Medicine summary

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Contents

L1: Acute coronary syndrome (ACS)

- Acute coronary diseases are the number one killers
- Risk factors for coronary diseases are: Diabetes mellitus, smoking, Hypertension, obesity, hyperlipidemia, age (>45 for males and >55 for females), Male gender and family history.
- Acute coronary syndromes are categorized into two main categories: ST-elevation acute coronary syndrome (e.g. STEMI) and non ST-elevation acute coronary syndrome (e.g. NSTEMI, unstable angina).
- To differentiate between NSTEMI and unstable angina, in NSTEMI cardiac enzyme are elevated while in unstable angina there is no cardiac enzyme elevation
- Creatine kinase MB (CK-MB) is cardio-specific and starts to rise 4-6 hours after onset of ischemia, then falls within 48-72 hours.
- Cardiac-specific troponin I and T are the most sensitive and specific markers for MI.
- Troponins T and I are released 4-6 hours and can still be found in blood up to 2 weeks.
- Aims of therapy in STEMI:

1) Open Artery and Improve oxygen supply:

- Supplemental O_2
- Coronary vasodilators (Nitroglycerine)
- Antiplatelet agents: Aspirin (ASA), P2Y12 inhibitors (Clopidogrel)
- Antithrombotic agents: Heparin (low molecular or unfractioned)

2) Reduce O_2 demand

- Beta-blockers and Analgesics.

3) Reperfusion therapy:

- Fibrinolytic therapy: ONLY for STEMI.
- Primary Percutaneous coronary intervention (PCI)

❖ Complications of MI:

- Arrhythmias (e.g. Ventricular fibrillation => Major cause of death)
- Heart failure (Pulmonary Edema)
- Cardiogenic Shock
- Mechanical complications (usually occurs late after MI)
- Pericarditis
- Embolism

L2: Arrhythmia

A. Atrial fibrillation (Afib)

- **Pathophysiology:** SA node lost its control to begin action potential and all heart cells instead produce AP in **Multiple circuits pattern**, which lead to Afib
- **Causes:** Mitral stenosis **"most common",** IHD, HTN, CHF, pneumonia, PE, COPD, hyperthyroidism

❖ **Signs & Symptoms:** palpation **"most common",** SOB, fatigue, Stroke, tachycardia

❖ **Recurrence of Afib is more likely when:**

1. Structure remodeling (fibrosis and enlargement of atrium due to previous Afib)
2. Electrical remodeling (decrease refractory period due to previous Afib)

❖ **Most dangerous complication of Afib is: Stroke** due to formation of clot in Left atrial appendage (LAA) ''because Afib cause stagnation in this muscular site''

- **Investigations:**

1. ECG of Afib: shows; **A.** Irregularly irregular rhythm **B.** absence of P wave **C.** Skip beat (AV node blocks beats to allow filing of atrium).

2. Transthoracic echocardiogram (TTE): to know if there is **mitral stenosis**

3. Holter monitor: it is 24h ECG, if you suspect Afib and to monitor treatment

4. Transesophageal echo (TEE): to know if there is any **clot in left atrium**

5. Chest x-ray: if you suspect **pneumonia or CHF** as a cause of Afib **6. Thyroid function:** to rule out hyperthyroidism

- **Classification of Afib by onset:**

1. Paroxysmal: when palpation appears and disappears spontaneously within 24 h

2. Persistent: when patient did cardio version in ER or palpation persist for more than 7 days

3. Permanent: when palpation is persist whole the year

- **Classification of Afib by etiology:**

1. Primary Afib:

A. Valvular Afib such as mitral stenosis - B. Non-valvular Afib due to many factors

2. Secondary Afib: due to acute attack such as injury

❖ **Management:**

Prevention of thromboembolism	Rate control	Maintenance & Restoration of sinus rhythm
- Use CHA2DS2VASc score "very important" **# exception from score is ''Afib with mitral stenosis''** **1. Warfarin (has narrow therapeutic index 2-3 INR & drug-food interactions)** **2. Aspirin less effective than warfarin** **3. Rivaroxaban (all ban family does not need INR monitor and no drug-food interactions) but has limitation with valvular Afib** **4- If all drugs fails remove LAA**	**- maintain heart rate below 110 during daily activities** 1. **beta blockers and calcium channel blocker** has the same effect But we avoid beta blockers in case of asthma and calcium channel blockers in case of ischemia 2. **digoxin** in case of hypotension 3. when all three fails use **peacemaker or ablation**	- **Maintenance of sinus rhythm** by using anti-arithmetic drug, the best is amiodarone but with worst side effect (e.g. pulmonary fibrosis, thyrotoxicosis) given for very old patient. **- Restoration of sinus rhythm by two ways:** 1. Immediate Cardio version which is used when symptoms start in less than 48 h 2. When symptom last for more than 48 h or the patient do not know the exact time when symptoms started we do TEE if there is clot start warfarin for 4 weeks then you do cardio version. In both cases patient must be on warfarin for 4 weeks after shock.

B. Atrial flutter

is re-entry circuits, **ECG**: shows ''saw-tooth'' appearance, **Treatment**: ablation

C. Supra ventricular tachycardia (SVT):

Benign conditions which treated electively by ablation, In AVRT (wolf-Parkinson-white syndrome) you will see delta wave in ECG due to accessory pathway

D. Ventricular arrhythmias:

VT: treat underlying cause or Shock if emergent**,** VF: treated by shock only then implant ICD

E. First degree block:

prolonged PR interval more than 200 ms

L3: Valvular Heart Disease

Mitral stenosis:

- Most common cause is rheumatic fever and more common in women.
- Clinical symptoms and signs: Dyspnea, Orthopnea, and PND, Hemoptysis, Systemic embolism "due to Afib", Hoarseness and dysphagia, Right-sided heart failure, Malar flush. Atrial fibrillation Loud S1, opening snap following S2, and mid-diastolic rumbling murmur.
- Management: diuretic and salt-restricted diet, digitalis and anticoagulant "in Afib", balloon valvulotomy "especially in pregnancy", mitral commissurotomy or valve replacement if balloon dilatation fails.

Mitral regurgitation:

- Most common causes are ischemia and myxomatous.
- Clinical symptoms and signs: Dyspnea, Orthopnea, and PND, Right-sided heart failure, atypical chest pain and palpitation. pansystolic murmur radiating to the axilla with thrill, mid-diastolic click , S3 gallop, distended neck veins in sever or acute MR.
- Management: after load reduction, mitral valve repair or replacement.

Aortic stenosis:

- Calcific aortic valve (usually in elderly) is the most common cause; bicuspid valve is the most common congenital cause.
- Clinical symptoms and signs: angina, syncope, CHF. Slow raising carotid pulse, radiate to the carotids. Systolic ejection murmur.
- Diagnosis: cardiac cath is the definitive diagnostic; > 0.8 cm^2 indicates severe stenosis.
- Management: valve replacement is the treatment of choice.

Aortic regurgitation:

- Causes: **Acute** → acute rheumatic fever, infective endocarditis, **chronic** → RHD, bicuspid valve, sever HTN, marfan's syndrome.
- Clinical symptoms and signs: angina, dyspnea. Mid-diastolic rumble at the apex, S3 and S4 gallop, duroziez's sign "femoral bruit", de musset's sign "head nodding with pulse".
- Management: **If the patient stable** → conservative like diurtics, salt restriction, and vasodilator. **Acute** → emergent valve replacement.

Tricuspid valve:

- Stenosis: IV drug abusers and carcinoid heart disease.
- Regurgitation: secondary to pulmonary HTN.

Mitral valve prolapse "Barlow Syndrome":

- Causes: large leaflets, abnormal papillary contraction.
- Clinical symptoms and signs: Atypical chest pain and palpitations. Mid-diastolic click, the most common sign. – Management: beta blockers for chest pain and palpitations.

