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Medicine

DBMCI · 2026

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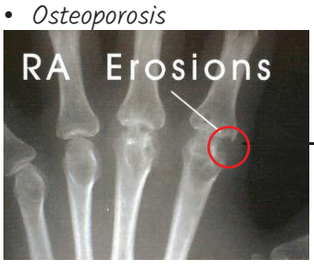
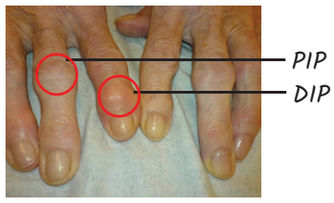
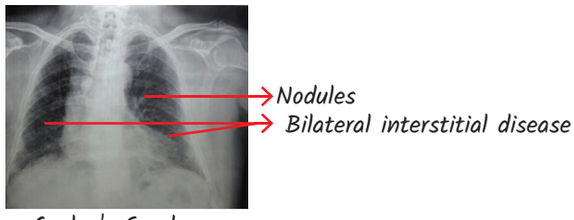
MEDICINE

“Once
you learn
the concepts,
you can’t help
but fall in love
with Medicine.”

- Dr. Thameem Saif

RHEUMATOLOGY

	Rheumatoid arthritis	Osteoarthritis
Joints involved	Involves smaller joints • MCP (Metacarpopharyngeal joints) - MC • PIP (Proximal inter-pharyngeal joints) • Wrist joint • Elbow • Shoulder • Knee • Ankle • MTP (Meta tarsal pharyngeal joint) • C_1C_2 vertebrae (Atlantoaxial joint)	Joints Involved : • Base of thumb (characteristic) • PIP - Bouchard's nodes • DIP - Heberden's node • Hip • Knee • MTP • Vertebrae - Lower cervical and lumbar
Joints spared	DIP	MCP and wrist
Symmetry	Bilaterally symmetrical	Bilaterally asymmetrical
Findings	• Bone erosions (also known as rat bite lesion) • Osteopenia • Osteoporosis 	• New bone formation (osteophyte) • Osteosclerosis • Cysts 

Features	Related points
Ocular	• Keratoconjunctivitis sicca • Episcleritis • Scleritis
Pulmonary	• Pleuritis • Pleural effusion (↓pleural fluid glucose levels) • Interstitial lung disease - Usual interstitial pneumonitis (UIP) pattern • Caplan's syndrome : - Pulmonary nodules - Pneumoconiosis  Caplan's Syndrome

Features	Related points
Cardiac	• Pericarditis • Restrictive cardiomyopathy ↓ leads to Heart failure with preserved ejection fraction (HFpEF) • Mitral regurgitation • ↑risk of ischemic heart disease
Blood abnormalities	• Anemia of chronic disease • Neutropenia • Lymphoma (Diffuse large B cell lymphoma)
Renal	• Membranous nephropathy • NSAIDs induced - Interstitial nephritis
Neurological	• Compressive neuropathy • Mononeuritis multiplex (rare) - Occurs due to vasculitis (d/t skin rashes)

DMARDs (Disease Modifying Antirheumatic Drugs)

- HCQ's - Hydroxychloroquine
  - Side effects - Retinopathy, Q-T prolongation
- Methotrexate (central / main drug)
  - Given once a week.

Side Effects

- Bone marrow affected (TLC / DLC monitored)
- Hepatotoxic - LFT derangement
- Teratogenic

Leflunomide

- Milder than methotrexate.
- Dihydro-orotate dehydrogenase inhibitor.
- Side effect - teratogenic.
  - Avoid pregnancy for 2 weeks after stopping the drug as Leflunomide metabolises through enterohepatic circulation.

Sulphasalazine

Side effects

- Bone marrow affected → Monitor TLC, DLC
- Azoospermia (reversible)

Triple Therapy

- Methotrexate + sulfasalazine + hydroxychloroquine

Anti-TNF Drugs

- Expensive
- Used if methotrexate ineffective
  a. Etanercept
  b. Adalimumab
  c. Golimumab
  d. Certolizumab
  e. Infliximab

- Side effects :
  a. Can cause reactivation of TB
  b. Neuropathy
  c. Drug induced lupus
  d. ↑ Risk of infections
  e. ↑ Risk of lymphoma and skin malignancy (may occur)

- Contraindicated in :
  a. SLE
  b. Hepatitis B

Other Drugs

1. Anakinra IL-1 antagonist
2. Toclizumab
3. Sarilumab
   } IL-6 receptor antagonist
4. Abatacept → Acts on CTLA-4
5. Tofacitinib
6. Baricitinib
   } JAK inhibitors

- Side effect : ↑risk of infections

SPONDYLOARTHRITIS

- Ankylosing spondylitis (Arial spondyloarthritis)
- Inflammatory bowel disease associated arthritis
  } Axial joints
- Psoriatic arthritis
- Reactive arthritis (Reiters)
  } Peripheral joints > axial joints

Common Features Of The Above Types :

- Sacroiliitis → backache.
- Uveitis
- Enthesitis → inflammation of ligament.
  - Achilles tendonitis
  - Plantar fasciitis
  - Anterior longitudinal ligament.
- Dactylitis → inflammation of digits.
  - Also known as sausage digits.

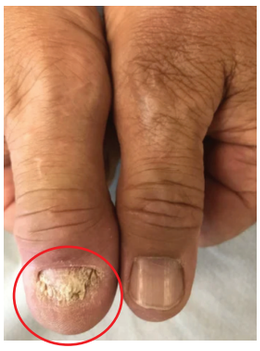
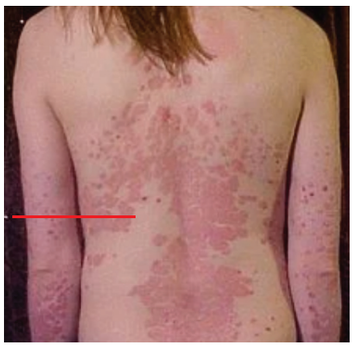
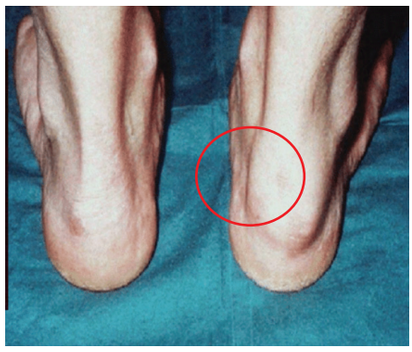
- Responds to NSAIDs.
- Family History +
- HLA-B27 +

PSORIATIC ARTHRITIS

Nail involvement is classic with peripheral arthropathy.

CASPAR Criteria

- Psoriasis
  - Currently present
  - Previous history
  - Family history
- Nail → pitting → splitting → dystrophic.
- Dactylitis
- Negative for Rheumatoid factor (RF).
- Bone → new bone formation + inflammation


Treatment :

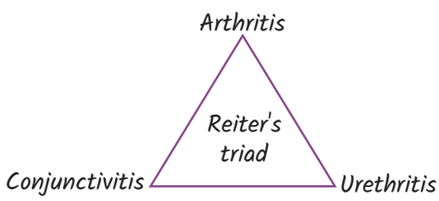
- Skin (psoriasis) + Peripheral arthritis → Methotrexate.
- Only arthritis → anti - TNF
- Secukinumab → IL 17 inhibitor
- Ustekinumab → IL 12/23 inhibitor
- Apremilast → PDE-4 inhibitors.

BIMEKIZUMAB - Dual - 17A + 17-F inhibitor

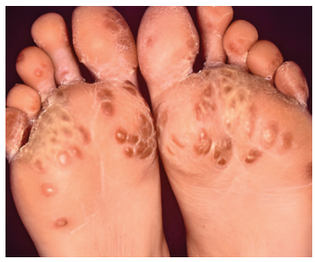
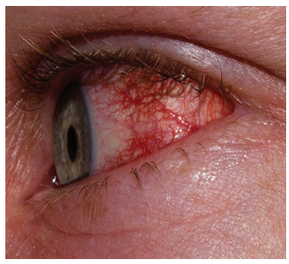
GUSELKUMAB, RISANKIZUMAB } IL-23 inhibitors

REACTIVE ARTHRITIS (REITER'S)

- Autoimmune process triggered by infection
  - Chlamydia
  - Shigella
  - Salmonella
  - Yersinia
  - Campylobacter
  - Mycoplasm genitalium
  - Ureaplasma urealyticum
  - HIV
- Peripheral joints all more commonly involved :
  - Knee, ankle.
  - DIP
  - Axial sacroilitis (asymmetrical).
  - Dactylitis
  - Uveitis
  - HLA-B27 positive



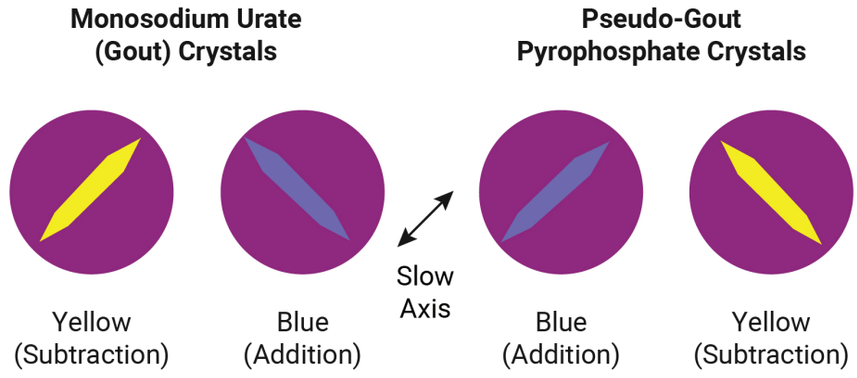
- Enteritis
- Skin lesions - Palms / soles → Keratoderma blennorrhagica.
  Vesicles progress to → crusts on palms / soles.
- Genitalia
  Penile → Balanitis circinata
- Oral ulcers (usually painless)
- Conjunctivitis

GOUT

Clinical Features :

- Usually monoarticular involvement. Rarely, oligoarticular (upto 4 joints involved).
- M/c joint affected : 1st metatarsophalangeal joint (Podagra).
- Other sites : Ankle joint, foot joints, small joints of hand.
- Does not involve warm joints like hip / shoulder joint.
- Typically, attacks occur in the night.



Synovial fluid analysis

- Negative birefringence.
- Needle shaped.
- Na urate crystals.

} 3 N's

PSEUDOGOUT

- Deposition of CPPD (Calcium Pyrophosphate Dehydrogenase crystals).
- M/c joint affected : Knee joint.

Associations

- Hyperparathyroidism.
- Hemochromatosis.

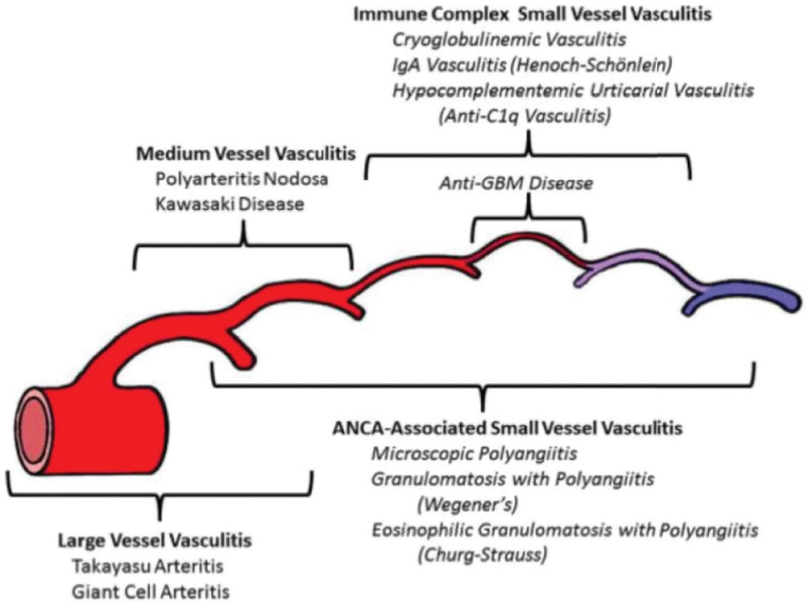
PRESENTATION

- Acute pain in the knee joint : Red, swollen.
- Calcification is seen in :
  - Ligaments.
  - Menisci of knee joint.
  - Triangular cartilage of wrist.
- Synovial fluid analysis :
  - ↑ TLC.
  - +ve birefringement (blue) rhomboid shaped CPPD crystals.

Vasculitis

Vessel size categories & representative examples

Vessel size	Affected vessels	Typical diseases	Key clinical features
Large vessel vasculitis	Aorta, its main branches (e.g. subclavian, carotid)	Giant cell arteritis (GCA), Takayasu arteritis	Limb claudication, diminished pulses, asymmetric BP, vascular bruits, ischemic symptoms, constitutional features
Medium sized vessels	Muscular arteries supplying organs, e.g. renal, mesenteric, coronary	Polyarteritis nodosa (PAN), Kawasaki disease	Organ ischemia/infarction (e.g. GI pain, renal ischemia), aneurysms, hypertension, mononeuritis multiplex, skin nodules/ulcers
Small vessel vasculitis	Arterioles, capillaries, venules	ANCA-associated (microscopic polyangiitis, granulomatosis with polyangiitis), IgA vasculitis, hypersensitivity vasculitis	Glomerulonephritis, pulmonary hemorrhage, skin purpura, alveolar capillaritis, neuropathy, immune complex deposition signs



TEMPORAL ARTERITIS

- Aka Giant Cell Arteritis (GCA).
- Age > 50 years.
- Temporal artery mainly involved but also involves aorta and it's branches, pulmonary vessels and external carotid artery.

CLINICAL FEATURES

- Headache (new onset) above 50 yrs.
- Scalp tenderness.
- Jaw claudication, trismus.
- Polymyalgia Rheumatica
- Visual complications : AION-Sudden onset of painless loss of vision, altitudinal filed defect (+)
- ESR>50/hr
- Next step steroids
- Confirm- temporal artery biopsy
- Treatment – steroids+Toclizumab, Upadacitinib (JAK 1i)
- Takayasu

CLINICAL FEATURES

- Below 40 yrs
- Asymmetric pulses (because of involvement of subclavian artery)

- Arm claudication.
- Unilateral Raynaud's phenomenon.
- Carotid involvement : Transient visual symptoms, TIA, stroke.
- Vertebral artery : Dizziness and visual changes.
- Aortic root → aortic regurgitation → CHF.
- Abdominal aorta → bowel ischemia → (Abdominal pain, diarrhea, nausea and vomiting, bloody stools.
- Renal artery : HTN, creatinine ↑↑
- Coronary artery : Ischemic heart disease.
- Pulmonary artery : Atypical chest pain and dyspnea.

