cyanosis/mottling	Prevention via Cytotoxic Cross-Match; Graft removal if occurs
Acute Tubular Injury (ATI/DGF)	Days 1-14 (Immediate)	Cold/warm ischemia-reperfusion injury during donor organ preservation	Tubular epithelial flattened/necrosis, loss of brush border; no interstitial infiltrate	Supportive care, maintain BP/perfusion; temporary dialysis if needed
Acute T-Cell Mediated Rejection (TCMR)	Days to months	Recipient T cells infiltrate donor tubulointerstitium/vessels (Direct pathway)	Interstitial lymphocytic infiltrate, Tubulitis (lymphocytes breaching TBM), arteritis	High-dose IV Methylprednisolone (500-1000 mg x 3d) → ATG if steroid-resistant [HY]
Acute Antibody-Mediated Rejection (ABMR)	Days to months	De novo donor-specific antibodies (DSAs) against donor endothelium	Peritubular capillaritis, microvascular injury, positive C4d IF staining, serum DSA+	Plasmapheresis + IVIG + Rituximab [HY]. (Eculizumab is NOT standard 1st line)
Acute CNI Nephrotoxicity	Any time (dose-related)	Afferent arteriolar vasoconstriction → ischemic GFR decline	Isometric tubular epithelial vacuolization, lack of inflammatory infiltrate	Reduce CNI dose; check trough levels; avoid CYP3A4 inhibitors
BK Virus Nephropathy (BKVN)	1-6 months (up to 2 yrs)	Polyomavirus reactivation due to over-immunosuppression	Intranuclear viral inclusion bodies in tubular cells ("Decoy Cells" in urine), positive SV40 stain	Judicious reduction of maintenance immunosuppression [HY]

The core idea

The renal tubulointerstitium is the metabolic engine of the nephron, highly perfused but uniquely vulnerable to toxic, metabolic, immunologic, and hypoxic insults. Primary glomerular disorders show heavy albuminuria from breakdown of the glomerular charge- and size-selective barrier. Primary tubulointerstitial diseases spare the glomeruli and renal vasculature relatively.

Damage to the tubular and interstitial components of the cortex and medulla mainly wrecks the kidney's concentrating, diluting, and acid-base regulation. Clinically this shows up as the classic tubulointerstitial triad: polyuria (impaired concentrating ability/acquired nephrogenic diabetes insipidus), non-anion-gap metabolic acidosis, and tubular (subnephrotic) proteinuria (<2 g/d). That triad is what separates these disorders from primary glomerulopathies.

Diagnostic step-ladder: working up tubulointerstitial disease

Logical stepwise approach needed to localize pathology in the tubulointerstitial compartment without wasting tests.

History & clinical exam

(screen for drug exposures, traditional herbs, systemic autoimmune signs)

|

v

Urinalysis & urine microscopy

(look for sterile pyuria, WBC casts, tubular proteinuria; rule out RBC casts)

|

v

Urine crystal analysis & chemistry

(identify pathognomonic drug/metabolic crystals under polarized light; measure FENa)

|

v

Renal ultrasonography

(assess kidney size/echogenicity: distinguishes acute bilateral swelling from chronic asymmetric scarring)

|

v

Post-Gastrectomy Syndromes

- **Early Dumping Syndrome** - 20-30 mins post-meal. Caused by **rapid gastric emptying of hyperosmolar chyme** into the jejunum with massive fluid shifts. Presents with tachycardia, palpitations, and diarrhoea.
- **Late Dumping Syndrome** - 2-3 hours postprandially. Caused by **reactive hypoglycaemia**.
- **Bile Reflux Gastropathy** - bilious vomiting and epigastric pain. Gold standard for documenting retrograde bile flow in refractory cases: **99mTc-HIDA Scintigraphy**.