L4: Heart failure

- **Heart failure** is a clinical syndrome resulting from the heart's inability to meet the body's circulatory demands under normal physiological conditions.
- Characterized by signs and symptoms of **high volume** and **low perfusion**:

High volume	Low perfusion
✓ Elevated JVP	✓ Fatigue
✓ Orthopnea	✓ Shortness of breathe
✓ PND	✓ Loss of conciseness
✓ hepatomegaly	✓ Cool extremities
	✓ hypotension

- Causes of HF:
  - Can be acute, such as: MI and myocarditis
  - Can be chronic (most common):
    - ✓ Coronary artery disease
    - ✓ Hypertension
    - ✓ Valvular heart disease
    - ✓ Dilated cardiomyopathy
    - ✓ Cor-pulmonale

- **Cardiomyopathy** is an abnormal function of the heart muscle:

Dilated cardiomyopathy	Hypertrophic cardiomyopathy	Restrictive cardiomyopathy
❖ Its dysfunction in contraction (ejection) ❖ **Causes:** ✓ **idiopathic** ✓ **Alcohol** ✓ **Chemotherapy**, such as: Trastuzumab and 5-Fluorouracil ✓ **Metabolic**: Thiamine deficiency, hypophosphatemia and Excess catecholamines (pheochromocytoma) ✓ **Skeletal myopathies:** Duchene and Becker ✓ **Peripartum cardiomyopathy:** appears in the last month before pregnancy or in the first 5 months after pregnancy (to the mother)	❖ Its dysfunction in relaxation (filling) ❖ It's about a reaction of the heart to increased BP. The heart hypertrophies to carry the load, but then develops difficulty in diastole (such as in long standing hypertension and highly trained athletes) ❖ Usually familial, with autosomal dominant inheritance ❖ May be genetic ❖ Clinical presentations usually start at puberty ❖ Most patients are asymptomatic. Occasionally, sudden death may be the 1st presentation ❖ How to diagnose? ✓ family history focusing on premature cardiac disease or death ✓ Echo	❖ Its dysfunction in **both**, contraction and filling ❖ **Causes:** ✓ **Sarcoidosis** ✓ **Amyloid** ✓ **Hemochromatosis** ✓ **Scleroderma**

L5: Heart Failure (H.F) Prognosis & Management

A- Systolic dysfunction:

- **Life style modification.**
- **Thiazide diuretics:** Hydrochlorothiazide, Metolazone. It blocks tubular reabsorption of Na and Cl. Indication: for symptoms relief of volume overload. Side effect: neutropenia, thrombocytopenia.
- **Loop diuretics:** Furosemide: Inhibit Cl reabsorption in ascending limb of loop of Henle. Indication: just for symptoms relief of volume overload. Side effect: hypokalemia.
- **K+ sparing agents:** reduces morbidity and mortality in patients with heart failure. It is contraindicated in renal failure. **Triamterene & amiloride:** act on distal tubule to decrease K^+ secretion. **Spironolactone:** aldosteron-antagonest (prolonged survival). **Eplerenone:** alternative to Spironolactone (doesn't cause gynecomastia).
- **ACE inhibitors:** reduces mortality. Block RAAS. Side effect: Renal insufficiency, Hypotension.
- Combination of ACE inhibitors + β-blockers: for patient with LVEF Less than 40%.
- **ARBs Inhibitors:** If the patient unable to take ACE inhibitors due to side effect.
- **β-blockers:** Carvedilol, metoprolol. Decrease Mortality.
- **Digitalis:** with Afib.
- **Hydralazine & isosorbide dinitrates:** in patient cannot tolerate ACE inhibitors.
- **Positive Inotropic Agents:** Dobutamine used in pulmonary edema and cardiogenic shock.
- **Anticoagulation & Antiarrhythmics**.

B- Diastolic dysfunction:

- **Beta-Blockers & Diuretics**.

- Most Common Cause of death from CHF is: Ventricular Arrhythmias.
- Standard treatment of CHF includes a loop diuretic, ACE inhibitor, and beta-blocker.
- Drugs Contraindicate in H.F: Metformin, thiazolidinediones, NSAIDs, and glitazone.

L6: Rheumatic Heart Disease

Acute Rheumatic Fever: A multisystem disease resulting from an autoimmune reaction to an infection with group A streptococcus. It is mainly a disease of children aged 5-14 and the **initial episodes** becomes less common with aging. **Recurrent episodes** become more common with aging.

- Up to 60% of patients with ARF progress to RHD. **The valvular damage is the hallmark of Rheumatic Carditis.**
- Susceptibility to Acute Rheumatic Fever is an inherited characteristic.

Rheumatic Heart Disease: is the **chronic valvular abnormalities** secondary to acute rheumatic fever.
Two theories for **pathogenesis:**

A) Molecular mimicry: Whereby an immune response targeted at streptococcal antigens also recognize the human tissues.
B) Streptococcal invasion: Invasion of the epithelial surfaces allowing it to become immunogenic.

Pathology:

- Fibrinoid degeneration seen in the collagen of connective tissues.
- **Aschoff nodules** seen only in the heart; composed of multinucleated giant cells surrounded by macrophages and T lymphocytes.

Clinical Features:

- The most clinical features are polyarthritis and carditis.
- Many patients report prior **sore throat, yet the infection is commonly sub-clinical** & it can only be confirmed by using streptococcal antibody testing.
- There is a latent period of (1-5) weeks.
- The mitral valve is almost always affected, sometimes with the aortic valve.
- Isolated aortic valve damage is rare.
- **Myocardial inflammation may cause electrical conduction abnormalities and prolongation of P-R interval due to 1st degree AV block and softening of the heart sound.**
- People with RHD are often asymptomatic for many years before their valvular disease progress to cause cardiac failure.

To confirm the diagnosis make a connection between the investigations and Jones' criteria:

Jones' Criteria	Investigations	Treatment and prevention
2 Major signs **or** 1 major and 2 minor. **Majors:** - Carditis. - Polyarthritis. - Sydenham Chorea. - Erythema Marginatum. - Subcutaneous nodules. **Minors:** - Fever. - Arthralgia. - Past history of rheumatic fever. - Raised CRP or ESR and leukocytosis. - Prolonged P-R interval.	- Throat swab culture. - Elevated antistreptolysin O (ASO) antibodies levels. - Cardiomegaly, pulmonary congestion. - **ECG** (prolonged P-R interval) - **Echo; cardiac dilation and valve abnormalities.**	**Treatment:** - Salicylate (aspirin): for arthritis. - Prednisolone: for carditis or severe arthritis. - Valve replacement. **Prevention of recurrent attack:** - Intramuscular Benzathine Penicillin G. - Oral Erythromycin for individuals who are allergic to penicillin and sulfadiazine.

L7: Infective endocarditis (I.E)

- It's inflammation of endocardium (internal structure, valve) by infection
- Increase turbulent of flow (e.g. VSD) → Trauma of endocardium → inflammation, it will cause Non-bacterial thrombotic endocarditis (NBTE) → if there is a bacteremia for any reason → will cause vegetation → I.E
- Bacterial come usually from mouth or urogenital
- Vegetation is the hallmark of I.E, it will cause valve destruction → H.F and stroke.
- Have high mortality, 20% of who develop I.E
- 2 g of penicillin as a prophylaxis for who at risk for I.E before mouth, or urogenital work
- Drug abuser (Right side I.E): They destructed their vein by needle causing NBTE, and then bacteria come from skin due to decrease hygiene → vegetation → to heart → to lung and cause pneumonia and septic embolism.
- Most common cause of I.E is Streptococcus viridans
- Most common cause of Right sided I.E is Staphylococcus aureus.
- Most common causes of I.E in prosthetic valve are: before 2 month is Staphylococcus aureus, while more than 2 month is Streptococcus viridans.
- Congenital heart disease, valvular replacement, previous endocarditis, and drug abuser are at risk to develop I.E
- Usually symptoms begin after 2 weeks of infection
- Symptoms are FROM JANE: Fever, Roth spot, Osler's nodes, Murmur, Janeway lesion, Anemia, Nail hemorrhage, Emboli. Some patient may come with H.F or sepsis.
- Investigation: CBC: anemia and ↑WBC.↓ESR. Blood culture: should be 3 cultures from 3 different sites, and 3 bottles (if 2 +ve out of 3 consider +ve test). Echo is a diagnostic test (vegetation will appear clearly).
- Duke criteria BE FIVE PM: Major criteria: +ve Blood culture (2 +ve out of 3 consider +ve test), +ve Echo (vegetation, abscess or valve dehiscence). Minor criteria: Fever, Immunoligical phenomna (Osler's, Roth), Vascular phenomna (Janeway lesions), Echo (suggestive but not definitive), Predisposition (heart condition or IV drug user), Microbiologic evidence (Positive blood culture but not meeting major criteria).
- To dignose one with I.E should have 2 major criteria, 1 major and 3 minor criteria, or 5 minor criteria
- Treatment: Antibiotic should be bacteriocidal, 2 g ampicillin and ceftriaxone for 4 to 6 weeks.
- Complication: CHF (most common complication), stroke, metastatic infection (Right side I.E goes to lung, Left side I.E goes to system like; meningitis, pyelonephritis).

L8: Cough

- The efferent pathway of cough goes through 4 phases:
  Inspiratory, compressive, expiratory and relaxation phase.
- Complications of cough include: Headache, dizziness, musculoskeletal pain, urinary incontinence (especially in females) and rib fracture.
- Cough is classified into 3 main categories based on duration:

1- Acute cough: < 3 Weeks

- Caused by upper respiratory tract infection (URTI) by viral or bacterial pathogen. (40%-50% of cases will have cough), URTI exacerbating already exist lung disease such as COPD or asthma, Pneumonia, Foreign body, aspiration, or Left ventricular heart failure.
- It is managed by only WATCHING (mostly resolve within 2 weeks)
- RED flags in acute cough:
  Symptoms: Weight loss, SOB, hemoptysis, fever, chest pain.
  Signs: Crackles, cyanosis, bronchial breathing, dull chest, tachypnea.

2- Sub-acute cough: 3 – 8 Weeks

- Likely due to: Post-infectious, asthma, bacterial sinusitis.
- Post-infectious cough: Begins with acute URTI and is not complicated by pneumonia (normal CXR and lung examination), due to postnasal drip or tracheobronchitis.

3- Chronic cough: > 8 weeks

- Differential diagnoses: Lung disease, Gastro esophageal reflux disease (GERD), Post nasal drip due to allergic rhinitis or bacterial sinusitis, ACEI, tobacco use or habit coughs.
- CXR is ALWAYS needed in chronic cough.

