ACS	Prinzmetal Angina	Stable angina	Unstable Angina & NSTEMI	STEMI
Initial	Risk factors modification.	**Risk factors modification:** (Smoking cessation, controlling HTN, DM, hyperlipidemia and obesity, diet and exercise)	**Acute treatment:** -Hospitalization -**Nitrates** -Oxygen supplement "To improve oxygen supply" -Morphine -Replace deficient electrolytes **After acute treatment:** **Reduce risk factors**: (Smoking cessation, weight loss and control DM, HTN and hyperlipidemia)	-Hospitalization Cardiac monitor (CCU) -**Nitrates** (Nitroglycerine) "Coronary vasodilators > Open artery" -Oxygen supplement "To improve oxygen supply" -Morphine
Medical	**Calcium channel blockers**, Nitrate	Aspirin "reduces morbidity & mortality", **Beta-blockers**, Nitrates, **Calcium channel blocker**	-Aspirin -Clopidogrel OR Prasugrel OR Ticagrelor -**Beta-blockers** (Atenolol OR Metoprolol) -**Glycoprotein IIb/IIIa inhibitors** (Abciximab OR Tirofiban) -Heparin OR Enoxaparin "**Anti-coagulant** > prevent progression of thrombus (decrease mortality)" **After acute treatment:** Continue Aspirin, **Beta blockers** and **Nitrates**.	-Aspirin "**Anti-platelets** > Reduces coronary reocclusion (Reduces mortality)" -Beta-blockers (Atenolol OR Metoprolol) "Block sympathetic stimulation > Reduces cardiac demand (Reduces mortality)" -**ACE inhibitors** (Enalapril OR Lisinopril) "**Anti-hypertensive** (Reduces mortality)" -**Statins** "**Anti-hyperlipidemic** > Lower cholesterol and stabilizes plaque (Reduce risk of further events)" -Clopidogrel "**Anti-platelets** (In pts undergo PCI)" -Heparin

Invasive		**1-Percutaneous coronary intervention PCI (angioplasty):** when Two or one vessels occluded. 2- **Coronary Artery Bypass Graft:** when 3 vessels occluded OR 2 vessels w/diabetes OR Left main artery	-Coronary angiography & Cardiac catheterization/Revascularization -**Percutaneous Coronary Intervention (PCI**) -90% of patients improve with medical regimen and don't need invasive management. Only if ischemia persist after 48 hours, then proceed directly to invasive procedures.	Thrombolysis (fibrinolytics) **only in STEMI** (fibrin specific: Alteplase, tPA or non fibrin specific: Streptokinase) (Greater benefit in anterior infarctions) **Percutaneous Coronary Intervention (PCI)**<90 min (Improve short/long-term outcomes) 1-One vessel occlusion 2-two vessel occlusion 3- no improvement with drugs **Coronary Artery Bypass Grafting (CABG):** 1-three- vessel occlusion 2-left main coronary artery occlusion 3-left ventricular dysfunction Only if complications OR Failure of PCI Benefits include low rates of events-free survival and reintervention-free survival

Arrhythmias			
-Bradyarrhythmias		**Tachyarrhythmias**	
Sinus Bradycardia	Atropine Blocking the vagal stimulation-	**Ventricular Tachycardia**	**Stable patient**: IV Amiodarone, Lidocaine or Procainamide. **Unstable patient** "Chest pain, SOB, Hypotension and confusion" Synchronized cardioversion.
AV Block (First degree, Second degree Mobitz 1)	No treatment required.	**Ventricular Fibrillation**	-Immediate defibrillation and CPR,if **persistent** IV epinephrine or vasopressin If **fails** antiarrhythmics. -Implantable defibrillator when cardioversion is successful.
AV Block (Second degree Mobitz 2, Third degree)	Pacemaker.	**Atrial Flutter**	**Hemodynamically unstable patient:** Synchronized cardioversion (shock) **Hemodynamically stable patient**: Rate control - Elective cardioversion-, Anticoagulation (depending on CHA2DS2VASc score) - Refer for Ablation
		paroxysmal Supraventricular Tachycardia	**Stimulate vagus delay** by carotid sinus massage (or breath holding / head immersion in cold water/ Valsalva maneuver) **followed by in stable pt IV Adenosine** (act by decreasing SA and AV nodal activity).CCB, b blocker, digoxin (prevention)

			-If episodes are recurrent and symptomatic →ablation is preferred
		Wolff-Parkinson-White Syndrome	Procainamide (Avoid digoxin / beta blockers /calcium channels blockers) or Radiofrequency catheter **1st line ablation.**
		Multifocal atrial tachycardia	calcium channel blockers, β-blockers, digoxin, amiodarone, IV flecainide, and IV propafenone. If LV function is not preserved, use digoxin, diltiazem, or amiodarone. -Electrical cardioversion is ineffective and should not be used.
		Atrial fibrillation (AF)	(1)Ventricular rate control. (2)Rhythm control. +Anticoagulation to reduce the risk of stroke in patients with AF >The CHA2DS2VASc Index

Acute AF			**Chronic AF**
Hemodynamically unstable patient → immediate electrical cardioversion to sinus rhythm.			A) **Rate control**: With Beta-blockers or• Ca -channel blockers •Digoxin if BP is low
Hemodynamically stable patient			B) **Anticoagulation** (use CHADSVASc score) -Patient with 'lone' AF under age 60 > no need for anticoagulation therapy (low risk of emboli). -Treat all other patients with chronic anticoagulation (warfarin).
Prevention of Thromboembolism "Stroke"(Depending on CHADVASc)	**Control of Heart Rate**	**Maintenance of Sinus Rhythm**	
AF present>48 hours, risk of embolization during cardioversion is significant. Anticoagulate patient for 3 weeks before and 4 weeks after cardioversion -To avoid waiting 3 weeks for anticoagulation, obtain a TEE to image the left atrium, if no thrombus is present, start IV heparin and perform cardioversion within 24 hours -An INR of 2 to 3 is the anticoagulation range	Determine the pulse in a patient with AF. if its too rapid, it must be treated	Use pharmacological cardioversion only if electrical cardioversion fails or is not feasible	
Pharmaceutical Warfarin, Aspirin Dabigatran, Apixaban Rivaroxaban avoid in renal impairment	**Pharmaceutical** • Ca-channel blockers In asthmatics •B-blockers In CAD •Digoxin	**Pharmaceutical**: most effective when initiated (within 7 days of AF onset). •Antiarrhythmic drugs – Class IA / IC – Class III: e.g. Amiodarone (**elderly**) •Flecainide •Propafenone (**young**)	
Non-Pharmaceutical: •Removal/isolation of left atrial appendage e.g. WATCHMAN device or surgery	**Non-Pharmaceutical** •Ablate/Pacing	**Non-Pharmaceutical** •Ablation •Surgery (MAZE) -Electrical shocks Direct-current cardioversion	