Visual Anchors

Condition	Visual / Radiographic Finding	Diagnostic Hallmark
Achalasia	"Bird-beak" tapering on barium swallow	Elevated IRP on manometry
Ménétrier's	Giant, tortuous folds sparing the antrum	Foveolar hyperplasia + Glandular atrophy
ZES	Enhancing mass in the "Gastrinoma Triangle"	Secretin Test: Gastrin rise ≥ 120 pg/mL
H. pylori	Spiral organisms in surface mucus (Steiner silver stain)	Urea Breath Test (UBT)
GAVE	"Watermelon Stomach" (longitudinal red stripes)	Gastric antral vascular ectasias
Emphysematous Gastritis	Curvilinear air in the stomach wall on CT	Sepsis + Gas-forming organisms
MALT Lymphoma	B-cell proliferation on biopsy	Regression (>80%) after Hp eradication
Dieulafoy's	Actively spurting caliber artery	Submucosal vessel without ulcer

Diagnostic Pathway: Obstruction

History (prior surgery?)



Physical exam

(tender hernia? bowel sounds?)



Abdominal X-ray

(staircasing air-fluid levels)



CT Scan

(differentiates ileus from mechanical blockage)

Acute Appendicitis

- **Pathogenesis: Luminal obstruction** (fecalith, lymphoid hyperplasia) is the initiating event
- **Obturator sign:** Pain on internal rotation of the flexed hip → **pelvic appendix**
- **Iliopsoas sign:** Pain on extension of the right hip → **retrocaecal appendix**

Radiographic and Visual Anchors

Condition	Visual / Radiographic Anchor	Diagnostic Hallmark
SBO	Valvulae conniventes span the full lumen width	Dilated loops >2.5 cm
LBO	Haustral markings do not cross the full lumen	Peripheral location
Sigmoid volvulus	"Coffee-bean" or "bent inner tube"	Sigmoid detorsion is first-line
Gallstone ileus	Rigler's Triad: pneumobilia + ectopic stone + SBO	Cholecystoduodenal fistula
Crohn's disease	"String sign" (narrowed ileum); "cobblestoning"	Skip lesions; transmural
UC	"Lead pipe" colon (loss of haustration)	Continuous mucosal inflammation
Whipple's	PAS-positive macrophages on duodenal biopsy	Tropheryma whipplei
Coeliac disease	"Scalloped" duodenal folds on endoscopy	Villous atrophy; crypt hyperplasia
Ischaemic colitis	"Thumbprinting" on CT (submucosal haemorrhage)	Occurs at watershed zones
Abetalipoproteinaemia	Lipid-laden enterocytes on postprandial biopsy	Defective chylomicron assembly

💡 Heads up: Even in diabetic patients, prophylactic cholecystectomy is not warranted for asymptomatic stones. This one comes up repeatedly.

💡 Heads up: Haemobilia presents with the triad of RUQ pain, obstructive jaundice, and GI bleeding (melaena). It usually follows iatrogenic trauma such as liver biopsy or percutaneous transhepatic cholangiography. Selective hepatic arteriography is gold standard - diagnostic and therapeutic via embolisation.

Chronic and Rare Biliary Conditions

Primary sclerosing cholangitis (PSC) - strongly associated with ulcerative colitis (60-80%). Produces a "beaded" appearance of ducts on imaging. High risk of cholangiocarcinoma

Porcelain gallbladder - complete intramural calcification. Previously thought to mandate surgery, but the malignancy risk is actually small; close surveillance is acceptable in asymptomatic patients

Cholesterosis ("strawberry gallbladder") - diffuse deposition of cholesteryl esters in macrophages; mucosal surface appears brick red with yellow flecks

Visual Anchors

Diagnosis	Radiographic Finding	Pathological Key
Acute cholecystitis	Failure to image the gallbladder on HIDA despite duct visualisation	Cystic duct obstruction
PSC	"Beaded" appearance on MRCP (multifocal stricturing)	Onion-skin periductal fibrosis
Choledochal cyst	Fusiform cystic dilation of the CBD, separate from the GB	Risk of cholangiocarcinoma
Porcelain GB	Thin, continuous rim of calcification outlining the wall on KUB	Chronic inflammation
Strawberry GB	Brick red mucosa with bright yellow flecks	Lipid-laden macrophages in lamina propria
Gallstone ileus	Pneumobilia (gas in biliary tree) + small bowel obstruction on KUB	Cholecystoduodenal fistula