- **GERD** and chronic cough: Mechanisms;

1. Aspiration to larynx or trachea: involve both cough symptoms (throat clearing and, worsen at night or with eating) and GI symptoms (heartburn, sour taste, regurgitation, and morning hoarseness).
2. Acid in distal oesophagus stimulates vagus and cough reflex: only cough symptoms (throat clearing and worsen at night or with eating) without GI symptoms.

- NSAIDs, Theophylline, ascorbic acid, alcohol, caffeine, fat, CCBs and beta-agonist reduce LES pressure and worsen reflux.
- Investigations: Esophageal pH monitoring for 24 hours, Endoscopy.
- Treatment: PPI + prokinetic e.g. domperidone or metoclopramide.

- **Post nasal drip:**

- Causes: Allergic rhinitis, non-allergic rhinitis or chronic bacterial sinusitis.
- Symptoms: 'something dripping', frequent throat clearing and nasal congestion or discharge.
- Treated by treating infection + nasal steroids.

L9: Bronchial asthma

- It is an abnormal bronchoconstriction of the airways. It is **reversible** (main difference between asthma and COPD).
- **Etiology:** Allergens, Infection, GERD, Emotional stress, exercise, Aspirin, NSAIDs, Beta blockers, histamine, Smoking.
- **Symptoms:** wheezing "**most common**", Dyspnea, cough, chest tightness, sputum, and Eczema.
- Symptoms often worsen at night or early in the morning.
- **Investigations:**

 # Best initial test in acute attack of asthma:

1- **Peak expiratory flow (PEF):** **Decrease** FEV1, FVC and FEV1\FVC ratio. **"Normal= 450-650 (men), 350-500 (women)"**

2- **ABG:** **Decrease** PaCo2 & **Increase** a-a gradient → asthma attack If Increase PaCo2 → Respiratory failure → **Intubation**

 # Most accurate test and for confirmation is Pulmonary function test (Spirometry):

- ✓ Decreased FEV1 and decreased FVC with a decreased ratio of FEV1/FVC
- ✓ **Increase** in FEV1 of more than 12% and 200 mL with the use of **bronchodilator (albuterol)**
- ✓ **Decrease** in FEV1 of more than 20% with the use of **bronchoconstrictor (methacholine or histamine)**
- ✓ **Increase** in the diffusion capacity of the lung for carbon monoxide (DLCO)

#Other tests: Chest x-ray to exclude other conditions (pneumonia, pneumothorax, foreign body).

- **Management:**

Acute severe asthma exacerbation	Chronic asthma
1- Initial therapy is **oxygen** combined with **inhaled short-acting beta agonists** such as albuterol and **a bolus of steroids**. 2- **Magnesium** helps relieve bronchospasm. Magnesium is used only in an acute, severe asthma exacerbation **NOT** responsive to several rounds of albuterol while waiting for steroids to take effect. 3- If the patient does **not respond** to oxygen, albuterol, and steroids or **develops respiratory acidosis** (increased pCO2), the patient may have to undergo **endotracheal intubation for mechanical ventilation.**	**Follow the steps if there is no response to previous step:** 1- Start with= **inhaled short-acting beta agonist (albuterol)** 2- Add= **long-term control agent Low-dose inhaled corticosteroids** 3- Add= **long-acting beta agonist (salmeterol)** 4- Increase= **corticosteroid to maximum (include previous steps)**

 # General notes for management:

- **Alternate long-term control agents other than steroids:** Cromolyn, nedocromil, Theophylline, montelukast.
- **Add Antibiotics if you suspect infection**

L10: COPD and bronchiectasis

I. **Chronic bronchitis:** Chronic productive cough for at least 3 months per year for at least 2 consecutive years.
II. **Emphysema:** permanent enlargement of air spaces distal to terminal bronchioles due to destruction of alveolar walls.

- The two often coexist.

Risk factors: Tobacco smoke and α_1 antitrypsin deficiency are the most important risk factors.

Sign and symptoms: Cough, sputum production, and dyspnea. Prolonged forced expiratory time (> 6 sec indicate obstruction), end-expiratory wheezing, using of accessory muscles, and hyper resonance in percussion, tachypnea, tachycardia and cyanosis.

Diagnosis:

- PFT: The definitive diagnostic test, decreased FEV and FEV1/FVC ratio (70% of predicted indicate mild disease, 50% or less indicate sever disease and in between is moderate).
- CXR: sever emphysema will show hyperinflation, flattened diaphragm, enlarged retrosternal space and diminished vascular marking. Useful in exacerbation to rule out complications.
- α_1 anti-trypsin level.
- ABG: chronic CO_2 retention decreased Po_2.

Management:

Smoking cessation (the most important), inhaled anti-cholinergic drugs (ipratropium), inhaled β_2 agonist (albuterol) or combination of both, oxygen therapy, vaccination (influenza, and strept. Pneumoniae), and pulmonary rehabilitation.

Management of acute exacerbation:

1. Bronchodilators are the first line.
2. Systemic corticosteroids (IV methylprednisolone).
3. Antibiotics (azithromycin or levofloxacin).
4. Supplement of oxygen.
5. Noninvasive positive-pressure ventilation.
6. Intubation if there is severe, acute respiratory acidosis and Co_2 retention.

Complications:

1- Acute exacerbation 2- secondary polycythemia 3- pulmonary HTN and cor-pulmonale.

III. **Bronchiectasis:** permanent, abnormal dilation and destruction of bronchial wall, with damaged cilia.

- Cystic fibrosis is the most common cause.
- **Clinical features:** chronic cough with large amount of mucopurulent foul smelling sputum, dyspnea, hemoptysis.
- **Diagnosis:** High resolution CT-scan is the study of choice.
- **Treatment:** Antibiotics (tobramycin, gentamycin), bronchial hygiene (hydration, chest physiotherapy, and inhaled bronchodilators).

L11: Pulmonary Embolism

- **PE** drives from **DVT** of large vessels of the legs 70% and pelvic veins 30%

Etiology of venous thrombosis (Virchows triad)	Etiology of DVT	Common presentations of PE
• Stasis of blood flow • Endothelial injury • hypercoagubality	• General anesthesia • Lower limb or pelvic injury or surgery • Congestive heart failure • Prolonged immobility • Pregnancy • Postpartum • Oral contraceptive pills • Malignancy • Obesity • Advanced age • Coagulation problems	• Sudden dyspnea • Pleuritic chest pain • hemoptysis

PE investigations	Notes and abnormalities
CXR	• best initiation, usually **normal**, but you may see atelectasis
EKG	• best initiation, usually shows sinus **tachycardia**
ABG	• best initiation, may shows **respiratory alkalosis and hypoxia**
Spiral CT	• the diagnostic tool
V/Q scan	• preferred in pregnancy than spiral CT
D-dimer	• it's very **sensitive**, but the **specificity is poor** (-ve test excludes a clot, but a +ve test doesn't mean anything)
Lower extremity Doppler study	• if It's positive, **no further tests is needed** • if it's negative, **don't exclude PE**
Angiography	• **rarely done,** because there is 0.5 mortality

Management

Heparin	Warfarin	Inferior vena cava filter (when?)
• best initial • Heparin-induced thrombocytopenia and thrombosis, so you need to do CBC every week	• Use it with heparin until the INR is done (2-3) • DON'T USE IT IN PREGNANCY	• Absolute contraindication to anticoagulation • Recurrent PE despite adequate anticoagulant therapy • Hemodynamic or respiratory compromise that is severe enough that another PE may be lethal

❖ **Massive PE:**

- ✓ Decreased cardiac output (shock)
- ✓ The patient dies within the one hour
- ✓ Thrombolytics is the treatment

L12: Community acquired pneumonia (CAP)

- **Pneumonia:** inflammation of the parenchyma of the lung (alveoli).
- **Types of pneumonia:**

1. Community-acquired pneumonia (CAP).
2. Healthcare-associated pneumonia (HCAP): Pneumonia that develops within 48 hours of admission.
3. Hospital-acquired pneumonia (HAP): Pneumonia after 48 hours of admission.
4. Ventilator-associated pneumonia (VAP): Pneumonia after 48 hours of intubation.

- **Types of CAP:**

1. Typical CAP:

- **Symptoms:** Acute onset of fever, shaking chills, productive cough, and pleuritic chest pain.
- **Signs:** Tachycardia, tachypnea, late inspiratory crackles, and dullness on percussion.
- **CXR:** Lobar consolidation.
- **Most common cause:** Streptococcus pneumonia (Gram +ve diplococci), especially if you did Splenectomy.
- Haemophilus influenza: Most common cause in children, and also important in splenectomy patient.
- Influenza is most important viral cause in adults, especially during winter months.

2. Atypical CAP:

- **Symptoms:** Headache, sore throat, fatigue, myalgias, dry cough, and fevers.
- **Signs:** Normal pulse in the setting of high fever, wheezing, rhonchi, crackles.
- **CXR:** Diffuse reticulonodular infiltrates.
- **Most common cause:** Mycoplasma pneumonia.
- **Coxiella burnetii:** Cause Q fever.
- **Francisella tularensis:** Cause tularemia, and transmitted from Rabbits.
- **Chlamydia psittaci**: transmitted from birds.