Valvular heart disease	Medical	surgical
Aortic Stenosis	Limited role	Aortic valve replacement Treatment of choice "indicated in symptomatic patients
Aortic Regurgitation	**Asymptomatic:** Medical therapy Serial echo check ups, salt restriction, diuretics,Vasodilators, afterload reduction "ACEI, arteriodilators",digoxin	**Symptomatic:** Aortic valve replacement Acute Aortic Regur: Medical emergency, perform emergent valve replacement
Mitral Stenosis	Diuretics (for pulmonary congestion and edema) oBeta blockers for palpitation and to decrease heart rate. oIf the patient has A-Fib treat them accordingly (digoxin and warfarin)	Percutaneous balloon valvuloplasty Open commissurotomy
Mitral Regurgitation	oPreload reduction therapy: vasodilators Start with ACE inhibitors if the patient has a cough go for AR Blockers still can't tolerate go for hydralazine oChronic anticoagulant if A-Fib is present oIABP as bridge to surgery for acute MR	oMitral valve repair or replacement (performed before left ventricular function is too severely compromised) **How to assess left ventricular function?** -EF<60% ->indication of surgery -End Ventricular Systolic Diameter : EVSD> 40mm ->indication of surgery
Mitral Prolapse	Beta blockers for chest pain Aspirin (TIA)	

Acute rheumatic fever		
arthritis	Improve with salicylates (aspirin)	
	Bed rest	
streptococcal infection	Antibiotics (penicillin)	To avoid recurrent RF and damage the heart. If allergic give (erythromycin / cephalosporin)
ARF	Salicylates: Aspirin	75-100 mg /kg/day given as 4 divided doses for 6 -8 weeks oAttain a blood level 20-30 mg/d
	Prednisolone "corticosteroids"	2mg/kg/day taper over 6 weeks, Given when there is severe carditis.
Complications Tx	Diuretics, ACEI	Heart Failure
	Valve replacement	Symptoms develop or LV dysfunction (valvular problems)

Prevention of RF	
Primordial Prevention	Social; housing, hygiene, overcrowding
Primary Prevention	Treatment of Sore Throat.

Secondary Prevention	Monthly Penicillin (Benzathine penicillin G IM, Penicillin V Oral, Sulfadiazine Oral) if penicillin & sulfadiazine allergic so give (Erthromycin)
Tertiary Prophylaxis	Medications, Balloon Valvuloplasty, Valve Replacement.

HF (systolic failure Tx)	**Use**	**MOA**	**ADRs**	**Extra**
loop diuretics (furosemide(lasix),torsemide , bumetanide) **thiazide diuretics** (hydrochlorothiazide)	**Initial therapy in systolic failure & volume overload**	relieve the symptoms by↓ preload. Does not reduce the mortality.		Combination of **diuretic and ACEI** should be initial Tx in symptomatic pts
ACE inhibitors	Initial therapy for left ventricular systolic dysfunction (LVEF less than 40%)	↓ afterload by vasodilatation and ↓ preload by absorption of the fluid and it reduces the mortality.	angioedema + cough	Switches: -Cough-> ARB -developed hyperkalemia or renal impairment or in pregnant→ Hydralazine(**arterial dilator**) with isosorbide dinitrate (**Venodilator**).
B-Blockers (metoprolol . bisoprolol . carvedilol)	Stable pt used in class 1,2 and 3	anti-ischemic, antiarrhythmic , ↓ heart rate (↓ oxygen consumption increasing filling time) & reduced remodeling of the heart → It decreases mortality		combination of **B blockers**(MNM: blocked symptoms =asymptomatic) **and an ACE inhibitors** required for asymptomatic patient with LVEF less than 40% -not given in high heart rate
spironolactone(potassium sparing diuretic)	-chronic CHF - aldosterone antagonist used in advance class 3 and 4 only	decreases mortality -prolong survival in selected pts		developed gynecomastia, impotence (cause its structurally similar to progesterone) switch to eplerenone.
Digoxin		+ve inotropic effect used in severe CHF/ Afib & only relieve symptoms with EF<40%have been continues despite (ACEi , BB , LD and	-Vagotonic, Arrhythmogenic. -toxicity: yellow vision, N/V	

		spironolactone) and does not improve the mortality.		

Acute hF	O2, **loop diuretics (furosemide)** most important drug that decreases the preload *Best initial therapy **, nitrate (IV)** that decrease the afterload **, morphine** **-if pulmonary edema continues add dobutamine** **don't use ACEI & b blockers**

lifestyle modification	**Correction of reversible causes**	**devices**
Sodium and fluid restriction , Weight loss ,smoking cessation ,Restrict alcohol and use Exercise program	Ischemia Valvular heart disease Thyrotoxicosis, Shunts, Arrhythmia	●**ICD (implantable cardiovascular defibrillator**)it's indicated for patients at least 40 days post-MI, EF <35%, and class II or III. ● **Cardiac resynchronization therapy (CRT**): biventricular pacemaker indications are similar to ICD except these patients also have prolonged QRS duration >120 msec

Diastolic Dysfunction	**B-Blockers, diuretics, ACE inhibitors& ARB** Management of Acute decompensated Heart Failure (Acute Pulmonary edema)
Digoxin and spironolactone should NOT be used.	Note : If pulmonary edema continuous despite these 4 so should added dobutamine (increased contractility & decrease afterload)

Contraindicated Medication on CHF
Metformin : may cause potentially fatal lactic acidosis. ★ **Thiazolidinediones** (glitazone): causes fluid retention. ★ **NSAIDs** : may increase risk of CHF exacerbation. ★ some of **Calcium Channel blockers** : may raises mortality.

IE	Bactericidal antibiotics
Initial therapy (before culture result)	Vancomycin and gentamicin **Resistant organism:** add aminoglycoside & extend Tx duration
After culture result	
Streptococcus viridans	Penicillin: Ceftriaxone (IV penicillin G/ IV amoxicillin) for 4 weeks or ceftriaxone 2 weeks followed oral amoxicillin 2 weeks
Methicillin resistant Staphylococcus Aureus	flucloxacillin
Staphylococcus epidermidis or Resistant Staphylococcus/ allergy	Vancomycin
Enterococci	ampicillin and gentamicin
Fungal	amphotericin and valve replacement
Surgery	Indicated in CHF, Uncontrolled infection and Prevention of embolism.
Prophylaxis (High risk people: dental procedure)	amoxicillin or ampicillin. If allergic->give clindamycin