Pituitary Gland & its Disorders

CELL	CORTICOTROPE	SOMATOTROPE	LACTOTROPE	THYROTROPE	GONADOTROPE
Tissue-specific transcription factor	T-Pit	Prop-1, Pit-1	Prop-1, Pit-1	Prop-1, Pit-1, TEF	SF-1, DAX-1
Developmental timing	6 weeks	8 weeks	12 weeks	12 weeks	12 weeks
Hormone	POMC	GH	PRL	TSH	FSH, LH
Protein	Polypeptide	Polypeptide	Polypeptide	Glycoprotein α , β subunits	Glycoprotein α , β subunits
Amino acids	266 (ACTH 1-39)	191	198	211	210, 204
Stimulators	CRH, AVP, cytokines	GHRH, ghrelin	Estrogen, TRH, VIP	TRH	GnRH, activins, estrogen
Inhibitors	Glucocorticoids	Somatostatin, IGF-1	Dopamine	T ₃ , T ₄ , dopamine, somatostatin, glucocorticoids	Sex steroids, inhibin
Target gland	Adrenal	Liver, bone, other tissues	Breast, other tissues	Thyroid	Ovary, testis
Trophic effect	Steroid production	IGF-1 production, growth induction, insulin agonism	Milk production	T ₄ synthesis and secretion	Sex steroid production, follicle growth, germ cell maturation
Normal range	ACTH, 4-22 pg/L	<0.5 μ g/L ^a	M <15 μ g/L; F <20 μ g/L	0.1-5 mU/L	M, 5-20 IU/L; F (basal), 5-20 IU/L

Feature	Type 1 AIT	Type 2 AIT
Mechanism	Jod-Basedow (iodine excess) in abnormal gland	Destructive thyroiditis (toxic effect)
Vascularity	Increased flow on Doppler	Decreased flow
Pathology	True hyperthyroidism	Follicle destruction
Treatment	Antithyroid drugs (ATDs)	Glucocorticoids

Visual Anchors -

Diagnosis	Visual / Radiographic Finding	Diagnostic Key
Graves'	Exophthalmos + pretibial plaque	TSI-positive; homogeneous high uptake
Hashimoto's	Firm, irregular goitre; "puffy" face	TPO antibodies; high TSH
PTC	"Orphan Annie Eye" nuclei on histology	Punctate microcalcifications (psammoma bodies)
Subacute Thyroiditis	Exquisitely tender small goitre	High ESR (>50) + low RAIU
AIT Type 2	Low vascularity on colour-flow Doppler	Destructive process in normal gland
Cretinism	Large tongue, global delay, goitre	Iodine deficiency (endemic)
Factitious	Normal-sized gland; undetectable Tg	Exogenous ingestion; low RAIU
Toxic Adenoma	"Hot" nodule on scintigraphy	Suppressed background gland activity

Drug Characteristic	Phentermine	Orlistat	Phen/TPM	NAL/BUP	Liraglutide	Semaglutide	Tirzepatide
Mechanism of action	Sympathomimetic, increases norepinephrine release in CNS	Gastrointestinal lipase enzyme inhibitor	Phen: sympathomimetic in CNS; TPM: modulates GABA receptors in the CNS	Nal: blocks opioid-mediated POMC autoinhibition; activates POMC in the hypothalamus	GLP-1 receptor agonist	GLP-1 receptor agonist	GLP-1/GIP dual receptor agonist
Route of administration, frequency, and dose	Oral, once to 3 times daily, doses of 8, 15, 30, and 37.5 mg	Oral, 3 times daily within 1 h of fat-containing meals	Oral, once daily. Start 3.75/23 mg/d × 2 weeks, then 6.5/46 mg/d; can escalate to max dose of 15/2 mg/d	Oral, 1 tablet (8 mg/90 mg) QAM × 1 week; 1 tablet in morning and evening × 1 week; 2 tablets in morning and 2 tablets in evening	Subcutaneous, once daily; initiate at 0.6 mg/d × 1 week; increase by 0.6 mg weekly to 3 mg/d	Subcutaneous, once weekly. 0.25 mg QW × 4 weeks, 0.5 mg QW × 4 weeks, then 1 mg QW × 4 weeks, then 2.4 mg QW	Subcutaneous, once weekly; 2.5 mg QW × 4 weeks, then 5 mg QW × 4 weeks, then 7.5 mg QW × 4 weeks, then 10 mg QW × 4 weeks, then 12.5 mg QW, then 15

Symptoms

Obstructive disease — described as "chest tightness" or "inability to get a deep breath"