- **Investigations of CAP:** CXR, CBC (shows neutrophils leukocytosis), blood culture, ABG, Sputum Gram stain, Acid-fast stain" for TB", and Silver stain for Pneumocystis carinii in HIV patients.
- **CURB-65 scoring system** helps guide antibiotic and admission policies.

L13: Pleural Effusion

- Definition: Presence of large amount of fluid in the pleural space.

	Transudate (usually bilateral)	Exudate (usually unilateral)
Patho-physiology	↑ Capillary pressure in visceral or parietal pleurae. ↓ Plasma oncotic pressure.	↑ Permeability of pleural surface. ↓ Lymphatic flow from pleural surface.
Causes	• Ascites. • Cirrhosis. • Congestive heart failure. • Pulmonary Embolism • Hypoalbuminemia. • Intra-abdominal fluid. • Malnutrition. • Nephrotic syndrome. • Peritoneal dialysis.	• Asbestosis. • Chylothorax • Parapneumonic causes (pneumonia, lung abscess, and bronchiectasis). • Collagen vascular disease. • Complications of abdominal surgery. • Empyema. • Infection. • Lymphedema • Malignancy (primary lung cancer, lymphoma, metastatic cancer) • Pancreatitis • Rheumatoid arthritis. • Ruptured esophagus. • Trauma.
Diagnosis	• Pleural fluid–to-serum protein ratio < 0.5 • Pleural fluid–to-serum LDH ratio < 0.6	• Pleural fluid–to-serum protein ratio > 0.5 • Pleural fluid–to-serum LDH ratio > 0.6
Manage-ment	A. Diuretics and sodium restriction. B. Therapeutic thoracentesis (only if massive effusion is causing dyspnea)	Treat the underlying cause.

Investigations

CXR	Ultrasound	CT-Scan	Thoracentesis
About 250 mL of pleural fluid must accumulate before an effusion can be detected.	More accurate than CXR. Show hypoechoic space with transudate, and the presence of moving floating densities suggest an exudate.	More reliable than CXR for detecting effusions (indicated where malignancy is suspected)	• Thoracentesis is useful if etiology is unknown. • Therapeutic: drainage provides relief for large effusions.

Symptoms

- Often asymptomatic.
- Dyspnea on exertion.
- Peripheral edema.
- Orthopnea and PND.

Signs

- Dullness to percussion (stony dull).
- Decreased breath sounds over the effusion.
- Decreased tactile fremitus.
- Signs of pleurisy (a pleural rub)

L14: Investigations of lung diseases

- Before doing any procedure, we should assess (ABC): Airway, Breathing, and Circulation.in addition to consent from the patient.
- Before doing a thoracentesis, you have to do: coagulation test and CBC.
- If the thoracentesis findings include Blood or Pus it is an emergency you have to act quickly.
- If the Gram stain test is positive or the pH below 7.20 (acidic), this indicates drainage by chest tube or it will leads to fibrosis.
- Bronchoscopy is a diagnostic and therapeutic procedure.
- Pulmonary function tests include :
- **1- spirometry:** assess the airways, diagnose obstructive diseases if FEV1/FVC <75 %.
- Normal or Suggest Restrictive disease if FEV1/FVC>75%.
- **2-Lung volume:** assess air trapping and diagnose Restrictive disease.
- **3-Respiratory muscle strength**.
- **4- Diffusing capacity:** Emphysema, Interstitial Lung diseases, and pulmonary vascular diseases like (Pulmonary embolism).
- High-resolution CT is used when interstitial Lung disease is suspected.
- CT-Pulmonary Angiogram: is with pediatric patients or with very ill patient.
- V/Q is a nuclear scanning test, good for pregnant patient, CHF and if the patient allergic to the IV contrasts.

L15: Hypertension (HTN)

- Hypertension (HTN) is the 4th most common cause of death and it is responsible for more than 20% of deaths worldwide.
- HTN can be primary (90-95% of cases) or secondary (due to renal, endocrine, sleep apnea or oral contraceptives)
- The main risk factors for primary HTN are: Obesity, excessive salt intake + low potassium intake, excessive alcohol intake, NSAIDs, sedentary lifestyle, polycythemia, and family history of HTN.
- According to the European Society of Nephrology, HTN is classifies into 3 grades: Mild (grade I), Moderate (grade II), severe (grade III)
- Diagnosis of mild HTN should not be made until the BP has been measured at least in 3-6 visits.
- HTN is a major risk factor for many cardiovascular and renal diseases.
- Complications of HTN include: Stroke (common), CHF, Intracerebral hemorrhage, LVH, peripheral vascular diseases.
- Myocardial fibers of the left ventricle undergo hypertrophy and become markedly thickened in patient with severe hypertension.
- Hypertensive emergency is DBP >120 + acute end organ damage (MI, stroke, AKI)
- Malignant (accelerated) HTN is characterized by marked elevation in BP (systole >180 OR diastole >120) causing encephalopathy, retinal hemorrhage, or papilledema.
- Hypertensive retinopathy progresses in severity as HTN grading increases: Minimal constriction of retinal arterioles (stage I), Arteriovenous nicking (grade II), Flame-shaped hemorrhage (grade III), Papilledema and hemorrhage (grade IV malignant HTN)
- HTN patients may present asymptomatic or with epistaxis, chest discomfort or headache. (some may present with complication symptoms)
- ECG, urinalysis, lipid profile, serum electrolytes, blood glucose and hematocrit are some of the routine lab tests to diagnose HTN.
- Treatment of HTN has shown the most benefit in preventing CHF and renal failure by about 50%.
- Lifestyle modification is required for patients with SBP= 130-139 or DBP= 85-89. IF more than that, Drug therapy must be initiated.
- Some of the lifestyle changes are: Salt restriction, weight reduction, increase consumption of vegetables, smoking cessation, and regular exercise.

- ACEI, ARBs, Thiazide-like diuretics and CCB are the best therapy for HTN.
- Some of the recommended combination therapy include: ARBs (or ACEI) + thiazide-like diuretics, or CCBs + ACEI.
- Combination of beta blockers + ARBs, or ARBs + ACEI is NOT RECOMMENDED.

L16: Acid-Base disorder

- **Normal arterial blood pH** = 7.35 – 7.45
- **$PaCO_2$** = 35-45
- **Serum HCO_3=** 22-26
- **Anion gap** = 8-12 *(in Medscape 8-16)*

Assess the acid-base disorder step by step:

- Is the primary disorder an acidosis (pH < 7.35) or alkalosis (pH > 7.45)?
- Is the disorder respiratory or metabolic?
- Is the disorder is acute or chronic "compensated"?

	pCO_2	HCO_3	condition
Normal pH	-	-	normal
	↑	↑*	Chronic respiratory acidosis
	↓	↓*	Chronic respiratory alkalosis
High pH	-	↑	Metabolic alkalosis combined with respiratory alkalosis *(no respiratory compensation)*
	↓	-	Acute respiratory alkalosis *(no kidney compensation)*
	↑*	↑	Metabolic alkalosis *(compensated-absence of combined respiratory alkalosis)*
	↓	↓*	Acute on top of chronic respiratory alkalosis *(high pH → acute, compensation → chronic)*
Low pH	-	↓	Metabolic acidosis combined with respiratory acidosis *(no respiratory compensation)*
	↑	-	Acute respiratory acidosis *(no kidney compensation)*
	↓*	↓	Metabolic acidosis *(compensated-absence of combined respiratory acidosis)*
	↑	↑*	Acute on top of chronic respiratory acidosis *(low pH → acute, compensation → chronic)*

❖ **Metabolic Acidosis:**

1. High Anion gap acidosis—due to addition of H+ acid.
2. Non-anion gap acidosis—due to HCO3 loss.
   - Calculating the anion gap: AG = Na – [Cl + HCO3] "**Normal AG = 8-12**"

Normal Anion Gap (USED CARP)	High Anion Gap Differential (MUDPILES)
• Ureterostomy	• Methanol
• Small bowel fistula	• Uremia
• Extra Chloride	• DKA
• Diarrhea	• Paraldehyde
• Carbonic anhydrase inhibitors	• Inborn Errors
• Addison's disease	• Lactic Acidosis
• Renal tubular acidosis	• Ethylene Glycol
• Pancreatic fistulas	• Salicylates
Treatment: Replace Bicarbonate	

❖ **Metabolic Alkalosis:**

1. **Saline responsive "Urine Cl < 20":** Diuretics, NGT, Vomiting, villous adenoma.
2. **Non-Saline responsive "Urine Cl > 20":** 1^o & 2^o hyperaldosteronism.

❖ **Respiratory Acidosis:**

1. **Hypoventilation**: brain stem injury by stroke or tumor, Guillain-Barre syndrome, Myasthenia gravis, Scoliosis, kyphosis, chest wall deformity, sedative drugs.
2. Retention of CO2 (COPD, pneumonia, foreign body, pulmonary edema).

