Medicine Team
439

Summary

Step by step approaches

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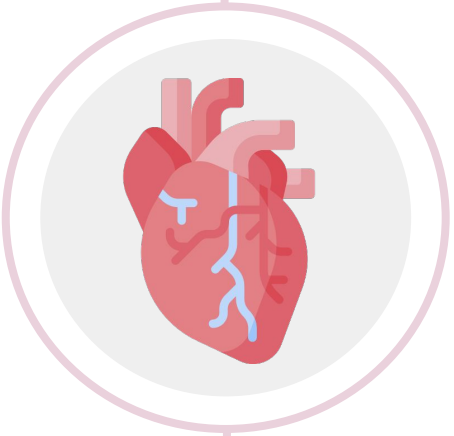
This file includes too many resources to reach the most optimal step by nor step approach (to organize your information) and not comprehensive alternative for the team lectures

Cardiology



Resources:

- Davidson’s principle and practice of medicine latest edition, 2018
- Kumar and clark’s Clinical medicine latest edition, 2021
- Current medical diagnosis and treatment, textbook, 2022 edition
- Doctors’ lectures and notes
- Master the boards, 2021 edition
- Kaplan USMLE Step 2 CK Internal medicine
- AMBOSS
- NICE guidelines on heart failure

Arrhythmia

Done by Naif Alsulais

AF symptoms?

Unstable?

Considered unstable if has:

- chest pain
- shortness of breath
- confusion
- systolic BP < 90 (Hypotension)

synchronized electrical Cardioversion (Shock) immediately

Contraindications:

- digoxin toxicity due to risk of ventricular arrhythmia
- Unstable AF more than 24 due to embolism

Stable?

ECG

Further investigations:

- **TSH:** if thyrotoxicosis suspected (unexplained weight loss)
- Other laboratory as needed: **Renal function**, **hepatic function**, **serum electrolytes** & **complete blood count**
- **TTE:** to identify size and functioning of atria and
- **CXR:** pulmonary pathology, detect congestive heart failure & enlargement of the chambers
- **Holter monitor:** diagnosis and follow up to see if the treatment is effective
- **TEE:** Not routinely used but useful for accurate assessment of risk of stroke, only reliable method to rule out left atrial appendage thrombus

Absent P-waves & Irregularly irregular rhythm?

Atrial fibrillation

Rate control[1]

Target heart rate for AF patients <110 bpm during activity and 60-80 bpm during rest

- **Medication:**
  - **Beta-blocker:** Atenolol, Metoprolol (Used in cases of Graves disease, CHF, IHD, HTN or LV systolic dysfunction)
  - **Calcium channel blocker (CCB):** Verapamil , diltiazem (Used if patient is Asthmatic)
  - **digoxin:** Used if patient is hypotensive
- **procedure:** Pace and ablate > we only use this if we can't control the rate with medication

Spontaneous conversion to sinus rythm?

No

Presenting <48 hrs?

Rhythm control[1]

Cardioversion

- **electrically**
- **pharmacologically** most effective when initiated within 7 days of AF (It's mainly used if electrical cardioversion fails or not feasible):
  - Absent heart disease: Class IC antiarrhythmics e.g. **IV Flecainide**, **Propafenone** Or Class IA e.g. **Procainamide**
  - Present heart disease: Class III antiarrhythmics e.g. **IV Amiodarone** Or **Dronedarone** (alternative)
- **procedure:** AF ablation first line (We can just do it upfront without medical therapy)

Anticoagulants for 4 wks

- **Warfarin:** Moderate or severe mitral stenosis or the presence of a mechanical heart valve regardless the score of risk
- **NOAC:** first line treatment, risk score **≥ 2**
- **aspirin:** controversial in risk score of **0-1**. young patients (<65 years) with lone AF

Risk factor	Score
Congestive heart failure/LV dysfunction	1
Hypertension	1
Age > 75	2
Diabetes mellitus	1
Stroke/TIA/thromboembolism	2
Vascular disease*	1
Age 65–74	1
Sex category (ie, female sex)	1
Maximum score	9

0-1: no prophylaxis
1: controversial
≥ 2: oral anticoagulant

Yes

Asses cause and follow up

Presenting >48 hrs?

TEE to check atrial thrombus
or
Give Anticoagulants for ≥3wks

saw-tooth baseline (F wave)?

Atrial flutter

Catheter ablation is first line

almost all patient with atrial flutter will undergo this procedure
Further management similar to Atrial fibrillation treatment

regular narrow-complex tachycardias?

Supraventricular tachycardia

P waves cannot be seen?

AVNRT

P waves seen after narrow QRS complexes?

AVRT

- **vagal maneuvers** (carotid massage or valsalva manoeuvre)
- SVT persists? **IV adenosine**
- SVT persists despite adenosine? **CCB, BB or flecainide**
- haemodynamic compromise? **DC cardioversion**
- recurrent SVT? **catheter ablation**

delta wave wide, bizarre QRS complexes?

Wolff-Parkinson-White Syndrome

- **Catheter ablation:** first-line treatment in symptomatic patients
- **IV procainamide or ibutilide:** The treatment of choice
- Avoid vagal maneuvers and AV nodal blocking agents (β-blockers, CCBs, adenosine, digoxin)

wide and bizarre QRS-complex, regular tachycardia?

Ventricular tachycardia

- **Treat the underlying cause**
- **Automatic implantable defibrillators**
- **with structural heart disease:** ICD to prevent VT then medication (BB, AA)
- **without structural heart disease:** medication (BB, AA)
- **treatment of choice:** catheter ablation in case of VT occurring in a normal heart and in VT associated with a myocardial infarct scar

no waves can be identified?

Ventricular fibrillation

- **Immediate defibrillation**
- **CPR resumed immediately for 2 minutes**
- **epinephrine:** given every 3–5 minutes during resuscitation
- **IV amiodarone:** if ventricular fibrillation or ventricular tachycardia re-initiates after successful defibrillation

1. There's no definite first step, it depends on the presence of symptoms and the acute onset. Rate control alone is considered for the patient who doesn't have symptoms or notices very little symptoms or chronic AF, while rhythm control to patient who immediately notices the arrhythmia and experiencing consequences of the arrhythmia

Acute coronary syndrome

Done by Naif Alsulais

ACS symptoms?

- **ECG**
- **Troponin** (troponin test after 6h if the pain started in less than 3h)
- **Aspirin** (best initial treatment)
- P2Y12 inhibitors (ticagrelor prefered over clopidogrel)
- Oxygen
- Insulin effusion if blood glucose more than 11 (STEMI)

ST segment elevation ACS?

Presenting <12 hrs from symptom onset?

No → **LMW heparin[1] Consider nitrate IV infusion**

Is primary PCI feasible within 120 mins?

No → Eligible[2] for thrombolysis?

No[3] → **LMW heparin[1] Consider nitrate IV infusion**

PCI + GP IIb/IIIa receptor antagonists IV
(PCI is the best therapy)

Thrombolysis IV (tPA, streptokinase..)
Should be given within 12 hrs of admission
Then fondaparinux or LMW heparin IV

UA or NSTEMI?

-ECG & -enzymes?

TIMI score "AMERICA"

one point for each

1. Age ≥ 65 y/o
2. Markers (Elevated cardiac biomarkers)
3. ECG (ST-segment deviation (≥0.5mm)
4. Risk factors (3 or more CAD risk factors)
5. Ischemic chest pain (2 or more angina events in < 24hrs)
6. Coronary stenosis (Prior stenosis of 50% or more)
7. Aspirin use in the past 7 days

0-1: Low risk | 2-3: Moderate risk | ≥ 4: High risk

High risk →

1. **Coronary angiography + GP IIb/IIIa receptor antagonists IV**
2. **Reperfusion therapy:**
   - **PCI**
   - **CABG:** clopidogrel should be withheld
     - PCI was unsuccessful
     - Three vessels disease
     - Left main coronary artery occlusion
     - Two-vessel disease in a patient with diabetes

Low risk →

Stress test

Positive →

Maintenance in-hospital medication

Patient must leave the hospital with these 6 drugs:

- DAPT (dual antiplatelet therapy: aspirin + ticagrelor) immediately upon arrival to the ER and stay on it for 12 months, then if the risk of bleeding low continue on DAPT up to 3 years, if high risk continue on single antiplatelet therapy
- LMW heparin[1] (stop after the PCI)
- statin
- b-blocker (contraindicated in hypotension, features of heart failure, bradycardia, RCA occlusion[4], cardiogenic shock (e.g., large LV infarct, low ejection fraction) or asthma)
- ACE inhibitor to reduce the risk of CHF development

Special cases:

- Nitrate (symptomatic relief)
- CCB (variant angina[5] or if BB can't be used)

If negative investigate for non-cardiac chest pain

1. Contraindicated in renal failure. In case of Heparin Induced Thrombocytopenia (HIT) give fondaparinux
2. Thrombolysis contraindications: Major bleeding or major trauma, recent surgery, Severe hypertension, pregnancy, peptic ulcer and nonhemorrhagic stroke within 3 months
3. If PCI cannot be performed within 120min for any reason, and thrombolysis is contraindicated, the procedure should be performed as soon as practically possible
4. cause hypotension and JVD treated by increasing Preload by IV fluid (BB, nitrate & diuretics are contraindicated)
5. Variant angina seen in young, smokers, healthy with no history cardiac disease at the night for 15 minutes

Rheumatic heart disease

Done by Hamad Almousa

Definition

- Acute rheumatic fever (ARF) is a sequela of streptococcal infection—typically following 2 to 4 weeks after group A streptococcal (strep. pyogenes) pharyngitis and has rheumatologic, cardiac, and neurologic manifestations. Usually affects children (most commonly 5-15 years) or young adults.

Diagnosis

A firm diagnosis requires: 2 Major manifestations OR 1 Major and 2 Minor manifestations along with Evidence of a recent streptococcal infection (Elevated ASO titer or Anti-Dnase B titer and a positive throat culture)

Major criteria: "J♡NES"
- Migratory polyarthritis (**J**oints)
- ♡ Carditis
- Subcutaneous **N**odules
- **E**rythema Marginatum
- **S**ydenham chorea

Minor criteria: "PEACE"
- **P**revious rheumatic fever
- **E**levated temperature (fever)
- **A**rthralgia
- Elevated inflammatory markers (ESR3, **C**RP4)
- **E**CG: First degree heart block (Prolonged PR interval)

Investigation

Evidence of a systemic illness
a. CBC: Leukocytosis
b. Raised erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP)

Evidence of preceding streptococcal infection
a. Throat swab culture (preferably before giving antibiotics) : group A beta-hemolytic streptococci
b. Anti-streptococcal serology:
(Repeat 10-14 days later if first test not confirmatory)
i. Elevated Antistreptolysin O antibodies (ASO titres)
ii. Elevated Ant-DNAse B titer.

Evidence of carditis
a. Chest X-ray: cardiomegaly; pulmonary congestion
b. ECG: First-degree AV block, Prolonged PR interval…
C. Echocardiography: Cardiac dilatation, Mitral regurgitation

Treatment

1. **Bed Rest**
2. **Salicylates**: Aspirin
3. **Antibiotics**: Like Procaine benzylpenicillin
4. **Steroids**: (Prednisolone)
5. **Heart Failure Treatment**: Like diuretics and ACEI.

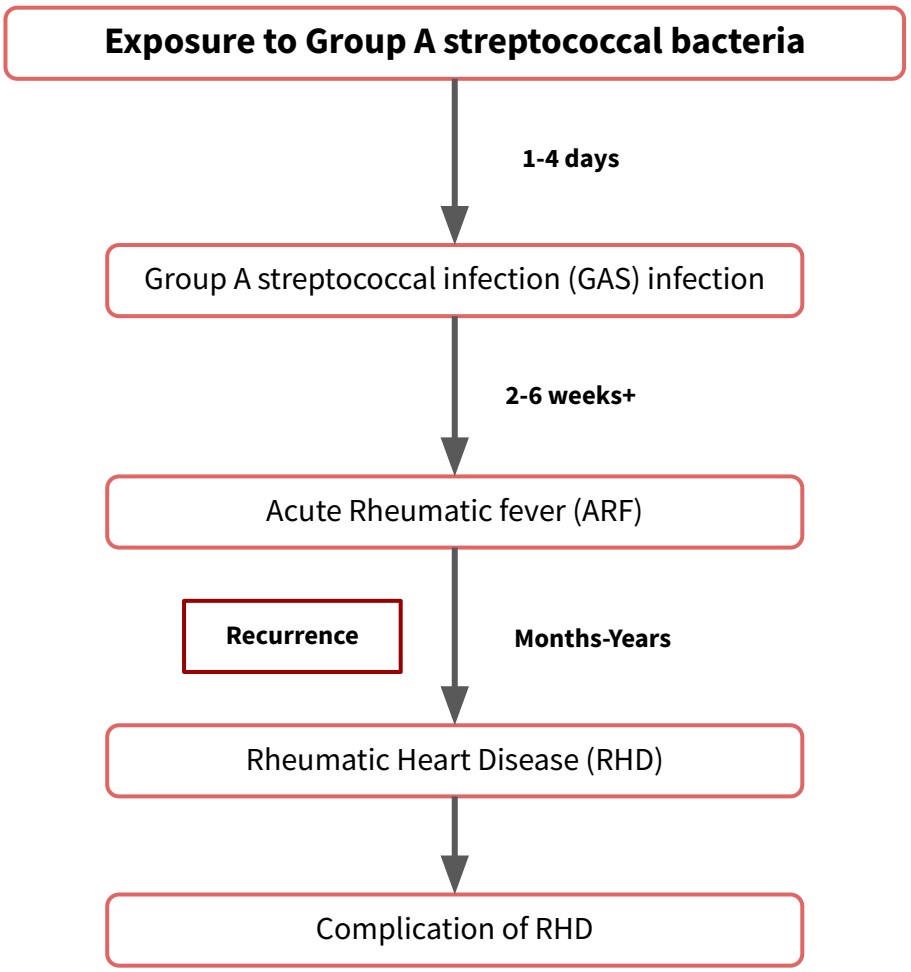
Primordial Prevention
- Reduce overcrowding
- Improved respiratory hygiene

Primary Prevention
- Aims to prevent ARF from developing as result of GAS infection
- Prompt antibiotic treatment of GAS pharyngitis with **BPG 1.2M units IM**

Secondary Prevention
- Antibiotics prophylaxis with IM penicillin G benzathine

- If the patient is penicillin allergic: Oral Sulfadiazine

- If the patient is penicillin and sulfadiazine allergic: use oral erythromycin



Valvular heart disease

Done by Naif Alsulais

Suspected valvular disease?

- **Echocardiogram** (Best initial test for all VHDs)
- **ECG** (define which chamber enlarged "axis deviation" or heart block)
- **CXR**
- **Cardiac catheterization** (Most accurate)

Systolic murmur?

Harsh Crescendo-decrescendo systolic ejection murmur

Heard at: aortic area
Increases with: squatting & expiration
Decreases with: standing valsalva, hand grip & inspiration
Radiate to: carotids

Aortic stenosis

Etiology:
- >65 years: Calcification
- <65: Bicuspid valve or rheumatic heart disease

Symptoms: SAD exertional
- **S**yncope (3 yr survival)
- **A**ngina (5 yr survival)
- **D**yspnea (2 yr survival)

Signs:
- Pulsus Parvus et Tardus: Low volume, slow rising, delayed upstroke carotid pulse
- Soft S2, reversed splittin & prominent S4
- Apex: Sustained Bifid LV impulse (Not displaced PMI)

Management:
- Aortic valve replacement only truly effective therapy
- Anticoagulants (for AF) & Cautious use of vasodilators (for hypertension) due to afterload reduction

Heard at: pulmonary area
Increases with: squatting & inspiration
Decreases with: standing valsalva & expiration
Radiate to: the back

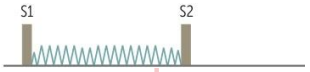
Pulmonic stenosis

Etiology:
- congenital
- RHD (rare)

Symptoms:
- Low CO

Management:
- balloon pulmonary valvuloplasty
- replacement by surgery

High pitched (blowing) holosystolic[1] murmur



Heard at: mitral area (apex)[1]
Increases with: squatting, hand grip & expiration
Decreases with: standing valsalva & inspiration
Radiate to: axilla

Mitral regurg

Etiology:
- Acute: Infective endocarditis, Papillary muscle rupture
- Chronic: Myxomatous (MVP) & RHD

Symptoms:
- Acute: HF symptoms & Cardiogenic shock
- Chronic: asymptomatic or AF, PHTN & HF symptoms

Signs:
- Acute: Soft & Short early systolic decrescendo murmur
- Chronic: Soft (muffled) S1, S3 (Prominent) in advanced stages

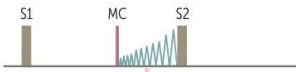
Management:
- Acute: All patients undergo surgery (repair preferred)
- Chronic: treat underlying cause, surgery when indicated

Heard at: tricuspid area[1]
Increases with: squatting, & inspiration
Decreases with: standing valsalva & expiration
Radiate to: the apex

Tricuspid regurg

Extremely rare and can be caused by infective endocarditis (especially IV drug users), rheumatic fever, or right ventricular heart failure.

Late systolic crescendo murmur with midsystolic click



Heard at: mitral area (apex)
Increases with: standing valsalva, hand grip & expiration
Decreases with: squatting & inspiration
Radiate to: axilla (if MR exist)

Mitral valve prolapse

Barlow syndrome

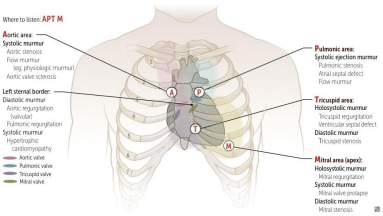
Etiology:
- Connective tissue disorders (e.g. Marfan's syndrome)
- thyrotoxicosis
- rheumatic or ischaemic heart disease

Symptoms:
- Mostly asymptomatic
- Atypical chest pain
- Palpitations
- Sudden cardiac death due to fatal ventricular arrhythmias

Signs:
- Mid-systolic click
- S3 If present with severe MR

Management:
- No treatment is required in most cases
- BB for the chest pain and palpitations with avoidance of caffeine
- Surgery: with severe MR

Diastolic murmur?



MANEUVER	CARDIOVASCULAR CHANGES	MURMURS THAT INCREASE WITH MANEUVER	MURMURS THAT DECREASE WITH MANEUVER
Standing Valsalva (strain phase)	↓ preload (↓ LV volume)	MVP (↓ LV volume) with earlier midsystolic click HCM (↓ LV volume)	Most murmurs (↓ flow through stenotic or regurgitant valve)
Passive leg raise	↑ preload (↑ LV volume)	Most murmurs (↑ flow through stenotic or regurgitant valve)	MVP (↑ LV volume) with later midsystolic click HCM (↑ LV volume)
Squatting	↑ preload, ↑ afterload (↑ LV volume)		
Hand grip	↑↑ afterload → ↑ reverse flow across aortic valve (↑ LV volume)	Most other left-sided murmurs (AR, MR, VSD)	AS (↓ transaortic valve pressure gradient) HCM (↑ LV volume)
Inspiration	↑ venous return to right heart, ↓ venous return to left heart	Most right-sided murmurs	Most left-sided murmurs

Important!

1. VSD has similar murmur to mitral and tricuspid regurg, however we can differentiate between VSD and mitral regurg by knowing that VSD heard at tricuspid area, so how can we differentiate between VSD and tricuspid regurg? Murmur in VSD described as **harsh** unlike tricuspid and mitral which is high pitched "blowing"

Valvular heart disease

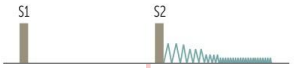
Done by Naif Alsulais

Suspected valvular disease?

- **Echocardiogram** (Best initial test for all VHDs)
- **ECG** (define which chamber enlarged "axis deviation" or heart block)
- **CXR**
- **Cardiac catheterization** (Most accurate)

Systolic murmur?