PE		Use	Complication
Oxygen		**hypoxemia**	
heparin	-Prevent further clots formation. (Low molecular weight Heparin or unfractionated heparin). -should continue for at least 5 days -Platelet count should be monitored at least every 3 d -**goal is an APTT of 1.5 to 2.5 times control**		**Bleeding**: -Stop heparin infusion -IV protamine sulfate **Heparin-induced thrombocytopenia and thrombosis:** -Stop-heparin for platelet counts <75,000. -Replace heparin with direct inhibitors of thrombin-like (desirudin, lepirudin, argatroban) / fondaparinux **Heparin-induced osteoporosis (therapy >1 mo):** -LMWHs may have lower propensity to cause osteoporosis as compared with unfractionated heparin
Oral anticoagulant (Started at the same time with the heparin)	-Oral anticoagulants are continued for 6 weeks to 6 months, -Anticoagulation(Heparin\warfarin) prevents further clot formation		
Warfarin	**monitor INR between 2 and 3** -**contraindicated in pregnancy**		**Bleeding**: -Stop therapy. -Administer vitamin K and fresh-frozen plasma
Rivaroxaban or dabigatran	No needs to monitor INR and reach the therapeutic effect in several Hours		
Thrombolytics			

streptokinase ,Urokinase,Recombinant tissue-plasminogen activator	lysis the clots in **massive PE**	● Hemodynamically unstable patient (hypotension and tachycardia) ● Right sided Heart failure ●hypoxia on 100% O2	
IVC Filter		-Anticoagulation contraindicated (eg, patients with multiple trauma, active bleeding) - Failure of antithrombotic therapy - Complications from anticoagulant therapy preclude further use - Prophylaxis against embolism from preexisting DVT in patients with poor cardiopulmonary reserve or in patients at high risk to develop DVT. - Patients with recurrent PE undergoing thromboendarterectomy.	-filter migration or misplacement, - filter erosion and perforation of IVC wall, and IVC obstruction due to filter thrombosis.
Thrombectomy			

asthma	
Causing asthma: Extrinsic asthma	**Aspirin, NSAID, B-blockers**
Conformity test	PFT: Bronchodilation (albuterol) Bronchoprovocation test: methacholine (muscarinic agent) or histamine
Mild asthma	Step1: Inhaled short acting B2 agonist (**SABA**) (albuterol (most common), pirbuterol or levalbuterol).
Moderate asthma	Step2: **SABA** +long term agent usually low dose Inhaled corticosteroids (**ICS**)-> (Leukotriene modifiers as montelukast, Or Cromolyn (nedocromil) Or theophylline.
Severe asthma	Step3: Add long acting B2 agonists (**LABA**) such as salmeterol or formoterol to low dose **ICS** and **SABA**. Step4: Increase the dose of ICS to maximum in addition to **LABA** and **SABA**. Step5: Add **anti-IgE** such as omalizumab to **LABG** , **ICS** AND **SABA**. Step6: Add **oral corticosteroids** such prednisone.
Acute exacerbation of asthma	1-**Oxygen** 2-Inhaled B2 agonist via nebulizer or MDI →Albuterol 3-**Steroids** I.V initially or orally. Third line agent includes **IV magnesium** →helps with bronchospasm in **acute cases**.

cough		
causing	ACE inhibitors	Bradykinin or Substance P increase →metabolized to PGE2 →PGE2 accumulates and caused vagal stimulation of cough.
	GERD: Drugs (Theophylline, oral B adrenergic agonists,NSAIDS,ascorbic acid, Calcium channel blockers)	
Tx		
GERD	Proton pump inhibitor (PPI) for minimum 8weeks (ex:Omeprazole)+ prokinetic (ex:domperidone or metoclopramide)	
Postnasal drip	●**Sinusitis**: 1-Antibiotics 2-Nasal corticosteroids. ●**Rhinitis** :Avoid irritants but if it was caused by a virus →medical options include: 1-Antihistamine (Diphenhydramine,promethazine,loratadine) 2- Nasal corticosteroid (beclomethasone, flunisolide), anti leukotrienes (montelukast) 3-Cromolyn Sodium 4-alpha adrenergic agonists(ephedrine, pseudoephedrine)	
ACE inhibitors	Switch to Angiotensin 2 receptor blockers(ARBS) in case of cardiac/ renal problems -Antitussive medications	
In acute cough	Dextromethorphan such as e.g. Benylin non-drowsy	●Have no benefit in the cough caused by smoking, asthma, or emphysema. ●Contraindicated in children(< 4 years) and in combination with paracetamol.

	Menthol	Steam inhalation, it has short duration of action.
	Sedating antihistamines	used in nocturnal cough. Major side effect is sleepiness.
	Codeine and Pholcodine	Not recommended as it has more side effects and less effectiveness comparing to Dextromethorphan.
	Guaifenesin and water	to improve the effectiveness of antitussive medications.

COPD & Bronchiectasis		
COPD	**stop smoking.**	
	Bronchodilators	a.Short acting bronchodilators for **mild disease** i.Beta 2 agonists: Salbutamol, Terbutaline b.Long acting bronchodilators for **moderate to severe disease** i.Beta 2 agonists: Salmeterol, Formoterol, Indacaterol ii.inhaled Anticholinergics (muscarinic antagonists) most effective in COPD. This is because it is believed that beta agonists might cause CVS side effects (tachycardia, etc.): Tiotropium bromide c.Oral bronchodilators can be given to patients **who cannot inhale efficiently/ refractory COPD** i.Theophylline : → Improves mucociliary clearance and central respiratory drive. not commonly used because it has lots of side effects and drug interactions
	Corticosteroids	a.Inhaled corticosteroids do not alter the natural history of the FEV1 decline i. Usually given in combination with long acting beta agonists b.Oral corticosteroids are useful in acute exacerbations (discussed later) i. Remember steroids have many side effects such as *osteoporosis and Cushing's syndrome*
	Pulmonary rehabilitation	Encourage patients to exercise (patients with dyspnea grade 3-5)
	Oxygen therapy	-Improves survival and quality of life in patients -Only in patients with hypoxia/cor pulmonale
	Vaccinations	a.Influenza vaccine yearly b.Strep pneumoniae vaccine every 5-6 years
	Surgery	a. **Bullectomy**. b. Lung volume reduction surgery (LVRS)→ predominantly upper lobe emphysema
Acute exacerbation of COPD	**Don't give 100% O_2**	might cause respiratory depression and worsens the patient's ventilation and acidosis
	first-line therapy. **Give nebulized short acting beta 2 agonist and an anticholinergic agent**	salbutamol and ipratropium
	Oral corticosteroids not inhaled	**Systemic** corticosteroids are used for patients requiring **hospitalization** (IV methylprednisolone is a common choice)
	Antibiotics (aminopenicillin or macrolide)	if there is increase in sputum color, volume, purulence, or breathlessness (bacterial infections)

	amoxycillin/Clavulanate, cephalosporin (e.g. cefuroxime), quinolone (ciprofloxacin, levofloxacin, moxifloxacin)	If worsening 2 out of 3 of the following: i. Shortness of breath ii. Amount of sputum iii. Purulency of sputum
Bronchiectasis	**airflow obstruction**	treat that same as COPD patients (inhaled bronchodilators and corticosteroids
	Physiotherapy	helps the patient to get rid of all the sputum in the respiratory tract
	Antibiotics	-usually same as COPD (aminopenicillin or macrolide) but usually **for longer durations and higher doses** -**pseudomonas colonization**, Nebulized antibiotic therapy (Gentamicin or Tobramycin twice daily - If culture shows Pseudomonas or Staph aureus, antibiotic therapy might be more difficult and should use sensitivity to guide antibiotic therapy -Surgical resection of focal lesions may be indicated.