❖ **Respiratory Alkalosis: (wash out CO2)**

Hyperventilation: anemia, anxiety, fever, hypoxemia, sepsis, pain, pregnancy, CHF, pneumonia, asthma, liver disease, pulmonary embolism.

❖ **Partial Compensation mechanism: (In full compensation pH will become normal)**

- **Acute respiratory acidosis:** 10-mmHg increase in PaCO2 → HCO3 increase by 1 mEq/l
- **Chronic respiratory acidosis:** 10-mmHg increase in PaCO2 → HCO3- increase by 3-3.5 mEq/l
- **Acute respiratory alkalosis:** 10-mmHg decrease in PaCO2 → HCO3- decrease by 2 mEq/l
- **Chronic respiratory alkalosis:** 10-mmHg decrease in PaCO2 → HCO3- decrease by 4-5 mEq/l
- **Metabolic acidosis**: 1 mEq/l decrease in HCO3 → PCO2 decreases by 1 mmHg
- **Metabolic alkalosis:** 1 mEq/l increases in HCO3 → PCO2 increases by 0.6 mmHg

❖ **Some important Cases:**

1. **In Asthmatic patient**: early stages => respiratory alkalosis in late stages => respiratory acidosis "to treat patient give him β_2 agonist"
2. **Abdominal pain, hypotension and very low pH:** Acute mesenteric ischemia lead to lactic acidosis "treat the patient give him IV fluids to treat hypotension".

L17: Electrolytes Imbalance I (Na^+ and Water)

- Osmolality: Number of particles per kilogram of solution.
- Tonicity: Whether the particles are effective or ineffective osmoles. (Point of comparison between the osmolality of fluids).
- EABV: Effective **Arterial** Blood Volume; the **amount** of arterial blood required to adequately fill the capacity of arterial circulation. It determined by C.O. and SVR.
- Low EABV does not in necessity indicate intravascular depletion, because there is a possibility that the blood is in the venous system due to heart failure.
- Before assessing the blood volume state, you asses the Na^+ conc. in urine.
- In normal physiological state, changes in Na conc. must be followed by changing in water volume.
- The water volume in the body is determined mainly by Na conc. so in hypovolemic/hypervolemic states it is an abnormality in Na conc.
- The Na conc. in the plasma is determined by water volume, so hypernatremic/hyponatremic states, it is most likely due to water intake or output abnormalities.
- Both of hypo- and hypernatremia may present with any of the following; confusion, disorientation, lethargy, seizures and in addition to coma in case of hyponatremia.

Disorders of Sodium homeostasis		
	Hypernatremia	Hyponatremia
Clinical presentation	- Occurs when there is a loss of free water. Could be due to **diabetes insipidus** (ineffective ADH), sweating, diarrhea, or even pneumonia (insensible losses from hyperventilation).	- Hyponatremia is characterized according to overall volume status of the body. **- Hypervolemia:** the most common cause of hyponatremia, with a hypervolemic state that are: A) CHF, B) Nephrotic syndrome, C) Cirrhosis. - This will raise ADH levels. **- Hypovolemia:** Sweating, burns, diuretics, diarrhea, pneumonia, etc. - They cause hyponatremia if there's a chronic replacement with free water. A little sodium and a lot of water are lost in urine. - Addison's disease or loss of adrenal function causes loss of aldosterone, leading to loss of sodium. **- Euvolemia:** The most common causes of hyponatremia in euvolemic state are; pseudohyponatremia (hyperglycemia, hyperalbuminemia), psychogenic polydipsia, hypothyroidism or SIADH.
Diagnostic Test	- Increase urine osmolality. (Decreased in diabetes insipidus). - Decrease urine volume. (increased in diabetes insipidus) - Decrease urine sodium.	- In case of SIADH, the urine has a high osmolality, and the accurate test in this state of disease is measuring ADH level.
Treatment	- Correct the underlying cause of fluid loss. - Calculate water deficit and administer it.	**- Mild hyponatremia ->** Restrict of fluid. **- Moderate hyponatremia** presented with minimal confusion **-> ** Saline and loop diuretics. **- Sever hyponatremia** presented with lethargy, seizures, coma **->** hypertonic saline, conivaptan or tolvaptan.

L18: Acute kidney injury (AKI)

❖ **Acute Kidney Injury** is a sudden decrease in kidney function over a period of hours to days, leading to sudden rise in urea and creatinine

Acute Kidney Injury causes		
	Prerenal	• **Hypovolemia:** ✓ Diuretics ✓ Burns ✓ Pancreatitis ✓ Decreased cardiac pump (CHF, PE and MI) ✓ Low albumin ✓ Cirrhosis
	Renal	• **Acute Tubular Necrosis:** ✓ Toxins (such as NSAIDs, Aminoglycosides and Cisplatin) ✓ Prolonged ischemia (Hypotension, Sepsis, Prolonged pre-renal state) • **Acute Interstitial nephritis:** ✓ Medications (such as Penicilin and Rifampicin) ✓ Infections (Direct infiltration OR Reactive to systemic infections such as Poststreptococcal infection) ✓ Systemic diseases (such as SLE) ✓ Idiopathic • **Glomerunephritis**
	Postrenal	• **Ureteric obstruction** • **Bladder neck obstruction** • **Urethral obstruction**

❖ **Important notes:**

- **Acute Tubular Necrosis** → **Muddy brown cast**
- **Acute Interstitial nephritis** → WBC cast
- **Glomerulonephritis** → **RBC cast**
- **Unilateral obstruction never ever causes AKI, UNLESS the person has one kidney**

Other causes of AKI		
Contrast nephropathy	**Rhabdomyolysis**	**Atheroembolic**
• Risk factors: ✓ CKD ✓ Old age ✓ DM • Creatinine starts to go up after 24-48 hrs (this is the distinguish word between contrast and atheroembolic) • Usually it has good prognosis	• Increase in serum CK (above 10,000) • Blood positive urine dipstick, with no RBC on microscopy • Pigmented granular casts • Common after trauma, burns, seizures, limb ischemia • Treatment is supportive care with IV fluids	• Commonly occur after intravascular procedures or cannulation • Creatinine starts to go up after one week • Usually it has bad prognosis