High-pitched (blowing) diastolic decrescendo murmur



Heard at: left sternal border[1]
Increases with: squatting, hand grip & expiration
Decreases with: standing valsalva & inspiration

Aortic regurg

Etiology:
- Acute: Aortic dissection, infective endocarditis & trauma
- Chronic: Marfan syndrome, rheumatic heart disease & bicuspid aortic valve

Symptoms:
- Acute: Sudden, severe dyspnea, chest pain & Symptoms of low CO & HF (Cardiogenic shock: hypotension & tachycardia)
- Chronic: Exertional dyspnea & chest pain

Signs:
- Acute: S3+ Murmur is early, short, faint and may be absent
- Chronic: Austin Flint Murmur, displaced PMI inferolaterally, high wide pulse pressure (150/50), collapsing pulse & Soft S1,2

Management:
- Acute: surgery (replacement) when symptomatic, medical (Vasodilators and inotropes)
- Chronic: surgery (when indicated), medical (Treat HTN)

Heard at: left sternal border
Increases with: squatting & inspiration
Decreases with: standing valsalva & expiration

Pulmonic regurg

Etiology:
- rheumatic heart disease

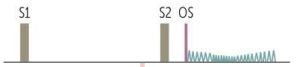
Symptoms:
- High CO

Signs:
- Graham Steel murmur: high-frequency decrescendo diastolic murmur

Management:
- if severe → valve replacement

Diastolic murmur?

Low pitched mid-diastolic rumble Follows opening snap



Heard at: mitral area (apex)
Increases with: squatting & expiration
Decreases with: standing valsalva & inspiration
Radiation : localised

Mitral stenosis

Etiology:
- rheumatic heart disease

Symptoms:
- Afib, Pulmonary hypertension & HF symptoms
- Stroke or peripheral Embolism due to AF
- Ortner's syndrome

Signs:
- Mitral facies
- Prominent a wave in JVP (absent in AF)
- Loud S1, S3 cannot be heard
- Tapping apex beat & parasternal heave
- Opening snap in early diastole (earlier OS indicates severe MS)

Management:
- Treat HF: only Diuretics
- Treat AF: BB (drug of choice) with warfarin
- Surgery:
  - we should do open heart surgery if area <1 cm2
  - PMBC (no calcification)
  - Open commissurotomy (mild calcification)
  - replacement (severe calcification)

Heard at: tricuspid area
Increases with: squatting, & inspiration
Decreases with: standing valsalva & expiration
Radiation : localised

Tricuspid stenosis

Etiology:
- most commonly caused by infective endocarditis (especially in IV drug users)
- carcinoid tumors
- rheumatic fever

Symptoms:
- Right heart failure

Other murmurs!

VSD murmur
- Holosystolic, **harsh**-sounding murmur
- Loudest at tricuspid area

ASD murmur
- Mid-systolic murmur at the pulmonic region with a diastolic rumble along the left sternal border
- The second heart sound is split and fixed relative to respiration area

PDA murmur
- Continuous machine-like murmur
- Best heard at left infraclavicular area

HCM murmur
- crescendo-decrescendo, midsystolic murmur
- heard best at the left lower sternal border & **doesn't radiate**
- Increases with valsalva
- Decreases with squatting

Carey Coombs murmur
- soft mid-diastolic murmur
- Occurs due to valvuliti in rheumatic heart disease

Carotid bruit
- continuous murmur in neck that increases in intensity during systole
- result of carotid artery stenosis which is a risk for stroke & TIA
- disappears with pressing on the sides of the neck

Chronic Heart failure

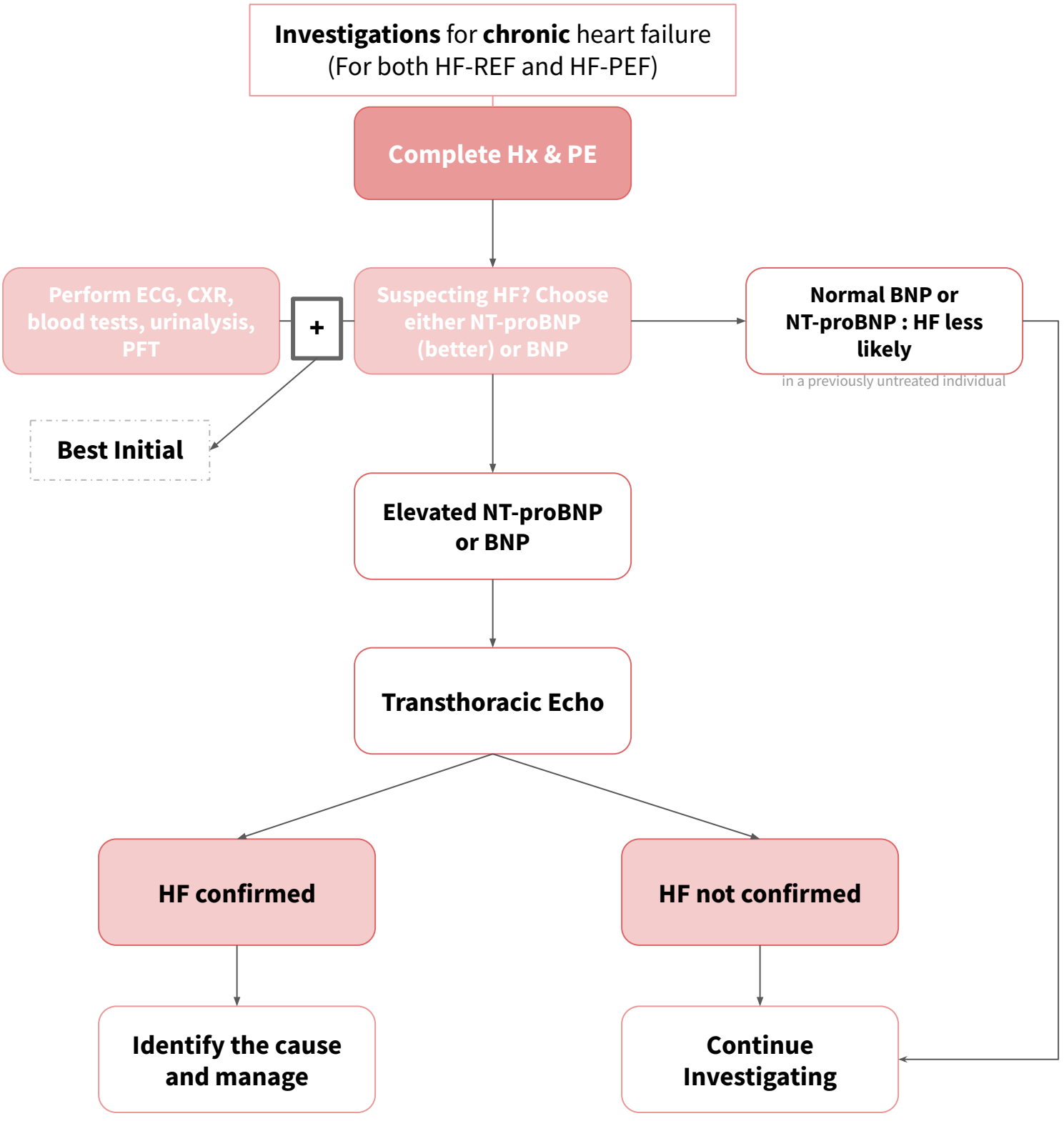
Done by Omar Alhalabi

Definition: Inability of the heart to perform its function to meet the body demands.

Causes:

- Most common: IHD, then cardiomyopathy, then hypertension
- Less common: valvular heart diseases, drugs, systemic and autoimmune diseases, renal and pulmonary diseases…

S & S: We all know it! Exertional dyspnea and fatigue, PND, Orthopnea, crackles palpitations, ankle swelling…



Chronic Heart failure

Done by Omar Alhalabi

Recall:
- ACE inhibitors ends with pril
- BB: cardio specific are MBC (Metoprolol, Bisoprolol, Carvedilol.
- ARBs ends with tan

Management of chronic HF-REF

First Line therapy

Use Loop diuretics for symptomatic relief (**does not improve mortality**)

+

ACE inhibitor and cardiac-specific BB (first Line)

Can't tolerate ACEI? Use an ARB instead

Can not tolerate neither ACEI nor ARB? Use Hydralazine and Nitrate

Still symptomatic, or developed hypokalemia due to Loop? Add **spironolactone** to this regimen

In addition to the already prescribed loop

Still symptomatic? Consult a cardiologist and choose one of the following

- **Replace ACE or ARB with the combination of sacubitril-Valsartan if EF <35%**
- **Add Ivabradine if he has normal sinus rhythm and HR >75 with EF <35%**
- **Add Hydralazine and Nitrate (specially if African-Caribbean)**
- **Add Digoxin for HF with Normal sinus rhythm to improve symptoms**

Management of chronic HF-PEF

- **Up to 2022, There are no treatments that improve mortality. So treatment is directed towards symptomatic relief, treatment of the underlying cause, and treating other comorbidities**
- **Diuretics are good In improving the symptoms of HF. The one that might have mortality benefit is spironolactone. Other diuretics are used for symptomatic relief. Also rehabilitation and exercise if appropriate.**

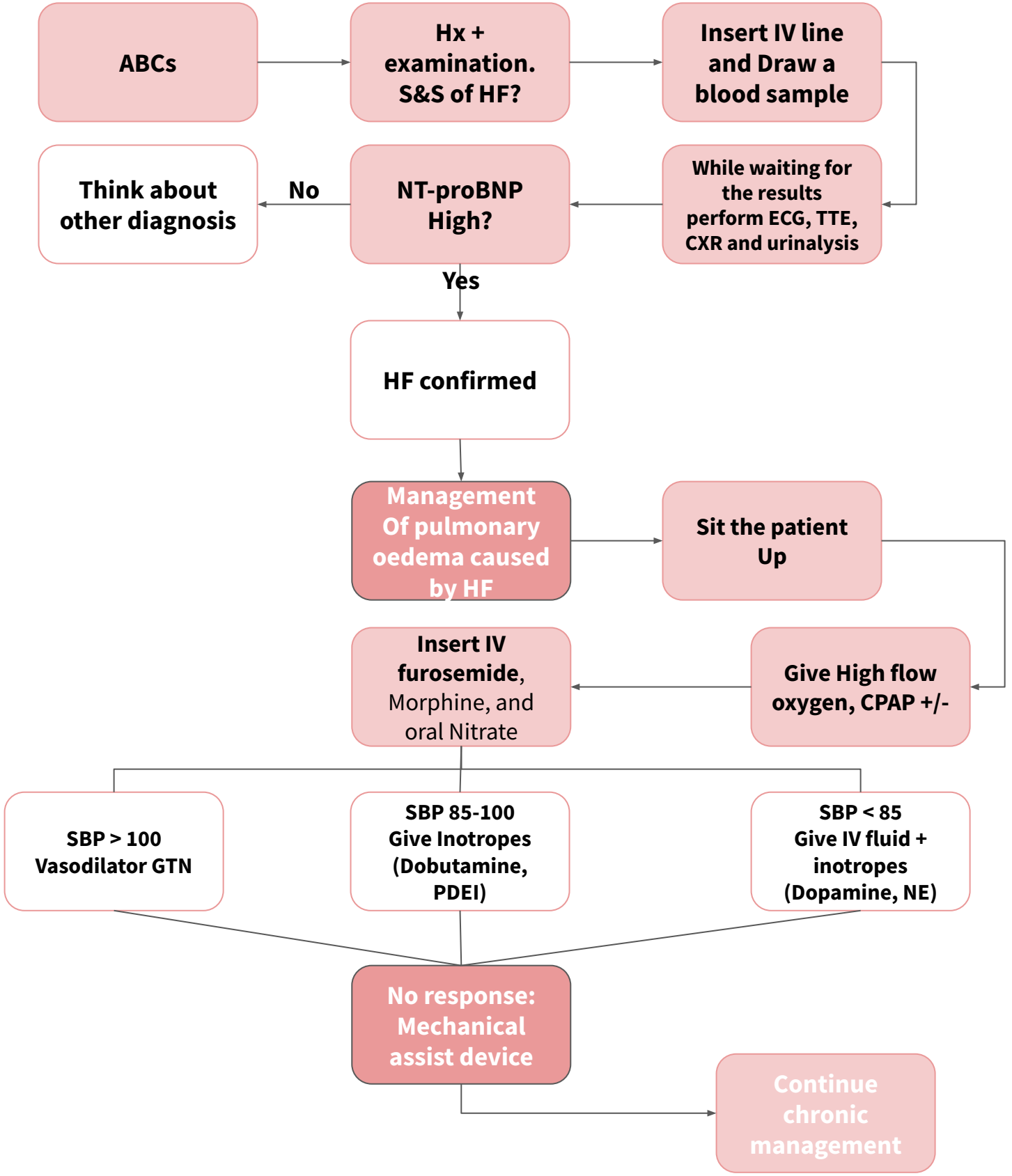
Acute Heart failure

Done by Omar Alhalabi

S & S: Acute onset worsening dyspnea at rest, Tachycardia, diaphoresis, cyanosis, signs of pulmonary oedema (ronchi, rales, frothy sputum), hypoxemia

Investigations and management:

You are going to do all the investigations of chronic HF but not in the same order, and you will need to rule out other differentials, such as PE



Infective endocarditis

Done by Hamad Almousa

Definition

- Infective Endocarditis is an infection of the endocardial surface of the heart, which may include; one or more heart valves (native or prosthetic), Chordae tendineae , a septal defect, AV shunt. leading to formation of bulky friable vegetations composed of thrombotic debris and organisms

Risk Factors

Patient factors:
Age: > 60 years
IV drug abuser (IVDU) ⟶ Staph. aureus mainly, Poor dentition dental procedure /infection ⟶ Viridans mainly

Comorbid conditions:
Structural heart disease, Valvular heart disease (VHD), Prosthetic heart valves, Congenital heart disease, Cardiac implantable electronic device

Prophylaxis

Antibiotic prophylaxis is reasonable before dental procedures requiring manipulation of gingival or periapical region of teeth or perforation of oral mucosa in patients with the following:

- Prosthetic cardiac valves including transcatheter valve or prosthetic material used for cardiac valve repair
- History of previous endocarditis
- Congenital heart defect(CHD) such as Ventricular septal defect (VSD), Patent Ductus Arteriosus (PDA) , Coarctation of aorta and Complex cyanotic disease (Tetralogy, Transposition, Single ventricle)
- Cardiac transplant with valve regurgitation due to a structurally abnormal valve

For dental procedure at risk :Amoxicillin or Ampicillin, 2 g (adults) 50mg/kg (children) orally or IV, single dose 30-60min before the procedure.

Causative Agent

Native valve IE: Streptococcus viridans
(in mitral valve prolapse, recent dental extraction)

IV drug abusers IE:
Staphylococcus aureus 60%

Prosthetic valve IE:
A) Early onset: (< 60d after surgery)
Staph. Epidermidis (30%) or Staph. Aureus (20%).
B) Late onset: (>60d after surgery)
Streptococcus viridans

Signs & Symptoms

Sub-acute:Fever, malaise, fatigue, night sweats, anorexia and weight loss

Acute: Congestive cardiac failure (CCF), new/changing murmur with severe systemic sepsis.

Immunological features: Osler's Nodes, Roth Spots, Glomerulonephritis, +ve Rheumatoid Factor
Vascular and septic Emboli: Splinter/Nail bed (Or subungual) Haemorrhages, Septic arthritis, Janeway Lesion, Anemia
Other: Splenomegaly, Petechiae

Diagnosis

Major criteria:
Positive blood culture, Endocardial involvement
Minor criteria
Fever, Echo findings (not involved in the major criteria), Vascular phenomena, Evidence from microbiology, Risk factors and predisposition, Immunological phenomena

Duke criteria
Definitive IE: Clinical criteria: Patients with 2 major, OR 1 major and 3 minor, OR 5 minor.
Possible IE: Clinical criteria: Patients with 1 major and 1 minor, OR 3 minor.

Management

Empirical therapy:

Acute: Blood culture and start treatment within 3 hours; Vancomycin and Gentamicin.

Sub-acute: Blood culture then antibiotic can be started within 3d: Amoxicillin with/without gentamicin

Prosthetic valve IE: Vancomycin, gentamicin and rifampicin

After identification of the causal organism:

Staphylococcus
Native valve
- MSSA: Flucloxacillin OR Naficillin OR Oxacillin for 4wks
- MRSA & Penicillin allergic Pts: Vancomycin for 4-6wks

Prosthetic valve
- MSSA: Flucloxacillin with gentamicin and rifampicin
- MRSA & Penicillin allergic Pts: Vancomycin, with gentamicin and rifampicin

Strep. viridans or bovis
Penicillin susceptible:
- IV Ceftriaxone once daily for 4 weeks (cure rate >98%)
- OR Ceftriaxone 2g for 2 weeks followed by oral amoxicillin for 2 weeks
- OR IV penicillin G OR IV amoxicillin for 4 weeks
- In B-lactam allergic patients: Vancomycin.

Penicillin resistant:
- Ceftriaxone with Gentamicin OR Penicillin G OR Amoxicillin.
- In B-lactam allergic patients: Vancomycin with Gentamicin

Infective endocarditis

Done by Hamad Almousa

Clinical Suspicion of Infective endocarditis

Transthoracic Echocardiography (TTE)

- Prosthetic valve or intracardiac valve → Transesophageal Echocardiography (TEE)
- Positive for Infective Endocarditis → Transesophageal Echocardiography (TEE)
- Non-diagnostic images → Transesophageal Echocardiography (TEE)
- Negative for Infective Endocarditis → Clinical suspicion of infective endocarditis
  - High → Transesophageal Echocardiography (TEE)
  - Low → Stop

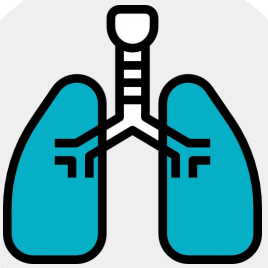
Infective endocarditis
Confirmed based on clinical features and echocardiography

Blood culture (at least 3 samples over 24hrs)

- Staphylococcus
  - Native valve
    - **MSSA:** Flucloxacillin OR Naficillin OR Oxacillin for 4wks
      MRSA & Penicillin allergic Pts: Vancomycin for 4-6wks
  - Prosthetic
    - **MSSA:** Flucloxacillin with gentamicin and rifampicin
      MRSA & Penicillin allergic Pts: Vancomycin, with gentamicin and rifampicin
- Strep. viridans or bovis
  - Penicillin susceptible:
    - **IV Ceftriaxone** once daily for 4 weeks (cure rate >98%)
    - **OR Ceftriaxone** 2g for 2 weeks followed by oral **amoxicillin** for 2 weeks
    - OR **IV penicillin** G OR IV amoxicillin for 4 weeks
    - **In B-lactam allergic patients: Vancomycin.**
  - Penicillin resistant:
    - Ceftriaxone with Gentamicin OR Penicillin G OR Amoxicillin.
    - In B-lactam allergic patients: Vancomycin with Gentamicin

Blood cultures: **It's the key diagnostic investigation** **(Best initial)**
Echo: **(Cornerstone of diagnosis)**
TTE; First-line non-invasive imaging test / TEE; Second-line invasive imaging test with greater sensitivity