Treatment – steroids and immunosuppressive therapy

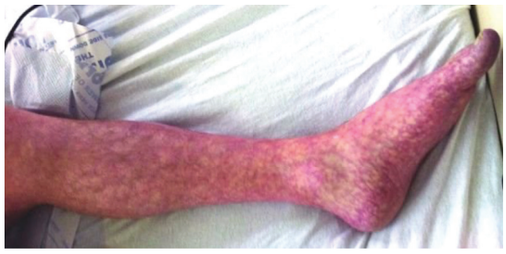
POLYARTERITIS NODOSA (PAN)

CLINICAL FEATURES

- Myalgia, arthralgia, fatigue, arthritis.
- Most common organ affected → Kidney (Renal artery branches > Renal artery)

  ↓

  HTN, S.creatinine ↑
- Hepatic artery : Liver dysfunction.
- Mononeuritis multiplex
  - Vasculitis → nerves.
  - Patchy asymmetric peripheral neuropathy pattern.
- Skin involvement : Purpura, nodules, livedo reticularis.
- Mesenteric artery : Bowel ischemia, abdominal pain, nausea, vomiting, diarrhea, blood in stool.
- Coronary arteries : Ischemic heart disease.
- Digital gangrene.



Livedo reticularis

Features not seen in PAN

- Pulmonary manifestations (bronchial arteries can be involved).
- ANCA association.
- Granulomas.
- Eosinophilia.
- Glomerulonephritis (no RBC casts)

Feature	GPA	MPA	EGPA
Respiratory	Cough ; lung nodules/cavitations;	Diffuse alveolar hemorrhage; Hemomptysis	Asthma with eosinophilia
Renal	Pauci-immune necrotizing crescentic GN (RPGN) common	Pauci-immune necrotizing crescentic GN (RPGN) very common	Pauci immune GN
ENT / Sinonasal	Prominent, Headache, sinus disease; saddle-nose deformity	Uncommon	Allergic rhinitis; nasal polcommon
Asthma / Eosinophilia	-	-	Asthma + peripheral eosin
Granulomas	Granulomas+	None	Granulomas+
ANCA pattern	PR3-ANCA (c-ANCA) most common	MPO-ANCA (p-ANCA) most common	MPO-ANCA (p-ANCA) in ~40are ANCA-negative
Neuropathy	Rare	Rare	Mononeuritis multiplex ve
Cardiac	Rare	Rare	Frequent: myocarditis, cardiomyopathy, coronary-vasculitis
Skin	Palpable purpura, nodules/ulcers	Palpable purpura, livedo, ulcers	Purpura, urticarial/angioe-lesions

TREATMENT - WEGENERS

- Rituximab + prednisolone + avocapan (C5 ⊖) - Ist line treatment
- MPA
- EGPA steroids/cyclophosphamide
- EGPA - Mepolizumab

HENOCH-SCHÖNLEIN PURPURA (HSP)

Tetrad

- Palpable purpura
- Arthralgia / arthritis + musculoskeletal pain / fatigue
- Bowel ischemia Nausea / vomiting / diarrhoea
  - Blood in stools
- IgA Nephropathy Hematuria + Proteinuria
- IgA Deposits on skin biopsy

Treatment

- Skin and joints - NSAIDs and colchicine
- GI - Severe disease - Steroids
- Rituximab
- Mycophenolate

KAWASAKI'S VASCULITIS

Criteria

- Fever atleast for 5 days plus 4/5 following criteria
  - Conjunctivitis
  - Oral changes of erythema and cracking of lips, strawberry tongue
  - Peripheral extremity changes - Swelling / desquamation.
  - Polymorphous rash-trunk
  - Cervical lymphadenopathy (larger than 1.5cm, usually unilateral)
- Coronary artery aneurysms present → LV dysfunction, MR

TREATMENT

- IV Ig
- Aspirin
- Resistant Kawasaki disease - Defined as having recrudescent or persistent fever at least 36 hours after the end of the first IVIG
  Repeat IV-Ig
  Still fever then
  - Steroids, anakinra, anti TNF drugs.

BEHCET'S VASCULITIS

- Recurrent oral ulcers plus 2/4
- Recurrent genital ulcers,
- Eye lesions- Anterior/posterior uveitis cells in vitreous or retinal vasculitis
- Skin lesions, - Erythema nodosum, nodular lesions
- Positive pathergy test
- Genital ulcers- Occur on the scrotum or labia
- Superficial thrombophlebitis often occurs in men and is associated with deep-vein thrombosis
- Arthralgia or arthritis (non erosive)
- Neuro involvement- subacute onset of severe headache, cranial nerve palsy, dysarthria, ataxia and hemiparesis
- GIT- ileoceacal ulcers occur- Clinical and endoscopic appearance of intestinal involvement can be similar to Crohn's disease

CRITERIA- SYSTEMIC LUPUS INTERNATIONAL COLLABORATING CLINIC CRITERIA (SLICC)

- Skin
  - Acute → Malar rash

Erythema

- Sub Acute → Annular psoriasiform Rash
  - Chronic → rash → Discolouration, discoid, scarring
- Oral / Nasal ulcers
- Non scaring Alopecia
- Synovitis (Non erosive arthritis)
- Serositis
- Renal+
- Neurologic
  - Seizures, Psychosis, Neuropathis, Myelitis, confusion etc
- Blood Abnormalities

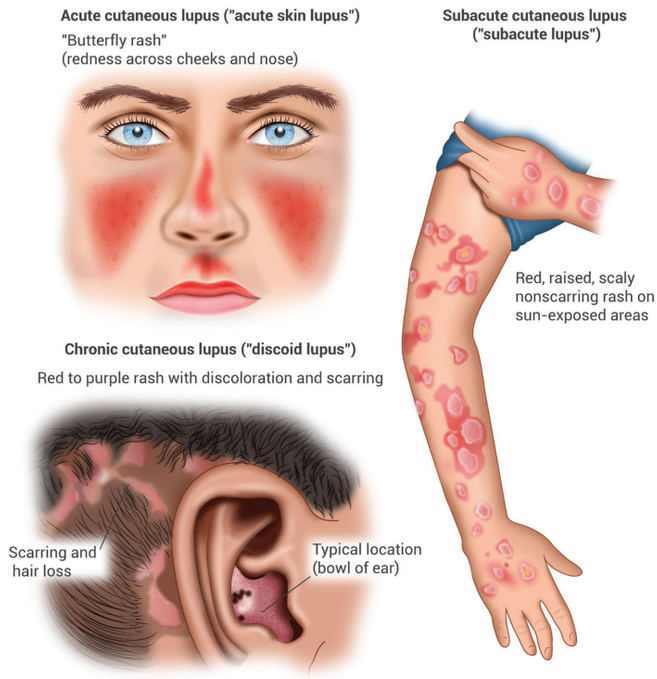
  a. Anemia

  i. Anemia of chronic diseases (most common)

  ii. Warm AIHA (Autoimmune hemolytic anemia)

  b. Neutropenia

  c. Thrombocytopenia
- Lab Criteria:
  - ANA Most sn
  - Anti ds DNA correlates disease severity +
  - Anti – smith
  - Anti phospholipid
  - Complement
  - +ve coomb's test (Absence of Hemolytic Anemia)

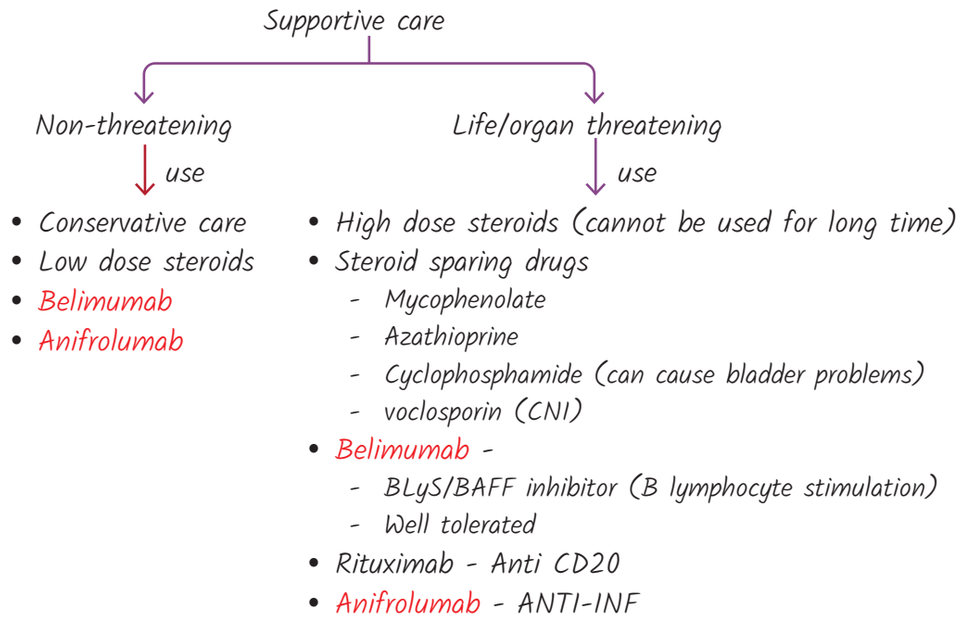


Antibody	Comments
ANA antibody	Most sensitive antibody
Anti-Smith antibody	Most specific antibody
Anti dsDNA antibody	Most accurate antibody High correlation with disease severity.
Anti - Ro (SSa), Anti - La (SSb)	Protective effect for kidney disease Anti Ro positive mothers causes: • Neonatal lupus • Heart blocks in neonate
UI - RNP antibody	Associated with MCTD (Mixed connective tissue disease) MCTD = Overlap syndrome + UI RNP antibody positive
Anti-neuronal antibody Anti phospholipid antibody Anti ribosomal P antibody	Associated with neuropsychiatric complications in SLE. Mainly psychiatric complication.

- Overlap syndrome
  - SLE
  - Myositis
  - Systemic Sclerosis
  - Rheumatoid arthritis

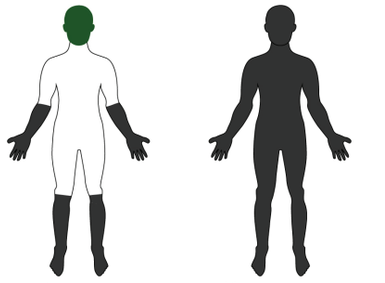
TREATMENT

- Hydroxy chloroquine (HCQ)



SYSTEMIC SCLEROSIS

CREST Syndrome	Features	Diffuse SSC
(+) Early precedes skin involvement (+) (+) (+) (+)	• Calcinosis • Raynauds phenomenon • Esophageal dysmotility • Sclerodactyly • Telangectasia	(+) (+) Simultaneous with skin (+) (+) (+)
Acral area involved	Typically occurs below nail base (periungual - early feature)	Trunk/proximal involvement
2%	Renal scleroderma crisis	15%
Anticentromere Ab	Antibodies : ANA +ve	• Anti - Scl 70 (Topoisomerase) • Anti-RNA pol III (RSC)



Limited cutaneous Systemic sclerosis | **Diffuse cutaneous Systemic sclerosis**

- GI-Dysphagia, GERD
- GAVE
- Pulm- NSIP
- Renal scleroderma crisis
- Htn
- Increased creat
- Proteinuria
- PBS- MAHA- schostocytes
- Treatment- Captopril, enalaprilat injection

Pulm-NSIP
- Nintedanib
- Toclizumab

SJOGREN'S

Clinical features

- Sicca manifestations
  - Dry eye → Keratoconjunctivitis sicca (Sandy/gritty feeling)
  - Dry mouth → Dental caries (loss of immune action of saliva), candidiasis
  - Dry vaginal mucosa

- Arthritis - MC extraglandular manifestation.
- Raynaud 's phenomenon
- Vasculitic skin rash
- Muscle cramps - Type I RTA / Interstitial nephritis → Hypokalemia + Metabolic acidosis
- Myalgia and arthralgia
- Mild cytopenias
- MGUS, Cryoglobulinemia.
- Lymphomas → Marginal zone B-cell lymphoma (low grade NHL)
- Interstitial lung disease (NSIP pattern, progressive-restrictive type)
  - Diagnosis - HRCT
  - Follow up - PFTs (Pulmonary Function Tests)
- Neurological manifestations
  - Peripheral neuropathy
  - Mononeuritis multiplex
  - Hepatomegaly splenomegaly

INVESTIGATION

- Schirmer's test, ocular staining - Dry eyes
- Antibodies
  - ANA : Present
  - Anti Ro (SSa)
  - Anti La (SSb)
- Biopsy → Labial biopsy/Inner lip biopsy (Minor salivary glands)

NOTES

NOTES

NEPHROLOGY

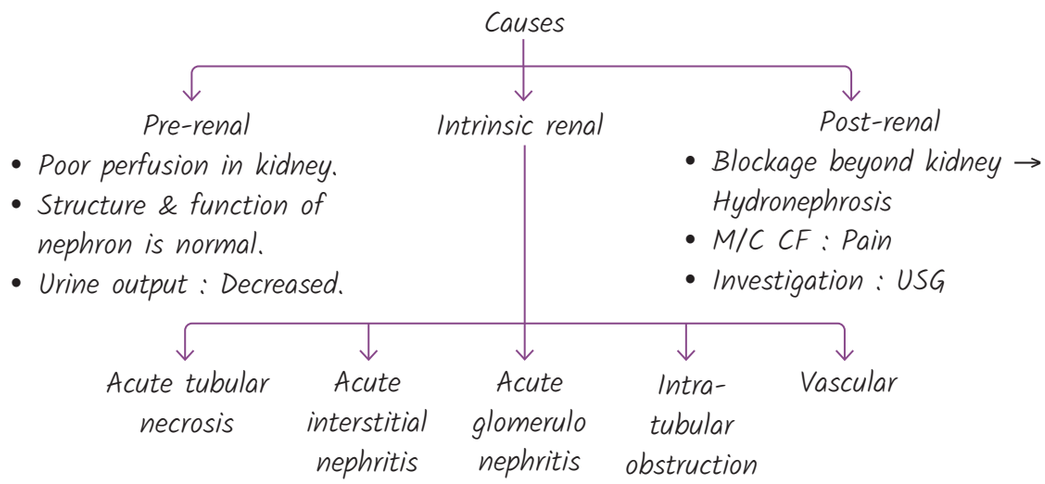
Spot Urine Analysis

- Show the Inspector creatinine ratio (ACR), take albumin in milligram & creatinine in gram
- ACR
  - < 30 mg alb/g creat - A1 - normal
  - 30-300 mg alb/g creat - A2 - microalbuminuria
  - >300 mg alb/g creat - A3 - macroalbuminuria