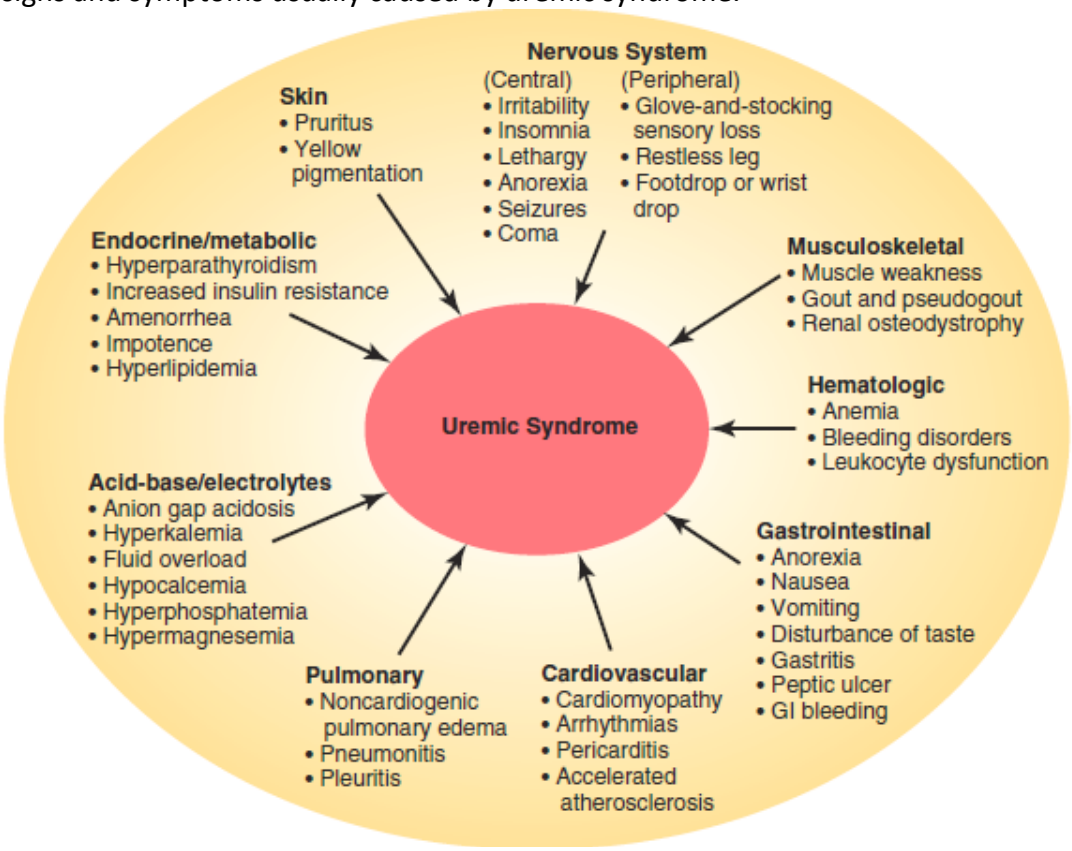
L19: Glomerular diseases

Nephrotic syndrome (NS)			
Clinical presentation	Anasarca (generalized edema), Hypoalbuminemia (serum Albumin <30 g/L), Heavy proteinuria > 3.5 g/24 hours of urine collection, Hyperlipidemia, lipiduria, N/V, fatigue.		
Complications	infection & sepsis (due to lose of immunoglobulin) + AKI		
Causes	Focal Segmental GlomeruloSclerosis (FSGS)	Minimal Change Disease (MCD)	Membranous Nephropathy (MN)
Notes about the cause	**- Primary FSGS:** sudden onset of heavy proteinuria and other symptoms of nephrotic syndrome. **- Secondary FSGS:** Proteinuria is less heavy + Renal impairment is commonly seen. **- Causes of 2^{ry} FSGS**: Massive obesity, Healing of prior GN, HIV, Anabolic steroid.	- No sclerosis. - **LM:** appears normal glomeruli, **EM:** shows foot processes effacement. - Main cause of Nephrotic syndrome in children. - **Primary:** (Idiopathic) or **secondary:** e.g. drugs like NSAIDs. - Sudden onset Edema.	- Most common cause of nephrotic syndrome in adults - Slowly developing. - Can be **Primary:**(Idiopathic), or **Secondary:** e.g. (SLE, malignancy)
Notes in NS	- Primary glomerular diseases: mostly immune system dysfunction. - Foot processes effacement of podocyte is the primary finding. - Normal liver synthesis 10g/day of albumin, NS patients lose 30g/day - Diagnosed by biopsy and the treatment for 1^{ry} is corticosteroid, 2^{ry} is treat underlying cause - Most common causes of secondary NS: D.M + Amyloidosis.		

Nephritic syndrome					
Clinical presentation	- Hematuria, proteinuria, HTN, edema, azotemia (elevation of BUN), oliguria. - **Urine analysis shows:** RBCs ,RBSs Cast ,dysmorphic RBCs , and Protein, these called Active Urinary Sediments (it means there is an active glomerular inflammation) - Creatinine may elevated = indicate AKI				
Causes	IgA Nephropathy	Post streptococcal glomerulonephritis (PSGN)	Anti-GBM antibody disease	ANCA vasculitis	Membrano-proliferative GN (MPGN)
Notes about the cause	- Most common type of Primary GN in developed countries. - Hematuria 1-3 days after upper respiratory tract infection (URTI). - You should control their blood pressure.	- Throat infection with Gram positive Group A beta-hemolytic Streptococcus (GAS). - Low serum C3 - Frank hematuria usually after one week of infection. - Positive (ASO). - Treatment is supportive.	- Linear Anti-GBM staining by IF - Goodpasture's disease is GN with pulmonary hemorrhage.	- ANCA=Anti-neutrophil cytoplasmic antibody - Have two types: 1- C-ANCA= Cytoplasmic type, more commonly causing Granulomatous with Polyangiitis (also called Wegner's). 2- P-ANCA= Perinuclear type, (e.g. Microscopic Polyangiitis & Churg-Strauss syndrome)	- Primary is idiopathic in children. - Secondary mainly in adult with Hepatitis B and C.
NOTES	- Lupus can cause nephritic syndrome - If crescentic glomerulonephritis developed, patient will have very bad prognosis (severe and rapid loss of kidney function).				

L20: Chronic Kidney Disease

- Persistent, progressive and irreversible loss of renal function.
- **Characterized** by GFR < 60mL/min/1.73m^2 for at least **3 months** to differentiate between it and Acute Kidney Injury.
- The **pathophysiology** involved two sets of mechanisms:
  A) The underlying etiology, (e.g. DM, congenital abnormalities etc.)
  B) A set of progressive mechanisms.
    1) Nephron Injury.
    2) Surviving nephrons try to adjust by increasing their filtration and excretion. (through increase blood flow and the size of the unaffected glomeruli)
    3) More reduction in renal mass, and the vicious circle starts again.
- **Clinical presentation;** patients with CKD may not have symptoms until advanced in which GFR < 15 mL/min and the signs and symptoms usually caused by uremic syndrome.



- **Diagnosis** is made through the detection of albumin urea or by reduction in the clearance of toxins by the kidney.
- Albumin-to-creatinine ratio (ACR) greater than 30 mg/g confirmed on several samples and without evidence of urinary infection raises concern for a diagnosis of CKD.
- **Treat** the underlying cause, slow the progression of CKD and manage its symptoms.
- Transplantation is the only cure.
- **Dialysis** is indicated in the following settings:
  A) Sever acidosis.
  B) Persistent hyperkalemia.
  C) Intoxication.
  D) Unmanageable hypervolemia.
  E) Uremia based on clinical presentations.
  F) Pericarditis.

L21: Electrolytic Imbalance II (Potassium & Calcium):

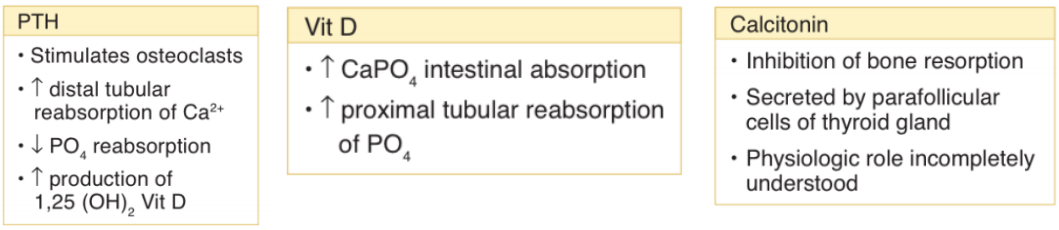
Potassium:

- Source of K+ is diet and excreted by renal clearance **(mainly)** and intestinal excretion
- It is the **most** abundant intracellular cation which maintained mainly by K+\Na+ pump
- Key role of K+ in body is **Maintains electrical gradient across cell membranes**

	Hyperkalemia	Hypokalemia
Cause	• NA/K ATPase dysfunction (acidosis & insulin deficiency) • Massive Cell breakdown (tumor lysis) • Decrease renal loss (Renal failure) • Aldosterone axis dysfunction (ACE inhibitors, Addison disease and spirolactone)	• Increase intestinal loss (vomiting & diarrhea) • Rapid transcellular shift (insulin, alkalosis & beta agonist) • Increase renal loss (high aldosterone, loop diuretics , renal artery stenosis & low Mg)
Symptom	Muscle weakness & arrhythmia **In ECG:** peaked T wave , wide QRS and short QT	Muscle weakness, arrhythmia and nephrogenic diabetes insipidus **In ECG:** Flat T wave + extra U wave
Management	- If ECG abnormal Calcium chloride to stabilize heart - **Push K into cells:** Insulin **with glucose** , Beta agonists - **Through kidney:** diuretics (loop diuretics) or dialysis - **Through gut :** K chelation (Ca resonium) with Laxatives	- Identify and treat the underlying cause - if Severe loss IV without dextrose containing fluid - if mild loss orally "safer" - Mg level should be corrected if it's not

Calcium:

- Increasing and decreasing in calcium parameters represent **total or free calcium**
- 98% of Ca stored in bones and 2% in blood – 51% of blood Ca are free and 40% is protein bound and 9% is ionized Ca with phosphate or oxalate



	Hypercalcemia	Hypocalcemia
Cause	- Hyperparathyroidism "primary or Secondary to renal" - Lung cancer "squamous cell carcinoma of lung" - Granulomatous disease "tuberculosis & sarcoidosis" - Vitamin D intoxication - Drugs "Thiazide diuretics and lithium"	- Hypoparathyroidism - Renal failure - Hungry bone syndrome - Small bowl resection - Drugs "loop diuretics and phenytoin"
symptoms	Confusion, vomiting, constipation, renal stones & nephrogenic diabetes insipidus **In ECG:** short QT interval	Cataract, Chvostek's sign, Trousseau's sign and seizures **In ECG:** Long QT interval
Manag-ment	1. In very severe cases, IV normal saline followed by furosemide "loop diuretics" 2. IV bisphosphonate "zoledronate or pamidronate" 3. You can add calcitonin in some cases 4. If patient has renal failure you can us dialysis	1. IV or oral calcium replacement and Vit D if needed 2. you have to correct Mg levels if it is low

L22: Gastrointestinal (GI) bleeding

- As the GI system is divided into upper and lower parts, GI bleeding above ligament of treitz (ligament separating duodenum and jejunum) is considered upper GI bleed, while below ligament of treitz is considered to be lower GI bleed.

❖ **UPPER GI BLEEDING:**
- ✓ **Causes:** Peptic ulcer (most common), Variceal bleeding, Malignancy, erosive mucosal lesions, Hemophilia, Aortoenteric fistula (following aortic surgery), Mallory-Weiss syndrome.
- ✓ **Clinical features**: IF high volume of blood loss => Hematemesis
  IF 100 ml of blood loss => Melena, IF 5-10 ml of blood loss => Occult-blood positive brown feces OR coffee ground emesis.

❖ **LOWER GI BLEEDING**
- ✓ **Causes:** Diverticulosis (most common), IBD, Colorectal CA, Colorectal polyps, Ischemic colitis, Hemorrhoids, anal fissures and small intestine bleeding.
- ✓ **Clinical features:** Hematochezia, Occult blood in stool, fresh rectal bleeding and anal pain during defecation.