Pulmonology

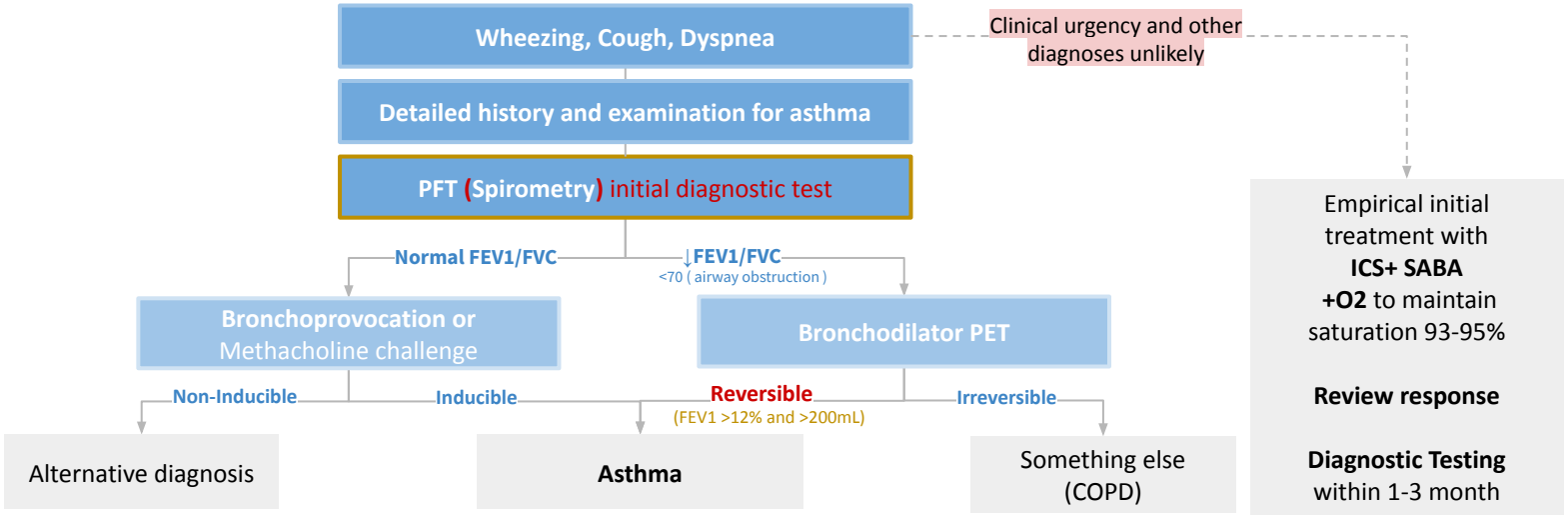


Resources:

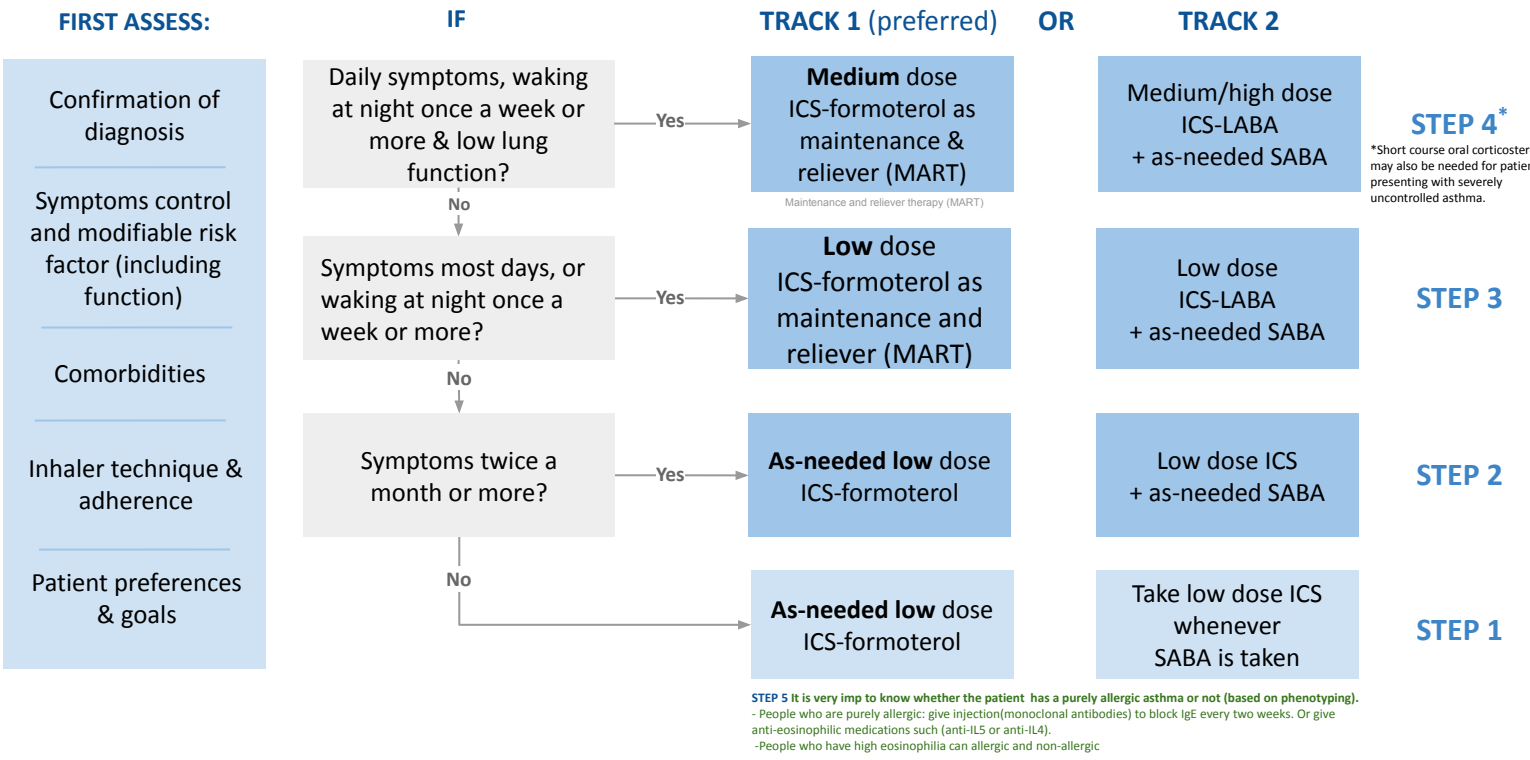
- Davidson’s principle and practice of medicine latest edition, 2018
- Kumar and clark’s Clinical medicine latest edition, 2021
- Doctors’ lectures and notes
- Master the boards, 2021 edition
- Global Initiative for Asthma 2021

Asthma

Done by Ghada Aljedaie

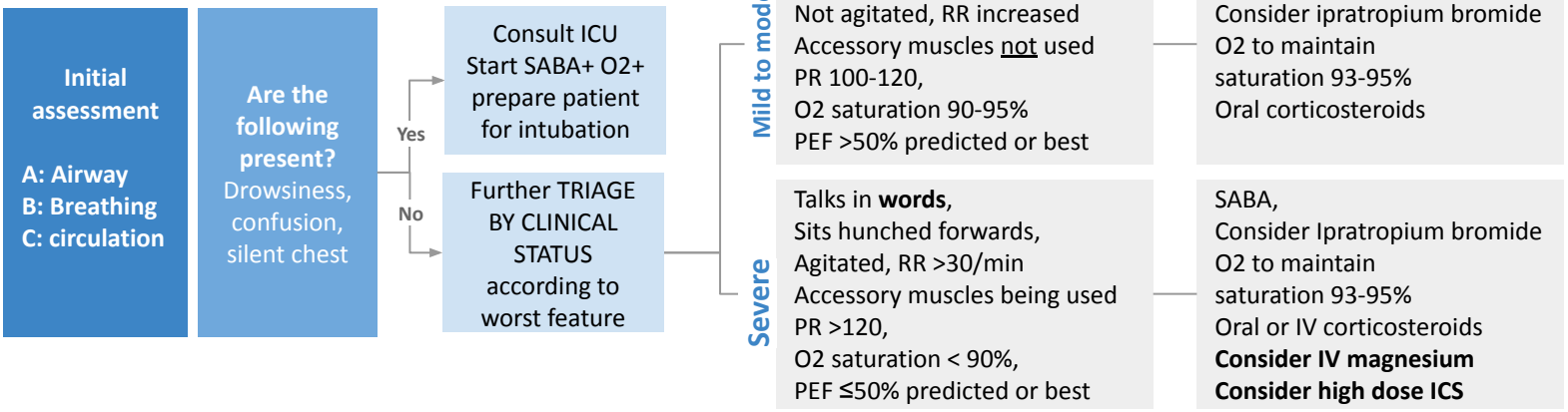


Treatment of Asthma:



STEP 5 It is very imp to know whether the patient has a purely allergic asthma or not (based on phenotyping).
- People who are purely allergic: give injection(monoclonal antibodies) to block IgE every two weeks. Or give anti-eosinophilic medications such (anti-IL5 or anti-IL4).
-People who have high eosinophilia can allergic and non-allergic

Management of asthma exacerbations in emergency department



Community acquired pneumonia

Done by Mohammad Aljumah

Pneumonia sign/ symptoms?

- **Symptoms:** Cough(productive with typical / unproductive with atypical), fever, chest pain, and SOB
- **Signs:** dullness, crackles, bronchial breath sounds(indicative for consolidation), increase in vocal and tactile fremitus and egophony
- **extrapulmonary manifestation:** (meningitis, bacteremia (septic shock), reactive arthritis (after hepatitis A infection)

Investigate

- **CXR** (best initial)
  - **Consolidation:** classical finding in pneumonia
  - **Cavity:** upper zone (staph), lower zone (anaerobes)
  - **Interstitial Infiltrates:** atypical organisms especially viral
  - **Parapneumonic Effusion:** blunting of costophrenic angle
- **CT:** If CXR is inconclusive
- **Blood culture (2 set):** marker of severity (w/ septicemia)
- **Serum/ urine antigen:** the next step If legionella suspected

CURB-65

ONE POINT FOR EACH

- **C**onfusion
- **U**rea: > 19 mg/dl (7 mmol/L)
- **R**espiratory Rate: ≥ 30
- **B**lood Pressure: Systolic <90 mmHg or Diastolic ≤ 60 mmHg
- **Age:** ≥ 65

0 or 1 Point?

Send home With azithromycin for 3-5 days OR high dose amoxicillin OR clindamycin If it's anaerobe

2 Points?

admit to hospital and treat with ceftriaxone + azithromycin

3 or more?

ICU and treat with Tazocin (Piperacillin/tazobactam) also called piptaz.

Special conditions

- **Aspiration Pneumonia:** clindamycin
- **Risk of Staph (MRSA):** Vancomycin
- **bronchiectasis (gram -ve like pseudomonas):** levofloxacin or ciprofloxacin.

Organisms + risk factors important!

Typical (60% - 70%)		**Strept .pneumoniae** most common cause of CAP (secondary[1] & primary)	**Haemophilus influenzae** most common in smokers and COPD	**Staphylococcus aureus** 2nd most common cause of secondary[1] increased risk with crowded living conditions	**Aerobic gram(-) bacteria** e.g. Pseudomonas aeruginosa (associated with cystic fibrosis & bronchiectasis) & Klebsiella pneumoniae (associated with aspiration lobar pneumonia in alcoholics)	**Anaerobes** Bacteroides, Prevotella, Fusobacterium, Peptostreptococcus Increased risk with **aspiration**: • loss/alteration in consciousness (stroke, seizure, anesthesia, drugs (opioids) or alcohol • Esophageal disorders (achalasia or uncontrolled GERD) • Vomiting
Atypical (30%-40%)	**Atypical Bacteria**	**Legionella spp** (hotel and traveling history) contaminated water, air,ventilation systems GI symptoms, headache, chest pain	**Mycoplasma pneumoniae** After trauma, splenectomy & in HIV	**Chlamydia pneumoniae** joints pain, headache, sinusitis & skin rash	**Chlamydia psittaci** birds	**Coxiella burnetii** farmers
	Respiratory virus	**Influenza A & B viruses**	**Respiratory syncytial virus** most common cause in infants	**Coronaviruses** COVID-19 or middle east respiratory syndrome coronavirus	**Rhinoviruses**	**Adenoviruses**

1. Secondary to viral pneumonia

COPD

Done by: Shaden Alobaid

Definition

- It is characterized by persistent respiratory symptoms and airflow limitations that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases. It is a combination of emphysema and chronic bronchitis.

Risk Factors

- **Tobacco smoking (most common cause)**
- Alpha-1 Antitrypsin deficiency (genetic)
- Environmental factors (influence the final disease)
- Family history

Emphysema (distal)

- Permanent enlargement of the airspaces distal to the terminal bronchioles (alveolar walls).
- Imbalance between elastase and anti elastase -> ↑ elastase -> destroys elastin -> impairs your lungs elastic recoil -> air trapping -> cannot get air out

Chronic bronchitis *(proximal=conducting zone)*

- Chronic productive cough that bursitis for at least 3 consecutive months in at least 2 consecutive years
- Chronic inflammation due to:
  - Stimulation of submucosal mucus secreting glands -> ↑ secretion of mucus in bronchi
  - Constriction of segmental bronchi or/and proximal bronchioles -> irreversible fibrosis

Signs and Symptoms

Presentation: Age ≥45 and a heavy smoker

- Chronic & progressive dyspnea
- Chronic productive cough +/- hemoptysis
- Expiratory wheeze & chest tight
- Cyanosis due to hypoxemia (with chronic bronchitis)
- Weight loss, cachexia & osteoporosis
- Barrel chest from air trapping (mostly with emphysema)
- Pursed lip breathing (mostly with emphysema)
- Hyperresonance & ↓breath sounds
- hepatosplenomegaly due to hyperinflated lungs (they push spleen & liver down giving a false impression of hepatosplenomegaly

Diagnosis & investigation

Pulmonary function Test (spirometry)
Only diagnostic investigation

- **Spirometry:**
  - **Reduced FEV1 and reduced FVC**
  - **Reduced FEV1:FVC ratio <70 %**
  - TLC : normal in chronic bronchitis and ↑ in emphysema
- DLCO : normal in chronic bronchitis and ↓ in emphysema
- Irreversible bronchoconstriction and inflammation separates wheezing of COPD from wheezing of asthma. Asthma can be reversed with bronchodilators b**ut not COPD**
- **Severity of COPD correlates with FEV1**

ABG

- Hypoxemia and hypercapnia are expected findings in patients with acute or chronic respiratory acidosis
  - Decreased pO_2: partial respiratory failure
  - Decreased pO_2 and increased pCO2: global respiratory failure

CXR
Best initial test

- SIgns of **hyperinflated lung** (barrel chest): decreased lung markings "hyperlucency", increase AP diameter, diaphragm pushed down and flattened , and sometimes presence of large bullae and infiltrations

Others

- Echocardiography: RA and RV hypertrophy
- CBC: ↑hematocrit Chronic hypoxemia ⟶ increased release of erythropoietin ⟶ increased erythropoiesis ⟶ secondary polycythemia
- Test for Alpha-1 Antitrypsin deficiency
- HRCT for characterizing beulla if found in X-ray

Management

Non-pharmacological

- Smoking cessation : most effective step to slow the decline in lung function
- Long term O2 therapy: proven to **reduce mortality. Aim: PaO_2: 88-92% Indicated in :**
  - PaO2 ≤ 55 mm Hg or SaO2 ≤ 88% at rest
  - PaO2 between 55 and 60 mm Hg or SaO2 of 88%, if there is evidence of pulmonary hypertension, congestive cardiac failure, or polycythemia
- **Vaccination:** Influenza and pneumococcal

Pharmacological

- Mild: SABA
- Moderate to severe: LABA (formo/salmeterol) & Inhaled anticholinergics (Tiotropiun bromide (LAMA), Ipratropium bromide (SAMA). **LABA is given before ICS**
- Pts who can't inhale -> oral bronchodilators (theophylline)
- PDE4-inhibitors (roflumilast) they open airway & reducing inflammation,for maintenance of COPD treatment
- Surgical management (Bullectomy and lung transplant)

COPD & bronchiectasis

COPD Exacerbation

COPD exacerbations are defined as: an acute worsening of respiratory symptoms that result in additional therapy

Classifications	1- Mild: Short acting bronchodilators (SABD) 2- Moderate: SABS+antibiotics and/or oral corticosteroids 3- Severe: hospitalization or ER
Management	1- Pharmacological: bronchodilators, corticosteroids, antibiotics, controlled O2 therapy 2- Respiratory support 3- Non-invasive ventilation

Bronchiectasis

Definition

Abnormal, and permanently dilated airways. Bronchial walls become inflamed, thickened and irreversibly damaged. The mucociliary transport mechanism is impaired and frequent bacterial infections ensue.

Characteristics

1- Persistent cough **2- Excessive sputum secretions** **3- Recurrent airway infection**

Etiology

Congenital:

- Kartagener's syndrome
- Hypogammaglobulinemia
- **Cystic fibrosis**
- Abnormal cartilage formation
- Pulmonary sequesterian

Acquired:

- **Recurrent pulmonary infection (TB, Severe pneumonia, MAC)**
- Bronchial obstruction
- **Childhood infection** (e.g. measles, pertussis)
- Aspiration
- Granulomas

Cystic Fibrosis	• A hereditary autosomal recessive disorder caused by defective CFTR protein due to mutation of this gene on the long arm of chromosome 7 • Clinical features: **Salty sweat, men infertility,** failure to thrive (Due to malabsorption) • Diagnosis: Sweat chloride test (a chloride concentration ≥60mmol/L

Clinical feature

- **Hemoptysis**
- **Clubbing of fingers**
- **Chronic cough (large amount of mucus)**

- Auscultation : Crackles and Wheezing
- Dyspnea
- Weight loss
- Pallor due to anemia

Diagnosis & investigation

High resolution - CT (HR-CT) scan **Gold standard**	• **Signet ring sign** • **Thickened, dilated bronchi** • **Cysts at the end of boncho**
Sputum Culture	• **Essential for adequate treatment**
CXR	Can be normal, but sometimes shows: dilated bronchi with thickened bronchial walls
Others	• Spirometry

Management

Chest physiotherapy / Postural drainage/ Inhaled bronchodilators & Anti inflammatory / Immunization /Nebulized saline /Mucolytics

Exacerbation of Bronchiectasis	• Recurrent bouts of pneumonia and acute bacterial infection of sections of dilated bronchi, Increased production of mucous above baseline +/- Low-grade fever • Management : 1- Empiric therapy: Amoxicillin, clarithromycin and ciprofloxacin for 14 days 2- Long term antibiotics: used for patients who have 3 or more exacerbation/yr (Nebulised antibiotics: gentamicin/tobramycin/colistin and Long term macrolides) 3- Antimicrobial agents

Pulmonary Embolism

Done by Raghad Albarrak

1. Suspected PE:

- Sudden onset dyspnea.
- Sudden Pleuritic chest pain.
- Haemoptysis.

2. Wells score:

- assess clinical probability

i **Box 29.4 Wells score for pulmonary embolism (PE) (two-level)**

Clinical feature	Points
Clinical signs and symptoms of deep vein thrombosis (DVT) (minimum of leg swelling and pain with palpation of the deep veins)	3
Alternative diagnosis less likely than PE	3
Heart rate >100 beats/min	1.5
Immobilization (for >3 days) or surgery in the previous 4 weeks	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Total score ≤ 4

PE unlikely

D-dimer

Negative

No PE

Positive

CT pulmonary angiography

Negative

No PE

Positive

PE confirmed

Total score > 4

PE likely

CT pulmonary angiography*

- Visible intraluminal filling defects of pulmonary arteries.
- Wedge-shaped infarction with pleural effusion.

Positive

PE confirmed

Negative

No PE

Treatment

Intiaital (0-7 days)

- Parenteral low-molecular weight heparin (LMWH):
  - normal renal function.
  - preferred in patients with cancer.
- Intravenous unfractionated heparin (UFH): In patients with renal failure.

💡 **Prompt initiation of anticoagulation (heparin) while awaiting investigations is prudent.**

💡 Not required if long-term anticoagulation is planned with rivaroxaban or apixaban (DOACs).

Long term (1 week - 3 months)

- Direct oral anticoagulant.
- Warfarin.

Extended (3 month - indefinite)

- Continue for 3 to 6 months or more, **depending on risk factors.** Some patients at significant risk for recurrent PE (e.g., malignancy, hypercoagulable state) may be considered for lifelong anticoagulation.

💡 Cancer = 6 months anticoagulants or as long as cancer is active.

Massive PE

For massive PE use thrombolytic (Recombinant tissue-plasminogen activator) and anticoagulant.
When are thrombolytics the right answer?

- Hemodynamic instability.
- Hypoxia on 100% oxygen.
- Right ventricular dysfunction by echocardiography.