Pleural Effusion	
Causes: Exudative pleural effusion	Drugs: Hydralazine, Cimetidine, Methotrexate (very common)
Tx	
Thoracocentesis	-Uncomplicated pneumonia: antibiotics - Hemithorax involved/empyema: tube thoracostomy +/- VATS - Malignant effusion: chest tube +/- **pleurodesis** (sclerosants) / VATS
Transudative effusion	Diuretics and sodium restriction
	Therapeutic thoracentesis (in **massive effusion**).
Exudative effusion	treat underlying cause.
Parapneumonic effusions	Uncomplicated: antibiotics alone.
	Complicated or empyema: ●Chest tube drainage and antibiotics ●Intrapleural injection of thrombolytic agents (streptokinase or urokinase); may accelerate the drainage. ●Surgical lysis of adhesions may be required.

Pneumonia			
Community-acquired pneumonia	**Out-Patients**	**Healthy & non Antibiotic past 3 months**	Macrolides (azithromycin or clarithromycin) or doxycycline
		Comorbidity or Antibiotic past 3 months	-Fluoroquinolones (levofloxacin or moxifloxacin) -Advanced macrolide + Beta-lactam (Amoxicillin)
		Continuous 5 days, stop when patient is afebrile for 48 hours	
	In-Patient	Fluoroquinolones (levofloxacin or moxifloxacin) Or Ceftriaxone and azithromycin	
	Streptococcus Pneumoniae	Penicillin G., Ceftriaxone, Azithromycin, Levofloxacin, Vancomycin (in allergy)	
	Haemophilus Influenzae	-Amoxicillin + Clavulanic Acid (Augmentin). - Ceftriaxone, Fluoroquinolones (FQ), or(TMP-SMX).	
	Pseudomonas Aeruginosa	-Ceftazidime -Cefepime, (Pip/Tazo), Amikacin, Tobramycin, Aztreonam, Ciprofloxacin, Carbapenems, or Polymyxin B.	
	Acinetobacter	Polymyxin B (colistin) or Tigecycline.	
	Coxiella Burnetii (Q-Fever)	Doxycycline, Rifampin or hydroxychloroquine.	
	Chlamydophila Psittaci (Psittacosis)	**1st**: Tetracycline or doxycycline Alternative: Macrolide.	
	Francisella Tularensis (Tularemia)	Streptomycin	
	Influenza	-Neuraminidase Inhibitors: Oseltamivir/ Tamiflu Zanamivir(Relenza) for→**Influenza A, B** -Adamantanes: Amantadine / Symmetrel Rimantadine / Flumadine for→ **Influenza A** **H1N1 is resistant to Adamantanes.**	
Hospitality acquired pneumonia	**Antipseudomonal caplaspitnes**	cefepime or ceftazidime	
	Antipseudomonal penicillins	piperacillin / tasopactem	
	Antipseudomonal Carbapenem	Imipenem (cause seizure) , meropenem or doripenem	
Ventilator Associated pneumonia	**Antipseudomonal beta-lactam**	Cephalosporins or penicillin or carbapenem	
	Second antipseudomonal agent	Aminoglycosides or fluoroquinolones	
	Methicillin- resistant antistaphylococcal agent	Vancomycin or linezolid	

Diabetic Nephropathy	
Management and Prevention	❑Control the glucose level. ❑Lower the BP to 130/80. ❑Reduce Cardiovascular risk. (because the atherosclerosis is highly accelerated among DM) ❑RAAS blockade, independent to BP by giving ACEi/ARBs. Both ARbs and ACEi can cause hyperkalemia, in the presence of renal artery stenosis it might cause marked deterioration in renal function, after initial dose check →electrolytes and renal function. Non-dihydropyridine calcium antagonist (diltiazem,verapamil)may be good alternatives. ❑Lower the LDL level to less than 100 mg/dL →give statins if needed. ❑Smoking cessation. ❑Lifestyle modification: Weight loss + exercise + dietrestriction (Slat + protein).

Hypertension			
Risk Factors	NSAID by salt and water retention through the kidney by inhibiting prostaglandin		
Etiology	Medications: oral contraceptives by direct increase of angiotensinogen from the liver.,decongestants, estrogen, appetite suppressants, chronic steroids, tricyclic antidepressants (TCAs).		
treatment	**Choice of therapy depends on**	1-Risk factors (mainly age and ethnicity) 2- Cause 3- Respond to initial therapy 4- Comorbid condition.	
	When to treat & what target?	●Low risk patients >160/100 target <140/90 ●Diabetic ,high risk patients and elderly treat when its > 130/80 target < 130-120/80. ●Moderate to high risk treat > 140/90	
	Goal of management is to reduce the incidence of adverse cardiovascular events,stroke and heart failure.		
1.Life style modification	●Weight loss (low BMI) ●Dietary management salt restriction + less fat and red meat and more vegetables & fruits and potassium. ●Exercise ●Tobacco cessation + Avoid alcohol consumption ●Stop unnecessary medications.		
2. Drug Therapy →divided into two states	**A. People without any conditions**	**People less than 60y (either black or white)**	thiazide , Calcium Channel blockers(CCB) , ACEi or ARB
		Black people above 60 y	thiazide or (CCB)
		Non black people Above than 60y	CCB or ACEi or ARB
	B. People with specific condition:	**DM**	ACEi or ARB (Inhibit the RAAS) and major side effect is Hyperkalemia, Thiazide, CCB
		Hyperthyroidism	BB (decrease both CO and Renin release) and has many side effects including bradycardia and bronchospasm.
		Benign Prostatic Hypertrophy	Alpha Blockers (decrease the arteriolar resistance)
		Osteoporosis	Thiazide (increase the Calcium reabsorption)
		CHF or CAD	BB , ACEi or ARB, Thiazide, Aldosterone antagonist
		Pregnant woman	BB (use first) or CCB or hydralazine or alpha methyldopa
		Migraine	BB, CCB

		Post Myocardial Infarction	BB, ACEi
		Chronic kidney disease	ACEi, ARBs, Thiazide
		Stroke	CCB +ACEi
Hypertensive crisis	1- IV labetalol or nitroprusside 2-I.V glycerol trinitrate 3-hydralazine 4- I.V sodium nitroprusside		Note: Don't lower the BP to normal because my provoke stroke .
Other modalities to treat resistant HTN	●Hypertension renal denervation → interrupt the renal sympathetic nervous system → stop RAAS system. ●Implantable device to activated baroreceptor → to reduce BP		
Refractory HTN causes	1- Non-adherence to therapy 2- Inadequate Tx 3- Failure to recognize under lying cause		

Acid Base Balance			
Respiratory acidosis			
Etiology	CNS Medication such as (Commonly sleeping pills, other: morphine, anesthetics and narcotics)		
Acute Respiratory Acidosis	Causes: Acute drug intoxication ●narcotics, ●sedatives		
treatment	1. Verify patency of airways. 2. Give supplemental oxygen : If PaO2 is low (<60 mmHg) 3.Treat underlying cause 4. Intubation and mechanical ventilation might be required for: ●Severe acidosis (PH <7) ●PaCO2 > 60 or inability to increase PaO2 ●Mental deterioration ●Impending respiratory fatigue		
Respiratory alkalosis			
etioloy	Drugs (aspirin)		
Treatment	●Treat the underlying cause. ●Breathe into paper bag to recycle the exhaled CO2 (especially who have anxiety).		
Metabolic acidosis			
etiology	1.High Anion Gap Causes:	Increase Lactate	Correct hypoperfusion
		Ketoacidosis	Insulin and fluids
		Uremia	Dialysis
		Oxalic acid	Fomepizole, Dialysis
		Formic acid	Fomepizole, Dialysis
		Salicylates	Alkalinize urine
	2.Normal Anion Gap	Decreased production of HCO3	amphotericin