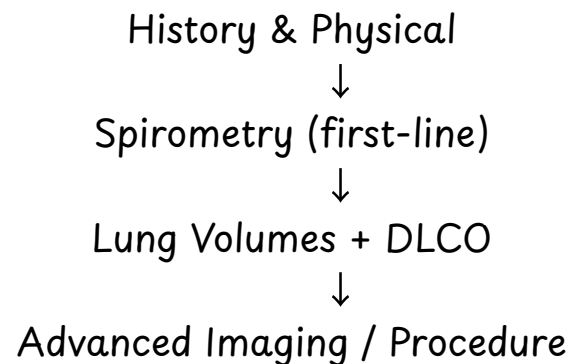
CHF — described as "air hunger" or a sense of suffocation

Chronic cough — defined as cough persisting >8 weeks

Chest pain — arises from the parietal pleura (richly innervated), not the parenchyma, which lacks pain fibres

Diagnosis

Step-ladder approach:



History & Physical: Screen for triggers (asthma), smoking (COPD), environmental exposures.

Spirometry:

$FEV_1/FVC < 70\%$ (or $< LLN$) → airflow obstruction

Reversibility = $\geq 10\%$ increase in FEV_1 % predicted (or $12\% + 200$ mL) after bronchodilator

Lung Volumes:

$TLC < 80\%$ → restrictive pathophysiology

High RV/TLC ratio → gas trapping due to premature airway closure

DLCO (Diffusing Capacity):

Decreased DLCO → parenchymal destruction (IPF, emphysema) or vascular disease (PE, PAH)

Management / Procedures

Interventional Pulmonology:

Rigid bronchoscopy is mandatory when there is high risk of severe bleeding (e.g., coagulopathy) — provides a secure airway and a large conduit for management
 Transthoracic Needle Aspiration (TTNA) — sensitivity >90% for peripheral lesions, but negative predictive value is only ~70%

Heads up: A negative TTNA result does not rule out cancer. NPV of ~70% means a significant proportion of negatives are false negatives. Don't be falsely reassured.

Radiology & Visual Anchors

HRCT:

Secondary pulmonary lobule — fundamental unit for HRCT analysis
 Centrilobular emphysema (CLE) — tends to appear in upper lung zones
 "Atoll Sign" (Reversed Halo) — characteristic of Cryptogenic Organizing Pneumonia (COP)
 "Eggshell" calcification — pathognomonic for Silicosis hilar lymphadenopathy

Flow-Volume Loop Shapes:

Pattern	Shape	Diagnosis
Scooped/Concave	Concave expiratory limb	Obstructive (asthma/COPD)
Miniature loop	Small overall loop	Restrictive (IPF)
Flattened (both limbs)	Flat inspiratory + expiratory	Fixed large airway obstruction (e.g., tracheal tumour)

Clinical Syndromes: Health Care-Associated Infections

Pathophysiology

Nosocomial infections = infections arising $\geq 48-72$ hours after hospital admission.

Core mechanism: **disruption of normal anatomic barriers** + alteration of the host's indigenous microbiota.

- Body is colonized by trillions of commensals that give **colonization resistance**
- Devices like **IV lines, surgical drains, endotracheal tubes, and Foley catheters** create artificial portals of entry
- Once barriers are breached, commensals (***S. aureus*, *S. epidermidis***) escape their normal habitats and colonize sterile sites
- Often helped along by **glycocalyx biofilms** forming on device surfaces — these protect pathogens from both host immunity and antibiotics
- **Antibiotic selection pressure** wipes out sensitive flora and lets **multidrug-resistant organisms (MDROs)** take over

Diagnosis / Definitions

- **VAP** — occurs ≥ 48 h after intubation
- **CLABSI** — central line lying in direct contact with bloodstream **triples the risk of in-hospital death**
- **CAUTI** — defined by $\geq 10^3$ CFU/mL in a symptomatic patient

CDI diagnostic step-ladder:

≥ 3 unformed stools in 24h



Review for antibiotic exposure



Contact Precautions (Gown/Gloves) [HY]



Stool NAAT or Toxin Enzyme Immunoassay



Environmental cleaning with sporicidal bleach

NTEP 2024 MDR-TB guidelines

Confirmed rifampicin-resistant TB → immediate transition to MDR regimens. Preferred: 6-month all-oral BPaLM (Bedaquiline, Pretomanid, Linezolid, Moxifloxacin).