*: Ventilation perfusion scan when CTPA in contraindicated: renal impairment and allergies to intravenous contrast agents.

- When V/Q scan showed intermediate probability do leg duplex for DVT, if +ive > treatment , if -ive > do CTPA.
- **Pulmonary angiogram:** Consider when noninvasive testing is equivocal and risk of anticoagulation is high, or if the patient is hemodynamically unstable and embolectomy may be required.

Pleural Effusion

Done by Raghad Albarrak

1. Symptoms:

- Dyspnea.
- Pleuritic chest pain.
- Dry, nonproductive Cough.
- Other symptoms related to cause.

2. Physical examination:

Can detected clinically if > 500ml:
- Asymmetric expansion.
- Tracheal shift away from the effusion.
- Stony dullness.
- Decreased tactile fremitus.
- Faint or absent breath sounds.
- Absent vocal resonance.

3. Investigations:

1. **Chest x-rays**
   Initial diagnostic test.
   Findings: blunting of costophrenic angle. (can be detected on X-ray when ≥ 300 mL of fluid).
2. US: 2nd diagnostic test after CXR (confirmatory + provides details about the nature of the effusion).
3. Thoracocentesis.

4.Diagnosis:

This is by pleural aspiration (thoracocentesis), which is necessary to determine the etiology of a pleural effusion.

Indications:
1. **Unknown etiology**, with >10mm depth on lateral decubitus CXR or US.
2. Concern for **empyema**.
3. **Air fluid level** in pleural space.
4. **Therapeutically** for symptomatic relief.

💡 may not be necessary if there is already a clear diagnosis.

5. Light's criteria:

Pleural effusion is exudative if one or more of the following:
- Pleural fluid protein : serum protein >0.5
- Pleural fluid LDH : serum LDH >0.6
- Pleural fluid LDH >2/3 upper limit of normal for serum (105–333 IU/L)

absence of all 3 criteria = Transudative

Transudative

Protein content: < 30 g/L
LDH: < 200 IU/L
Glucose: ≥ 60 mg/dL
Appearance: Clear

Main causes

- **CHF.**
- **Nephrotic syndrome.**
- **Hepatic cirrhosis.**
- **Hypothyroidism.**

Exudative

Protein content: > 30 g/L
LDH: > 200 IU/L
Glucose: < 60 mg/dL
Appearance: Cloudy

Main causes

- **Pneumonia and empyema.**
- **Malignancy.**
- **PE.**
- **Inflammatory.**
- **Connective tissue disease.**
- **TB:** diagnosed by pleural biopsy.

Character	Empyema	Complicated Parapneumonic effusion	Uncomplicated parapneumonic effusion	Chylothorax
Color	Pus	Turbid		Cloudy,milky
Pleural fluid parameter	pH is < 7.2		pH : normal	- Triglycerides > 110 mg/dL. - Total cholesterol 55–200 mg/dL.
Treatment	Chest tube + Antibiotics		Antibiotics	-

Management is of the underlying condition unless the fluid is purulent (empyema), in which case drainage is mandatory and antibiotics.

Investigation of lung disease

Done by Noura Alsalem

Imaging

Chest X-ray	• Performed on the majority of patients suspected of having chest disease. (Initial test).
HRCT (high resolution CT scan)	• Interstitial lung diseases e.g. **sarcoidosis, pulmonary fibrosis.** • **Bronchiectasis.**
CT angiogram	• **In pulmonary embolism** **CT angiogram is contraindicated in: - Pregnancy - allergy to contrast - renal failure**

Thoracentesis

In pleural effusion

- Before performing it check the patient's **CBC** and **coagulation profile**, to exclude any bleeding problems and **confirm pleural effusion by ultrasound** or decubitus film.
- NEVER do thoracentesis for a patient with collapsed lungs → you will cause pneumothorax on top of collapse. Instead, do bronchoscopy.
- If any of the following features are present (Gross appearance of **pus**, Gram stain **positive,** pH below 7.20) do chest tube

- **Chest tube indications:**

1. Empyema 2. Complicated parapneumonic effusion 3. Symptomatic pleural effusion 4. Hemothorax 5. Pneumothorax

Pleural biopsy

- 1- In **Granulomatous disease e.g. TB** 2- Malignancy

Pulmonary function tests

Spirometry	• **Diagnose obstructive lung disease, suggest** restrictive lung disease • Measures: forced vital capacity (FVC), forced expiratory volume in 1st sec (FEVL1)	• obstructive lung: - FVC <70% reduced - FEV1 <70% reduced more than FVC - **FEV1/FVC <70% reduced** • Restrictive lung: - FVC <70% reduced -FEV1 <70% reduced less than FVC -FEV1/FVC increased or normal
Lung volume	• **Diagnose restrictive lung disease** • Measures: Total lung capacity (TLC), Residual volume (RV)	
Diffusion capacity(DC)(DLco)	• to distinguish emphysema from chronic bronchitis or chronic asthma. • **Low DLco indicates Emphysema.** • Normal DLco in chronic bronchitis	

Bronchoscopy

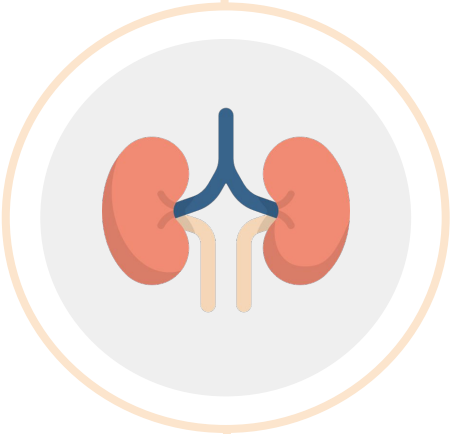
- Diagnostic indications: suspected lung cancer , collapsed lobes or segments seen in CXR .
- Therapeutic indications: **Remove foreign bodies,** remove abnormal endobronchial tissue, difficult endotracheal tube intubation, endobronchial stent placement.

Scintigraphic imaging (V/Q) scan

- It is used in **pulmonary embolism** when patient is allergic to contrast or in **pregnancy** although it only gives a probability.

Disease	Bronchiectasis	Pulmonary fibrosis	Pulmonary embolism	Pleural effusion	COPD	asthma
History/Exam	**Chronic productive cough (mucopurulent copious foul smelling sputum)**, hemoptysis, clubbing, basal crackles, having cystic fibrosis.	Chronic dry cough, SOB Cyanosis, clubbing, bilateral inspiratory crackles.	**sudden onset of SOB, pleuritic (severe) chest pain, tachypnea, tachycardia,** haemoptysis.	SOB, chest pain, cough, **stony dullness**, absent breath sound, heard bronchial sounds, **trachea shifted away from affected side.**	Chronic and **progressive** dyspnea, cough, sputum production, barrel chest, **Does not improve with bronchodilator** .	Recurrent intermittent episodes of cough, **wheezing**, SOB triggered by allergen exposure, exercise and cold air.

Nephrology



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- AMBOSS
- Master the boards, 2021 edition

Diabetic nephropathy

Done by Abdullah Alanzan

Diabetic nephropathy

Urine dipstick + Urine Analysis

Microalbuminuria

ACR : 30-300 mg/1 g Creatinine
3-30 mg/1 mmol creatinine
Urine dipstick can't detect it

Macroalbuminuria

ACR : >300 mg/1 g creatinine or
>30 mg/1 mmol creatinine
Or
+ve Urine dipstick

Repeat the test
2 +ve out of 3 needed to confirm

In type 1 DM check for retinopathy

-ve

Most likely albuminuria is due to other cause ⟶ Do **Biopsy** to confirm

+ve

RAAS blockade
by giving (**ACEi or ARBs**) independent of BP

Glycemic control HbA1C < 7%

Aggressive reduction of cardiovascular risk factors by :

1- BP control : (BP < 130/80)

By ACEi or ARBs or Diuretics

control the BP also important to **prevent progression End Stage Renal Disease (ESRD)**

2- Lowering lipid : LDL < 2.0 mmol/L

Lifestyle modification
Dietary restriction of proteins and Na, Weight loss and exercise.

Type 2 DM patient ?

New agent: SGLT inhibitor

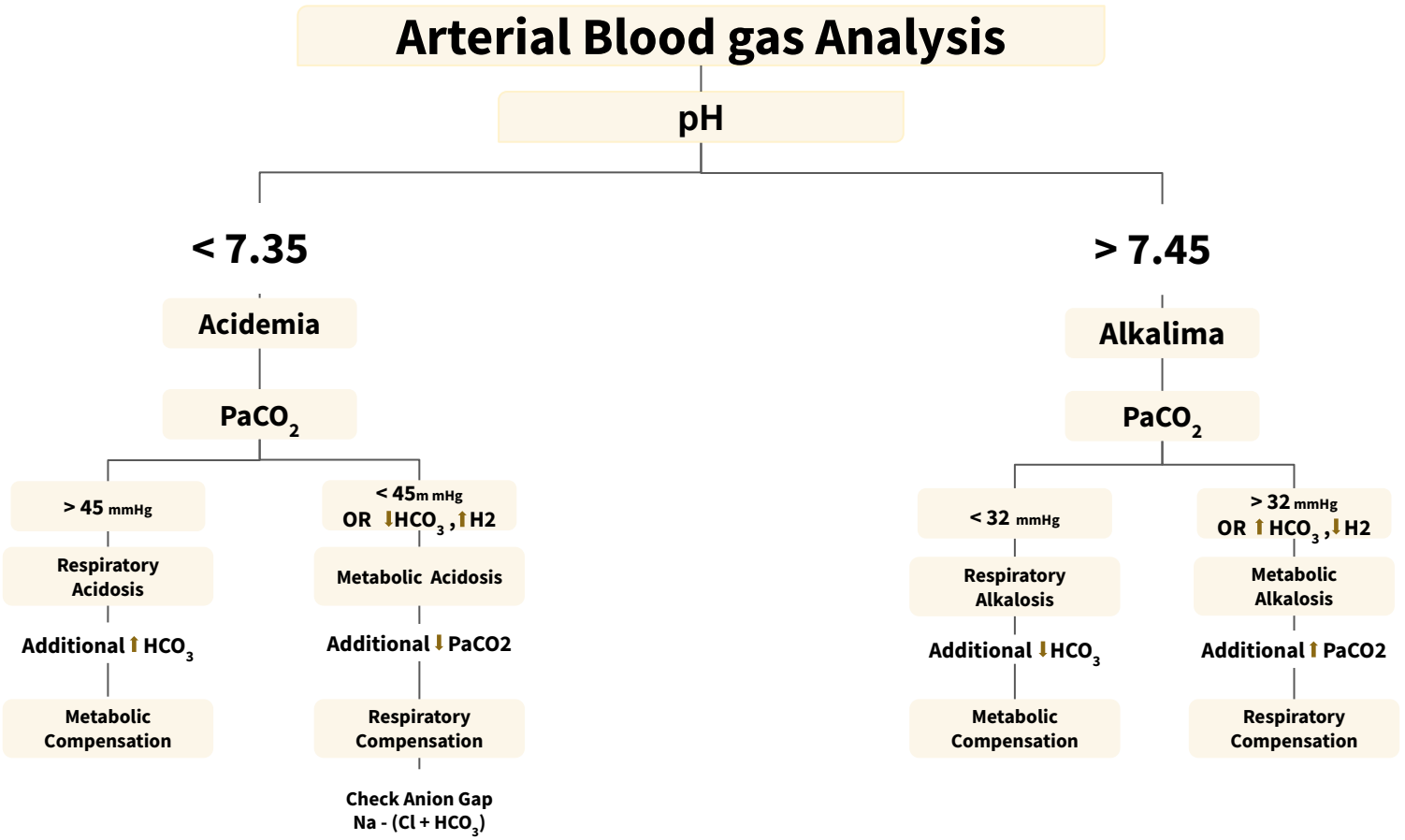
Never for Type 1 DM it may cause **Euglycemic diabetic ketoacidosis (DKA)**
And patient with **recurrent UTI**

Dialysis
should be considered in **ESRD**

Renal transplant
If there is complete Renal failure

Acid-Base disorders

Done by Hamad Almousa



Normal PH doesn't mean there's no disturbance, always look at bicarbonate and CO2 ⟶ mixed disturbance

Respiratory Acidosis

- Most probably caused by Hypoventilation;
Causes can be: (Opiate overdose) (Obstructive lung disease) (No muscular strength) (Obstructive sleep apnea)

Respiratory Alkalosis

- Most probably caused by Hyperventilation;
Causes can be: (Pain) (Anxiety) (Fever) (Hypoxemia) (Basically anything that causes fast respiratory rate)

Metabolic Acidosis

- First look at the anion gap ; (> 12 = Anion Gap Acidosis) / (< 12 = Non Gap Acidosis)
A. Anion gap acidosis causes can be **(MUD PILES)** (M=methanol, U=uremia, D=DKA, P=propylene glycol, I=isopropyl alcohol, L=lactic Acidosis, E=ethylene glycol, S=salicylate)
B. Non gap acidosis ⟶ **check urine anion gap**, (if pos+ = RENAL TUBULAR ACIDOSIS) (if neg - = Diarrhea)

Metabolic Alkalosis

- First look at the Urine chlorine (to see if volume responsive or not)
Volume responsive (treated by IV normal saline) ⟶ Urine chloride will be <10 ; causes can be (Volume depletion) (Emesis) (Diuretics)
NOT volume responsive ⟶ Check BP ; -**Hypertensive** = (Increase Aldos , cortisol)
-**normal BP** = Genetic Disease or Tubular defects or massive Bicarbonate ingestion

Acid-Base disorders

Done by Hamad Almousa

Step 1 History & physical examination:
look for clues that may lead to the abnormalities in pH
● Vomiting: causes loss of acid and gastric contents, which suggests development of alkalosis
● Diarrhea ● Hypoventilation ● Respiratory disease ● Medications (laxatives, diuretics, etc) ● Diabetes

Step 2 Look at the pH
Determine if it is : ● Normal 7.35 – 7.45 (No abnormality or presence of mixed acidosis and alkalosis)
● Low <7.35 (acidemic) ● High >7.45 (alkalemic)

Step 3a Determine the primary abnormality that is causing the abnormal pH
● If the pH is acidemic (<7.35), then look for Low HCO3 (Metabolic) or High PCO2 (Respiratory)
● If the pH is alkalemic (>7.45), then look for High HCO3 (Metabolic) or Low PCO2 (Respiratory)

Step 3b If pH is normal, that doesn't rule out mixed acidosis and alkalosis (Determine what is being mixed)
● Look for high or low PCO2= Low PCO2 suggests respiratory alkalosis/High PCO2 suggests respiratory acidosis
● Look for high or low HCO3= Low HCO3 suggests metabolic acidosis/High HCO3 suggests metabolic alkalosis

Step 4 check for compensation

Compensation is the mechanism by which the body adapts to either acidosis or alkalosis, it will not fully correct the abnormality; example:
● A patient has diabetic ketoacidosis, pH is 7.29, HCO3 is 15 (hence, it is metabolic acidosis)
● Use the metabolic acidosis formula: Expected PCO2 by using Winter's formula PCO2 = 1.5 x HCO3 + 8 (±21) = 1.5 x 15 + 8 = 30.5
● So: you expect the PCO2 in this patient to be in the range of 28.5– 32.54
● Now, determine whether there is a compensation or an additional disorder:
○ If the PCO2 in this patient is higher than 32.5 ⟶ consider additional respiratory acidosis
○ If the PCO2 in the patient is lower than 28.5 ⟶ consider additional respiratory alkalosis

Primary disorder		Expected compensation
Metabolic acidosis		● $PaCO_2 = 1.5 \times HCO_3 + 8 \pm 2$ ● $\downarrow PaCO_2 = 1.2 \times \Delta HCO_3$ ● $PaCO_2$ ~ last two digits of pH
Metabolic alkalosis		● $\uparrow PaCO_2 = 0.7 \times \Delta HCO_3$
Respiratory acidosis	**Acute**	● $\uparrow HCO_3 = 0.1 \times \Delta PaCO_2$
	Chronic (COPD)	● $\uparrow HCO_3 = 0.35 \times \Delta PaCO_2$ ● $\downarrow pH = 0.003 \times \Delta PaCO_2$
Respiratory alkalosis	**Acute**	● $\downarrow HCO_3 = 0.2 \times \Delta PaCO_2$
	Chronic	● $\downarrow HCO_3 = 0.4 \times \Delta PaCO_2$

Step 5 Calculate the anion gap

anion gap (AG): AG = Na – (Cl + HCO3)

● Normal anion gap = 6-12
● Albumin is the main unmeasured anion. To overcome the effects of hypoalbuminemia on the AG, the corrected AG can be used which is AG + (0.25 X (40-albumin)) expressed in g/L.
● If there is a reduction of albumin the bicarb and Cl- will increase
● An increase in anion gap that means there's additional acids like lactic acid and keto acid.

Acute Kidney Injury

Done by Hamad Almousa

Acute Kidney Injury

- **Clinical history** (HPI,PMH,medication,allergies): compare **baseline creatinine** to current creatinine levels to specify which type of kidney injury does the patient have (acute/ chronic/ acute in top of chronic).
- **Physical examination** (BP, weight,fluid status,urine output)
- **Lab tests** (Urinalysis) / - Diagnostic test (Renal imaging, biopsy)

Pre-renal AKI

Volume depletion
- Hemorrhage, Gi loss, Renal losses, Cutaneous losses, Pancreatitis

Decreased cardiac output
- Heart failure, PE, Acute MI, Severe valvular heart disease, Abdominal compartment syndrome

Drugs

Intrinsic AKI

Vascular damage
-HTN, Renal artery/vein thrombosis, atherothrom boembolism

Glomerular damage
-Nephrotic/ Nephritic, glomerulone phritis, autoimmune disease.

ATN
-Ischemic
-Toxic

AIN
-Drugs
-Infection

Post-renal AKI

Bladder neck obstruction
- BPH, Cancer of the prostate, Neurogenic bladder, Bladder (tumor, tone disease, hemorrhage/clot)

Ureteric obstruction
- Stone disease, Tumor, Ligation during pelvic surgery

Urethral obstruction
- strictures, tumor

Acute kidney Injury identified

Decreased urine output and/or increase in serum creatinine by 26.4 umol/L (0.3 mg/dL) within 48 h

Identify the cause (prerenal, renal, postrenal)

- Reserve precipitant cause e.g. sepsis or hypovolemia
- Grade severity of AKI
- Evaluate complication of AKI

- Determine appropriate level of care
- Determine need for renal replacement therapy

Does not require renal replacement therapy

1. Monitor as appropriate for clinical condition
2. Discuss with specialist nephrologist if necessary
3. Maintain euvolemic state
4. AKI is resolved once cause has been addressed and creatinine is on a downward trend with no significant biochemical abnormalities
5. Patient is at risk for CKD. Therefore, follow-up appointment at latest 3 months after AKI to assess creatinine levels

Require renal replacement therapy (dialysis)
Indication for RRT may include:
a. Refractory hyperkalemia
b. Refractory metabolic acidosis
c. Symptoms of uremia e.g. encephalopathy
d. Uremic pericarditis
e. Refractory volume overload

1. Resuscitate patient with a focus on emergent biochemical abnormalities
2. Discuss with specialist nephrologist if necessary
3. Stabilize patient as feasible until RRT is available

Acute Kidney Injury

Done by Hamad Almousa

1- Kidney Injury identified (determine the type)

Acute

- Deterioration of renal function over a period of hours to days.

- Acute: >26.4 µmol/L increase in creatinine within 48 hours

Chronic

- Chronic occurs when the patient has (an abnormal baseline that is elevated) for over 3 months

- e.g. baseline: 280 and current creatinine is 285

Acute on top of chronic

- Acute on top of chronic occurs when there is an increase of creatinine levels within a short period of time over with an abnormal baseline that is elevated)

- e.g. baseline is 150 and current creatinine is 285

2- Determine the cause

BP ?

Normal ?
Exclude pre-renal & ATN

Urinalysis ?