		Carbonic anhydrase inhibition	Due to diuretics as acetazolamide.
treatment	1.Treat the underlyingcause. 2.Sodium bicarbonate is sometimes used in severe acidosis (esp. in normal AG acidosis). 3.Mechanical ventilation might be needed if the patient is fatigued (esp. in DKA).		
Metabolic alkalosis			
etiology	Saline –Responsive U(cl.) <20		Diuretic use→ These decrease the ECF volume
treatment	1.Treat the underlying cause. 2.Give normal saline plus potassium in saline responsive 3.Spironolactone (K+ sparing diuretic) might be considered in saline resistant.		

Acute Kidney Injury			
NSAIDs	decrease prostaglandins →decrease GFR		
ACE inhibitor	decrease angiotensin II → decreased hydrostatic pressure in the glomerular capillary →decreased GFR		
Pre- renal	- Treat underlying disorder -Give Normal Saline to maintain euvolemia and restore blood pressure. **Normal saline is not given for patients with ascites or edema.** -Important to stop antihypertensive medications. (Eliminate any offending agent NSAID or ACEi)		
Post - renal	- Relieving the obstruction You must secure an IV line in order to replace the fluid that the patient will urinate. IF you did not do so, the patient would lose lots of fluids and would go into hypovolemic shock.		
Renal- AKI	**tubular**	**Caused** by: toxic (Drugs such as aminoglycosides, amphotericin B, lithium, etc) **Treatment**: supportive (correct BP, avoid nephrotoxic drugs, etc.)	**Treatment** of **ATN** is supportive care: ➔ Maintenance of euvolemia (with diuretics, IVF, as necessary). ➔ Avoidance of hypotension. ➔ Avoidance of nephrotoxic medications (including NSAIDs and ACE-I). ➔ Dialysis, if necessary.
		Rhabdomyolysis	**treatment** is IV calcium gluconate to protect the cardiac membrane, and give IV insulin with 5% dextrose to push K+ into the cells
		Contrast nephropathy	Decrease the risk of this condition by giving patient IV fluids before and after the exposure to contrast and also avoid nephrotoxic drugs -1/2 NS 1 cc/kg/hr 12 hours pre/post - N-acetyle cystein 600 BID pre/post (4 doses) - Monitoring of urine output, Creatinine and electrolytes
		calcium oxalate	**Caused** by medication that increases oxalic acid level in body (ethylene glycol)
	Interstitial	Most commonly caused by drugs (penicillin & NSAIDs, steroids)	**Treatment**: stop the offending agent (the drug causing this problem), supportive treatment.
	Acute Glomerulonephritis		➔General: supportive therapy ➔Disease specific: Steroid - Immunosuppressive agents - Plasmapheresis
When to do dialysis?	1.Refractory hyperkalemia 2.Refractory metabolic acidosis 3.Refractory volume overload 4.Symptoms of uremia (uremic pericarditis, encephalopathy)		

Electrolyte imbalance (Na, H2O)		
(Heart Failure with reduced Ejection Fraction	ACE inhibitors	
Central Diabetes insipidus	desmopressin	
Nephrogenic Diabetes insipidus	thiazide diuretics	interfere with the kidney's ability to dilute urine → NDI patients will pass concentrated urine.
Treatment of hypernatremia	Treat the underlying cause	
	hypervolemic	give diuretics (furosemide) and D5W to remove excess sodium. Dialyze patients with renal failure.
	hypovolemic	Stabilize him with NS after ⇒that correct the hyponatremia by replacing the free water deficit -1/2 NS, D5W, oral H2O
	euvolemic	replace the free water deficit* and patients with central diabetes insipidus require desmopressin, amiloride for lithium induced NDI
Treatment of hyponatremia	Remember that if you quickly correct hyponatremia you can damage the CNS (central pontine myelinolysis) ●Do not exceed 9 mEq/L in 24 hours (Uptodate)	
	symptomatic	3% hypertonic saline
	asymptomatic	correct slowly, and treat the underlying cause
	Both symptomatic & asymptomatic long term Tx (H2O restriction, demeclocycline (ADH antagonist)	

Electrolyte imbalance (K, Ca)		
What keeps the Intracellular K high?	•Insulin , Beta agonists enhance the K/Na pump function •Beta Blockers inhibit the pump function	
Hyperkalemia causes	Potassium-sparing diuretics (spironolactone) ACE inhibitors. Rapid administration of β-blocker.	
Hyperkalemia treatment	*If the hyperkalemia is severe, or if ECG changes are present, first give IV calcium.	✓Calcium stabilizes the resting membrane potential of the myocardial membrane— that is, it decreases membrane excitability. ✓Use caution in giving calcium to patients on digoxin.(Hypercalcemia predisposes the patient to digoxin toxicity.)
	Shift potassium into the intracellular compartment	✓Glucose and insulin (Glucose alone will stimulate insulin from β-cells, but exogenous insulin is more rapid) Give both to prevent hypoglycemia.

		✓Inhaled beta agonist. ✓Sodium bicarbonate -Increases pH level, shifts K+ into cells. -An emergency measure in severe hyperkalemia.
	*Remove potassium from the body	✓Kayexalate—GI potassium exchange resin (Na+ /K+ exchange in GI tract) absorbs K+ in the colon, preventing reabsorption (passed in stool). ✓Hemodialysis. -Most rapid and effective way of lowering plasma K+. -Reserved for intractable hyperkalemia and for those with renal failure. ✓Diuretics (furosemide).
Hypokalemia causes	Laxatives. Excessive glucocorticoids Epinephrine (β 2-agonists)	
Hypokalemia treatment	Identify and treat the underlying cause.	
	*Discontinue any medications that can aggravate hypokalemia	
	Oral KCl is the preferred (safest) method of replacement and is appropriate in most instances.	
	IV KCl can be given if hypokalemia is severe (less than 2.5)or if the patient has arrhythmias secondary to hypokalemia.	○Give slowly to avoid hyperkalemia. ○Monitor K+ concentration and monitor cardiac rhythm when giving IV potassium. ○Infusion pearls. • Maximum infusion rate of 10 mEq/hr in peripheral IV line. • Maximum infusion rate of 20 mEq/hr in central line. • May add 1% lidocaine to bag to decrease pain (potassium burns!)
	As with calcium, it is difficult to correct the potassium level if any hypomagnesemia is not corrected first	
Hypercalcemia causes & treatment	**Causes**: thiazide diuretics	**Treatment**: Increase urinary excretion: 1- IV fluids (NS) 2- Diuretics Inhibit Bone resorption 1-Bisphosphonate 2-Calcitonin
Hypocalcemia causes & treatment	**Causes**: loop diuretics	**Treatment:** Symptomatic: IV calcium Long term: oral calcium