Linezolid toxicity (myelosuppression/neuropathy) requiring permanent discontinuation → entire BPaLM regimen must be discontinued.

BPaLM strictly contraindicated in severe extrapulmonary or spinal TB (Pott's disease) — these need an individualized longer MDR-TB regimen, 18–20 months.

Heads up: If linezolid has to be stopped permanently for toxicity, you don't just swap it out, the whole BPaLM regimen gets discontinued.

NTM

M. kansasii: Isoniazid + Ethambutol + Rifampin. Clarithromycin is not used. MAC: macrolide-based daily regimen.

Heads up: *M. kansasii* gets INH + Ethambutol + Rifampin, not clarithromycin. Easy mix-up with MAC treatment.

Leprosy reactions

Type 1 (Reversal): slowly tapered systemic prednisolone.

Type 2 (ENL): prednisolone and thalidomide in severe/recurrent cases. MDT must be continued through the reaction.

5. Diagnostic Step-Ladders (as flowcharts)

Syphilis monitoring principle

Treat syphilis
|
v
Track response with quantitative VDRL/RPR titers

ICU Scoring Systems

System	Primary Purpose	Components	Key Clinical Differentiator [HY]
qSOFA	Rapid Screening	RR ≥ 22 , AMS, SBP ≤ 100	Not diagnostic; identifies patients at risk for poor outcomes
SOFA	Sepsis Diagnosis	Six Organ Systems (Resp, Coag, Liver, CV, CNS, Renal)	Sepsis = ≥ 2 point increase from baseline in the setting of infection
APACHE II	Mortality Prediction	Age, 12 physiologic variables, chronic health	Uses the worst values from the first 24 hours of ICU admission

Heads up: SOI scores like qSOFA, SOFA, and APACHE II are population-level tools built for research and quality assessment. They are not validated for making precise treatment calls or predicting exact outcomes in an individual patient at the bedside. Don't lean on them that way.

Monitoring the Critically Ill: Physiology of Perfusion Hemodynamic Surrogates vs True Volumes

Traditional markers like Central Venous Pressure (CVP) and Pulmonary Artery Occlusion Pressure (PAOP) are pressure-based estimates often used to infer preload.

Heads up: CVP and PAOP are pressures only [HY], not true ventricular volumes or cardiac output. Use them as trend indicators, not absolute measures of volume status.

Gold standard for CO: Thermodilution via a pulmonary artery catheter
Gold standard for core temperature: Pulmonary Artery temperature measurement

Secondary Prevention and ICDs

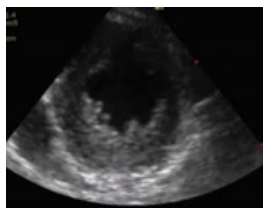
Secondary Prevention: indicated for survivors of VT/VF with no reversible cause [HY]

Primary Prevention: for LVEF $\leq 35\%$ (NYHA II-III) or LVEF $\leq 30\%$ (NYHA I), at least 40 days post-MI

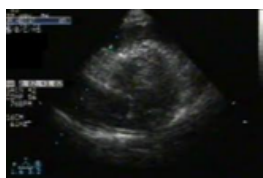
Medication: Beta-blockers are the only class consistently shown to reduce SCD risk across multiple settings [HY]

Visual Anchor: Mechanical Complications

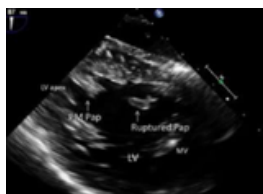
Ventricular Septal Rupture (VSR): new harsh pansystolic murmur and palpable thrill, typically 24 hours post-infarction. Echo shows a left-to-right shunt on Color Doppler.



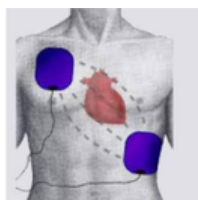
LV Free Wall Rupture: leads to Beck's Triad (hypotension, JVP distention, muffled heart sounds) from tamponade. On echo, look for a new pericardial effusion with echodense clotted blood [HY].