	Nephrotic Syndrome	Nephritic Syndrome
Proteinuria	>3.5gm/day	<3.5gm/day
Microscopic hematuria	±	+++
RBC casts	-	+
Onset	Insidious	Acute/subacute
Edema	++++	+, Pulmonary edema
BP	Normal/↑	HTN +++
JVP	Normal/↑	↑↑↑
S. Albumin	↓↓	↓
AT III, Protein C, Protein S, Immunoglobulins	↓ levels leading to • ↑Risk of infection • ↑Thrombosis	May or may not be decreased
Renal conditions	• Minimal Change Disease (MCD) • Membranous nephropathy (MN) • Focal Sclerosing Glomerulosclerosis (FSGS) • Membrano-Proliferative Glomerulonephritis (MPGN)	• Post Streptococcal Glomerulonephritis (PSGN) • IgA nephropathy • ANCA associated Glomerulonephritis • Anti GBM disease • MPGN
Systemic conditions	Diabetes Mellitus (DM), Amyloidosis (SLE)	SLE, HSP

Stage	Urine Output	S. creatinine
Stage 1	< 0.5 ml/kg/hr for 6-12 hours	↑ by ≥ 0.3 mg/48 hrs or 1.5 - 1.9 times over 7 days
Stage 2	< 0.5 ml/kg/hr for 12-24 hours	↑ by 2-2.9 times the baseline value
Stage 3	• < 0.3 ml/kg/hr for > 24 hours or • Anuria for 12 hours	• 3 times the baseline value or • ≥ 4 mg/dl or • Patient is on dialysis

- Volume status : ↑/↓
- Electrolyte abnormalities
  - Hyperkalemia (K^+ ↑)
  - Hyperphosphatemia (PO_4 ↑)
  - Metabolic acidosis
  - Hypocalcemia (S. Calcium ↓)
  - BUN ↑
  - S. creatinine ↑
- Urine output ↓
- Uremic complications :
  - Altered sensorium
  - Pericarditis
  - Bleeding



INDICATIONS

A : Acidosis refractory to treatment.
E : Electrolyte abnormality: K+ ↑ despite treatment
I : Ingestion: Lithium, Metformin, Salicylates, Ethylene glycol, Theophylline
O : Overload: Pulmonary edema
U : Uremia: Altered mental status, pericarditis, bleeding (platelet dysfunction)

CAUSES

1. Diabetic nephropathy (M/C cause)
2. Hypertension-associated nephropathy
3. Chronic glomerulonephritis
4. Ischemic causes
5. Adult polycystic kidney disease

STAGES OF CKD

Based on GFR

Stage	GFR (in ml/min)
1	>90
2	60-89
3a	45-59
3b	30-44
4	15-29
5	0-14 (Renal failure)

Based on Albuminuria

Grade	Albuminuria (mg of albumin/gram of creatinine)
A1	0-30
A2	30-300
A3	>300

- ACEI/ARB- to decrease proteinuria and control BP
- Calcium Channel Blockers
  Added when BP cannot be controlled by only ACE inhibitors/ARBs.
- SGLT2 Inhibitors
  - Eg : Empagliflozin
- Non-Steroidal Mineralocorticoid Receptor Antagonist- Finerinone
- Atrasentan, Sparsentan
  - Newer drug
  - MOA : Endothelin-1 receptor antagonist
- Glycemic control in DM
- Sodium bicarbonate = slow progression of CKD
- GLP-1RA

Anemia

- Check s. ferritin. Correct Iron and only then give EPO
- ROXADUSTAT, DAPRODUSTAT (HIF-PHI-hypoxia inducible factor-prolyl hydroxylase i)
- Hb-Target- 10-11.5 g/dl

Hyperkalemia

K+ binders - Decrease potassium absorption in the gut.

a. Patiromer

b. Zirconium

c. Sodium Polysterene Sulfonate (SPS) - Not preferred.

- Calcium free phosphate binders
  - Sevelamer
  - Lanthanum
- Cinacalcet

Polycystic Kidney disease

AD PKD :

- PKD – 1 = Defect in gene chromosome – 16 severe (ESRD - 55YR)
- PKD – 2 = Defect in gene on chromosome – 4- milder (ESRD - 75YR)

Renal Manifestations

- Flank Pain
- Recurrent UTI
- Renal stones
- Hematuria
- Chronic kidney disease
  - Hypertension
  - Anaemia
  - Bone mineral disorder
- End stage renal disease → needs dialysis.

Extra Renal Manifestations

- Hepatic cysts (can become infected - complications)
  (M/C extrarenal manifestation)
- Diverticulosis
- Pancreatic cysts, meningeal cysts, arachnoid cystsSeminal vesicle cysts
- Seminal vesicle cysts
- Mitral valve prolapse
- Aortic root dilatation (AR)
- Berry aneurysms
- Abdominal wall hernias

Treatment

- Hydration
- V2 Rs blocker
- Control BP (target <120/80)

RTA-1	RTA-2
Distal tubule defect	Proximal tubule defect
Defect - Acidification of urine	Defect - Reabsorption of bicarbonates
Urine pH > 5.5 (inability of kidney to excrete acid)	• Urine pH ≤ 5.5 - Urine acidified since distal tubule is normal. - Bicarbonate has reached a steady state. - On taking high bicarbonate meal, urine pH > 5.5
Causes : - Sjögren's syndrome - SLE - Cirrhosis - Amphotericin-B - Medullary Sponge Kidney - Hyperparathyroidism - Sarcoidosis	Causes : - Fanconi's syndrome - Plasma cell abnormalities - Amyloidosis - Drugs → Cisplatin, Ifosfamide, Tenofovir, NRTIs
Hypokalemia	Hypokalemia

TYPE 4 RTA

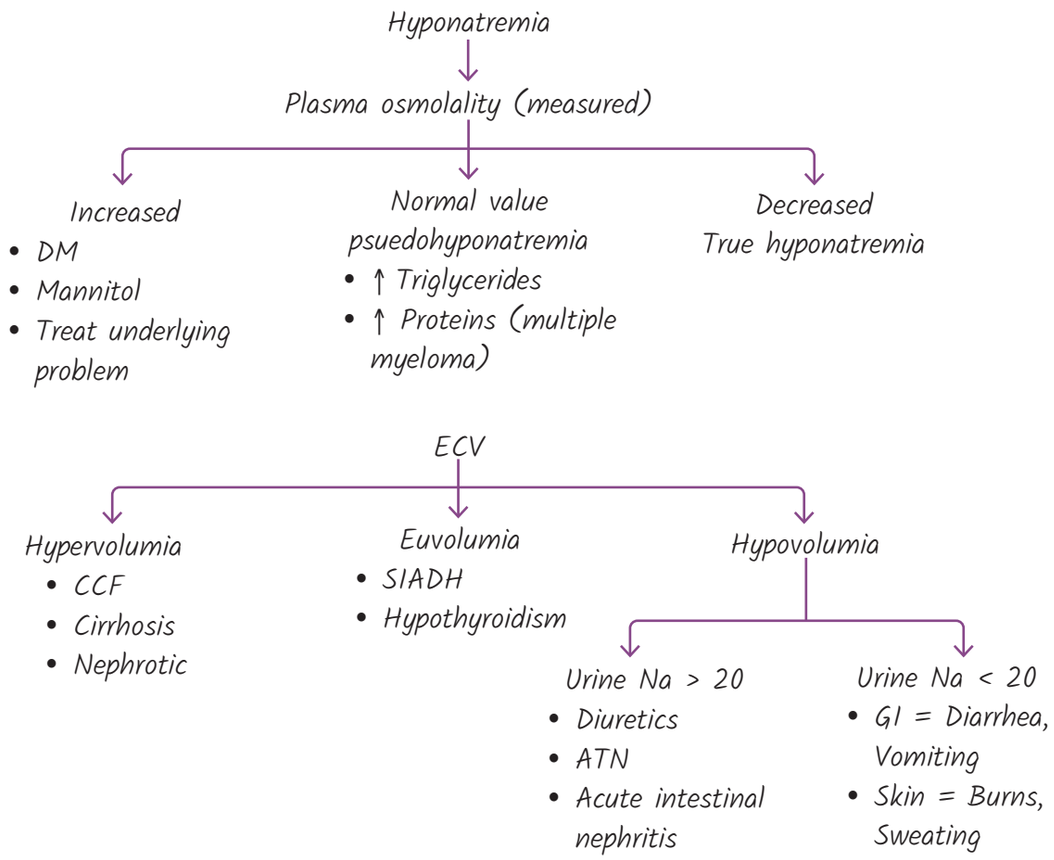
Pathogenesis

- ↓ secretion of renin & aldosterone due to :
  - Diabetic Nephropathy
  - ACE inhibitors
  - ARBs
  - Spironolactone
  - Addison's disease
  - Obstructive uropathy

Features

- Hyperkalemia
- Normal Anion Gap Metabolic Acidosis (NAGMA)

HYPONATREMIA



- Sodium deficit = Total body water (Desired sodium - Actual sodium)

ACUTE HYPONATREMIA

- Rapid correction : 100 ml of 3% saline.
- Calculate Na deficit and correct carefully (Overcorrection to be avoided)
  - Check Serum sodium post correction.
  - Repeat 100 ml of 3% saline until increases by 3 to 4 mEq/: then slow correction can be done.

CHRONIC HYPONATREMIA

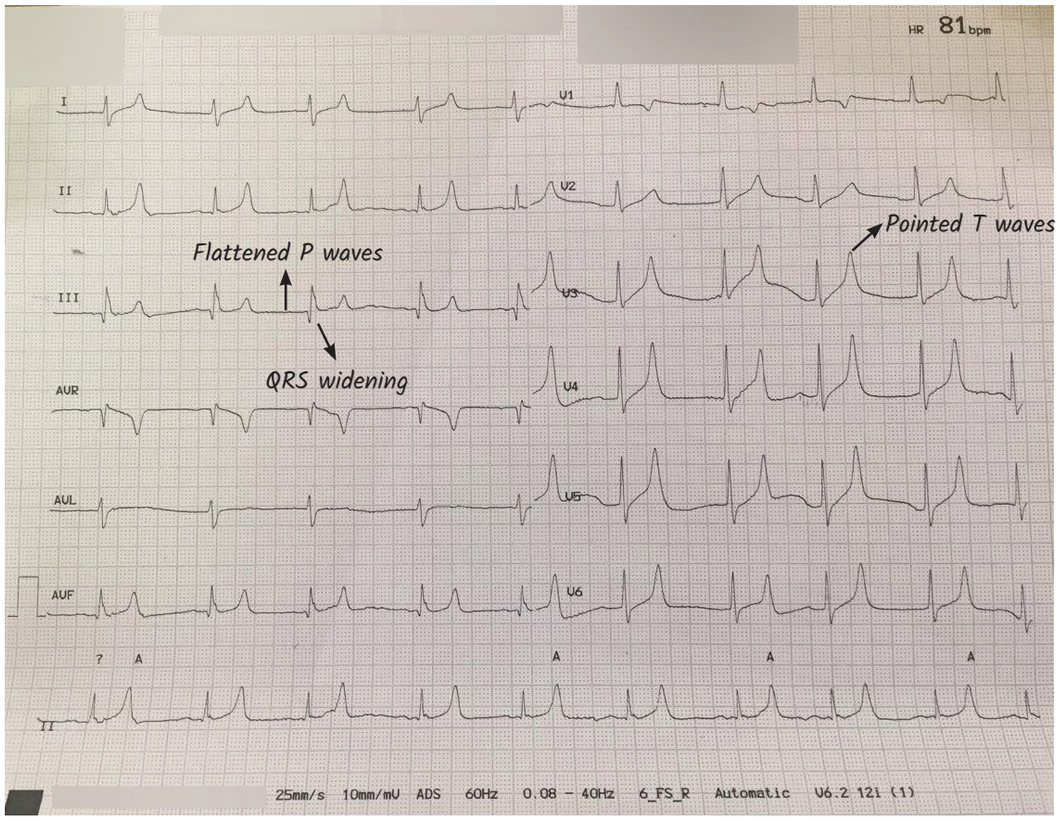
- Slow correction to be done (to avoid pontine myelinolysis).
- Fluid restriction → normalizes Na - H_2O ratio (done in mild hyponatremia)
- In case of volume overload (Eg : CCF, Cirrhosis) to prevent pulmonary edema, diuretic given.
- Important in hyponatremia occuring slowly
  - Ist 6 hours - Correction of 4 - 6 mEq
  - After 24 hrs - 8 mEq correction/day (should never be > 10mEq/day)

} Desmopressin Rescue

S Na > 145

1. Calculate water deficit.
2. Calculate ongoing loss → Urine → Free water clearance.
3. Calculate insensible loss.

HYPERKALEMIA



TREATMENT

1. Stabilize the membrane by giving calcium:
   - Calcium does not reduce K^+ but only stabilizes the membrane.
   - Calcium can be given as → Calcium gluconate (Slow-IV)
     → Calcium chloride (Rapid-IV)

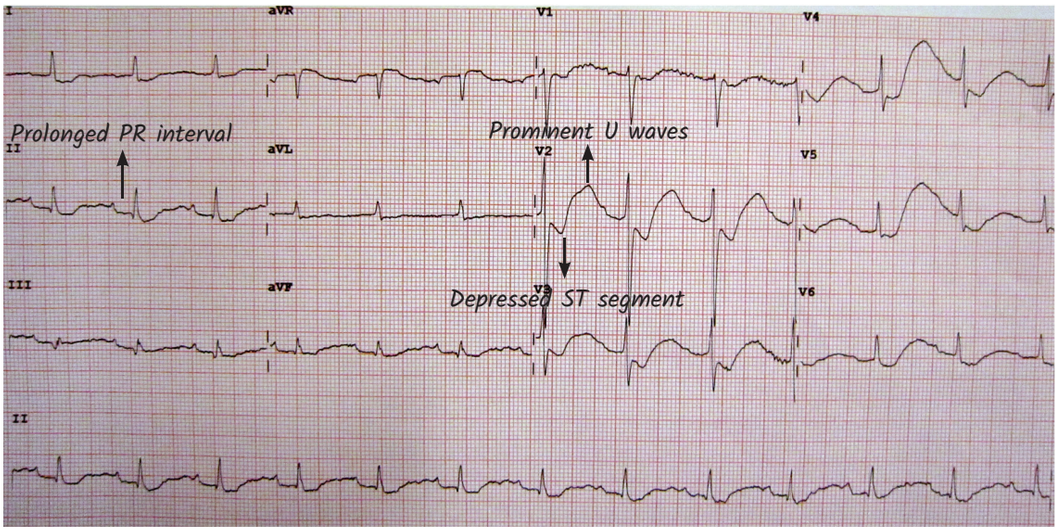
2. Insulin + Glucose → Shifts K^+ into cells and prevents arrhythmias.
   - Insulin → 10 units of regular insulin
   - Glucose (25 gm) → 50% dextrose (50 ml).

3. β_2-Agonist → Shifts K^+ into cells (salbutamol nebulisation).