GLOMERULAR DISEASES		
Primary FSGS	Usually first line treatment with corticosteroids , second: immune suppressing medications such as ciclosporin, cyclophosphamide and mycophenolate mofetil	Progression to CKD is common in patients who do not respond to steroids
	Cause: Drugs: Interferon, Pamidronate, Heroin	

Secondary FSGS	treat the primary cause and add supportive measures to protect the kidneys, e.g. keeping blood pressure well controlled with ACEi.	not typically treated with Immunosuppression,
Primary MCD	Corticosteroids plus Cyclophosphamide or cyclosporine and May be Rituximab	In children; typically is corticosteroid responsive in > 90%
Secondary MCD	Cause: Drugs (NSAIDs, Lithium, Sulfasalazine, Pamidronate, D-penicillamine, some antibiotics)	
	Secondary MN: Mainly target the primary disease that caused MN, and treat the Nephrotic syndrome manifestations	
-IgA Nephropathy (Berger diseases)	Most important treatment is to control the blood pressure which also decreases the proteinuria. •Treated in severe diseases with ACE inhibitors and steroids	There is really no effective immunosuppressing therapy except in severe cases where it can be tried.
Post streptococcal glomerulonephritis (PSGN)	fluid and sodium restriction with diuretic and hypotensive agents is usually adequate antibiotic might be added depending on case	
-Lupus Nephritis	depends on the findings in renal biopsy. It usually involves high degree of immunosuppressing medications.	
ANCA vasculitis	immunosuppression that includes corticosteroids and cyclophosphamide.	
Anti-GBM antibody disease,(Goodpasture's Syndrome)	If Linear Anti-GBM positive always started immediately to remove the antibodies by Plasmapheresis and preventing further antibodies production by giving heavy immunosuppression that includes corticosteroids and cyclophosphamide.	

Chronic Kidney Failure (for only to know treatment skip to last row)	
ESRD: advanced CKD (Stage-5)	requiring dialysis or kidney transplantation
Factors contributing to the Progression of CKD	Drugs (NSAID)
salt losing nephropathy	require stepwise increases in Nacl and fluid intake.
Management	
Nutrition	restriction intake of: -protein; not less than 0.8mg/kg/day -Phosphate -sodium -potassium
Salt and water retention	-Salt intake restriction "daily Na+ < 100 meq -fluid restriction 1 – 1.5 L/day -Loop diuretics -RAS inhibition (ACEi, ARB) if HTN w proteinuria
Hyperkalemia	**Medications that ↑K+**: ACEI, ARB, NSAID, K- sparing diuretics, B-Blockers, andheparin. **Treatment of hyperkalemia:** -IV calcium gluconate 10 cc of 10%

	-Followed by 25 ml of 50% dextrose solution with 5-10 units regular insulin -B2-adrenergic agonist nebulizer (salbutamol) -NaHCO3 IV/oral -calcium resonium or sodium kayexalate to catch the k in the capillaries of the colon and get it into the stool
Hyperphosphatemia and secondary hyperparathyroidism	a. Reduce phosphate intake to < 10 mg/kg/day b. Phosphate binders: Calcium carbonate, Sevelamer (Renagel), Lanthanum carbonate c. Vitamin D (Calcitriol) 0.125 mcq/day -Must be withheld until **s. phosphate concentration < 6mg/dl because it may cause severe soft tissue calcifications.** -Vitamin D compounds can cause hypercalcemia and hyperphosphatemia, which may increase coronary calcification, so parcicalcitrol (Zemplar) is an analogue that inhibits PTH synthesis without elevation of calcium/phos. d. Indication for parathyroidectomy: PTH > 800 pg/ml with symptoms of bone disease (myopathy, bone pain) persistent hyperphosphatemia soft tissue calcifications.
Hyperlipidemia	the goal is to keep low density lipoprotein cholesterol < 100 mg/dl by diet control and statin group.
Anemia	A. Oral iron -in non-dialysis patients (CKD stages 1-4): 100-200 mg elemental iron should be given daily in 2-3 days, either one hour before meals or two hours post. (1 tab Ferrous fumerate, 200 mg contains 66 mg elemental iron) -In dialysis patients (CKD 5):IV iron should be given as ongoing iron losses tends to be higher B. IV iron - 1 gr of iron saccharate (ferrosac) divided into 10 doses of 100 mg given with each dialysis session. C. Erythropoietin types: Short acting and long acting ➔ Short acting eprex during hemodialysis ➔ Long acting Darbepoetin peritoneal dialysis **Recombinant Erythropoeitin–epoeitin alfa (eprex):** - patients on: starting dose 120 – 180 IU/kg/week, IV - pre-dialysis patients and PD patients: 80-120 IU/kg/week subcutaneously weekly dose - Hb/Hct monitoring every 4 weeks - **the most common side effects:** headache, HTN, arthralgia, and diarrhea - resistance to epoeitin: 1. inadequate Epo dose 2. anemia of chronic disease (infection, inflammation) 3. functional iron deficiency 4. secondary to hyperparathyroidism 5. carnitine deficiency 6. hemoglobinopathies 7. aluminum toxicity 8. B12/folate deficiency 9. Malnutrition

	D. Darbepoetin Alfa (Aranesp) - Recombinant Epo - Half-life: three folds longer IV and two folds longer S/C than that of epoetin - Recommended starting dose 0.45 mcg/kg S/C weekly or double the dose every 2 weeks
Medications	Loop diuretics (fursemide) (Salt and water retention). -RAS inhibition (ACEi, ARB) if **HTN with proteinuria**. -Phosphate binders(Calcium carbonate, Sevelamer (Renagel), Lanthanum carbonate). given with meals. -Statin (hyperlipidemia). -Parcicalcitrol (Zemplar)**inhibits PTH synthesis without elevation of Ca2+/PO43**- (vitamin D compounds →Hypercalcemia + Hyperphosphatemia →coronary calcification). Parathyroidectomy when PTH 800 pg/ml + bone disease symptoms (myopathy, bone pain) + persistent hyperphosphatemia soft tissue calcifications. -Hyperkalemia (temporary protect the heart from arrhythmia by shifting the K+ into the cell): IV calcium gluconate 10 cc of 10% →25 ml of 50% dextrose solution with5-10 units regular insulin →B2-adrenergic agonist nebulizer (salbutamol) →NaHCO3 IV/oral. -Erythropoietin for **anemia** (Hemoglobin should not go back to normal but around 11-12, if more than 12 high chance of strokes and cardiac problems) -Oral Iron(100-200mg) if **not on dialysis**, IV Iron if on **Dialysis** divided into 10 dosesof 100 mg given with each dialysis session

Acute Viral Hepatitis	
Autoimmune disorder	prednisone and or azathioprine.
Hepatitis A	-Hepatitis A vaccine (pre-exposure) "Active vaccine" - Immunoglobulin (pre- and post-exposure) "passive" resolve spontaneously over a few weeks and are almost always benign conditions
Hepatitis B	-vaccine -only treated chronically with interferon or lamivudine.
Acute hepatitis C	in the few cases in which it is detected, should be treated with Interferon and ribavirin and either boceprevir or telaprevir.
Chronic HCV	Interferon and ribavirin or direct acting anti virus which depends of specific HCV genotype: genotype 1 →ledipasvir and sofosbuvir orally for 12 weeks, other genotypes include →sofosbuvir and ribavirin orally.