Papillary Muscle Rupture: blowing systolic murmur at the apex radiating to the axilla. Echo shows a flail mitral leaflet prolapsing into the LV.



ACLS Pad Placement: one pad below the right clavicle, other on the left lateral chest below the axilla (anterolateral), for optimal current through the myocardium.



Bone metastasis follows the "Seed and Soil" theory — tumor cells (the seed) get recruited to the bone marrow (the soil) via the CXCL12/CXCR4 chemokine pathway.

Clinical Etiology and Risk Factors

Environmental and modifiable factors drive an estimated 60–90% of cancer cases.

Tobacco smoking is the biggest driver. A "Current Smoker" is defined as someone who has smoked at least 100 cigarettes in their lifetime and currently smokes ≥1 cigarette per day. Smoking induces a higher tumor mutational burden (TMB), which increases mortality but can actually make some tumors (like lung cancer) more responsive to PD-1/PD-L1 immunotherapy, since higher TMB means more neoantigen expression.

Comparative Clarity: Major Modifiable Risk Factors [HY]

Risk Factor	Most Strongly Associated Cancer	Key Exam Fact
Obesity	Endometrial Cancer	RR is far higher than other obesity-related cancers.
Alcohol	Mouth, Pharynx, Larynx, Esophagus, Breast	Any amount of alcohol increases risk; higher intake adds stomach/liver risk.
Smoking	Lung, Oropharynx, Bladder	Multiplexes risk of second primary cancers if continued during CT/RT.
UV Radiation	Melanoma, BCC, SCC	Melanoma has the highest mutational burden of any studied malignancy.

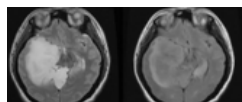
Indolent vs Aggressive

Low-grade gliomas: slow-growing, giving the brain time to adapt, which explains why seizures are the presenting symptom in up to 90% of cases [HY]

High-grade gliomas (Glioblastoma): highly infiltrative with rapid expansion, presenting with headaches and focal deficits

Diffuse Gliomas (Adult-Type)

Tumor Type	Key Molecular Features	Clinical Context
Astrocytoma, IDH-mutant	IDH1/2 mutation; 1p/19q intact	Grades 2-4; vorasidenib emerging therapy
Oligodendroglioma	IDH mutation + 1p/19q co-deletion [HY]	"Fried egg" appearance; very chemosensitive
Glioblastoma (GBM)	IDH-wildtype [HY]; TERT mutation or EGFR amp	Grade 4; ring-enhancing on MRI



Heads up: MGMT promoter hypermethylation silences the DNA repair enzyme MGMT. This is a positive prognostic factor because it makes the tumor more sensitive to Temozolomide [HY]. MGMT overexpression, on the other hand, predicts resistance. Don't mix these two up.

CNS Diagnostic Approach

Neurologic Exam
(focal deficits, signs of increased ICP: morning headache, nausea)



MRI with Gadolinium
(preferred diagnostic test)

Hemolytic Anemias: Intracorpuseular vs Extracorpuseular Defects

Pathophysiology

Hemolysis occurs when red cell life span (<120 days) is shortened beyond marrow compensatory capacity.

- **Intracorpuseular** defects: inherited membrane, enzyme, or hemoglobin abnormalities
- **Extracorpuseular** factors: immune, mechanical, or infectious triggers

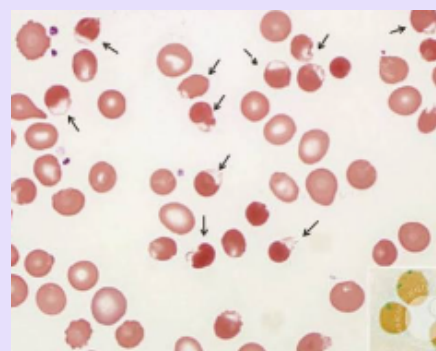
Inherited Enzymopathies and Membrane Defects

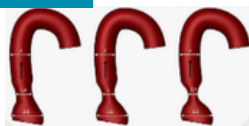
Hereditary Spherocytosis (HS): defective radial connections (e.g. ankyrin, band 3) lead to membrane vesiculation and spherocyte formation.

💡 **Heads up:** Increased MCHC (>34 g/dL) and RDW (>14%) with a negative Coombs test is highly suggestive of HS [HY]. Worth checking these together before jumping to other workups.