4. To decrease K^+ in the body :
   - K^+ Binders
     - Patiromer
     - Zirconium
     - Sodium Polystyrene Sulfonate (SPS) - can cause intestinal necrosis.
5. Dialysis
6. Diuretics

7. Sodium bicarbonate → Not a preferred treatment for hyperkalemia.
   - Used to correct severe acidosis & pH < 7.1.
   - Reduces K^+ very slowly.

HYPOKALEMIA



In the absence of abnormal K+ redistribution, the total deficit correlates with serum K+, such that serum K+ drops by ~0.27 mM for every 100-mmol reduction in total-body stores Total body potassium deficits are typically large (>200 mEq for each 1 mEq/L decrease in plasma potassium below 3.5)

- *Oral KCl → 75 meq tablets or syrups*
- *IV K+ :*

- *Maximum limits :*
  - *Peripheral IV line → 10-15 meq/hr*
  - *Central IV line → 20 meq/hr*
  - *Maximum limit in a day → 240 meq/day*
- *Check for serum Mg before giving K+ :*
  - *If a patient has Mg2+ deficiency then*

ROMK channels are open and they keep secreting K+ in urine

Body K+ ↓

- *So magnesium should be corrected along with K+*

HYPOMAGNESEMIA

- *Normal serum magnesium = 1.8-2.4 meq*
- *If Serum magnesium <1.8 meq → hypomagnesemia*

Treatment

- *If symptomatic → IV MgSO4 → 1-2 g slow IV*
- *Infusion over 3-6 hours → oral supplementation*

PULMONOLOGY

SPIROMETRY

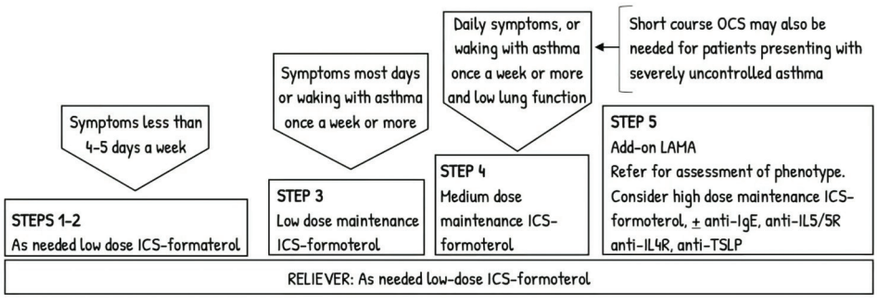
- FEVI : Amount of air a person can breathe out in 1 second with force
- FVC (Forced Vital Capacity) : Amount of total vital capacity breathed out
- PEFR : Peak expiratory flow rate; it can also be measured

Obstructive Lung Disease	Restrictive Lung Disease
• Example - Ashma - COPD • $FEV_1 \rightarrow \downarrow\downarrow$ • $FVC \rightarrow \downarrow/N$ • $\frac{FEV_1}{FVC} = \downarrow$	• Parenchymal or extraparenchymal • $FEVI \rightarrow \downarrow$ • $FVC \rightarrow \downarrow/\downarrow\downarrow$ • $\frac{FEV_1}{FVC} = N/\uparrow$

DLCO

Anemia	
Polycythemia	
Asthma	
Bronchitis	
Emphysema	
COPD	
L → R shunt	VSD / ASD / PDA (↑ Blood flow)
R → L shunt	TOF (↓ Blood flow)
ILD	D/t thick membrane
Alveolar Hemorrhage	CO is captured by alveolar haemorrhagic blood

ASTHMA



Agent Name	Mechanism of Action	Patient Population
Omalizumab	Anti-IgE	Elevated IgE level (30-1000 IU/mL) & allergen sensitivity
Mepolizumab & Reslizumab	Anti-IL 5	Peripheral eosinophilia (150-400 eosinophils/µl)
Dupilumab	Anti-IL-4 (blocks IL-4 and IL-13 signal-ling)	Glucocorticoid-dependent severe asthma; better with eosinophilic disease
Tezepelumab	Anti-thymic stromal lymphopoietin (TSLP)	Moderate-severe asthma, non- eosinophilic disease
ITEPEKIMAB	IL-33i	Severe asthma [smokers]

BRONCHIAL THERMOPLASTY

Bronchoscopic therapy using alar probe under sedation.

- Principle : Use radio-frequency wave to heat the distal airway muscles → reduction in thickness of airway muscles. → decreases hospitalization.

COPD

Exacerbations

Mild

- 0/1 exacerbation/year
- No hospital admission

Severe

- >2 exacerbation/year
- Atleast 1 exacerbation, required → hospital admission

Severe exacerbations	E		FeV_1 grade 3,4
Mild exacerbations	A	B	FeV_1 grade 1,2
	mMRC 0-1 CAT <10	mMRC >2 CAT ≥ 10	

Category	Treatment
A	Bronchodilator
B	Long acting β_2 agonist (LABA) + Long acting anti-muscuranic agent (LAMA), both as inhalers
E	Higher dose of LAMA + LABA ↓ inhaled corticosteroid (ICS) if eosinophils > 300 Symptoms persist ↓ Start Roflumilast (Phosphodiesterase-4 inhibitors) / Azithromycin

- Update - dupilumab
- Ensifentrine (PDE-3,4i)

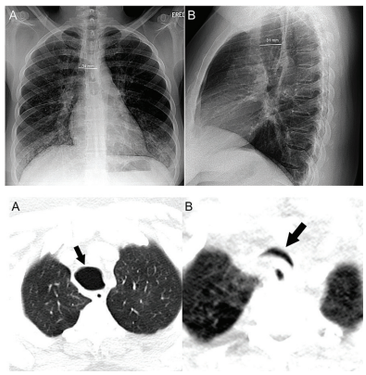
Treatment	Related points
Vaccination	• Yearly administration • Vaccines - Influenza - Pneumococcal vaccine
Lung volume reduction surgery	• Done only in cases of : - Upper lobe emphysema - Exercise intolerance - FEVI > 20% - DLCO > 20%. • Benefits of surgery - Creates space - Lung works better
Lung transplant	• Done when: - BODE index : 5 to 6 - PaO2 < 60 - PaCO2 > 50 - FEVI < 25%
Supplemental Oxygen	• Long term O2 therapy (LTOT) • Indications: - PaO2 ≤55 SpO2 ≤88 - PaO2 <60, SpO2 ≤89 { • Provided patients have • Polycythemia (Hct >55) • Cor pulmonale } • 16-18 hours of administration needed for optimum efficacy

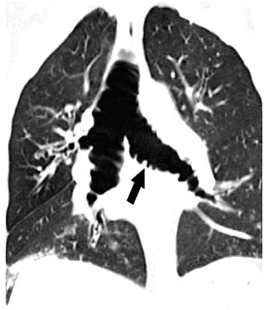
- BODE Index comprises of :
  - Body Mass Index (BMI)
  - Obstruction (FEVI)
  - Dyspnea (MMRC scale)
  - Exercise capacity (6-minute walk distance)

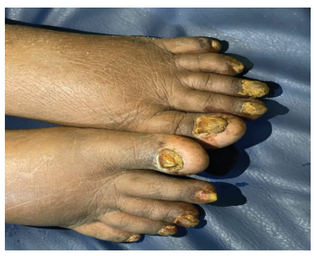
BRONCHIECTASIS

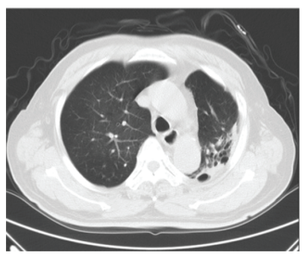
Causes of Bronchiectasis

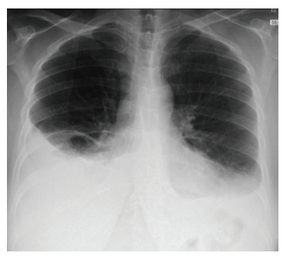
- Post TB > Post infective > Idiopathic > ABPA
- Cystic Fibrosis - MC cause in developed countries
- PCD - Kartagener syndrome, Young's syndrome
- William Campbell syndrome - 4 to 6th division of bronchi → cartilage defect
- Munier Kuhn syndrome - Tracheobronchomegaly (Trachea + Proximal bronchi are dilated)
- Yellow Nail Syndrome - Lymphatic Problems :
  - Lymphedema
  - Chylous effusion - (Triglycerides > 110 mg/dl in pleural fluid)
  - Bronchiectasis
  - Yellow nail
- Young's Syndrome - Sinusitis + Bronchiectasis + Obstructive azoospermia
- α_1-Anti trypsin deficiency

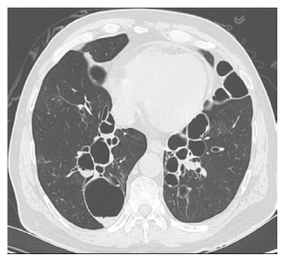


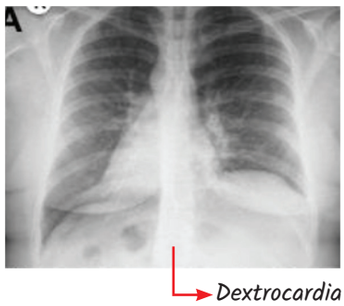


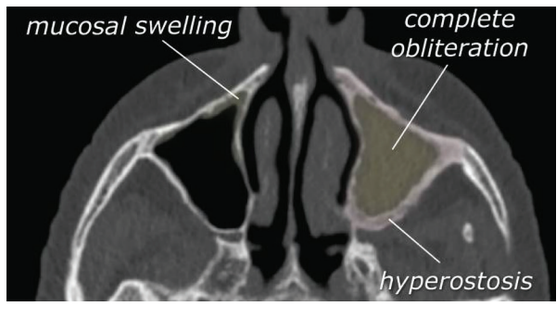












COMPLICATIONS OF BRONCHIECTASIS

- Pneumonias
- Metastasis Abscess
- Amyloidosis
- Empyema
- Sepsis

ARDS

Direct (Alveolar)	Indirect (Endothelial)
• Pneumonia	• Sepsis: M/c/c
• Aspiration	• Transfusion reaction
• Pulmonary Contusion	• Severe trauma
• Near drowning	• Pancreatitis
• Toxic gas inhalation	• Post-Cardiopulmonary bypass
	• Burns

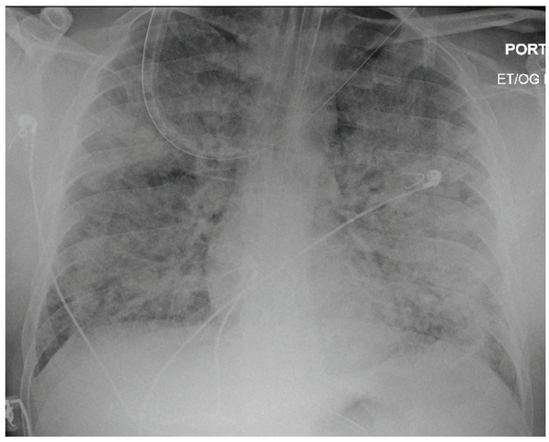
	Exudative Phase	Proliferative	Fibrotic
Time:	4-7 days	≥ 7-21 days	≥ 21 days

Berlin's 2012

- $\frac{PaO_2}{FiO_2} < 300 \rightarrow$ < 300 - Mild
  < 200 - Moderate
  < 100 - Severe

- B/L
- Acute - (<1 week)
- Pulm. Edema → Not due to hydrostatic cause (Cardiac)
  → Echo

New definition criteria	Classification		
	Mild	Moderate	Severe
Time to Installation	Up to seven days - known risk factors(s)		
Pulmonary Edema	Not explained by cardiogenic edema or intravascular volume overload		
Radiologic Features	Bilateral infiltrates on chest X-ray or CT or lung ultrasound by a trained professional. (not explained by nodules, pleural effusion, or atelectasis)		
Hypoxemia $PaO_2 : FiO_2$	201-300 with NIV/CPAP PEEP ≥5 or HFNO >30 L/min	101-200 with PEEP ≥5	≤100 with PEEP ≥5
Hypoxemia SpO_2/FiO_2	≤315 with SpO_2 ≤97%		



Treatment

- Ventilators- Volume Cycled
- Low TV 6ml/Kg
- PEEP (Positive End Exp. Pressure)
  Pplat < 30 cm H_2O
  BP – Maintained
- Early NM – Blockade (For severe ARDS)
- Prone position ventilation
- ECMO
- I : E ratio – Reversal

Do not give

- Surfactant : No clinical benefits in adults.
- Beta Agonists : No efficacy, potential harm if given IV.
- High-Frequency Oscillatory Ventilation: May increase mortality.
- Steroids are (Moderate /severe ARDS)

SHOCK

	Distributive Shock	Hypovolemic Shock	Cardiogenic / Obstructive Shock	
			Cardiogenic	Obstructive
Causes	• Sepsis (MC cause) • Neurogenic shock • Anaphylactic shock • Addisonian shock	• Blood loss • Dehydration - Polyuria - Vomiting - Diarrhea	• Acute HF • Acute MI • Acute AR / MR • Arrhythmias	• Pulmonary embolism • Tension pneumothorax • Dissection of aorta (Blood accumulates in false lumen → narrowing of lumen → obstruction) • Cardiac tamponade

SEPSIS

- Life threatening organ dysfunction caused by dysregulated lost response to infection (SOFA $\geq$ 2)
- Septic Shock → Sepsis + Vasopressors requirement + Lactate > 2 mmol/L despite fluids

SOFA SCORE

- SOFA : Sequential Organ Failure Assessment score.

Components

- Respiration : PaO_2 / FiO_2 (kPa).
- Coagulation : Platelets count.
- Liver : Bilirubin.
- Cardiovascular - MAP.
- Glasgow coma scale - EVM.
- Renal : Creatinine and urine output

Components (qSOFA)

- Hypotension : SBP < 100 mmHg.
- Altered mental status : GCS < 15.
- Tachypnea : RR > 22 / min.

} If 2/3 is (+) : Perform SOFA score.