Gastrointestinal Bleeding	
Always ask patient if they take	NSAIDs/aspirin, clopidogrel or anticoagulants
Steps of management	
1- Hemodynamic status and resuscitation	●IV fluid resuscitation [initial assessment]. ●Supplemental oxygen. ●Draw blood for hemoglobin and hematocrit, PT, PTT and platelet count ○Monitor the hemoglobin every 4 to 8 hours until the patient is hemoglobin stable for at least 24 hours. ●Has been shown to significantly decrease mortality.
2-Blood transfusion	●Type and crossmatch adequate blood ●Don't rush into transfusion ●Should be administered to a patient with a hemoglobin level of 70 g/L or less ●Rarely indicated when the level is > 100 g/L ●**Almost always indicated when the level is < 60 g/L.** ●Target level of 70 to 90 g/L ●The role of transfusion in clinically stable patient with mild GI bleeding remains controversial, with uncertainty at which hemoglobin level transfusion should be initiated. ●The restrictive RBCs transfusion had significantly improved survival and reduced rebreeding. ●Transfusion as the clinical demands: shock, patient with cardiopulmonary disease.
3-Risk stratification	A. Low vs. high risk B. Early identification C. Appropriate intervention D. Minimizes morbidity and mortality
4-Pre-endoscopic therapy	Nasogastric tube: Evaluvation of aspirate PPI treatment before endoscopy
5-Endoscopic therapy	Timing of endoscopy: ●Definition of early endoscopy Ranges from 2 to 24 hours AFTER INITIAL PRESENTATION May need to be delayed or deferred: ○Active acute coronary syndromes ○Suspected perforation
6-Post-endoscopy	●PPIs: ○Reduce: rebleeding and need for surgery. ○Has no benefit on overall mortality (improve mortality if patients at highest risk) ○Intermittent PPI (IV boluses) therapy is comparable to the recommended continuous IV infusion in patients with High risk bleeding ulcers. ○There is no difference in clinical outcomes between oral and intravenous PPI. ○Patients receiving oral PPI have a shorter hospital stay.➔ Compared to H2Ras with or without endoscopic therapy they reduce: rebleeding, surgery but not mortality
Hospitalization	●It takes 72 hours for most high-risk lesions to become low-risk lesions AFTER endoscopic therapy. ●60% - 76% of patients who had rebleeding within 30 days AFTER endoscopic hemostasis PLUS high-dose PPI therapy did so within the first 72 hours.

ICU	For at least the first 24 hours on the basis of risk or clinical condition: –Hemodynamic instability –Increasing age –Severe comorbidity –Active bleeding at endoscopy –Large ulcer size (>2 cm)
After discharge	a prescription for a single daily-dose oral PPI for a duration as dictated by the underlying etiology.
Management of continued or recurrent bleeding	●**Percutaneous or trans catheter arterial embolization** ●Technical success range from 52% to 98% ●Recurrent bleeding in about 10% to 20% ●Complications include: -Bowel ischemia -Secondary duodenal stenosis -Gastric, hepatic, and splenic infarction ●A second attempt at endoscopic therapy remains the preferred strategy oAngiography: Where available, percutaneous embolization can be considered as an alternative to surgery for patients for whom endoscopic therapy has failed.
Management of Esophageal varices	1. Patients with medium/large varices: Beta blockers (Propranolol, nadolol) or Endoscopic variceal ligation 2. Patients with high-risk small varices: Beta blockers 3. Patients with small varices without signs of increased risk: Beta blockers
H pylori	•Patients with bleeding peptic ulcers should be tested for H. pylori –Receive eradication therapy if present –Confirmation of eradication •Negative H. pylori diagnostic tests obtained in the acute setting should be repeated
When to do surgery	●Hemodynamically unstable patients who have not responded to IV fluid, transfusion, endoscopic interventions, or correction of coagulopathies. ●Sever initial bleed or recurrence of bleed after endoscopic treatment. ●Continued bleeding for more than 24 hours. ●Visible vessel at base of ulcer (30% to 50% chance of rebleed). ●Ongoing transfusion requirement (five units within first 4 to 6 hours)

kkJaundice (causes)	
Unconjugated Hyperbilirubinemia (indirect) Decreased hepatic intake of bilirubin or impaired conjugation	Drugs such as rifampicin, penicillin, sulfonamide and radiocontrast agent
Conjugated Hyperbilirubinemia (direct) Decrease intrahepatocellular excretion of bilirubin. (ALT/AST elevated)	Drug induced: Oral contraceptive, Tylenol OD, idiosyncratic reaction or toxins as cocain.

Liver Cirrhosis			
Varicosity of veins (esophageal & gastric) **variceal without bleeding: give** nonselective BB **(**propranolol **or** nadolol **)**	(Active bleeding)		
	ABC	IV fluids (normal saline) to maintain BP , PRBC if hemoglobin level is low , platelets if low , plasma if PT or INT is high .	
	IV prophylactic antibiotic	(ceftriaxone or ciprofloxacin) : to prevent infection because bleeding is good media to bacteria	
	IV vasopressin	octreotide or somatostatin 3 to 5 days. It causes vasoconstriction of the splanchnic vessels →the varices to collapse.	
	upper GI endoscopy	either Endoscopic variceal ligation (EVL) or Endoscopic sclerotherapy	
	Nonselective BB (propranolol or nadolol)	to prevent recurrence of bleeding	
	Transjugular intrahepatic portosystemic shunt (TIPS)	artificial channel between portal system and hepatic vein used If the above drugs can't control the bleeding	
ASCITES	●Treat the underlying cause ●Salt dietary restriction ●Diuretics (Combination of Spironolactone and Furosemide) ●Recurrent tapping : done when asites not respond to above ●TIPS : done when asites not respond Recurrent tapping ●Liver transplantation : last option when asites cannot control	**Treatment of refractory ascites ~10%** 1. Serial therapeutic paracenteses + intravenous infusion of albumin if draining > 5L fluid 2. Transjugular intrahepatic portosystemic shunt 3. Liver transplantation 4. Peritoneovenous shunt	
Spontaneous bacterial peritonitis (SBP)	●IV third generation cephalosporin (ceftriaxone or cefotaxime) ●Albumin		
Hepatic encephalopathy	Exacerbation factors	Drugs (like narcotics and , diuretics sedative drugs.)	
	Treatment	●Lactulose (**First line of treatment**) : changes the colonic PH, making it acidic by forming NH4 →.. prevents absorption of ammonia and promote excretion . ●Rifaximin/ metronidazole: kills the flora → decrease the ammonia production	
Hepatorenal syndrome	●Correct underlying cause ● Albumin ● Vasoconstrictors of splanchnic vessels (Terlipressin, octreotide, midodrine, epinephrine) ● HD (Hemodialysis) ●Liver Transplantation in (decompensated cirrhosis)		
Hepatocellular carcinoma (Hepatoma)	Different scoring systems, Famous system(Barcelona Clinic Liver Cancer Staging Classification (BCLC) 1. Liver Transplantation 2. Surgical resection 3. Ablation (alcohol, RFA, Microwave) 4. Transarterial chemoembolization or Radioembolization (injection of a chemotherapeutic agent and lipiodol into the hepatic artery) 5. Systemic therapy (very limited role)		
Coagulopathy	Fresh frozen plasma		