G6PD Deficiency: an intracorpuseular defect triggered by oxidative stress (fava beans, infections, drugs like **rasburicase**).

Visual Anchor: smear shows "**bite cells**" (blister cells) and **Heinz bodies** (precipitated denatured hemoglobin).





Disseminated Intravascular Coagulation (DIC)

Widespread thrombin generation triggered by Tissue Factor (TF) release.

Heads up: D-dimer elevation carries the highest single-point weight in the ISTH scoring system for DIC.

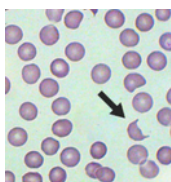
Differential — DIC vs liver failure:

Low Factor VIII → suggests DIC (consumed)

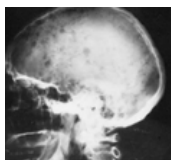
Normal/Elevated Factor VIII → favors Liver Failure (endothelial synthesis continues)

Visual Anchor Summary

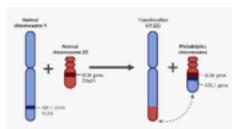
TTP / HUS / DIC: look for Schistocytes (fragmented RBCs) on peripheral smear.



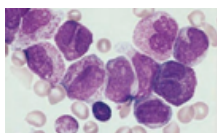
Multiple Myeloma: "Punched-out" lytic lesions on skull X-ray; "Church spire" M-spike on serum protein electrophoresis.



CML: Philadelphia Chromosome t(9;22) creating the BCR-ABL1 fusion protein.



APML: Abundant Auer rods and procoagulant granules in leukemic promyelocytes.



Heads up: Treat APML urgently with ATRA to differentiate the cells and resolve the DIC. Time matters here.

Heads up: Chronic PPIs are linked to fractures, pneumonia, C. diff, hypomagnesemia, and B12 deficiency — but NOT renal tubular acidosis. PPIs cause acute interstitial nephritis and CKD, not RTA. Don't get tricked into picking RTA on this one.

Sleep issues

Insomnia — chronic benzos (diazepam, alprazolam, clonazepam) are inappropriate in older adults: daytime sedation, worse cognition, worse delirium, doubled fall/MVA risk. Melatonin [HY] is the safer choice for sleep-onset insomnia, doesn't impair cognition or raise fall risk.

Restless Legs Syndrome (RLS) — irresistible urge to move legs in the evening, relieved by movement. Iron deficiency [HY] is a key reversible risk factor (low brain iron impairs dopaminergic pathways). Always check ferritin [HY] and correct if low.

Delirium in hospitalized patients

CAM criteria (need 1 + 2, plus either 3 or 4):

Acute onset and fluctuating course [HY]

Inattention (the hallmark)

Disorganized thinking

Altered level of consciousness

Look for reversible precipitants: hyponatremia, hypercalcemia, infections (pneumonia, UTI), thyroid dysfunction, fecal impaction (check with DRE) [HY].

Heads up: Hyponatremia and hypercalcemia are classic electrolyte causes of delirium. Hypokalemia [HY] is not — don't list it as a direct cause on an exam.

Managing agitation, step by step:

First line: non-drug measures (reorientation, sleep-wake cycle, sensory aids).

Second line: if patient's a danger to self/others (pulling lines, climbing out of bed) — low-dose risperidone (0.25–0.5 mg) [HY] short term.

Third line: avoid benzos routinely, they worsen confusion and prolong delirium. If one is truly needed (e.g. alcohol/sedative withdrawal), lorazepam [HY] is preferred — it's cleared by direct hepatic glucuronidation with no active metabolites, so it doesn't accumulate in older adults. Avoid alprazolam (fast dependence).

Thrombocytopenia (platelets $<100,000/\mu\text{L}$) [HY]

Renal insufficiency (creatinine $>1.1\text{ mg/dL}$)

Impaired liver function (AST/ALT $\geq 2\times$ upper limit of normal)

Pulmonary edema

New-onset cerebral/visual disturbances (severe headache, blurred vision)

sFlt-1/PlGF ratio [HY] predicts severe preeclampsia. A ratio ≥ 40 [HY] is highly predictive within 2 weeks [HY] and needs close monitoring.