TREATMENT

- IV fluids : RL 30 ml / kg. (Guided by dynamic fluid responsiveness)
- Start antibiotics + source control
- Vasopressor

↓

1st vasopressor of choice : NE (Nor-Epinephrine)

↓ BP not maintained

2nd vasopressor : Vasopressin

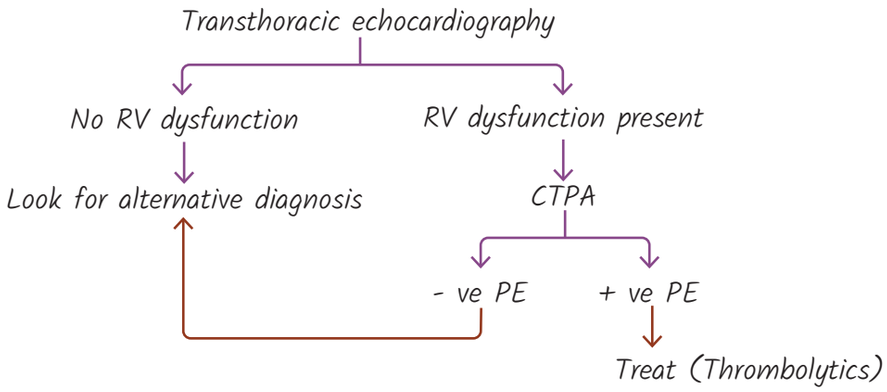
↓ + steroids

3rd vasopressor : Epinephrine

PULMONARY EMBOLISM

- Recent major surgery (Especially orthopedic hip/knee, pelvic, abdominal, neurosurgery)
- Major trauma (Long bone fractures, spinal cord injury, pelvic fractures)
- Prolonged immobilization (E.g., bedridden patients, long-haul flights, ICU stay)
- History of prior VTE (DVT/PE)
- Active cancer / malignancy
- Pregnancy and postpartum state
- Estrogen therapy (Oral contraceptives, HRT), especially with smoking/obesity
- Inherited thrombophilias (Factor V Leiden, antiphospholipid syn)

Hemodynamically unstable

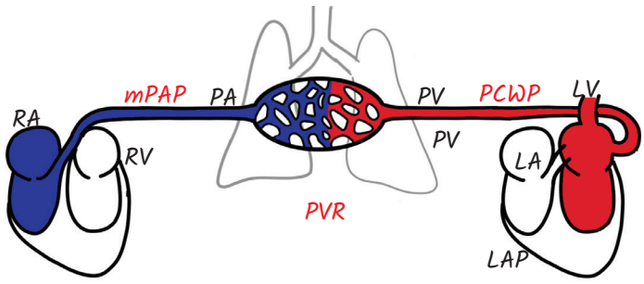


Hemodynamically stable

- Low Risk - D - Dimer
- High Risk - CTPA

PULMONARY HYPERTENSION (PH)

- Pulmonary hypertension is defined as mean Pulmonary arterial pressure ≥ 20 mmHg.



1	Pulmonary arterial hypertension (includes idiopathic and heritable, and disease related to drugs and schistosomiasis and portal hypertension)
2	Pulmonary hypertension due to left-sided heart disease
3	Pulmonary hypertension due to lung disease and/or hypoxia
4	Chronic thromboembolic pulmonary hypertension and other pulmonary artery obstruction
5	Pulmonary hypertension with unclear or multifactorial causes

Group 1

- Idiopathic – F : M = 3:1
- Familial Bone Morphogenic protein Receptor – 2 (BMPR – 2)
- CTD -Scleroderma, SLE
- Drugs - Methamphetamines, Anorexia
- Schistosomiasis

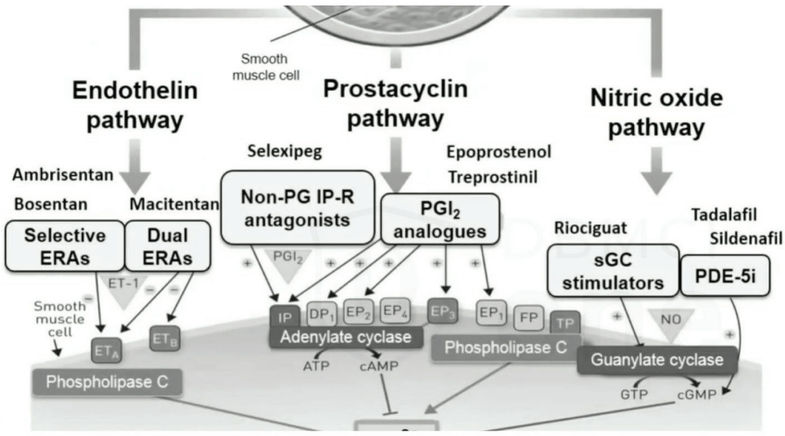
Clinical Features

- Dyspnoea
- Fatigue
- Chest pain
- Syncope
- Pedal edema

- O/E - RHF
- PFT- FEV1-N, FVC-N, Decreased DLCO, SaO2 decreases on saturation

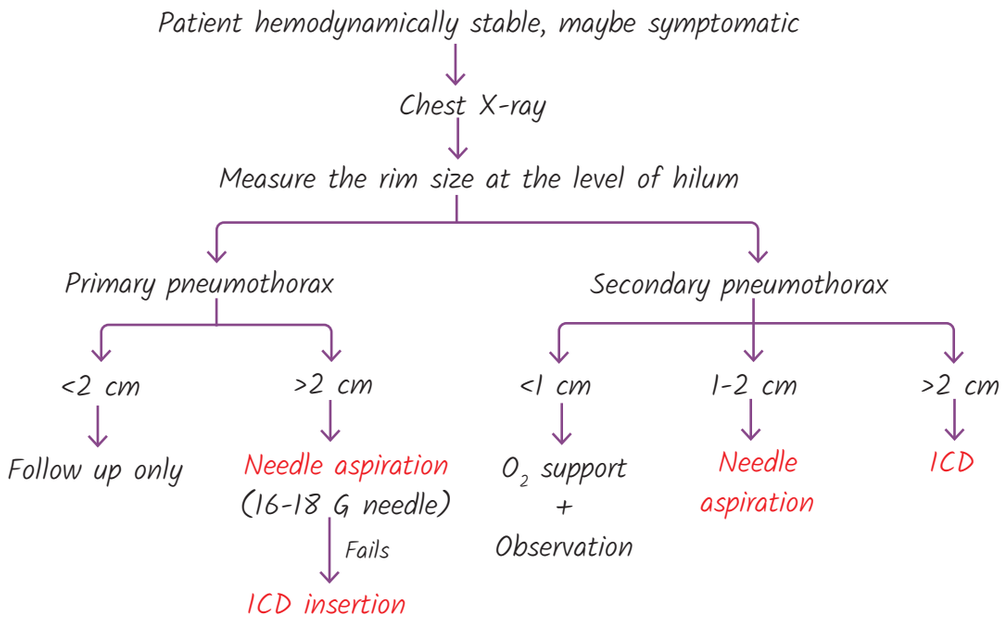
Treatment

- Vasoreativity test
- If +ve - use CCBs
- If -ve use - ERB + PDE5 inhibitor +/- IV prostacyclin analogues



- SOTATERCEPT→ Activin signalling inhibitors → ↓ PAH
- SELEXIPAG → Prostaglandin IP receptors agonist

PNEUMOTHORAX



PLEURAL EFFUSION

Light's Criteria

1. Pleural fluid / serum protein > 0.5
2. Pleural fluid / serum LDH > 0.6
3. Pleural fluid LDH > 2/3rd upper normal serum LDH

Any 1 positive - exudate

Transudative Effusions

- MC cause - cardiac failure
- Low oncotic pressure - Nephrotic syndrome
- Portal Hypertension
  - Cirrhosis → ascites → pleural effusion (hepatic hydrothorax)
- Peritoneal dialysis
  - Dialysate has no protein / LDH → transudative effusion
- SVC obstruction (High hydrostatic pressure)
- Urinothorax : low pH transudate
  - Clinical clue : Smell of urine during aspiration.
- Urine has no LDH / protein → looks like exudate based on Light's criteria.
- Atelectasis - collapsed lung → creates -ve pressure in pleural space → transudate

Exudative Effusions

- Parapneumonic effusion → pneumonia + effusion
- TB → Hypersensitivity to Ag in pleura
  - ↑ protein, ↓ glucose, lymphocytes ↑
  - ADA > 40 IU/L
  - IFN-γ > 140 pg/ml
- Empyema (frank pus)
- Malignancy
- Rheumatoid arthritis

  } Pleural fluid glucose ↓↓↓
- Pulmonary embolism → Hemorrhagic effusion, eosinophils (+/-)
- Esophageal rupture
- Pancreatitis

  } Pleural fluid amylase ↑
- Chylous effusion → Triglycerides ≥ 110 mg/dl
- Pseudochylous effusion → cholesterol ↑
- Vasculitis
- SLE
- Renal failure

INTERSTITIAL LUNG DISEASES

- IPF

CLINICAL FEATURES

1. Progressive dyspnea on exertion
2. Non-productive cough
3. Begin in the lower lobe and progress upwards.

DIAGNOSIS

Examination

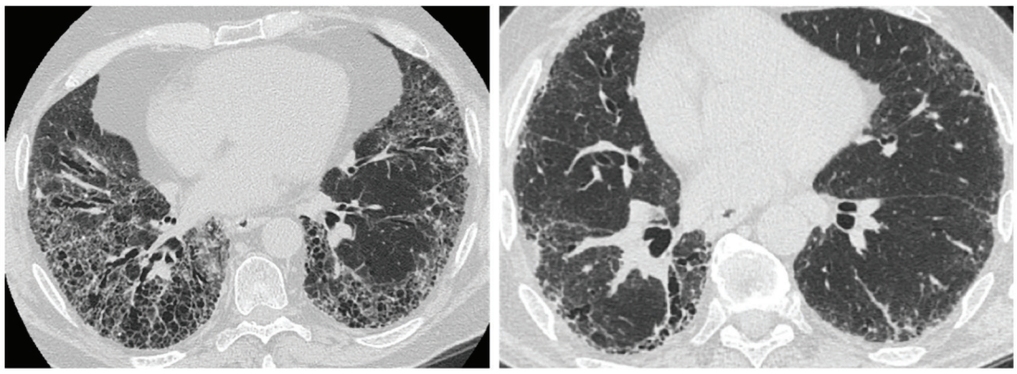
1. Clubbing
2. Cyanosis
3. SaO_2 ↓, Tachypnea
4. Rapid shallow breathing (to compensate for restricted lung volume)
5. B/L basal crepitations (hallmark finding) - Velcro crepitations
6. Exercise desaturation (on 6 minutes walk test)

Patho- UIP (Heterogenous pattern)

- Treatment
- Nintedanib
- Pirfenidone
- Antacids

HRCT

- Sub-pleural reticular opacities
- Lower lobes affected
- Fibrosis
  - Honeycombing
  - Traction bronchiectasis
  - Septal thickening



NON-SPECIFIC INTERSTITIAL PNEUMONITIS

- Age of occurence - slightly younger population (< 60 years)
- Seen in non-smokers
- Idiopathic
- Associated with CTDs (except Rheumatoid Arthritis → UIP pattern).
- Drugs
- Infection

CLINICAL FEATURES

1. Dyspnea on exertion
2. Non-productive cough

DIAGNOSIS

Examination

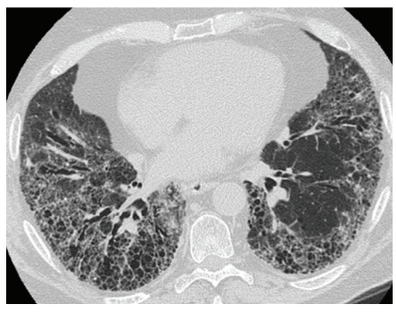
B/L crepitations in lower lobes

Investigations

HRCT

- Ground-glass opacities (GGO)
- Site - lower lobes
- Minimal fibrosis
- Honeycombing can occur (minimal)

} progressive



TREATMENT

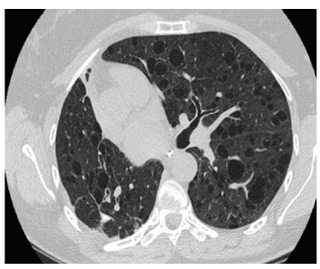
- Treat underlying cause
- Steroids + immunosuppressive therapy
- Nintedanib

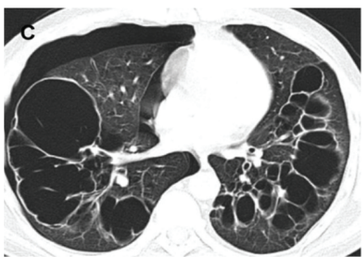
CYSTIC DISEASES OF THE LUNG

1. Lymphangioleiomyomatosis (LAM)
2. Pulmonary Langerhans Cell Histiocytosis (PLCH)

- Lymphangioleiomyomatosis (LAM)
- Females 3rd - 4th decade
- Pneumothorax +
- -ve (Non smokers)
- Cysts Thin walled
- Angiomyolipomas
  - Kidney
  - VEGF-D- ↑
  - Rx
  - Sirolimus (Rapamycin)

- Pulmonary langhans cell Histiocytosis (PLCH)
- Male
- Usually 20-40 yrs
- Pneumothprax +
- Smoking +
- Cysts Thick walled
- Rx
  - Smoking cessation
  - Supportive
  - Steroids + chemo Rx
  - Cladribine
  - Cyclophosph





PULMONARY ALVEOLAR PROTEINOSIS (PAP)

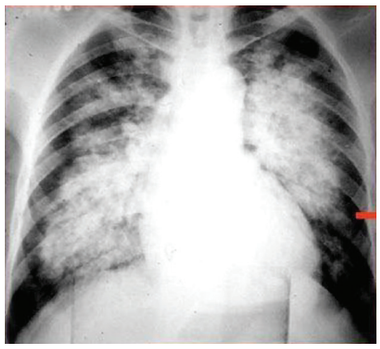
- Granulocyte macrophage colony stimulating factor (GM-CSF) → maturation of alveolar macrophages → degrades surfactant.

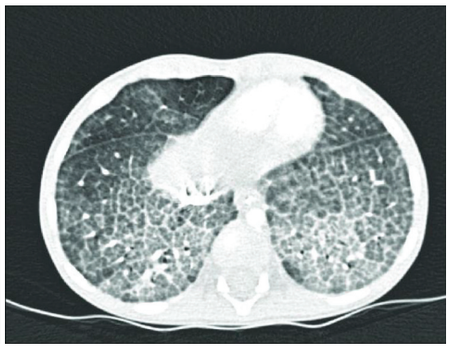
Primary PAP

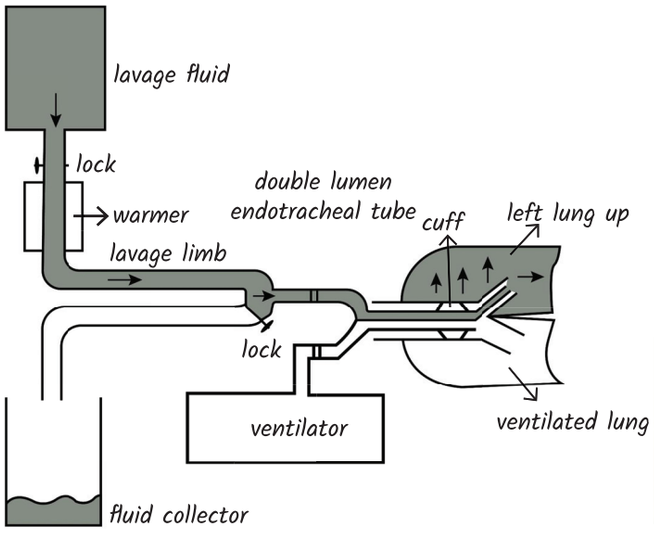
- Autoimmune PAP - Antibodies (IgG) attack GM-CSF.