Abnormal

WBC's/ eosinophils

AIN
A toxic medication is usually mentioned in history

Look for offending agent +/- steroid

RBC's/ Proteinuria

GN

Further investigations:

BUN, serum creatinine, CBC, U/S kidneys, **serology**, **kidney biopsy**

Normal

US → Look for **obstruction**

Caused by catheter ? Remove it

Other causes ? → treat them

Hypotension

Specific Gravity/ Concentration of urine ?

High/Concentrated

Kidney is **functioning well** → exclude renal causes

Check for Na → Normal ? → **Pre-renal cause**

IV Fluids

Low/Diluted

Kidney is **abnormal**

Renal cause due to prolonged ischemia (**ATN**)

- Maintain blood volume
- Avoid the cause
- Monitor the patient

Na & water balance

Done by Mohammed Aljumah

Confirmed hypernatremia >145 mmol/L?

Determine the etiology through clinical evaluation

Etiology unclear?

Check urine osmolality

>300 mOsmol/kg?

Implies extrarenal mechanism

<300 mOsmol/kg?

Implies renal mechanism

Hypovolemic? (H_2O loss)

GI loss:
- Diarrhea
- vomiting
- NG tube

Skin loss:
- Burns
- fever
- excessive sweating

Euvolemic? (Low H_2O intake)

Impaired thirst:
- Hypothalamic lesion
- Dementia

Lack of access to H_2O:
- Immobility
- Intubation

Hypervolemic? (Excessive salt intake)

Iatrogenic:
- Hypertonic fluids
- TPN (total parenteral nutrition)

Ingestion
- Salt water ingestion
- Salt water emetics
- Soy sauce

Hypovolemic?

Diuretics (esp. loop)

osmotic diuresis:
- Hyperglycemia

Diabetes insipidus★:
- Central
- Nephrogenic

Post AKI diuresis:
- Therapeutic ATN phase
- Post-obstructive diuresis

Euvolemic?

Diabetes insipidus★:
- Central
- Nephrogenic

Hypervolemic?

Conn syndrome (1ry hyperaldosteronism)

Cushing syndrome (1ry hypercortisolism)

Oliguria (<3 L/day)

Polyuria (>3 L/day)

Normal urine output

★ **Diabetes insipidus:**

Polyuria, Polydipsia & Low urine osmolality?

Fluid restriction for 8 hours

Urine osmolality remains low

Administer desmopressin

Urine osmolality becomes high

Suspect primary polydipsia (hyponatremia)

Urine osmolality becomes high

Central diabetes insipidus
- Caused by trauma & tumors
- Management: desmopressin

Urine osmolality remains low

Nephrogenic diabetes insipidus
- Caused by: hypercalcemia, hypokalemia, lithium & amphotericin B
- Management: thiazides or NSAIDs (consider amiloride if caused by lithium)

Na & water balance

Done by Mohammed Aljumah

Confirmed hyponatremia <135 mmol/L?

Determine the etiology through clinical evaluation

Etiology unclear?

Check serum osmolality

<280 mOsmol/kg?

Hypotonic hyponatremia (true)

Check urine osmolality

- Low = ADH independent (diluted)
- High = ADH dependent (concentrated)

High >100 mOsmol/kg?

Check urine sodium

- Low = active RAAS
- High = inactive RAAS

High >20-30 mEq/L and/or Fe_{NA} >1%?

Renal sodium loss

Hypovolemic?

- Diuretics (esp. thiazides)
- Mineralocorticoid deficiency (1ry adrenal addison disease)
- Salt wasting nephropathy

Euvolemic?

- SIADH*
- Hypocortisolism (2ry adrenal disease)
- Hypothyroidism
- Antidepressants

Hypervolemic?

- Renal failure

Low <20-30 mEq/L and/or Fe_{NA} <1%?

Extrarenal sodium loss

Hypovolemic?

- Diarrhea & vomiting
- Third spacing (eg. pancreatitis)
- Burns

Hypervolemic?

- Heart failure
- Liver cirrhosis
- Nephrotic syndrome

280-295 mOsmol/kg?

Isotonic hyponatremia (pseudo)

- TURP syndrome
- Multiple myeloma
- Hyperlipidemia

>295 mOsmol/kg?

Hypertonic hyponatremia

- Hyperglycemia
- IV mannitol
- IV radiocontrast

Low <100 mOsmol/kg?

Euvolemic?

- 1ry polydipsia
- Low solute intake (eg. tea & toast diet)
- Beer potomania

★ SIADH **(HIVE)**:

H: Hypoosmolar Hyponatremia (Posm <275 mOsm/Kg H2O)

I: Inappropriate urine concentration (Uosm >100 mOsm/Kg H2O)

V: Euvolemia No diuretic use

E: Endocrine = normal Thyroid, adrenal and renal function

+

- Urinary sodium >40 mmol/L with normal dietary salt intake
- Fe_{urea} >55%
- Elevated vasopressin levels despite hypotonicity and clinical euvolemia

Na & water balance

Done by Mohammed Aljumah

Hypotonic hyponatremia		
Hypervolemic	**Euvolemic**	**Hypovolemic**
Water: ↑↑ Sodium: ↑	water:↑ Sodium: N	water: ↓ Sodium: ↓↓

Hypernatremia		
Hypervolemic	**Euvolemic**	**Hypovolemic**
Water: ↑ Sodium: ↑↑	water:↓ Sodium: N	water: ↓↓ Sodium: ↓

Feel lost?

To know what's happening in each one:

1. Determine the volume status:
   a. **Hypervolemic:** give one arrow (↑) to sodium and water
   b. **Hypeovolemic:** give one arrow (↓) to sodium and water
   c. **Euvolemic:** give one arrow (↑in hyponatremia or ↓in hypernatremia) to water only
2. See the condition either:
   a. **Hyponatremia:** caused by water excess > always make the water more and give an extra to water If (↑) and to sodium If (↓)
   b. **Hypernatremia:** caused by water deficit > always make the water less and give an extra to sodium If (↑) and to water If (↓)

		Management	Rate of correction	Overcorrection
1st correct volume (Dysvolemia = Na disorder)	**Low volume (low Na)**	**Normal saline** (standard resuscitation fluid)	-	-
	High volume[1] (high Na)	**Diuretics** In case of renal failure consider hemodialysis instead	-	-
2nd correct sodium (Dysnatremia = water/tonicity disorder)	**Water deficit (hypernatremia)**	**Hypotonic solutions (oral water, D5W or ½ NS)**	**Acute (<48 hrs):** Up to 1 mEq/L/hour **Chronic (>48 hrs):** 0.5 mEq/L/hour (appx 10 mEq/day)	**Cerebral edema/herniation** "From high to low, your brain will blow"
	Water Excess[1] (hyponatremia)	• **Water restriction** • **Hypertonic solutions (3% NaCl)** First step for acute symptomatic hyponatremia (seizures, coma or suspected/known intracranial pathology)	**Acute (<48 hrs):** 6-8 mEq/L/day **Chronic (>48 hrs):** 8-12 mEq/L/day	**Osmotic demyelination syndrome** "From low to high, your pons will die"

SIADH management:

- **Restriction of all fluids:** (PO, IV or medications) to be <1000 mL/day , Ideally (daily fluid should be 500 mL less than daily urine output)
- Persistent hyponatremia? vaptan drugs or low dose loop diuretics

$$\textbf{Target TBW} = \frac{\text{current TBW } (0.6 \times \text{body weight}) \times \text{current Na}}{\text{Target Na } (140)}$$

- **Water deficit =** Target TBW - current TBW
- **Water excess =** current TBW - Target TBW
- **Sodium deficit =** current TBW x 4 (x2 if 3% NaCl)

1. For hypervolemic hyponatremia consider vaptan drugs (esp. heart failure patients)

K-Ca imbalance

Done by Abdullah Alanzan

K-Ca imbalance

- **Calcium Disorder**
- **Potassium Disorder**

Hypokalemia (<3.4 mmol/L)

Causes

↑ Intestinal loss :
Diarrhea, Laxatives abuse, Vomiting

↑ Renal loss :
Diuretics (loop & thiazide diuretics)
↑ aldosterone (hyperaldosteronism)

Rapid transcellular shift :
↑ **Insulin**, B- agonists (e.g: **salbutamol**), Alkalosis

Clinical manifestations

Generally asymptomatic but may present with :

- **Muscular weakness, Tiredness**
- **Serious arrhythmias**
- U wave ST Depression
- Paralytic ileus

Investigation

Route of K+ loss

- (If Urine K^+ > 30 mmol/24 hrs) ⟶ kidney
- (If Urine K^+ < 20 mmol/24 hrs) ⟶ GI tract

Urine chloride

- (< 30 mmol/L) ⟶ Vomiting
- (> 40 mmol/L) ⟶ suggests diuretic therapy, Bartter's or Gitelman's syndrome.

Treatment

1- Stop the loss (Treat the underlying cause)
2- Replace lost K with K

Urgent ?

- **(IV) Potassium chloride**
  -Rate of administration should not exceed 10 mmol/h
  -Potassium bicarbonate can be given in case of accompanied metabolic acidosis

Not Urgent

- **Oral (PO) Potassium chloride**

Hyperkalemia (>5.5 mmol/L)

Causes

Na/K ATPase dysfunction:

- Beta Blocker
- Digoxin
- ↓ Insulin
- Aspirin

Massive cell breakdown
Rhabdomyolysis
Tumor lysis syndrome

Aldosterone axis dysfunction
Adrenal deficiency (e.g: Addison's disease), Aldosterone resistance

Clinical manifestations

- **Progressive muscular weakness**
  but sometimes there are no symptoms until cardiac arrest occurs
- **ECG changes** not always present
  Peaking of the **T wave**
  widening of the **QRS complex**
  prolonged **PR interval**

The most urgent test in severe hyperkalemia is **ECG**

Treatment

ECG abnormality ?

- **-ve**
- **+ve**

+ve

Ca gluconate
Only protect the heart , does not lower K^+ level

ECG become normal ?

-ve

Push K into cells: **Insulin**, Beta agonists.

Remove the K load:
Through the kidney: diuretics (loop),
Through the gut: Laxatives, K chelation (Ca resonium)

Refractory ?

- **Dialysis**

K-Ca imbalance

Done by Abdullah Alanzan

K-Ca imbalance

- **Calcium Disorder**
  - Hypocalcaemia
    - **Clinical manifestations**
      - Chvostek's sign
      - Trousseau's sign
      - **Prolonged QT interval.**
      - Seizure (In severe)
    - **Differential diagnosis /Etiology**
      - Decreased intake or Malabsorption
      - ↓ PTH or ↓ **Vitamin D**
      - Loop diuretics
      - **Hungry bone syndrome (Post parathyroidectomy)**
    - **Check Phosphate level (PO_4)**
      - **High (PO_4)**: CKD (↑ PTH); Hypoparathyroidism (↓PTH)
      - **Low (PO_4)**: Vitamin D deficiency
  - Hypercalcaemia
    - **Clinical manifestations**
      - **Renal "Stones"**: Nephrolithiasis, Nephrocalcinosis.
      - **Skeleton "Bones"** : **Bone pains.**
      - **Abdominal "Moans"** : Nausea, vomiting, Constipation, Pancreatitis.
      - **CNS** : Impaired concentration and memory, **Seizure**
      - **CVS**: **ECG (shortened QT interval).**
    - **Parathyroid Hormone (PTH)**
      - **High PTH (Dependent)**
        - **Check Phosphate level (PO_4)**
          - **High (PO_4) + Renal Impairment**: Suggest tertiary hyperparathyroidism
          - **Low (PO_4)**: Primary hyperparathyroidism; Multiple endocrine neoplasia.
      - **Low PTH (Independent)**: Increased intake; Increased Vitamin D; **Thiazide diuretics**; **Osteoclastic bone metastasis.**; immobilization
- **Potassium Disorder**

Glomerular diseases

Done by Ghada Aljedaie

Renal failure
Hypertension
Edema
proteinuria

urine dipstick

- ≥ **3+** proteins → **Urine analysis**
  - **-Heavy proteinuria > 3.5 g/day***
    -Lipiduria *3.5g = 3500 mg
    -fatty casts with Maltese Cross appearance
    -Renal tubular epithelial cell casts
  - **Blood tests**
    - ↑Cr and/or ↑BUN ,↓Na
      ↓total protein,
      ↓albumin (< 30 g/L)
      Hyperlipidemia (↑LDL↑TG)
    - **Nephrotic Syndrome**
- Hematuria → **Urine analysis**
  - **-Hematuria**
    -Dysmorphic RBCs (Acanthocytes)
    -Red blood cell casts
    -Mild to moderate proteinuria of > 150 mg/24 h but < 3.5 g/24 h
    -Sterile pyuria +/- WBC casts
  - **Blood tests**
    - ↑ Creatinine,
      ↓ GFR
      Azotemia with↑ BUN
    - **Nephritic Syndrome**

Membranoproliferative GN (MPGN)
It may present with Nephritic picture or Nephrotic syndrome

Biopsy to confirm the type
Light and Electron microscopy, IF

	Diagnosis/Management :	Associate with:
LM: Diffuse thickening of the glomerular capillary **EM:** Foot process effacement ,subendothelial immunoglobulin deposits (spike and dome appearance) **IF:** deposits of (IgG and C3)	**Membranous nephropathy** Tx:Corticosteroids + Cyclophosphamide	**Idiopathic:** Associated with PLA2R autoantibodies **Caucasian adults** **Drugs:** Penicillamine, NSAIDs, **heavy metals** **Hepatitis** B,C virus **Malignancy:** prostate, lung, GI, breast and lymphoma. (imp to screen) **SLE**
LM: segmental sclerosis with hyalinosis **EM:** diffuse Foot process effacement	**Focal Segmental Glomerulosclerosis** Tx: 1st line: corticosteroids 2ed: cyclosporine or tacrolimus	West African adults previous focal glomerular injury **HIV infection** Heroin misuse Morbid obesity Chronic hypertension Sickle cell disease
LM: normal glomeruli **EM:** only diffuse foot processes effacement	**Minimal Change Disease** Tx: 1st line:corticosteroids 2ed: cyclosporine or Cyclophosphamide	90% **children** biopsy not commonly done Allergies Drugs, most commonly NSAIDs **Haematological malignancies:** hodgkin lymphoma,non hodgkin lymphoma and leukemia
LM: Thickening of the glomerular basement membrane (increased permeability) Eosinophilic nodular glomerulosclerosis (Kimmelstiel-Wilson nodules) , Mesangial matrix expansion	**Diabetic nephropathy** Tx: **1st line: ACEI or ARBS** 2ed:diuretics or CCB to control HTN Dietary modification	**General treatment of Nephrotic syndrome:** ● Dietary sodium restriction and a loop diuretic ● Prophylactic anticoagulation ● Pneumococcal vaccine ● HMG-CoA reductase inhibitor ● ACEI and/or ARB for any patients with proteinuria **Treatment of Secondary causes** ● Secondary causes treated by targeting the cause ● In FSGS given ACE with treatment of cause
LM: Mesangial proliferation, Subendothelial and/or subepithelial immune complex deposition Thickening of the capillary walls (appear as wire loops) Congo red stain: amyloid deposition in the mesangium showing **apple-green birefringence** under polarized light Nodular glomerulosclerosis **EM:** amyloid fibrils	**Amyloid nephropathy** Treatment of underlying disease (e.g., bone marrow transplantation may be used for multiple myeloma)	

Glomerular diseases

Done by Ghada Aljedaie

Renal failure
Hypertension
Edema
proteinuria

urine dipstick

≥ 3+ proteins → **Urine analysis**

-**Heavy proteinuria (> 3.5 g/day)** *3.5g = 3500 mg
-Lipiduria
-fatty casts with Maltese Cross appearance
-Renal tubular epithelial cell casts

Blood tests

↑Cr and/or ↑BUN ,↓Na
↓total protein,↓**albumin** (< 30 g/L)
Hyperlipidemia (↑LDL ↑TG)

Nephrotic Syndrome

Membranoproliferative GN (MPGN)
It may present with Nephritic picture or Nephrotic syndrome

Hematuria → **Urine analysis**

-**Hematuria**
Dysmorphic RBCs (Acanthocytes)
-Red blood cell casts
-Mild to moderate proteinuria of > 150 mg/24 h but < 3.5 g/24 h
-Sterile pyuria +/- WBC casts

Blood tests

↑ Creatinine,
↓ GFR
Azotemia with↑ BUN

Nephritic Syndrome

1-Serological test. 2-Biopsy:
\+ Complement,PR2,MPO, ANCA, and anti-GBM AB

+ glomerular **immune complex** IF: **granular** Appearance

↓ Serum C3 (Compliments mediated)

+*ANA* > **SLE**
+*ASO* > **PSGN****
+ *blood culture*> **IE**
+ *Cryoglobulin* > **Cryoglobulinemia MPGN**

Pt present with cola like hematuria usually after **1 to 3 weeks from the start of infection

Treatment

- **SLE:** depends on the findings in renal biopsy It usually involves high degree of immunosuppressive.
- **PSGN:** supportive (wait & see)

Normal C3 (Antibody mediated)

+ IgA immunostain

post URT infection*

IgA nephropathy (Berger)
*Pt present with hematuria **<1** week of Clinical URT infection

+ skin rash +arthralgia

HSP (It's a Systemic vasculitis)

Treatment

- Most important treatment is to control the blood pressure which also decreases the proteinuria.
- ACEi or ARB For proteinuria.
- immunosuppressive only in severe cases

+ Circulating **anti-GBM** IF: **liner** deposit of IgG & C3

No Lung involvement → anti-GBM GN
+Lung involvement → Goodpasture GN

Treatment

- Plasmapheresis + corticosteroids & cyclophosphamide

+ Circulating **ANCA** IF: Negative (**Paucity** of staining)

immunofluorescence

+ MPO
+ p-ANCA

+Granulomas → Microscopic polyangitis PAN
+Granulomas
+Asthma
+Eosinophilia → Chugr-strauss syndrome

+ PR3
+ c-ANCA → Granulomatous Polyangiitis (Wegener)

▶ **TIP** When asked about the "**best test**" for Wegener, lung biopsy is better than renal biopsy with sinus biopsy being the least accurate. When all 3 are in the choices, choose lung biopsy.