- **Signs of GI bleeding:** Signs of volume depletion, Signs and symptoms of anemia. (FIRTST SIGN IS POSTURAL HYPOTENSION)

❖ **Diagnosis of GI bleeding:**
- ✓ **HISTORY:** Nature of bleeding, associated symptoms, drugs intake list …
- ✓ **EXAMINATION:** Vital signs, Consciousness level, ABC.
- ✓ **LABS:** CBC, Coagulation profile, Liver profile, Crossmatch, Renal profile.
- ✓ **ASESS DEGREE OF HYPOVOLEMIC SHOCK.**
- ✓ **Diagnostic modalities:** **ENDOSCOPY: the most accurate test for diagnosis and has therapeutic uses, Nuclear bleeding scan (detect low volume bleeding), Angio (Rarely used), Capsule endoscopy (newest modality to visualize small intestine).

❖ **MANAGEMENT:**
- ✓ If patient is hemodynamically unstable=> **IV fluid resuscitation,** 2 Large bore IV lines.
- ✓ **Blood transfusion:** Indicated if Hb <7 without comorbidities, Hb <10 with comorbidities OR unresponsive hypovolemic shock.
  - Target level is 70-90 g\L.
  - Correction of coagulopathy is recommended for patients receiving anticoagulation therapy (delay endoscopy if high INR).
- ✓ **Pre-endoscopic PPI:** NO effect on rebleeding NOR mortality, has a supportive and safe profile. (may be used)
- ✓ **Endoscopic management**: Early endoscopy (2 to 24 hours AFTER INITIAL PRESENTATION) is needed in patients with ACTIVE BLEEDING (predicted by Hb<80 g\L, fresh blood in NGT … etc.)
  - It takes 72 hours for most high-risk lesions to become low-risk lesions AFTER endoscopic therapy
- ✓ **Pharmacological therapy: PPI**
  - When compared to H_2 antagonists OR placebo WITH or WITHOUT endoscopic therapy => PPIs reduces REBLEEDING ONLY.
  - When compared to placebo or H_2 antagonists WITH endoscopic therapy => PPIs reduces REBLEEDING and MORTALITY.
- ✓ **WHO TO ADMIT FOR MONITORING (For at least the first 24 hours)?**
  - Hemodynamic instability, Increasing age, Severe comorbidity, Active bleeding at endoscopy, Large ulcer size (>2 cm).

L23: Inflammatory bowel disease (IBD)

- **IBD** is a chronic inflammation of GI tract (Either CD, UC, or indeterminate colitis "between them").
- NOD2/CARD15 "It was thought this genetic mutation was only found in Ashkenazi Jews, however this has been proven wrong. Even patients in KSA have the same genetic mutation".

	Ulcerative colitis (**UC**)	Crohn's disease (**CD**)
Definition	Disease of colon only, start from rectum (50% of patients limit to rectum) and extend proximally in a continues*[1] fashion.	May involve any part of GI, particularly **ileocecal region**, in a skip fashion.
Prevalence	It increasing in Saudi Arabia (UC more common than CD)	
Sex	Male = female	
Appendectomy	Reduce symptoms	Worse symptoms
Smoking	Reduce symptoms	Worse symptoms
Symptoms	**Urgency to defecate, bloody mucosy diarrhea**, rectal bleeding, crampy abdominal pain, and tenesmus.	**Diarrhea**, Vomiting, anemia, fatigue, Crampy abdominal pain, weight loss, fever, and perianal disease.
Site	• Proctitis: rectum. • Proctosigmoiditis: rectum and sigmoid colon. • Left-sided colitis: from rectum to splenic flexure. • Extensive colitis: beyond the splenic flexure. • Pancolitis: whole colon	Any part of GIT
Extra intestinal manifestations	Uveitis /Episcleritis, Erythema Nodosum, Pyoderma Gangrenosum, Arthritis, Ankylosing Spondylitis, **Sclerosing cholangitis "more common with UC and associated with colon cancer"**	
Gross feature	Loss of blood vessels, **colonic pseudopolyps**, more ulceration, diffuse inflammation	Thickening of bowel wall with creeping fat, cobblestones appearance.
Microscopic feature	Inflammation only in mucosa, crypt abscesses with branching, loss of mucin in goblet cells atrophies of glands.	**Transmural inflammation** (all layer), focal ulceration, Noncaseating granuloma (If caseating thinks of TB).
Investigations	Combined investigation especially **Colonoscopy & Imaging studies** **Antibody tests : pANCA) "more common in UC" & ASCA "more common in CD"**	
Complication	Toxic megacolon (very dilated colon accompanied by abdominal distension) ,Perforation (they need surgery), Hemorrhage, Stricture.	**Fistulas, abscess**, perforation, perianal disease, malabsorption, gallstone*[2], renal stone, amyloidosis and **stricture**
Risk of colorectal cancer	May occur in long duration (usually > 10 years of begin of the disease)	Very low
Treatment	1. **Rule out infection (most important step)**. 2. **5 ASA therapy " 5-aminosalicylic acid":** Sulfasalazine, Mesalamine • **Oral with Rectal formulations** are used in Left-sided colitis and Pancolitis • **Rectal formulations** are used in ulcerative proctitis and proctosigmoiditis 3. **Corticosteroids:** Prednisolone, cortisone • **Local acting:** Hydrocortisone enema or suppository 4. **Immunomodulators:** Azathioprine, Methotrexate 5. **Anti TNF therapy:** Infliximab, Adalimumab	1. **Rule out infection (most important step).** 2. **No 5 ASA therapy** 3. **Corticosteroids:** Prednisolone, cortisone • **Local acting:** Budesonide 4. **Immunomodulators:** Azathioprine, Methotrexate 5. **Anti TNF therapy:** Infliximab, Adalimumab

1. The only type present without continues inflammation is **cecal patch** (which is here the rectum affected then there is a normal mucosa then there is affected base of the cecum near the appendix).
2. Due to the malabsorption of bile in the ileum.

L24: Acute Viral Hepatitis

- Acute Hepatitis is the inflammation of the liver lasting less than 6 months.
- **The most common causes** are HAV and HEV and they **rarely** progress into chronic liver disease.
- HAV and HEV transmitted through fecal-oral route and contaminated water.
- **HBV** in infected individual is present in all body fluids thus it's **highly infectious**.
- HCV is transmitted through blood, sexual route is uncommon.
- **HDV** it has a small defective RNA, and **only propagates** in an individual who has a **coexistent HBV**. Transmitted in the same manner of HBV.
- Acute Viral Hepatitis begins with a prodromal phase lasting several days characterized by **GIT symptoms**.
- Jaundice soon appears (**Icteric hepatitis**), often accompanied by an improvement in the patient's well-being.
- Many patient could be asymptomatic or symptomatic without jaundice (**Anicteric hepatitis**). In such instances, medical attention is not sought.
- In serology, **presence of IgM** antibodies against any of the previous viruses **indicates acute or recent infection**, while **presence of IgG indicates acute, previous, or chronic infection.**
- Acute HAV and HEV diagnosed by anti-HAV and anti-HEV IgM.
- Acute HBV diagnosed by anti-HBc and anti-HBe. Anti-HBs may be present in acute or chronic infection, or may indicate immunity.
- Acute HCV, HDV and HEV diagnosed by **PCR** assay for their RNAs.
- **Complications include;** Cholestatic Hepatitis (**usually during HAV infection**), acute liver failure, chronic hepatitis (**usually due HBV and HCV**).
- Chronic hepatitis diagnosed through persistence elevated levels of ALT and AST, viral antigens **OR** nucleic acids beyond 6 months.
- **Rare complications;** aplastic anemia, pancreatitis, myocarditis, pericarditis, pleural effusion, Guillain-Barre syndrome, aseptic meningitis and encephalitis, polyarteritis nodosa (HBV), and glomerulonephritis (HBV and HCV).
- HAV, HBV and HEV are usually self-limited.
- Prevention of HAV and HEV is through hygiene and **vaccination against HAV**.
- HBV is prevented through hepatitis B immunoglobulin (HBIM) and HBV vaccine.
- HCV is prevented through screening of the blood unit before transfusion.