Investigations

- Chest X-Ray → Bat wing appearance
- HRCT → Crazy pavement appearance
- BAL (bronchoscopy) → Surfactant positive
  - Milky exudates
  - Eosinophilic lipoprotein (PAS +ve)







ACUTE INTERSTITIAL PNEUMONITIS (AIP)

- Aka Hamman Rich Syndrome
- Unknown trigger
  - Infection may cause AIP.
- Acute presentation
- Rapid onset dyspnea
  - $SpO_2 \downarrow$
  - Tachypnea, tachycardia
- Treatment → Ventilator support + Supportive care
- Poor prognosis

RB-ILD AND DIP

- Associated with smoking (30-50 pack years)
- RB-ILD (Restrictive Bronchiolitis with ILD) - Involve upper lobe
- DIP - Start at lower lobe → involve entire lung

HYPERSENSITIVITY PNEUMONITIS

Working Industry	Antigen	Causative Organism
• Farmer	Mouldy hay	Thermophilic Actinomycetes
• Sugarcane	Bagasse	Thermophilic Actinomycetes
• Malt Worker	Contaminated barley	Aspergillus Clavatus
• Wood Dust	Wood dust	Bacillus subtilis
• Birds	Bloom antigen	Pigeon feathers
• Cheese Workers	Cheese	Penicillium
• Tobacco	Mould in tobacco	Aspergillus
• Hot Tub	Mould	Cladosporium

CLINICAL EXAMINATION OF HP

1. Exposure history & temporal Association
2. Clubbing is rare
3. Identifying the offending antigen (more sensitive)
4. Presence of precipitating antibodies (present in normal individuals exposed to the same antigens too)
5. Recurrent episodes of symptoms.
6. Symptoms within 4-8 Hours of exposure to antigen
7. Cough Independent of inspiratory crackles
8. Weight loss

BAL

Lymphocytes CD8 > CD4

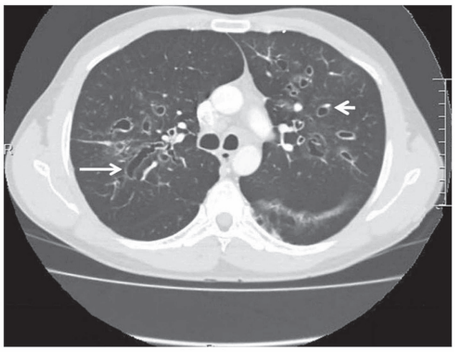
HRCT

- Centrilobular nodules.
- Mosaic Pattern: Ground glass opacities

ABPA

Minimal Diagnostic criteria - ABPA.

- Asthma
- Blood - eosinophilia, ↑ IgE, precipitates antibodies against aspergillin.
- Central bronchiectasis.
- Derma - skin test to aspergillus.



Treatment

- Steroids + itraconazole / Voriconazole ± IL-5i

SARCOIDOSIS

- Multi system, granulomatous, chronic inflammatory disease
- Unknown etiology
- Involved : Lungs, skin, eye, lymph nodes, Reticulo-endothelial system
- Granuloma : Well defined, clear, non-caseating
- It is a diagnosis of exclusion (TB, fungal infections are differentials)

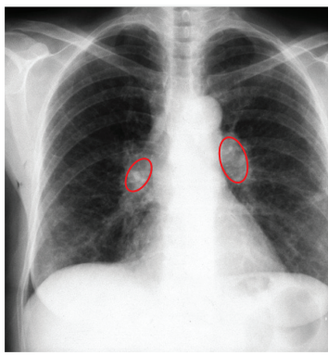
PATHOLOGY

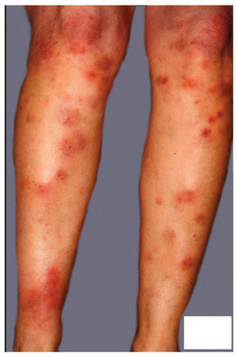
- Hallmark : Non caseating, well defined granulomas
- Histo-Pathological Examination : Inclusion bodies seen :
  - Schaumann bodies
  - Asteroid bodies
  - Residual bodies
  - Crystalline inclusion bodies

CLINICAL FEATURES

- Constitutional - fever, weight loss, fatigue, arthralgia, arthritis
- Lofgren's presentations :
  - Arthritis/Polyarthritis - ankle, knee
  - Skin - Erythema nodosum - painful lesions in lower limb

- Fever
- Chest X-ray : suggestive of bilateral hilar lymphadenopathy.
- Treatment - NSAIDs (No role of steroids)



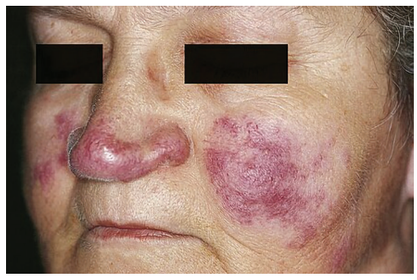


SCADDING Stages for pulmonary involvement :

Stage :

1. B/L Hilar Lymphadenopathy (B/L HLN)
2. B/L HLN + Parenchymal involvement (infilterates in interstitium in upper and mid zone) (ILD)
3. B/L extensive parenchymal infilterates
4. Fibrosis + Fibrotic bands + Traction Bronchiectasis

- Upper respiratory tract involvement -
  - Hoarseness of voice
  - Sinusitis
  - Saddle nose deformity
- Heerfordt's syndrome : Uveo-parotitis
- Endocrine :
  - Hypopituitarism
- Cardiac :
  - Arrhythmia, heart blocks
  - Cardiomyopathy (dilated or restrictive) (patchy disease - Cardiac MRI - helps)
- Reticuloendothelial system :
  - Hepatomegaly, Jaundice : LFT - Abnormal
  - Lymphadenopathy
  - Spleenomegaly
- Blood :
  - Anemia, thrombocytopenia, leukopenia
  - Increased Calcium - in serum and urine
  - Lupus Pernio
- ACE levels - Increased
- Lymph node biopsy - well defined, non caseating granulomas



Treatment

Steroids used in

1. Progressive pulmonary disease
2. Skin - Disfiguring lesions
3. ↑ Calcium (persistent).
4. Renal/Liver dysfunction.
5. Uveitis not responding to topical steroids.
6. Hepatospleenomegaly.
7. Cardiac involvement.

- Methotrexate
- Azathioprine
- Anti TNF
- Rituxumab

NOTES

Causes of Giant 'a' Waves

- Tricuspid stenosis
- Pulmonary stenosis
- Pulmonary hypertension
- RV - Diastolic dysfunction

Cannon 'a' Waves

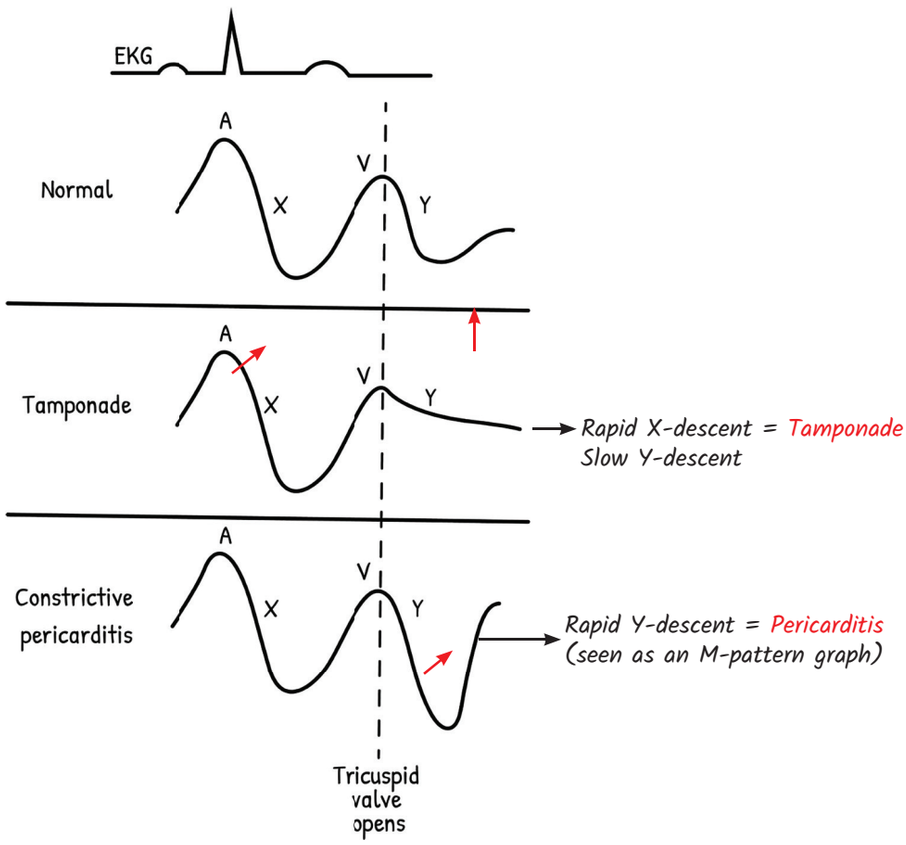
- A-V Dissociation
- Complete heart block
- Ventricular tachycardia
- Ventricular ectopics

cv - Waves (Absent x' and CV merges)

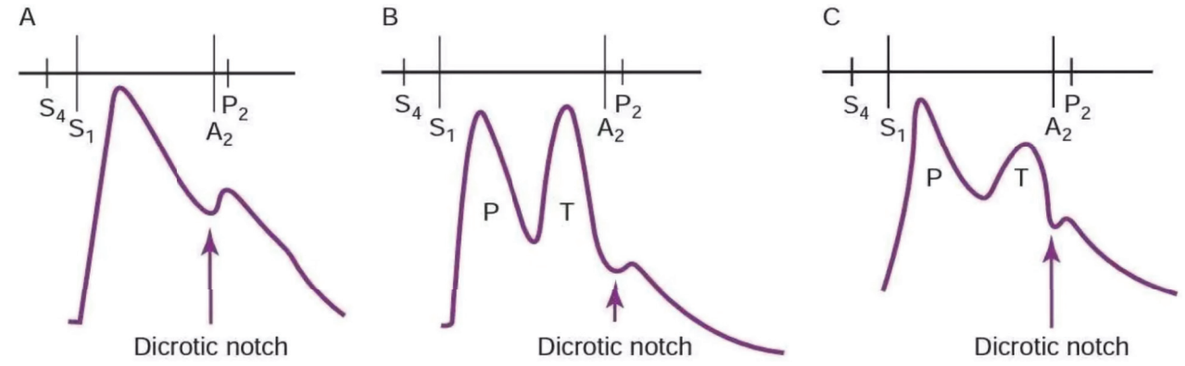
- Tricuspid regurgitation

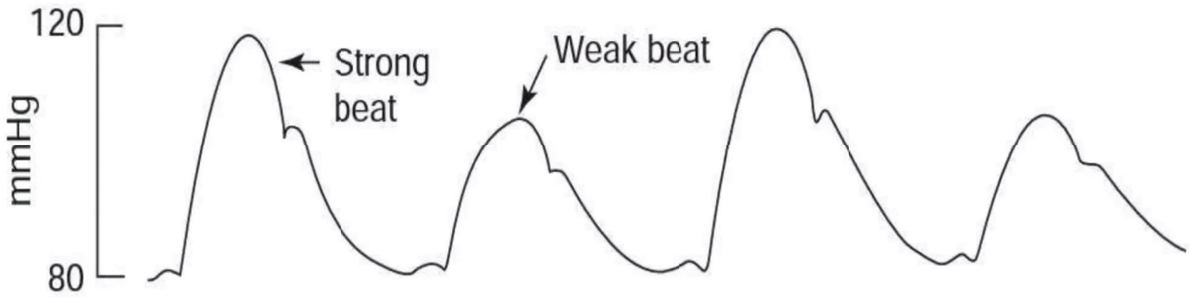
Giant 'v' Waves

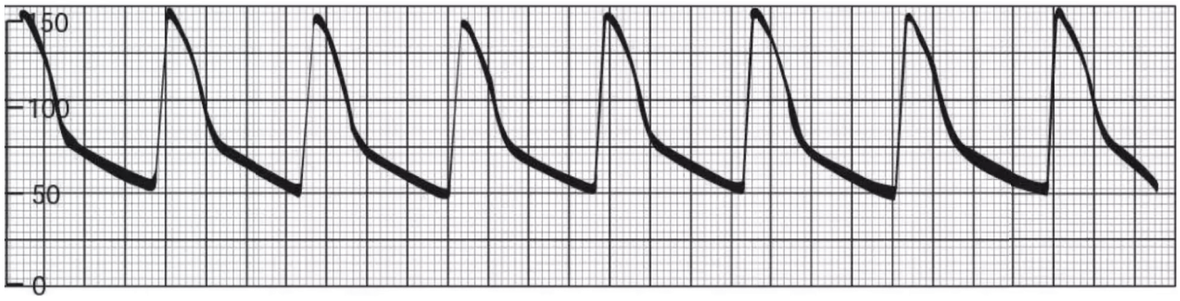
- Tricuspid regurgitation
- ASD (Initially giant 'a' waves)



Pulse







Collapsing pulse causes

- *Fever*
- *Thyrotoxicosis*
- *Exercise*
- *Aortic regurgitation*
- *Anemia*
- *Pregnancy*

HYPERTENSION

Stage	SBP		DBP
1	≥ 140 mm Hg	or	≥ 90 mm Hg
2	≥ 160 mm Hg	or	≥ 100 mm Hg
3	≥ 180 mm Hg	or	≥ 110 mm Hg