Management of IBD				
1.Medical Therapy	**Drugs**	**MOA**	**use**	**Side effects**
5-ASA **(5-aminosalicylic acid compounds or** aminosalicylates**)**	Sulfasalazine, Mesalamine compounds Oral (Asacol, pentasa. Rectal(Canasa, Rowasa)	Induce and maintain remission by anti-inflammatory effect (inhibit prostaglandins and leukotrienes)		(of sulfasalazine): Crystalluria ,BM depression, Megaloblastic anemia,Folic acid deficiency
Metronidazole			if no response to 5-ASA	
Corticosteroids	Systemic:Prednisolone. Local (rectal): Budesonide		Acute exacerbation and if there is no response to metronidazole (used to Induce remission)	
Immunomodulators	Azathioprine methotrexate	Azathioprine "Inhibit purine synthesis", methotrexate "folic acid antagonist"	in conjunction with steroids if the patient does not respond to the previous drugs	
Anti-TNF therapy	infliximab, Adalimumab, Certolizumab	Inhibit TNF-α and it has higher response in CD than UC	in fistula or severe disorder unresponsiveness to	
2. Nutrition therapy(crohn's)	Bile acid sequestrant (cholestyramine, colestipol)			
3. SURGICAL THERAPY	Used For complications ,Failure of medical therapy and Severe disability ❑ crohn's →segmental resection with anastomosis ❑ UC →total resection			

Dysphagia		
Achalasia	Drugs	Antimuscarinic agents / Nitroglycerin / Calcium channel blockers
	Endoscopic (Pneumatic) dilatation	effective in 80-85% of patients thus the treatment of choice
	Botulinum toxin injection	
	Surgical	Surgical sectioning / Heller myotomy
Esophageal diverticula	Zenker	Surgery: Cricopharyngeal myotomy/ Diverticulectomy is of secondary importance
	epiphrenic	Surgery: Esophagomyotomy / Diverticulectomy is of secondary importance.

GERD	Major factors	drugs (eg: Anticholinergics, Calcium Channel blockers &Nitrates),
	treatment	A- PPIs, which are usually effective in resolving symptoms and healing esophagitis B- domperidone, when dysmotility features are prominent C- antacids and alginates can also provide symptomatic benefit D- H2-receptor antagonist drugs also help resolving symptoms without healing esophagitis
	Barrett's esophagus	1-Endoscopic therapies, such as: ● radiofrequency ablation or photodynamic therapy used only for those with dysplasia or intraconal cancer like patients with CLO without dysplasia should undergo endoscopy at 3–5-yearly intervals ● low-grade dysplasia at 6–12-monthly intervals 2- esophagectomy or endoscopic therapy with a combination of endoscopic resection(ER) ● For those with high-grade dysplasia (HGD) or intraconal carcinoma.
	Benign esophageal stricture	long-term therapy with a PPI drug
Esophageal Cancer	Tx	1. **Palliation** is the goal in most patients 2. Surgery (esophagectomy) 3. Chemotherapy plus radiation before surgery has been shown to prolong survival more than surgery alone.

Abdominal pain & IBS		
Abdominal pain	History: Medications (aspirin or NSAIDs)	
Peptic Ulcer	Acid suppression: - H2 blockers "accelerate healing of ulcer" - PPIs. (most effective) - Antacids. "Symptomatic relief" ● Eradicate H. pylori: - With triple or quadruple therapy (PPI, bismuth and 2 antibiotics). ● Cytoprotection: - Sucralfate. "facilitates ulcer healing." - Misoprostol. "Can cause GI upset (common side effect)"	
Acute pancreatitis	Causes	Drugs : diuretics, NSAIDs
	Treatment	●NPO ●IV hydration ●Painkiller If more than 30% of the pancreas is necrosed, prophylactic antibiotics (imipenem
IRRITABLE BOWEL SYNDROME (IBS) Management	●Fiber and diet ●Anti spasmodic agent (hyoscyamine Or dicyclomine) ●Tricyclic and antidepressant ●Anti Motility : ❑ diarrhea →Loperamide ❑ Constitution →cisapride ●Tegaserod maleate (Zelnorm) is a serotonin agonist recently introduced for the treatment of IBS	

Chronic diarrhea & Malabsorption

Malabsorption TX	Treatment of causative disease	●A gluten-free diet helps treat celiac disease. ●Similarly, a lactose-free diet ●Protease and lipase supplements are the therapy for pancreatic insufficiency. ●Corticosteroids, anti-inflammatory agents, such as mesalamine, and other therapies are used to treat Crohn's ●Whipple disease: 2 weeks with Antibiotics to cross BBB:Ceftriaxone, or Penicillin G + streptomycin… - THEN Septra 1 year (Trimethoprim/sulfamethoxazole) ●SBBO: Metronidazole, Amox/Clav, Tetracycline, Doxycycline, Amoxicillin (May need to cycle Abx to avoid resistance) - Bowel cleanse with PEG (Polyethylene glycol) -Prokinetic to stimulate motility > bacterial cleansing - Octreotide – at low dose, has a promotility effect; (causes hypomotility at higher doses) - Probiotics – minimal evidence ●Tropical Sprue: 1) Folate & B12 Supplement 2) Tetracycline for 3-6 months. ●Bile Acid-Induced Diarrhea: Cholestyramine (it binds to bile acids thus reducing its irritant effects)
	Nutritional support	●Supplementing various minerals calcium, magnesium, iron, and vitamins. ●Caloric and protein replacement also is essential. ●Medium-chain triglycerides can be used for lymphatic obstruction. ●In severe intestinal disease, such as massive resection and extensive regional enteritis, parenteral nutrition .
Chronic diarrhea	Osmotic	Caused by: osmotic laxatives
	Carcinoid	therapy is octreotide
	Ab associated diarrhea	Clindamycin or any Ab + C.diff
	Treatment	➢Rehydrate; monitor electrolytes and replace if necessary ➢Treat the underlying cause (stop or change the medication, advice lactose free diet). Consider atrial of NPO. ➢Consider antibiotics in infectious diarrhea→5 day course of ciprofloxacin. Antibiotics are definitely recommended in these following conditions: ●Patients has fever, bloody stools, or severe diarrhea- give quinolones. ●Positive stool culture ●Patients has traveler's diarrhea. ●C.Difficile infection- Give Metronidazole. ➢Loperamide (Imodium) is an antidiarrheal agent that should only be given is mild to moderate and not recommended in patient with fever of bloody diarrhea.