	Microscopic polyangitis PAN	Chugr-strauss syndrome	Granulomatous Polyangiitis (Wegener)
Associate with:	• Usually only mild respiratory symptoms • Can cause pauci-immune RPGN	• Asthma • Allergic rhinitis • Purpura • Peripheral neuropathy • Can cause pauci-immune RPGN	• **Pulmonary and nasopharyngeal involvement is common** • May manifest with hemoptysis & nasal ulcers. • Can cause pauci-immune RPGN
Treatment	• glucocorticoids and cyclophosphamide		• high dose corticosteroids & cyclophosphamide > fail > Rituximab

Hypertension

Done by Omar Alhalabi

- There are more than 8 guidelines for the diagnosis and management of hypertension. We are going to use the one mentioned by the doctor in the lecture

Values:

BP category	Systolic mmHg	and/or	Diastolic mmHg
Normal	Less than 120	and	Less than 80
Elevated	120 - 129	and	Less than 80
★ Stage 1 HTN	130 - 139	or	80 - 89
Stage 2 HTN	140 - or Higher	or	90 or Higher

Diagnosis:

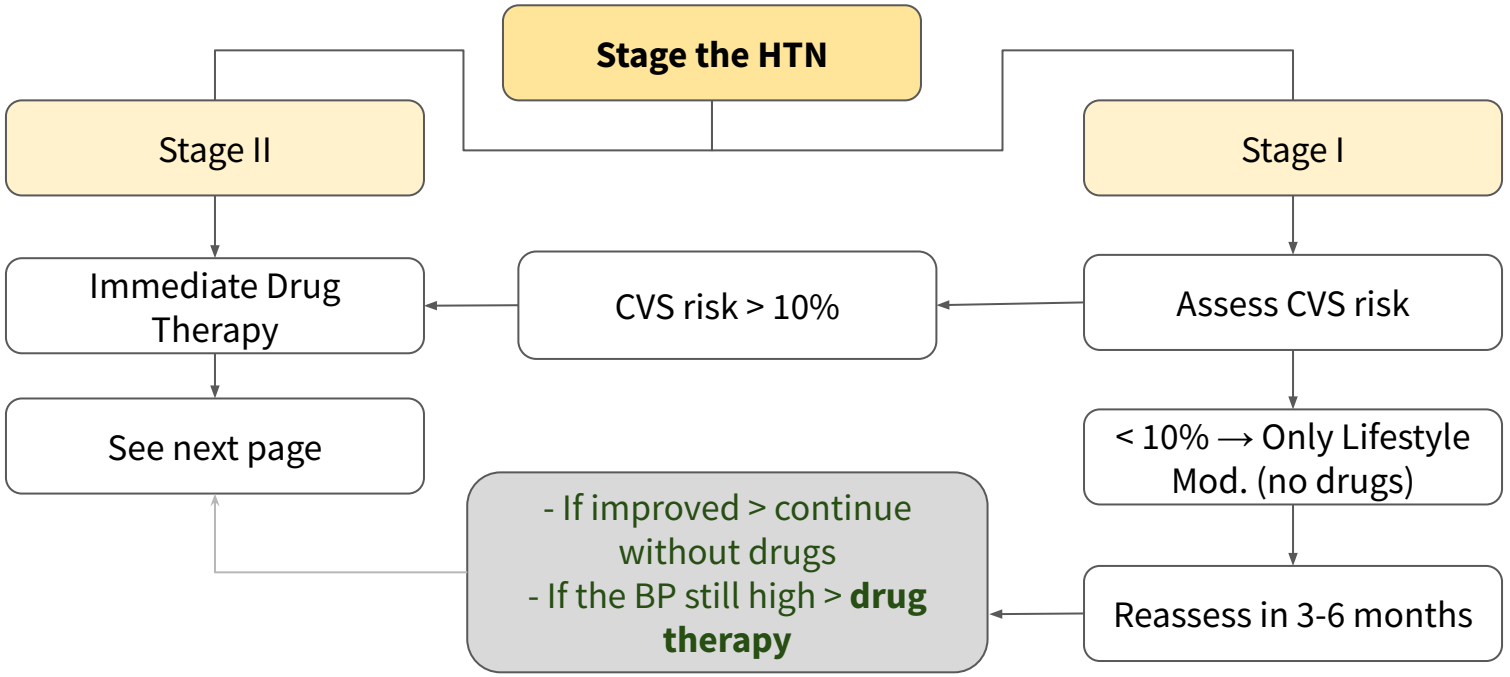
Office (Best) : take 3 measures and take the average of the least two measures for 2-3 visits. If the average is > 135 mmHg → Confirm Dx

Home or ambulatory : if the average is > 135 mmHg → Confirm Dx

Q:What is the Only case in which you shouldn't use Automatic BP measuring device and you should use the manual one with stethoscope? AF

Management

1. **Exclude secondary causes**
2. **Lifestyle modification is always recommended**



Hypertension

Done by Omar Alhalabi

Causes of HTN

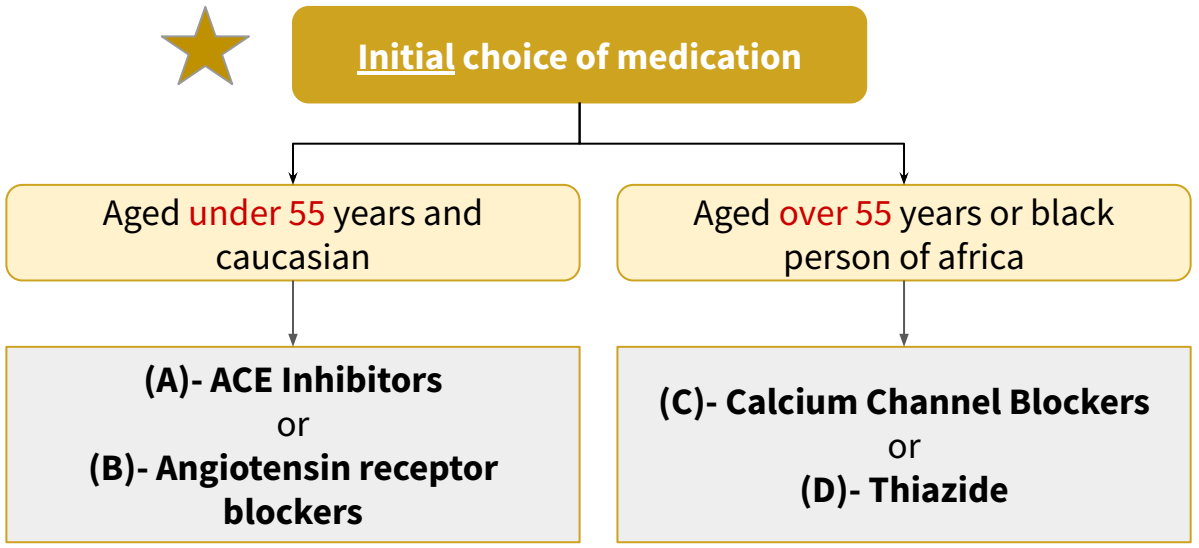
1. **Primary: Idiopathic**
2. **2ndary:**
   a. Renal artery stenosis and many other renal diseases
   b. OSA
   c. Pheochromocytoma
   d. Hyperaldosteronism
   e. Cushing syndrome
   f. Coarctation of the aorta…

Investigations after the diagnosis

1. **Routine:**
   a. ECG
   b. Urinalysis
   c. Electrolytes
   d. Blood glucose + Lipid profile

2. **Optional** (if suspecting 2ndary causes:
   a. CXR
   b. Renal angio. (renal artery sten.)
   c. Urinary catecholamines (Pheo.)
   d. Urinary cortisol (Cushing)

Drug Management:



- **Step 2: If BP > 20/10 mmHg above goal (140/90),** may start with **2** BP lowering medications → **A(B)+C or A(B)+D** (ONE PILL DUAL COMBINATION)
- **Step 3: A(B) + C + D**
- **DO NOT start with B-Blockers (Used as last option)** Unless indicated by co-morbid condition
- **DO NOT use ACEI and ARBs together**

BP target of less than 130/80 mmHg is recommended "To reduce risks of complications".

Hypertension

Done by Omar Alhalabi

Drug Management:

Management in special Conditions	Preferred therapy
Congestive heart failure	Thiazide, **ACEI(Best)** , Aldosterone antagonist, BB
Post Myocardial Infarction	BB, ACEi
Diabetes mellitus with proteinuria	ACEi, ARBs, NO
Diabetes mellitus without Proteinuria	Thiazide, CCB, ARBs, ACEi
Chronic kidney disease	ACEi, ARB, Thiazide
Stroke	CCB+ACEi
Benign prostatic hyperplasia	α antagonists e.g. prazosin, terazosin, doxazosin
Pregnancy	Hydralazine (vasodilator), nifedipine (CCB), Aldomet (methyldopa), labetalol **Oral labetalol** is first-line therapy during pregnancy. Second Line agents are methyldopa and nifedipine. **In breastfeeding,** ACE inhibitors, beta-blockers and nifedipine are safe. Methyldopa should be avoided because of the risk of depression.

Hypertensive crisis + urgency

Hypertensive crisis: BP ≥ 180 systolic or ≥ 120 diastolic with end organ damage

Hypertensive Urgency: same as crisis but without end organ damage

Management of HTN emergency: complex and depends on the affected organs. Generally, **First line is IV labetalol**, or sodium nitroprusside. The later is less preferred due to side effects. Click Here to see detailed management (we are not supposed to know the detailed management)

Chronic kidney failure

Done by Abdullah Alanzan

Chronic Kidney Failure
(presence of kidney disease for at least 3 months)

How to Identifying CKD ?

- Compare to baseline if known
- Presence of complications of CKD
- Kidney imaging changes (US)
- Biopsy features of chronicity

After confirming CKD : do **Cr clearance** to estimate GFR and urinalysis (+ve proteinuria may require ACEI or ARB therapy)

CKD Complication

Metabolic and electrolytes abnormalities

- Hyponatremia
- **Metabolic acidosis**
- Hyperkalemia

You need to rule out other causes

If confirmed due to CKD give .. **NaHCO3 IV/oral**

(target: serum bicarb > 22)

ECG abnormality ?

-ve

- **Insulin** + 50% dextrose
- **Beta agonists (salbutamol)**

+ve

- Ca gluconate

Mineral and Bone disorder (CKD- MBD)

- **Hyperphosphatemia**
- **Hypocalcemia**
- Secondary **hyperparathyroidism**
- Hypovitaminosis D

First correct **Hyperphosphatemia** by :

1. **Start by (Diet control) < 10 mg/kg/day**
2. **Phosphate binders :**
   - **Calcium carbonate** (Only If Ca+ is Low)
   - **Sevelamer**

Then correct **Hypocalcemia** by correcting **Vit D** and **PTH :**

- **Vitamin D (Calcitriol)** correction should not start until phosphate level is normal

PTH correction

If Ca + PO4 high → Cinacalcet

If Ca+ low → Vit D (Calcitriol)

Persistent ↑ PTH ?

Parathyroidectomy
Side effect : hypocalcemia

Cardiovascular abnormalities

- **Hypertension** (most common cause of 2ry HTN)
- Cardiomyopathy

First line of therapy is **RAAS blockade**

SBP should be lower than 120

Pericarditis → **intensive dialysis**

Hematologic abnormalities

- Normochromic anemia
- Platelet Dysfunction

Anemia found ?
Hb < 13 for males
Hb < 12 in females

Target **Hb/Hct**
Hb **10-12** (10-11.5) g/dL,
Hct 33-36%

Do all Anemia work up before labeling them as Anemia of CKD

Anemia of CKD confirmed ?

First step to achieve the target is :

Tackle **iron deficiency** by supplementing Oral iron

Target T-Sat > 0.3.
Target S. ferritin > 500 ng/ml.

iron targets achieved and anemia persisted ?

Recombinant Erythropoietin **(EPO)**
(e.g. darbepoetin, epoetin)

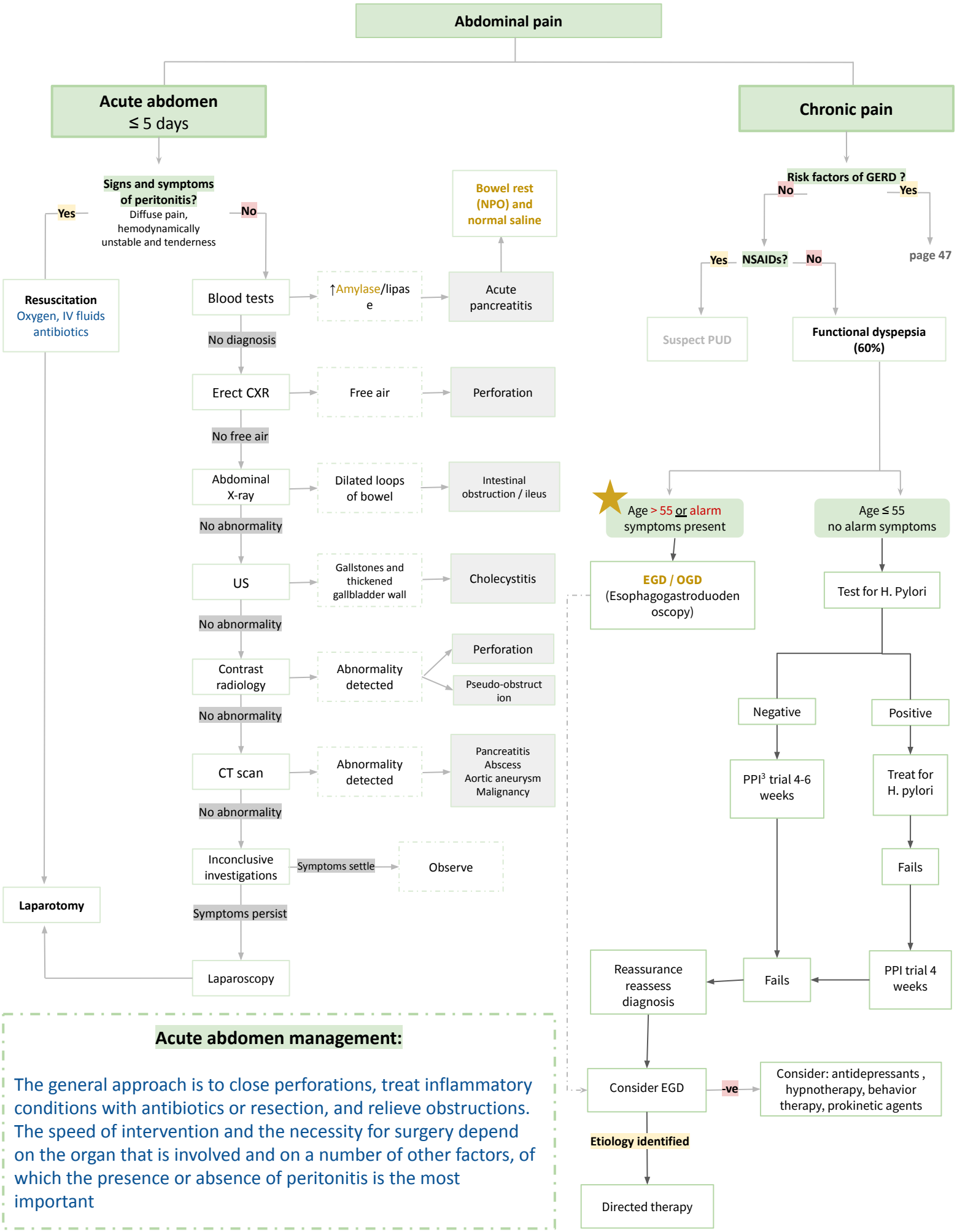
Gastroenterology

Resources:

- Davidson’s principle and practice of medicine latest edition, 2018
- Kumar and clark’s Clinical medicine latest edition, 2021
- Current medical diagnosis and treatment, textbook, 2022
- Harrison’s principles of internal medicine, latest Ed. 2018
- Doctors’ lectures and notes
- Master the boards, 2021 edition
- American Journal of gastroenterology
- AMBOSS
- American gastroinsteninalogical association

Abdominal pain

Done by Ghada Alabdi

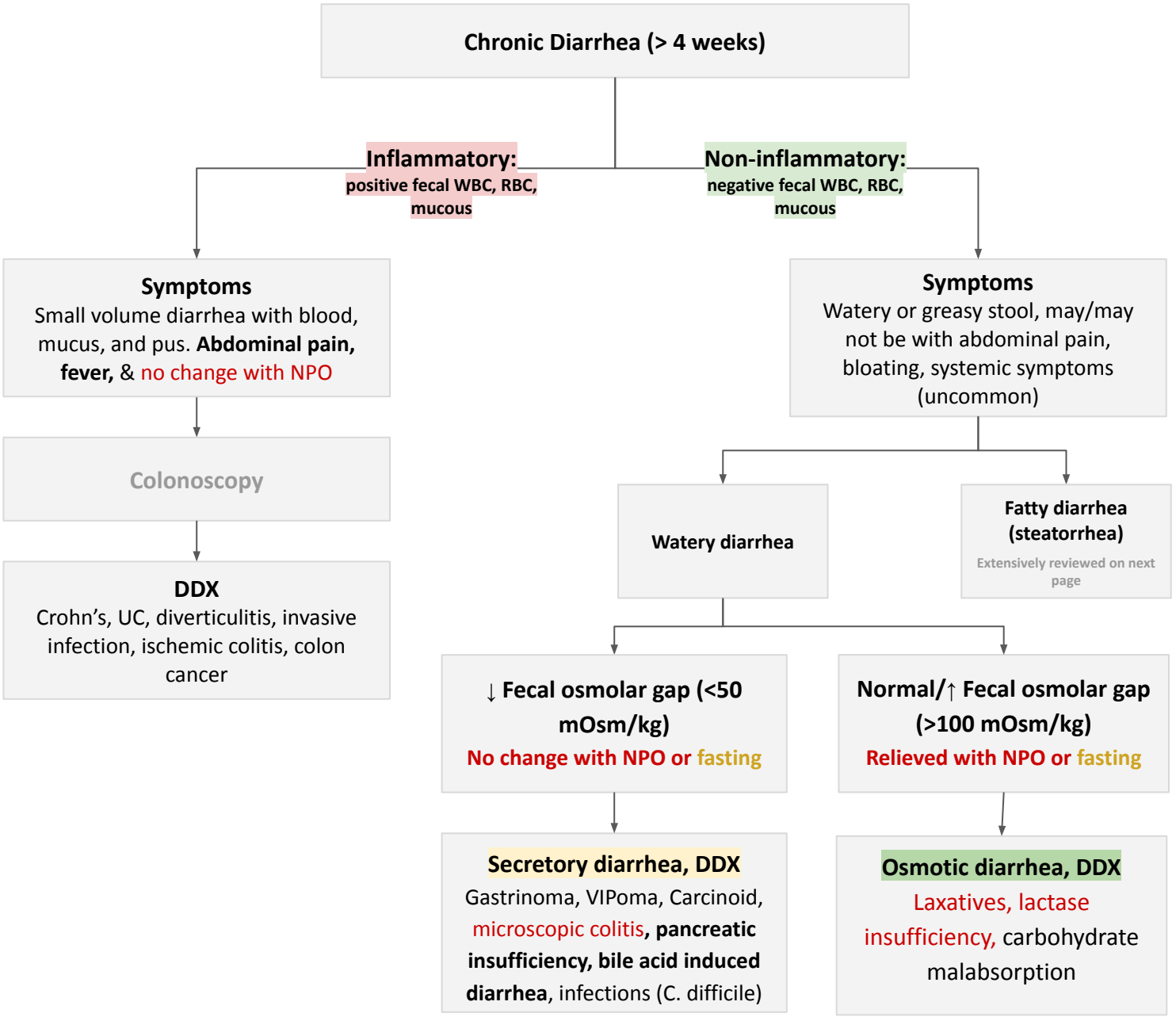


Acute abdomen management:

The general approach is to close perforations, treat inflammatory conditions with antibiotics or resection, and relieve obstructions. The speed of intervention and the necessity for surgery depend on the organ that is involved and on a number of other factors, of which the presence or absence of peritonitis is the most important

Chronic diarrhea

Done by Sarah Alobaid



The workup for diarrhea includes a detailed patient history, physical examination, and laboratory tests to assess severe cases.

Laboratory Tests:

- **CBC**: to show anemia or leukocytosis
- **Chemistry screen:** fluid/electrolyte status, nutritional status, serum protein/globulin
- **Stool analysis:**
  - **Fecal leukocytes** (or marker for neutrophils: lactoferrin or calprotectin) and **fecal occult blood**
  - **Stool electrolytes** (fecal osmolar gap) and **stool pH** (for carbohydrate malabsorption)
  - **Fecal fat test** (48h or 72h quantitative or Sudan stain) and **stool weight**
  - **Laxative screen** (if positive, repeat before approaching pt)
- **Stool culture** (more useful only for acute), An ova and parasite (O&P) exam, **Giardia Ag, C diff,** Coccidia, Microsporidia, Cryptosporidiosis.