HTN EMERGENCY

Neuro

Indicated

- Labetalol
- Nicardipine
- Clevidipine

Contraindicated

- Nitroprusside
- Nitroglycerine
- Methyldopa
- Clonidine

Cardiac- Ischemia

- NTG + labetalol / esmolol
- Nicardipine + esmolol

Contraindicated

- Hydralazine
- Minoxidil
- Nitroprusside
- Diazoxide

ISCHEMIC HEART DISEASE

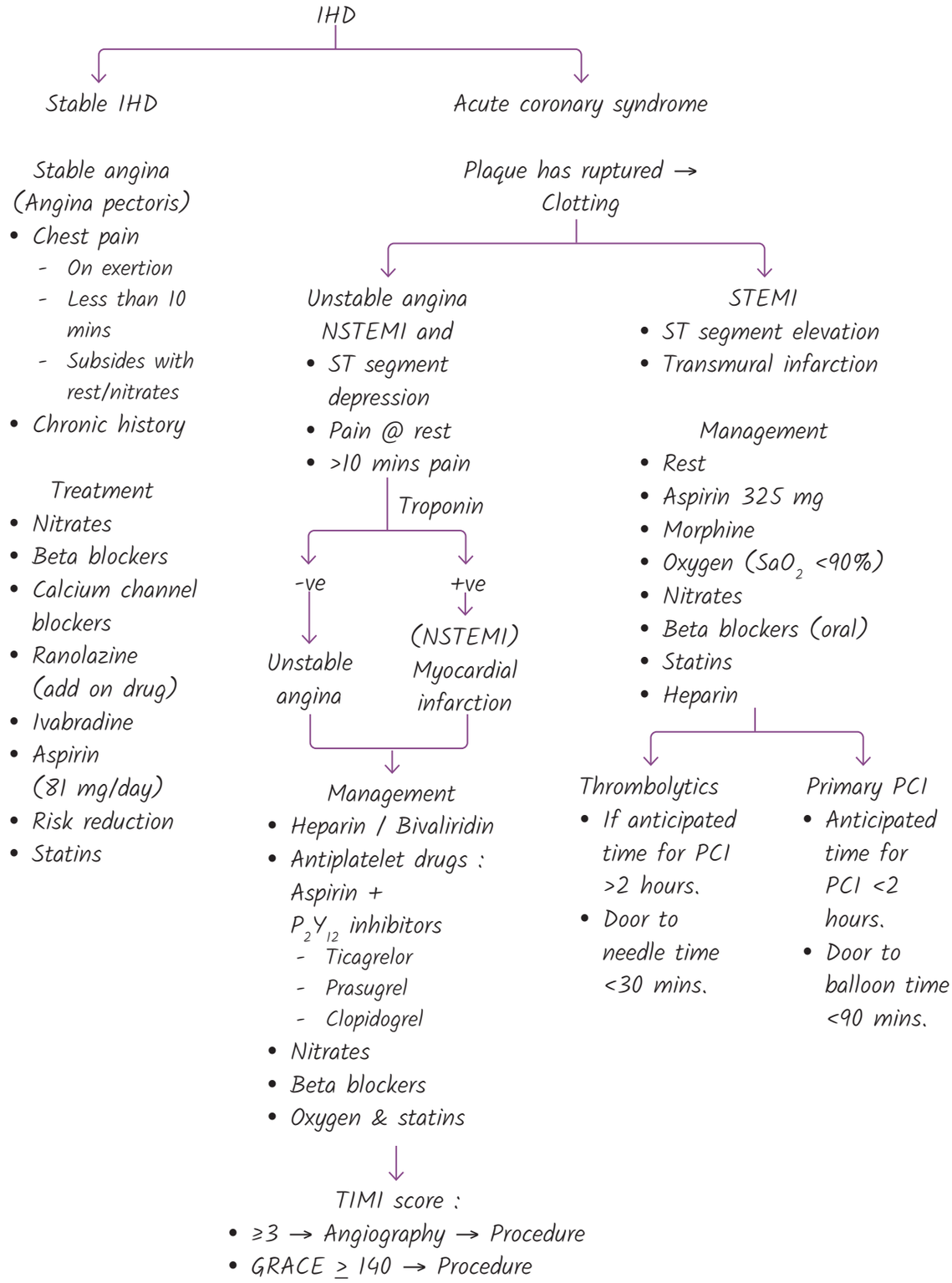
Dyslipedemia

- Bempedoic acid- an ATP citrate lyase inhibitor

Inclisiran

- MOA-RNA interference (RNAi) to silence the gene for PCSK9, a protein that degrades LDL receptors

Colchicine
Reduces inflammation
Decreases MACE post MI and also beneficial in stable CAD

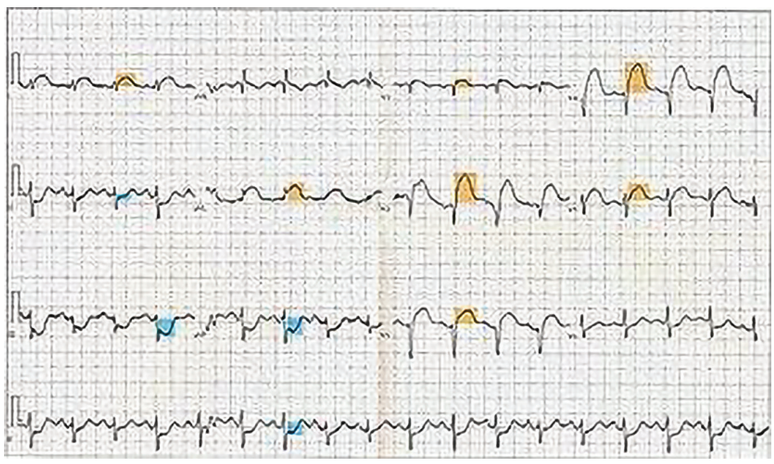

IHD
Stable IHD
Acute coronary syndrome
Stable angina (Angina pectoris)
• Chest pain
- On exertion
- Less than 10 mins
- Subsides with rest/nitrates
• Chronic history
Treatment
• Nitrates
• Beta blockers
• Calcium channel blockers
• Ranolazine (add on drug)
• Ivabradine
• Aspirin (81 mg/day)
• Risk reduction
• Statins
Plaque has ruptured → Clotting
Unstable angina NSTEMI and
• ST segment depression
• Pain @ rest
• >10 mins pain
Troponin
-ve
+ve
Unstable angina
(NSTEMI) Myocardial infarction
Management
• Heparin / Bivaliridin
• Antiplatelet drugs : Aspirin + P_2Y_{12} inhibitors
- Ticagrelor
- Prasugrel
- Clopidogrel
• Nitrates
• Beta blockers
• Oxygen & statins
TIMI score :
• ≥3 → Angiography → Procedure
• GRACE ≥ 140 → Procedure
STEMI
• ST segment elevation
• Transmural infarction
Management
• Rest
• Aspirin 325 mg
• Morphine
• Oxygen (SaO_2 <90%)
• Nitrates
• Beta blockers (oral)
• Statins
• Heparin
Thrombolytics
• If anticipated time for PCI >2 hours.
• Door to needle time <30 mins.
Primary PCI
• Anticipated time for PCI <2 hours.
• Door to balloon time <90 mins.

Leads ST Elevated	Anatomic Location	Coronary Artery
II, III, aVF	Inferior	RCA >> LCx
V_2-V_4	Anterior	LAD
V_1-V_4	Anteroseptal	LAD
I, aVL, V_5, V_6	Lateral	LCx > LAD

POST MI

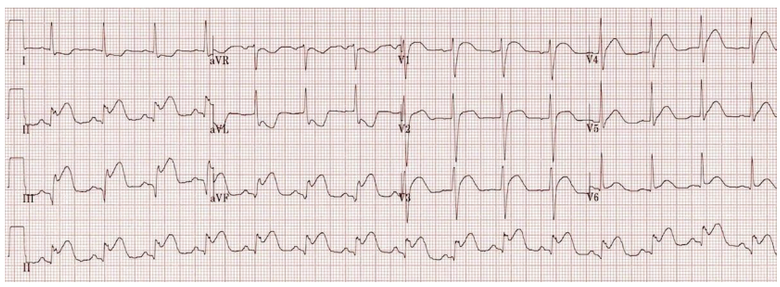
- DAPT- aspirin + clopidogrel / Ticagrelol / Prasugrel
- If atrial fib occurs- then stop aspirin and add a DOAC
- Statins, ACEI and Betablockers

ALWMI

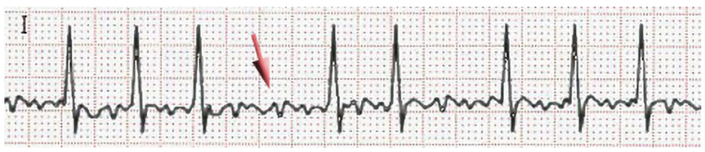


IWMI

- Avvsd
- Nitrates
- Diuretics



ARRYTHMIAS



Rate control

- Beta blockers
- CCB (diltiazem / verapamil)
- Digoxin

Rhythm reverters

- Ibutilide
- Dofetolide
- Amiodarone
- Dronaderone
- Procainamide

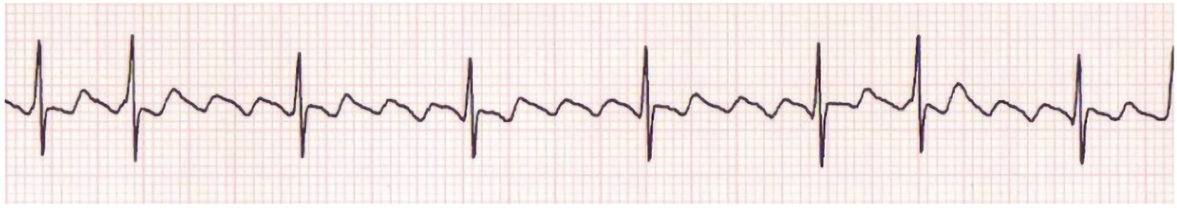
Anti Coagulation

- If cause is valvular A-fib (MS or prosthetic valve disease) - Warfarin
- If cause is non valvular A-fib - CHADVASC score (gives risk of stroke)
  - If score >2 : Anticoagulation (DAOC)
  - If 0 or 1 : No treatment

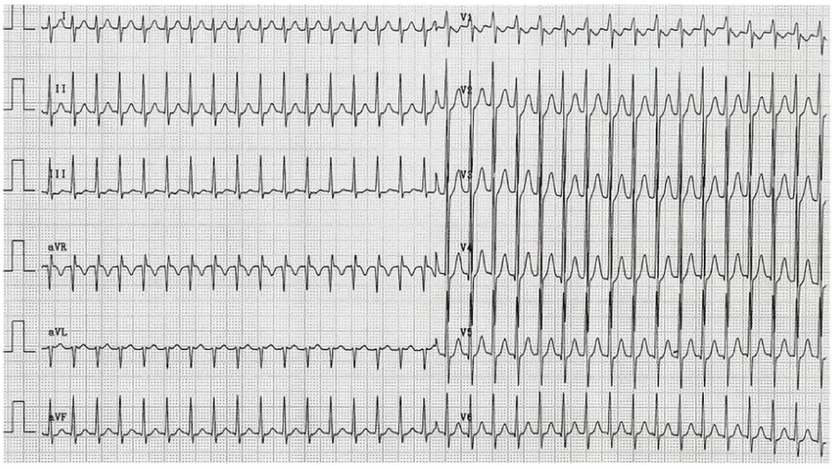
Risk Factor	Points
Congestive heart failure	1
Hypertension	1
Age ≥ 75 yrs	2
Diabetes mellitus	1
Stroke / TIA / Embolus	2
Vascular disease	1
Age : 65-75 yrs	1
Sex : Female	1

In patients with atrial fibrillation duration of greater than 48 hours (or unknown duration), a minimum of 3 weeks of anticoagulation. And post shock- 4 weeks anticoagulation

A Flutter



SVT

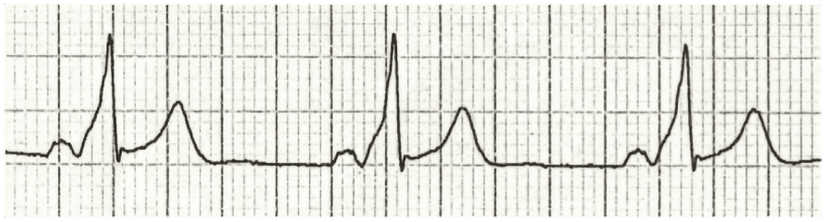


Treatment

- Unstable - Shock
- Stable
- Carotid massage
- Inj. Adenosine 6mg IV - 12mg IV

- β-blockers
- CCBs (Diltiazem / Verapamil)

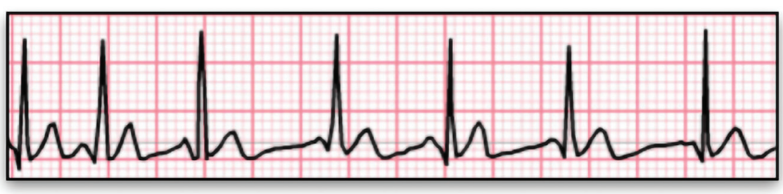
WPW



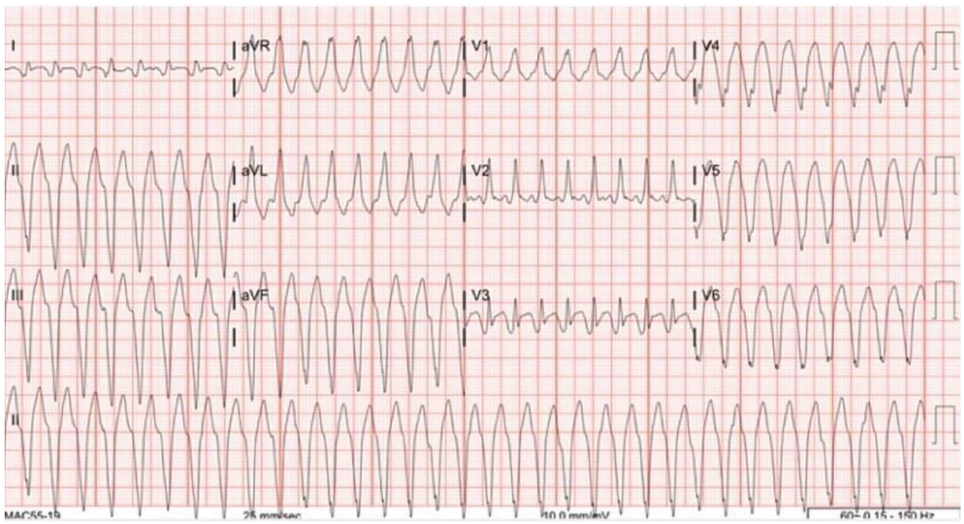
Treatment

- Class Ia, class Ic & class III anti arrhythmic drugs / A-fib + WBW (Avvsd- AV NODE blocker)
- Radiofrequency ablation of accessory pathway.