L25: Liver Cirrhosis complications

	Pathophysiology	How to diagnose?	Management	Notes
Varices	Portal hypertension leads to reduction in the portal blood to the liver, then to the development of the collateral vessels.	Presents with: - Hematemesis. - Melena.	General management: - 2 IV lines, Type and cross match and Resuscitation. Specific management: - Octreotide (vasopressin). - Banding and sclerotherapy. - Surgical shunt and TIPSS.	We can prevent the non-bleeding varices by: - Treat the underlying cause. - Prophylactic banding. - Beta blockers. - Liver transplantation.
Ascites	PTH leads to nitric oxide production (NO). NO causes vasodilation and reduction in BP. This results in activation of the Renin-Angiotensin system, aldosterone and increased sympathetic activation. The BP will normalized, but there will be salt and water retention.	Physical examination: - Shifting dullness. Investigation: - Ultrasound. - SAAG: > 11 mg/dl means PTH or CHF. < 11 mg/dl means nephrotic syndrome, cancer, or infections.	General treatment (GT): - Treat the underlying cause + salt restriction + loop diuretics + Spironolactone. If GT doesn't work: recurrent tapping. If recurrent tapping doesn't work: TIPSS. - Liver transplantation.	Spontaneous Bacterial Peritonitis: - Infection of ascitic fluid. - Usually by E.coli. - Dx by ascetic tap (will show more than 250 PMN. - Treatment: 3rd generation cephalosporin.
Hepatic encephalo-pathy	Liver is damaged, so there is no detoxification of blood, leading to ammonia (neurotoxin) existing in the brain.	Presents with: - Reversal of sleep pattern. - Personality changes. - Fluctuating. - flapping tremor (due to the neurotoxins prevent the nerve conduction).	- Identify and treat precipitation factor. - Treat underlying liver disease. - Normal protein diet. - Antibiotics (Neomycin, metronidazole). - Lactulose. - Transplantation.	Precipitating factors: - Sedatives. - Infection. - Hypokalemia. - Protein dietary. - Constipation (therefore we use lactulose). - GI hemorrhage.
HCC	HCC is usually caused by liver cirrhosis. However, HCC is rarely caused primarily.	Investigations: - Screened by US. - Diagnosed by CT.	----	Liver cirrhosis patients must be screened by US every 6 months to detect any small HCC. Because HCC has poor prognosis, so we have to prevent it from its early stages.

L26: Esophageal disease:

I. **Dysphagia** is a difficulty in swallowing, sensation of obstruction of food passage.

- Dysphagia can be:

1- Oropharyngeal (Transfer Dysphagia): arising from: Upper esophagus, Pharynx, Upper esophageal sphincter.

- Characterized by: problems at initiating a swallow, and if they did, they developed coughing, chocking, and Nasal regurgitation.
- Causes are diseases of striated muscles: CVA, Polymyositis, and Myasthenia gravis.

2- Esophageal dysphagia: arising from: Esophageal body, Lower esophageal sphincter, and cardia.

- Classified as:

A. Mechanical dysphagia: problems of swallowing Solids.

- Due to: large food bolus, Intrinsic narrowing: e.g. esophageal ring (usually intermittent) + (young patient), or Extrinsic compression: thyroid mass.

B. Motor dysphagia: problem of swallowing Solids and liquids, due to Smooth muscles disorder: Scleroderma, Achalasia or spasm.

II. **GERD** is a damage of the esophageal mucosa.

- Heart burn + Regurgitation are the most common manifestation.
- Diagnosed by Endoscopy
- Complication: bleeding, and Barrett's esophagus

III. **Achalasia** is a motor disorder: High LES pressure + absent peristalsis.

- Symptoms: Dysphagia (for both solids and liquids) and Regurgitation
- Diagnosed by Barium swallow (it shows Bird's beak like) and Manometry.

IV. **Infectious Esophagitis**: can be viral, bacterial, or fungal.

- Symptoms: Odynophagia (Painful swallowing), and dysphagia.
- Diagnosed by Endoscopy and biopsy.

V. **Diverticula** are Outpouchings of the wall of the esophagus.

- If the Diverticula occur in upper part = Zenker's diverticulum, or in lower = epiphrenic diverticulum.
- Symptoms: asymptomatic or have regurgitation, dysphagia, cough, and halitosis (smelly breath).

VI. **Esophageal cancer**: more in male over 50 years.

- **Risk factor**: Excess alcohol, and smoking (They risk for squamous cell carcinoma). Barrett's esophagus (risk for adenocarcinoma).
- Most cases are Squamous cell carcinoma.
- Symptoms: Weight loss, progressive dysphagia, and odynophagia.
- Diagnosed by: Barium swallow, endoscopy, and biopsy.

L27: Chronic diarrhea and malabsorption

I. **Chronic diarrhea:** is decrease in fecal consistency (weight of stool) more than 4 weeks.

- **Chronic diarrhea** can be due to IBD, IBS, malabsorption syndromes (e.g. celiac disease), or chronic infection.
- **Crohn's:** effect young people, cause non-bloody diarrhea, LIF pain, weight loss, high ESR and CRP.
- **IBS:** effect young people, can cause diarrhea, intermittent diffuse abdominal pain which improve with defecation, no weight loss or loss of appetite, and normal ESR and CRP.
- **Celiac:** effect young people, cause abdominal floating, diarrhea, itchy skin rash, +ve anti TTG antibody.
- **Fatty diarrhea** can be caused by chronic pancreatitis or celiac disease.

Classification of diarrhea			
Type	**Osmotic diarrhea**	**Secretory diarrhea**	**Motility diarrhea**
Description	Non absorbable solute which pull water to intestinal wall	Damage of intestinal wall, so the absorption will decrease	No enough contact time between food and intestinal membrane due abnormal muscle contraction.
Example	Lactase deficiency	Bacterial infection	IBS, Gastric/ intestinal resection

II. **Malabsorption:** can be **global** (diffuse mucosal disease e.g. celiac) or **partial** (interfere with specific nutrient e.g. B_{12} deficiency due to intrinsic factor deficiency).

- **Symptoms:** Weight loss, steatorrhea, edema, muscle atrophy, anemia, tetany (In general it varies and depends on nutrient deficient).

Malabsorption causes				
I. Luminal phase defect			**II. Mucosal phase defect**	**III. Transport phase defect**
1- Reduced nutrient availability	**2- Impaired fat solubilization**	**3- Defect nutrient hydrolysis**		
- **Cofactor deficiency** (e.g. pernicious anemia, gastric surgery) - **Nutrient consumption** (e.g. bacterial overgrowth)	- **Reduced bile salt synthesis** (e.g. hepatocellular disease) - **Impaired bile salt secretion** (e.g. chronic cholestasis) - **Bile salt inactivation** (e.g. bacterial overgrowth) - **Impaired CCK release** (e.g. mucosal disease) - **Increase bile salt losses** (e.g. terminal ileal disease or resection)	- **Lipase inactivation** (e.g. ZE syndrome) - **Enzyme deficiency** (e.g. pancreatic insufficiency or cancer) - **Impaired mixing or rapid transit** (e.g. resection, bypass, hyperthyroidism)	- **Extensive mucosal loss** (e.g. resection or infarction). - **Diffuse mucosal disease** (e.g. Gluten, Crohn's, infection). - **Enterocyte defect**.	- **Vascular** (e.g. vasculitis, atheroma) - **Lymphatic** (e.g. lymphangiectasia, radiation, nodal tumor)

- **Investigation:** CBC, CRP, serology test (e.g. Anti TTG ab), endoscopy, imaging studies (to know the cause).
- **Treatment:** You should treat the underlying cause (e.g. celiac treated by gluten free diet etc.) and nutritional supplement.

L28: Abdominal pain include IBS

- 12 weeks separate acute from chronic abdominal pain

Acute abdominal pain:

- Two syndromes consider as surgical abdomen: 1- obstruction (frequent vomiting with abdominal pain), 2- peritonitis. They may associated with unstable vital signs, fever, and dehydration
- Acute right upper quadrant pain → usually biliary stone
- Severe epigastric pain radiate to back, and relief by lean forward → Acute pancreatitis. Gallstone is the most common cause of acute pancreatitis in Saudi Arabia. Give them a lot of fluid (7 L/day), to prevent necrosis, and keep them NPO. No antibiotic, it increases morbidity and mortality (unless there is a clear infection).

Kinds of pain	
Visceral pain	Parietal pain
Involves hollow or solid organs, usually midline pain, and poorly localized.	Involves parietal peritoneum, localized pain, cause tenderness, guarding which progress to rigidity, and rebound

- Guarding: voluntary abdominal muscle contraction due to painful palpation (you still can press), while rigidity is involuntary (very rigid you can't press).
- Referred pain: Based on developmental embryology (e.g. lower MI cause epigastric pain, urethral obstruction cause testicular pain).
- You should ask which came first, pain or vomiting, if pain came first usually it's surgical disease (e.g. obstruction), if vomiting came first, they need medical treatment (e.g. gastroenteritis).
- In acute abdomen pain we order X-ray to answer two questions: obstruction (dilation bowel) and perforation (air under diaphragm), if perforation not treated may cause peritonitis.

Chronic abdominal pain:

- Usually they have functional disorder rather than organic (e.g. IBS)
- In general IBS is the most common cause of abdominal pain
- IBS affect 20% of adult (70% of them are women). Usually in young adult. 50% of IBS patient who seek medical care have psychiatric disorder (e.g. depression, anxiety)
- Pathophysiology of IBS: Visceral hypersensitivity + psychosocial problem.
- Dx of IBS: by Rome III criteria + exclude alarm feature that suggest organic disorder (e.g. Unstable vital signs, weight loss, fever, dehydration, electrolyte abnormalities, GI blood loss, anemia, malnutrition)

Rome III criteria for IBS	Recurrent abdominal pain or discomfort at least 3 days/month in last 3 months, associated with at least 2 of the following:
	1- Improvement with defecation
	2- Change in frequency of stool
	3- Change in stool form (Appearance)

- No cure from IBS, only manage the symptoms.

Good luck in your exam ☺