Imaging:

- Small bowel series
- CT/MRI or CT/MR enterography.
- To identify the cause/confirm it:
  - **Endoscopy** with small bowel biopsy and aspirate for quantitative culture. (Esp. for celiac disease)
  - **Colonoscopy**, including random biopsies. (Esp. for IBD)

Chronic diarrhea

Done by Sarah Alobaid

Chronic diarrhea, steatorrhea, abdominal bloating, +/- weight loss, vitamin (A,K,E,D) deficiency

Other: iron & B12 deficiency, normal/high folate, deconjugated bile acid

D-xylose absorption test
(requires intact intestinal mucosa)

Not absorbed | **Absorbed**

Serology (Initial test)

Anti-TTG (IgA), Anti-gliadin (IgG) in case of IgA def. patients

in case of positive/negative serology:
Endoscopy w/ biopsy (gold standard)

Foamy, (PAS)–positive macrophages in the lamina propria containing non–acid-fast gram-positive bacilli (organism: Tropheryma whipplei)

Dx: Whipple disease

- Intraepithelial lymphocytic infiltration
- crypt hyperplasia, villous atrophy
- scalloping of mucosa, ulceration and masses

Dx: Celiac disease (Most common)
Associated with **dermatitis herpetiformis**
(itchy rash over elbows, knees, shoulders, scalp)

Exocrine Pancreatic Insufficiency
Sx: steatorrhea, abdominal pain, high alcohol intake
Dx & Tx: give pancreatic enzymes (therapeutic and diagnostic)

Endoscopy w/ biopsy

Consider Bile Salt Malabsorption
Cause: idiopathic or as a complication of bowel resection (common scenario: pt with crohn's who had ileal resection
Dx: ↑ serum 7α-hydroxycholestenone, SeHCAT scan
Tx: cholestyramine (bile acid sequestrants)

Jejunal culture and biopsy (gold standard) → >10^4 microbe conc.

Schilling's test (to distinguish between malabsorption causes)

Therapeutic antibiotic trail

Dx: Small Bowel Bacterial Overgrowth

Celiac Disease

Sx: in addition to **anemia (iron deficiency anemia** or macrocytic anemia as it associated with hyposplenism**)**, osteoporosis
Associated with: T cell lymphoma, small bowel adenocarcinoma
At risk of celiac: IgA deficiency (2-3%), Autoimmune thyroid disease (6-8%), Type 1 DM (3-6%), Connective tissue disease, Down's syndrome.
Tx: education (1st), gluten-free diet, correction of nutritional deficiencies

Tropical Sprue

Sx: in addition to **both** B12 & folate deficiency, anorexia, cramps, not improved with gluten-free diet
Dx: Jejunal aspirate and biopsy→crypt hyperplasia, villous atrophy
Tx: tetracycline for 3-6m, folate & B12 supplements
Note: similar **symptoms and endoscopy presentation to celiac**, but only tropical sprue responds to antibiotics

Whipple Disease

Caused by bacterial and macrophage-predominant inflammatory cell infiltration of small intestinal mucosa and **obstruction of mesenteric lymph nodes.**
Sx: abdominal pain, polyarthralgia, cardiac and neurological symptoms, lymphadenopathy, fever
Tx: IV Ceftriaxone daily for 2 weeks THEN oral TMP/SMX for 1 year

Small Bowel Bacterial Overgrowth (SBBS)

Sx: B12 deficiency and elevated/normal folate **(unlike tropical sprue),** neuropathy, abdominal pain, hypoproteinemia, hypoalbuminemia
Tx: treat underlying cause, replace fat soluble vitamins, 7 -10 days of Abx (metronidazole, tetracycline)

Differential Results of The Schilling Tests in Several Diseases Associated With Cobalamin Malabsorption

Cause of Fat Malabsorption	^{58}Co-Labeld Cobalamin	With Intrinsic Factor	With Pancreatic Enzymes	After 5 Days of Antibiotics
Pernicious Anemia	Reduced	**Normal**	Reduced	Reduced
Chronic Pancreatitis	Reduced	Reduced	**Normal**	Reduced
Bacterial Overgrowth	Reduced	Reduced	Reduced	**Normal**
Ileal Disease	Reduced	Reduced	Reduced	Reduced

Gastrointestinal Bleeding

Done by Ghada Aljedaie

Suspected variceal haemorrhage

Start IV terlipressin and antibiotic prophylaxis

Urgent upper gastrointestinal endoscopy[2]
2 to 12 hrs[3]

Varices present?

Yes / No

Endoscopic therapy (variceal banding, sclerotherapy)

Stop terlipressin treat as for non variceal haemorrhage

Haemostasis?

Yes / No

-Continue **terlipressin to 72 hr**
-Introduce **beta blocker** as secondary prophylaxis
-Abx: ceftriaxone
-Enter patient into **endoscopic banding programme** to obliterate varices

Further endoscopic therapy or Balloon tamponade or **Emergency TIPSS***
*Transjugular intrahepatic portosystemic stent shunt

Suspected upper GI bleeding
hematemesis, melena, or select patients with hematochezia (i.e.,hemodynamic compare and risk factor for upper GI bleeding)

ABCDE Approach as needed
Protect airway, crystalloid fluids resuscitation, **blood** transfusion[1] when the patient is **actively bleeding**

Assess the risk by scoring system.
>0 ? Yes >> High risk

Start basic management
- Keep patient NPO
- CBC, type & screen,Coagulation profile & LFT
- **Suspected variceal haemorrhage**
- Anticoagulant reversal if INR >2.5
- **Conceder IV PPI** or octreotid

Table 1 | Glasgow–Blatchford score assessment criteria

Risk factors at presentation	Threshold	Score
Blood urea nitrogen (mmol/l)	6.5–7.9	2
	8.0–9.9	3
	10.0–24.9	4
	≥25.0	6
Hemoglobin for men (g/l)	120–130	1
	100–119	3
	<100	6
Hemoglobin for women (g/l)	100–120	1
	<100	6
Systolic blood pressure (mmHg)	100–109	1
	90–99	2
	<90	3
Heart rate (bpm)	>100	1
Melena	Present	1
Syncope	Present	2
Hepatic disease	Present	2
Cardiac failure	Present	2

Total score (0–23). Patients with scores >0 are considered to be at high risk. Permission obtained from Elsevier Ltd © Blatchford, O. *et al. Lancet*

Hemodynamically stable patient?

No / Yes

Unstable patient

EGD[2]
Endoscopic hemostasis if source of the bleeding is identified

Refractory hemodynamic instability

Consult interventional radiology and surgery

CTA +Angioembolization or surgery hemostasis
depending on individual evaluation

Stable patient

EGD[2]
2 to 24 h[3]
Source of the bleeding identified?

Yes

No

EDG negative Bowel prep possible

Colonoscopy
source of the bleeding of the identified?

Yes

No

Endoscopic treatment

Consider evaluation of a small bowel bleeding
e.g., Push Enteroscopy, Meckel scan, capsule Enteroscopy, Angiography

IV PPI

Treat underlying cause after hemostasis
peptic ulcer disease, esophageal or gastric cancer, diverticular disease, IBD

1. 2-4 units of blood should be cross-matched if a significant bleed is suspected:
   - PRBC: if Hb level <7 g/dl or <10 g/dl in patients with preexisting CVD
   - Fresh frozen plasma: if INR is elevated
2. Provide erythromycin I.V 30 minutes prior to endoscopy & High-dose I.V PPI
3. Delay endoscopy in case of ACS or suspected perforation

Liver Cirrhosis & its Complications

Done by Ghada Alabdi

◀ History:

Presenting symptoms	• **Asymptomatic** mainly. • Nonspecific constitutional symptoms, such as fatigue, weakness, and weight loss. • Symptoms of decompensation : Abdominal distension, Coffee-ground vomitus and black stool (**melena**) secondary to GI hemorrhage, Altered mental status in hepatic Encephalopathy, Lower extremity swelling, Jaundice, pruritus. • Hepatocellular carcinoma is the only complication that can happen even with compensated liver cirrhosis
Past and drug history	History of liver disease (all chronic liver disease can lead to cirrhosis), Surgery and dental, Metabolic syndrome, Drugs (Methotrexate, amiodarone, amoxicillin/clavulanate etc..).
Family history	Wilson Disease, Hemochromatosis, Apha1-antitrypsin deficiency, Viral hepatitis.
Social history	Risk-taking behaviors: IV drug use, sexual contact, and tattoos, Alcohol (amount, type & duration), Travel history.

◀ Clinical features

Hand and nails	Clubbing, Leukonychia, Palmar erythema, Bruising, Cholesterol deposits, Dupuytren contracture, Cyanosis: in patients with hepatopulmonary syndrome, Asterixis in hepatic encephalopathy
Chest wall features	Gynecomastia in men, Telangiectasia, Spider naevi.
Facial features	Muscle wasting, Telangiectasia, Bruising, Parotid gland swelling, Jaundiced sclera, Xanthelasma.
Abdominal features	Collaterals, Bruising, Hepatomegaly & Splenomegaly, Abdominal distension, Hepatic bruit, Loss of secondary Sexual hair, Testicular atrophy in men

◀ Investigations:

Liver Function Tests:	**ALT:**	**Moderately elevated** aminotransferases (often with an AST:ALT ratio >1)
	ALP:	Elevated (2 to 3 times the upper limit of normal).
	Others:	• Elevated GGT suggests alcohol consumption • Elevated ammonia may cause hepatic encephalopathy
CBC:	• **Thrombocytopenia, Leukopenia/neutropenia** and **Anemia.**	
Investigate the cause of cirrhosis	• Hepatitis: HbsAg, Anti-Hbs, Anti-Hbc, Anti-HCV • Wilson: Ceruloplasmin • A1ATD: serum levels of a1-antitrypsin • PSC and PBC: Cholestasis parameters • AIH: serum ASMA and AMA levels and hypergammaglobulinemia. (ASMA=Anti-Smooth Muscle Antibody)	
Radiological studies	**Mild-moderate disease**	• Surface nodularity and Hypertrophy of the caudate or the left lobes. • Increased echogenicity (ultrasound). "If ALP+ ALT+ AST were elevated and ASMA was negative, we have to do **Abdominal US**"
	Advanced disease	• Ascites. Splenomegaly , Portosystemic collateral, HCC.

Liver Cirrhosis & its Complications

Complications :

1. Ascites: Accumulation of fluid in the peritoneal cavity (most common)		
Investigation	Diagnostic paracentesis. **Routine:** 1. Cell count and differential 2. **Albumin** and total protein To measure SAAG.	
Management	1. **Dietary salt restriction.** 2. Diuretics (Spironolactone & Furosemide combination).	
★ **SAAG**	**High albumin gradient (SAAG>=1.1g\dL)**: Portal HTN is the most common cause • **Cirrhosis** / Alcoholic hepatitis. • Heart failure / Constrictive pericarditis.	**Low albumin gradient (SAAG<1.1g\dL):** • Peritoneal carcinomatosis • Lymphoma. • Nephrotic syndrome
Complications	**Spontaneous bacterial peritonitis:** Infection of ascitic fluid (**E.coli & Klebsiella**) **Diagnosis:** Ascitic fluid cell count→ **PMN count** (>250 cells/mm3) & a positive ascitic fluid culture **Treatment: Cefotaxime** + Albumin	

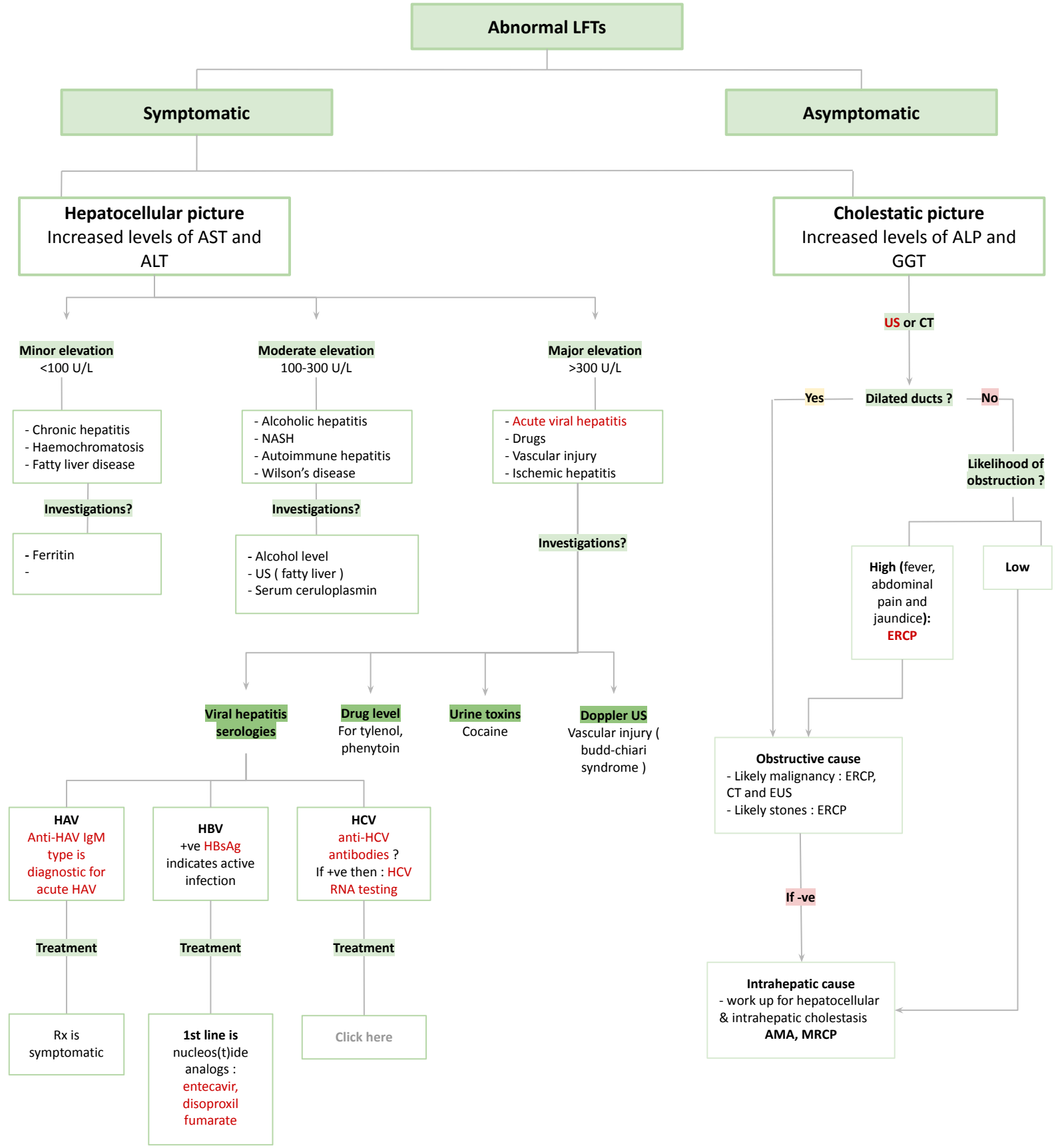
2. **Hepatic encephalopathy:** is a reversible brain dysfunction caused by liver insufficiency and PS shunts.	
Pathophysiology	Neurotoxin (ammonia) → Cross BBB → Activation of inhibitory neurotransmitter systems→ Impairment of excitatory neurotransmitter systems → Enhanced neural inhibition.
Clinical Features	(Flapping tremor).
Precipitants	Drugs, Increased ammonia, Dehydration, Portosystemic shunts, Vascular occlusion, HCC.
Treatment	1. **Lactulose or lactitol** (decrease absorption of ammonia) 2. **Rifaximin** or metronidazole (decrease GI bacteria that produce ammonia).

3. Hepatocellular carcinoma (Hepatoma) HCC	
Investigation	• **Blood tests:** (Alpha Fetoprotein AFP). • **Radiology:** Dynamic CT and MRI (See tumor density with time after IV bolus contrast. Requires both arterial enhancement and washout) • **Biopsy.**

Other Complications of liver cirrhosis	
Portal hypertension	- Develops as a complication of cirrhosis. it is the beginning and requirement for most cirrhosis complications - Cirrhosis: causes at least 90% of cases of portal hypertension - Complications: Variceal bleeding, Renal failure, Iron deficiency anaemia, Ascites & hypersplenism, Hepatic encephalopathy, Congestive gastropathy
Hepatorenal syndrome	Development of functional **acute kidney injury** in a patient who usually has **advanced liver disease** either cirrhosis or alcoholic hepatitis. **Treated by** hemodialysis +/- liver transplant
Portopulmonary Syndrome:	The presence of pulmonary hypertension in the coexistent portal hypertension.
Hepatic Hydrothorax:	Pleural effusion in a patient with cirrhosis and no evidence of cardiopulmonary disease.
Hepatopulmonary syndrome (HPS):	**Triad of:** Liver disease , Increased alveolar-arterial gradient , Evidence for intrapulmonary vascular abnormalities. **Treated by** O2 supportive therapy + liver transplant

Abnormal Liver Enzymes

Done by Ghada Alabdi



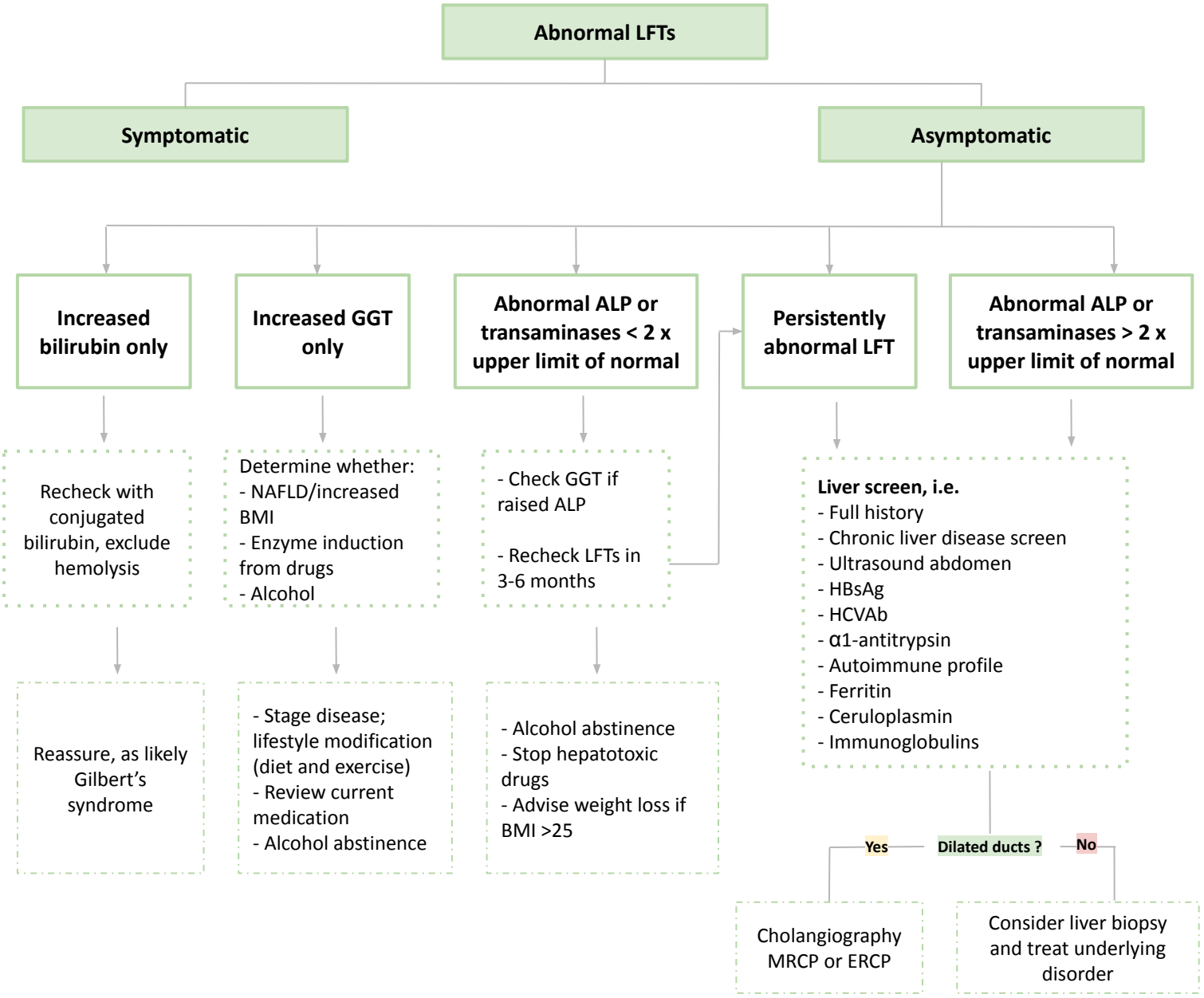
If all tests were -ve/ couldn't identify the cause then take a liver biopsy

AST & ALT normal range: 0-35 U/L.