Management timing:

Without severe features $\rightarrow$ expectant, deliver at 37 weeks [HY].

With severe features $\rightarrow$ deliver at ≥ 34 weeks [HY] (earlier if things deteriorate).

Managing severe hypertension

Acute severe hypertension ($\geq 160/110$) is an emergency — needs urgent treatment to avoid maternal intracranial hemorrhage.

First-line: IV Labetalol, IV Hydralazine, oral immediate-release Nifedipine [HY].

Chronic hypertension target per the CHAP Trial: $<140/90\text{ mmHg}$ [HY] using pregnancy-safe drugs.

Heads up: ACE inhibitors (enalapril) and ARBs (losartan) [HY] are absolutely off the table [HY] in pregnancy, full stop. Blocking the fetal RAAS causes fetal renal dysgenesis [HY], severe oligohydramnios [HY] ($\rightarrow$ pulmonary hypoplasia, limb contractures), skull ossification defects, and fetal death.

HELLP vs AFLP

Both are severe third-trimester emergencies that look similar on the surface but come from different pathways.

HELLP syndrome — microangiopathic hemolytic variant of preeclampsia.

Endothelial injury $\rightarrow$ fibrin deposition $\rightarrow$ shears RBCs (schistocytes on smear) and eats up platelets. Hepatic sinusoidal obstruction $\rightarrow$ ischemia, high transaminases, stretched Glisson's capsule $\rightarrow$ severe RUQ/epigastric pain [HY].

AFLP (Acute Fatty Liver of Pregnancy) — metabolic emergency from microvesicular fatty infiltration of hepatocytes, often tied to autosomal recessive fetal LCHAD deficiency. Fetus can't oxidize fatty acids, so medium/long-chain fatty acids build up toxic in maternal liver $\rightarrow$ rapid hepatic failure. Presents with encephalopathy, severe coagulopathy (INR >2.0), leukocytosis, and severe hypoglycemia (glucose $<42\text{ mg/dL}$) [HY].

2. Severe Immune-Mediated Type B Adverse Drug Reactions

DRESS / DIHS

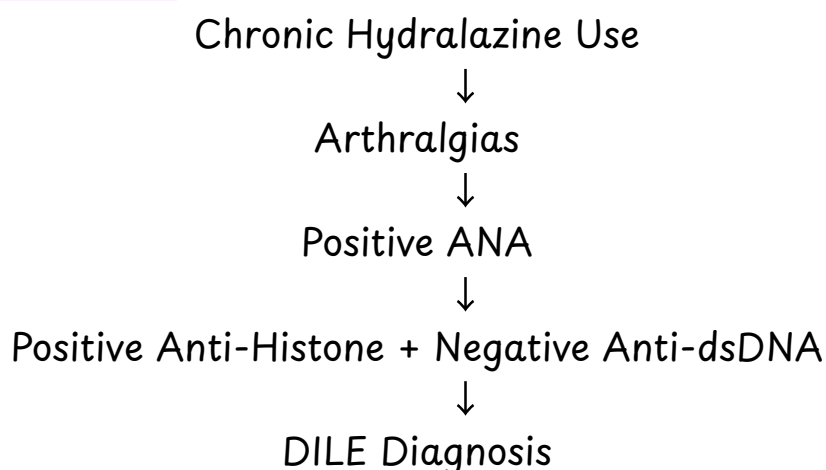
Delayed T-cell-mediated (Type IV) severe cutaneous adverse reaction, 2–8 week latency, triggered by aromatic anticonvulsants (carbamazepine, phenytoin, phenobarbital). Uniquely driven and amplified by reactivation of latent HHV-6, causing high-grade fever, facial edema, diffuse maculopapular rash, lymphadenopathy, eosinophilia, atypical lymphocytosis, and severe hepatitis. Highly persistent, needs a prolonged, slow systemic corticosteroid taper.



Drug-Induced Lupus Erythematosus (DILE)

Classically triggered by hydralazine, procainamide, isoniazid (INH), occurring predominantly in NAT2 slow acetylators. Classic triad: arthralgia, fever, myalgia plus serositis. Renal and CNS involvement are rare (this is what distinguishes it from idiopathic SLE).

Diagnostic step-ladder:



Immunofluorescence