ALP normal range: 36-92 U/L.

Abnormal Liver Enzymes

Done by Ghada Alabdi



Clinical situation
Action
Management

- Isolated **ALP** increase with normal AST, ALT & GGT = pregnancy or bone disease

Fatty Liver

Done by Naif Alsulais

NAFLD: Liver disease, where there is accumulation of excess fat in the liver cells, in people who drink little or no alcohol

Metabolic-Associated Fatty Liver Disease (MAFLD): the presence of hepatic steatosis together with one or more of the following:

- Overweight or obesity
- Type 2 diabetes
- Two or more other metabolic risk abnormalities

Suspected NAFLD

- Most are asymptomatic, commonly identified as an incidental:
- patients with progressive NASH may present late in the natural history of the disease with complications of cirrhosis and portal hypertension, such as variceal haemorrhage, or with hepatocellular carcinoma.
- Identified by risk factors (NAFLD is considered the hepatic manifestation of the metabolic syndrome):
  - Insulin resistance
  - Obesity, diabetes, dyslipidemia
  - Male
  - Medications (e.g Tamoxifen)
  - sedentary lifestyle
  - western diet
  - Hypertension
  - Ethnicity (Asians, hispanics)

Best initial: Ultrasound & LFT

Enlarged liver & Increase echogenicity (bright liver) on ultrasound

LFT normal in most cases, Raising Liver enzymes indicates NASH (Elevated bilirubin, AST, ALT, AP, GGT)

More A**L**T than AST (AST/ALT) if the **L**iver is infiltrated with **L**ipids

reversed as it progress into cirrhosis (AST more than ALT) and the fat reduced

Further investigations:

- CT (hypodense), MRI, MR spectroscopy
- Liver biopsy (GOLD standard)
- Liver elasticity (e.g fibroscan). Degree of fibrosis is the most important factor in prognosis

Criteria

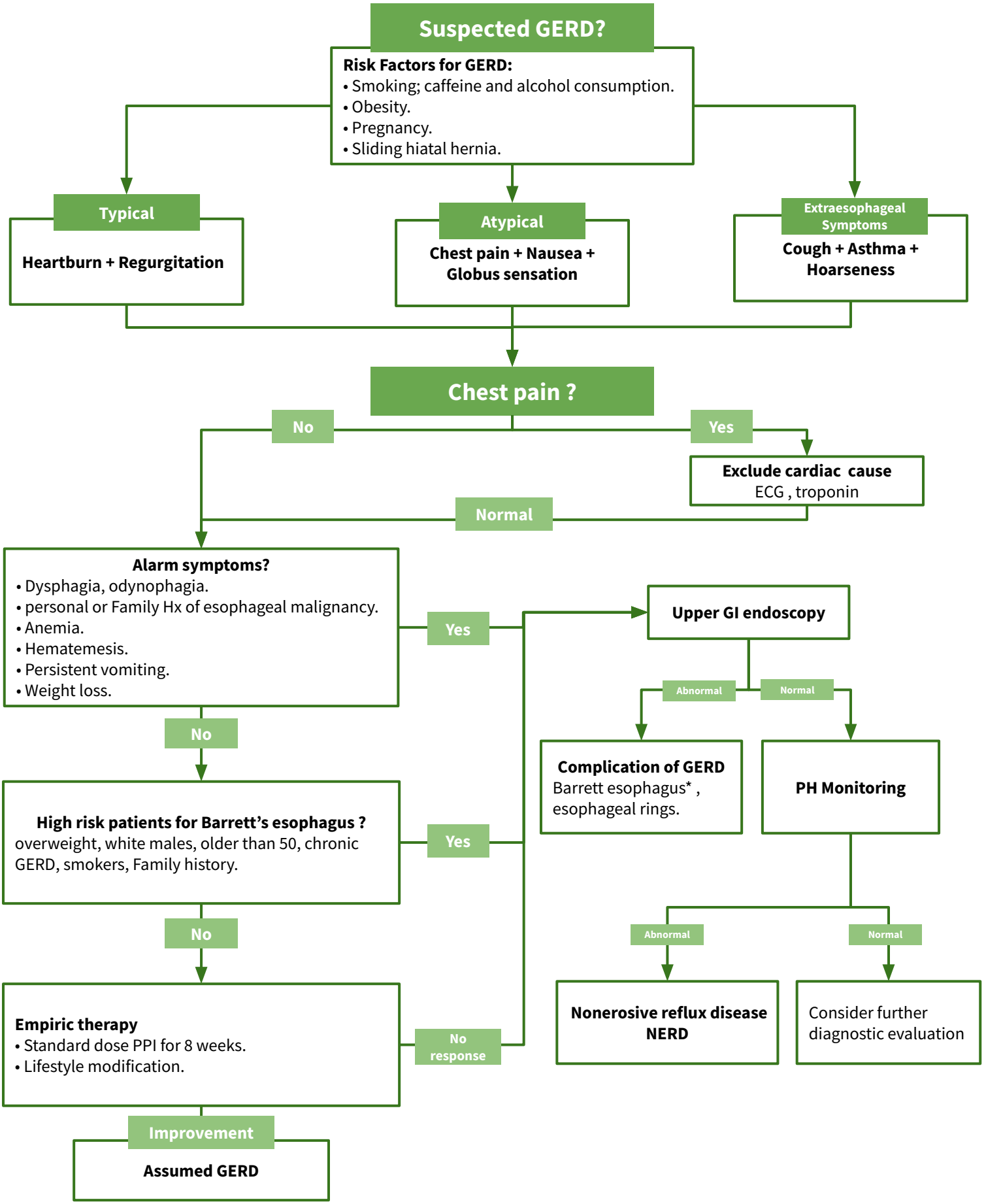
- **Liver fat > 5%:** Estimated by
  - Cross-section on histology
  - Non-invasively by MRI (more sensitive)
- **Lack of secondary causes of hepatic fat accumulation, such as:**
  - Significant alcohol consumption (daily alcohol consumption >30g for men and >20 g for women)
  - Long-term use of a steatogenic medications
  - Monogenic hereditary disorders

Management

- **Lifestyle modifications:** Cornerstone management
  - Weight loss most important: ≥ 5% improves steatosis, ≥ 7% improves NASH & ≥ 10% regress Fibrosis
  - Dietary modification (beneficial without weight loss): Low glycemic food & Avoid of high fructose containing food.
  - Exercise: Aerobic & Resistance activity independently
- **Medication:**
  - Diabetics: Pioglitazone or Liraglutide
  - Non-diabetics: Pioglitazone or Vit E
- **Surgery:**
  - Bariatric Surgery: should be avoided in those with advanced cirrhosis and portal hypertension

Esophageal diseases

Done by Raghad Albarrak

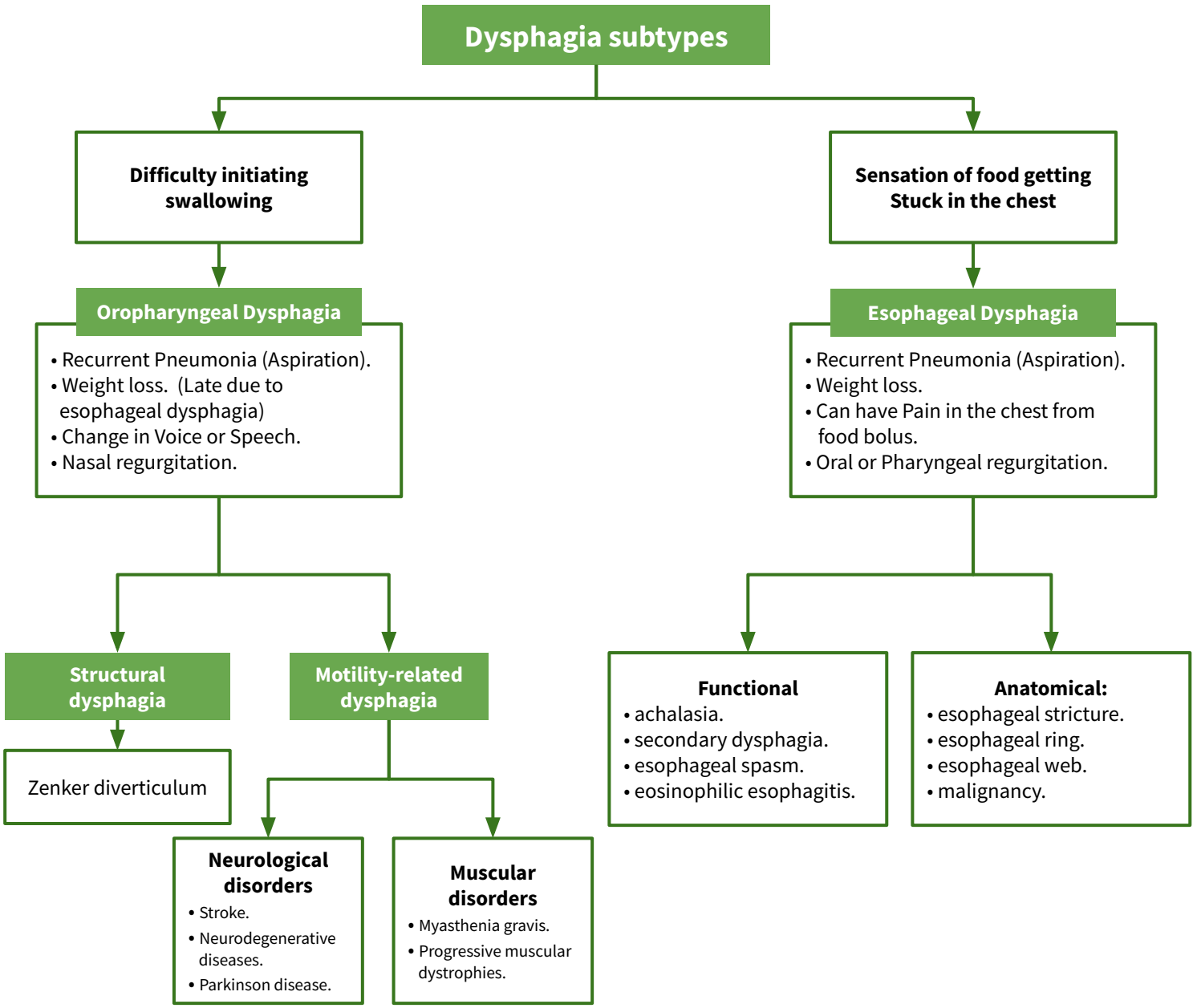


*: Metaplastic columnar epithelium (gastric and intestinal) replaces the stratified squamous epithelium. It is the most likely risk factor of Adenocarcinoma.

- Management of Barrett esophagus: - low-grade dysplasia > give high dose PPI with OGD in six months.
  - highly grade with no visible mass > give high dose PPI with OGD in three months.
  - highly grade with visible mass > do esophageal mucosal resection.

Esophageal diseases

Done by Raghad Albarrak

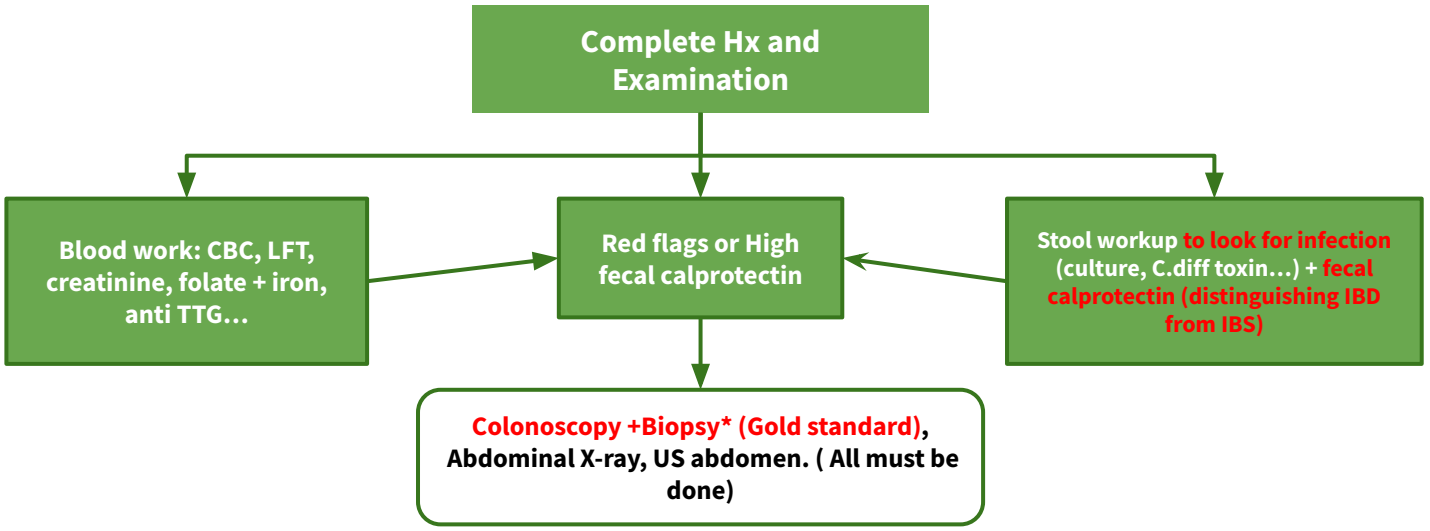


*: Dysphagia predominantly with solid (or initially to solids that progressed to liquids) food should raise suspicion for an underlying structural disorder, including malignancy. Dysphagia predominantly with liquids (or liquids and solid food) is suggestive of an esophageal motility disorder.

Inflammatory Bowel Disease

Done by Omar Alhalabi

	Ulcerative Colitis	Crohn's disease
Wall involvement	**Mucosal**, submucosal ulcers	**Full-thickness** inflammation with knife-like **fissures**
Location	Begins in **rectum** and can extend proximally up to cecum	**Anywhere** from mouth to anus with **skip lesions** **Terminal ileum** is the most common site
Symptoms	**Left** lower quadrant pain (rectum) with **bloody diarrhea**	**Right** lower quadrant pain (ileum) with **non-bloody diarrhea**
Inflammation	**Crypt abscess** with neutrophils	Lymphoid aggregates with **Granulomas**
Gross appearance	Pseudopolyps	**Cobblestone mucosa**, **creeping fat** and strictures
Complications	Toxic megacolon, Carcinoma	Malabsorption with nutritional deficiency, Calcium oxalate nephrolithiasis, fistula formation and Carcinoma
Smoking	Protects against UC	Increases risk
Positive antibody	pANCA	ASCA

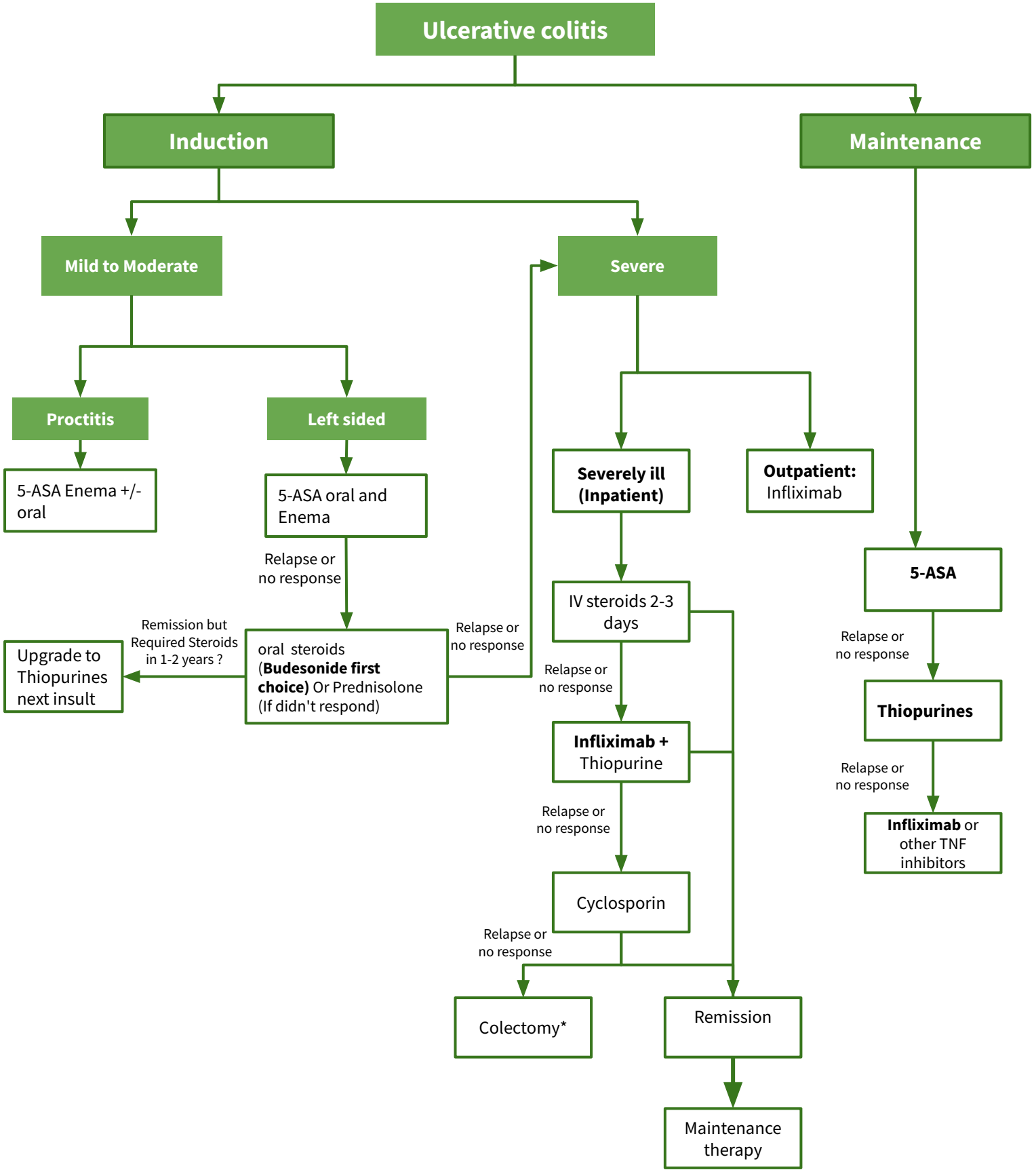


Very Important Notes:

- **You always have to rule out infection (even in a previously diagnosed with IBD)**
- **IBD is hard to diagnose, you will at least need all the previously mentioned tests to hopefully reach a diagnosis**
- *** In those who are suspected to have crohn's, and if you think from Hx and examination that it involved an upper GI segment that colonoscopy can not reach, you have to look for the upper GI by the following method:**
  - Upper GI endoscopy: if oral or oesophageal complications are suspected
  - Small Bowel imaging: Is ALWAYS mandatory in suspected crohn's pts, Technique:
    - MRI enterography (Best initial if available)
    - Other methods, such as Barium follow through, CT with contrast… depending on the availability
    - If radiology was not conclusive → capsule Endoscopy of small bowel

Inflammatory Bowel Disease

Done by Omar Alhalabi



- *Indications for surgery:
  - Failure of medical treatment
  - Complications eg: toxic megacolon, severe hemorrhage, perforation & acute dilatation
  - Dysplasia on endoscope

Inflammatory Bowel Disease

Done by Omar Alhalabi

Crohn's disease

Induction

- Oral or IV corticosteroids
  - Ileal? Budesonide. No response within 2 weeks? Switch to Prednisolone
  - Colonic? Prednisolone + Ca & Vit D

No response?

- Anti TNF (infliximab)

Maintenance

- Azathioprine or Mercaptopurine or methotrexate

Relapse?

- Anti TNF (infliximab)

Perianal disease

- Surgical drainage (first)
- Ciprofloxacin +Metronidazole + Azathioprine + Anti TNF (infliximab)

- Indications for surgery:
  - Failure of medical therapy
  - Complications eg; toxic megacolon, obstruction, perforation, abscess or fistulas
  - Failure to thrive in children