MAT



Monomorphic VT

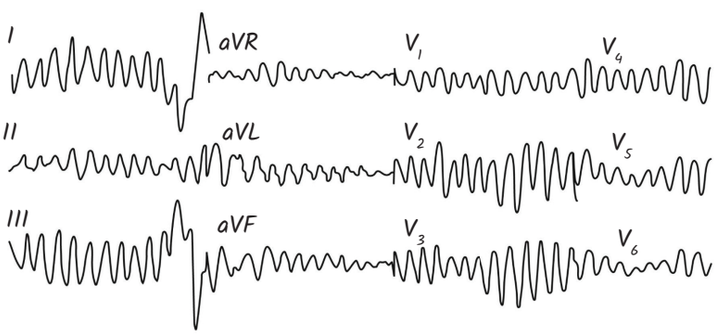


Treatment

- Hemodynamically unstable patient → Shock the patient
- Hemodynamically stable patient →
  1. Amiodarone
  2. Procainamide
  3. Lignocaine (faster action)
  4. Alternative treatment : Shock the patient after giving anaesthesia

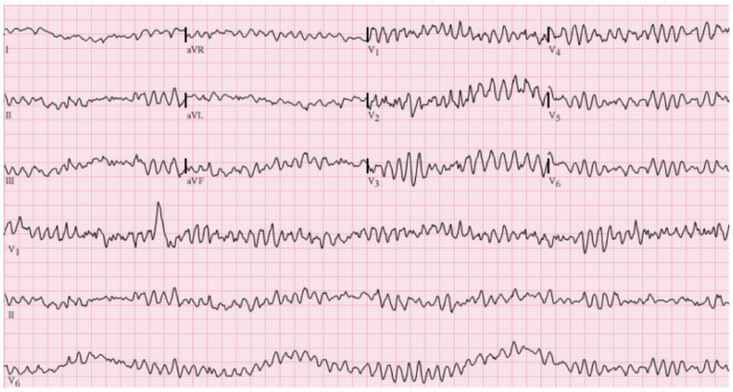
Polymorphic VT

- Causes of long QT
  - Macrolide and fluoroquinolone antibiotics (especially Moxifloxacin)
  - Terfenadine and Astemizole (antihistamines)
  - Antipsychotic and antidepressant medications
  - Methadone
  - Anti fungal medications - Ketoconazole
  - Class Ia and class III antiarrhythmics
  - Hypokalemia
  - Hypomagnesemia
  - Alcohol



Treatment

- Shock in hemodynamically unstable patient.
- IV $MgSO_4$: Main drug in both stable & unstable patient



V Fibrillation

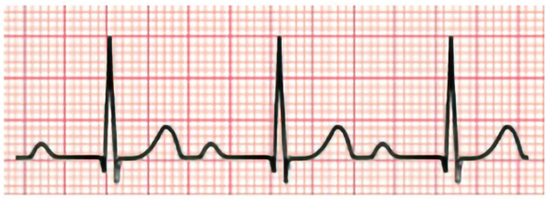
- Multiple re entry circuits in ventricle.
- ECG - wavy baseline

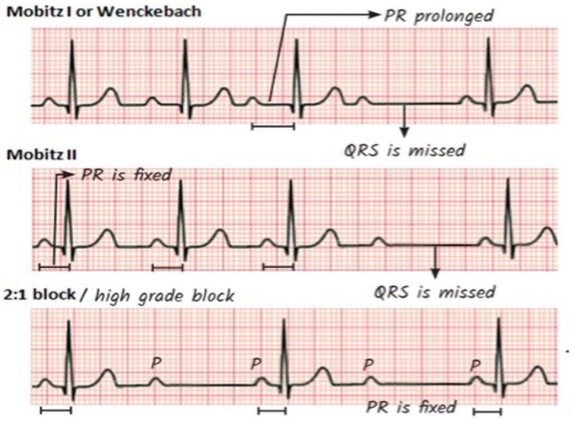
- Treatment : Shock

CPR

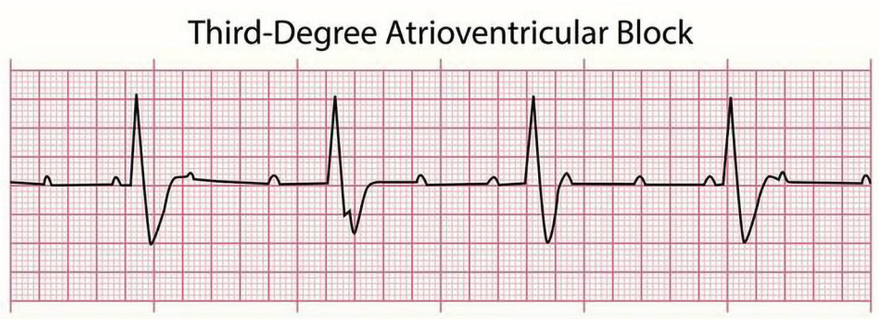
- Chest compression : Ventilation = 30:2
- Rate : 100-120 / minute. Allow for recoil
- Depth = 5-6 cm (2-2.4 inches)

First degree AV block





- Complete Heart Block



HEART FAILURE

Can be systolic heart failure or diastolic heart failure.

HFrEF = SV(↓)/EDV = <40% Systolic Heart Failure

Heart Failure with reduced EF (HFrEF)

HFpEF = (EDV-ESV)/EDV = SV(↓)/(EDV(↓ ↓) = >50%) EDV

Heart Failure with preserved EF (HFpEF) - Diastolic Heart Failure

Heart Failure with Midrange Ejection Fraction (HFmEF) = 40-50%.

Heart Failure with improved Ejection fraction (HFiEF)

TREATMENT

Stage C - For HFrEF (systolic heart failure).

- Medications :
  - Diuretics.
  - Beta Blockers.
  - Angiotensin Receptor Blocker + Neprilysin Inhibitors (ARNI).
  - Aldosterone receptor antagonist.
  - SGLT-2 Inhibitors.
  - Hydralazine + sorbitrate (renal dysfunction patients - ↓ GFR).
  - Ivabradine. (Isosorbide)
  - Vericiguat.
  - Digoxin.
  - Fantastic 4 → βB + ARNI + MRA + SGLT-2i

CARDIAC RE-SYNCHRONIZATION THERAPY

Used when QRS > 0.15 sec.

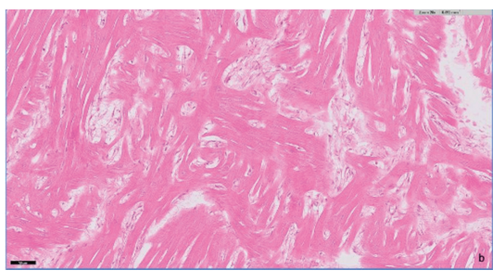
Treatment of HFpEF

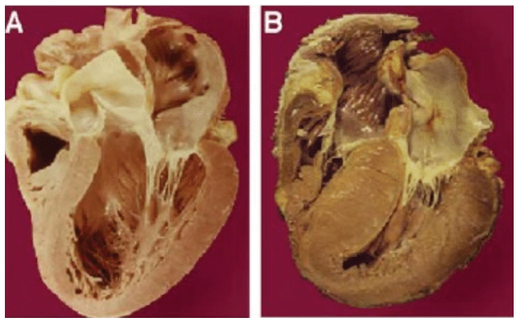
- SGLT 2 inhibitor - main drug
- Mineralocorticoid receptor antagonist (spironolactone)
- Angiotensin receptor-neprilysin inhibitor - sacubitril + valsartan
- Angiotensin receptor blocker (candesartan)
- Control BP, treat A fib, use cautious diuretics

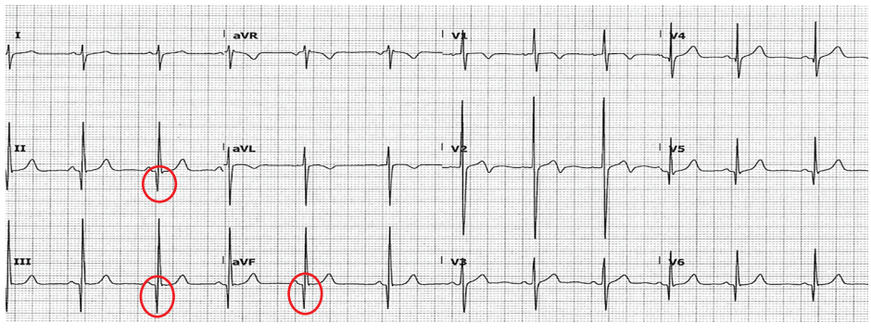
CARDIOMYOPATHY

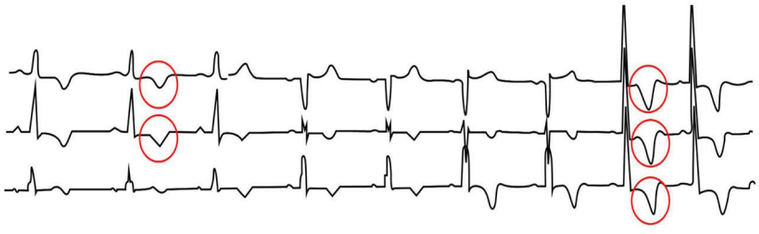
Hypertrophic Cardiomyopathy

- Sudden Cardiac death (SCD) rate : 0.8-1% /year.
- AD inheritance variable penetrance
- Genetic defect :
  - β myosin - Heavy chain.
  - β myosin Binding protein C.
- Septum is asymmetrically enlarged.
- Anterior & inferior septum most commonly involved.
- Yamaguchi ⟶ Apical involvement (ECHO- spade like and ecg diffuse T wave inversions)









Drugs

- Drug of choice - β-blocker
- If contraindicated - Verapamil
- 2nd line drug : Disopyramide (negative inotrope).
- New drug : Mavacamten ⟶ reduces mortality.
- Mavacamten : reversibly inhibiting cardiac myosin ATPase, which reduces the number of actin-myosin cross-bridges that form in the heart. This decreases excessive muscle contraction (hypercontractility)
  - Better relaxation of muscle
  - ↑ LV filling - reduces obstruction

  } New drug → AFICAMTEN → Myosin inhibitor
- If refractory to above drugs : Mechanical therapy

TAKOTSUBO CARDIOMYOPATHY

Synonyms

- Broken heart syndrome - triggered by emotional stress
- Neurogenic myocardial stunning

CLINICAL FEATURES

- Acute presentation (mimics STEMI).
  - Chest pain.
  - No/mild coronary artery occlusion.
- More common in post menopausal women.

Echo - Basal Hyperkinesia and mid and apical hypokinesia

Treatment

- Cardiogenic shock - Intra-aortic balloon pump (IABP)
- Avoid inotropic agents
- Anticoagulation - for LV thrombus (poor contraction → stagnation of blood).
- Treat ventricular arrhythmias (if present on monitoring).
- ACEI and Betablockers

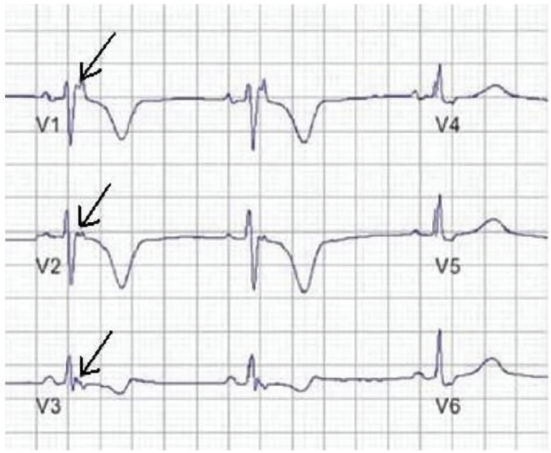
Arrhythmogenic cardiomyopathy

- Definition & Pathophysiology
- Genetic myocardial disease with fibrofatty replacement → arrhythmias, SCD, progressive dysfunction.
- Involves desmosomal (PKP2, DSP, DSG2, DSC2, JUP) and non-desmosomal genes.
- Broader concept than ARVC: includes right-dominant, biventricular, and left-dominant phenotypes.

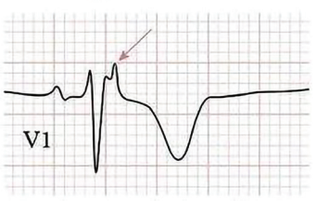
Clinical Features

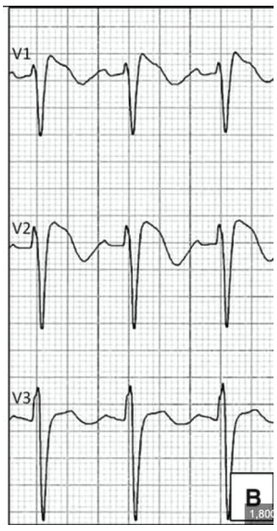
- Arrhythmias: VT, VF, palpitations, syncope, sudden cardiac death.
- Symptoms: palpitations, presyncope, syncope, HF symptoms late.
- Course: May progress to ventricular dilation and heart failure.

ECG- T wave inversion in lead III, VI-V3
Epsilon wave in VI , V2



ARVC





Brugada

Brugada

- Na channel defect
- Increase risk of ventricular arrhythmias
- Fever increase risk of ventricular arrhythmias
- Rx- ICD
- Quinidine

Treatment → ARVC

- Avoid competitive/strenuous athletics.
- Drugs: β-blockers, sotalol, amiodarone; HF therapy if dysfunction.
- ICD: cornerstone for preventing SCD in high-risk patients.
- Ablation: for recurrent VT; not curative.
- Advanced/novel: STAR (Stereotactic arrhythmia radioablation)

RESTRICTIVE CARDIOMYOPATHY

- Diastolic dysfunction in a patient who does not have enlarged muscle.
- Muscle is not hypertrophied (as in hypertrophic cardiomyopathy).
- Discreet muscle makes it non-stretchable (Restrictive physiology).
  - Diastolic dysfunction.
  - Non-compliant ventricle, inner cavity small.
  - Filling defect.

Myocardial	Endomyocardial

• Noninfiltrative
  - Idiopathic
  - Familial
  - Scleroderma
• Infiltrative
  - Amyloidosis
  - Fabry's
  - Sarcoidosis
• Storage disease
  - Gaucher's
  - Haemachromatosis
	• Endomyocardial fibrosis
	• Hypereosinophilia
	• Carcinoid heart disease
	• Metastatic cancer
	• Radiation
	• Anthracycline toxicity
	• Drugs (ergotamine)
	• Prior cardiac surgery

Clinical Features

- RHF features > LHF features
- RHF (prominent)
  - Elevated JVP
  - Pedal edema
  - Tender hepatomegaly
  - Ascites

GENERAL TREATMENT FOR RCM

- Cautious use of diuretics, low dose β-blockers.
- Avoid digoxin.
- Treat the underlying cause.
- Wild ATTR – Tafamidis / ACORAMADIS
  - Stabilizes tetramer (Prevents breaking and deposition between heart cells)
- Eplontersen (anti-sense oligonucleotide) - ↓ TTR production (RNAi)

Dilated cardiomyopathy

- Systolic Heart Failure

Causes

- Idiopathic/Genetic (Familial - m/c)
  - Titin
  - ß- myosin heavy chain (MYH7)
- Viral infection
- Alcohol
- Drugs - Doxorubicin, SFU, Trastuzumab
- Peripartum cardiomyopathy.
- Sarcoidosis.
- Hemochromatosis.
- Infectious or myocarditis.
- Eosinophilic cardiomyopathy.

Treatment

Treat like HFrEF

Peripartum CM- Bromocriptine

NOTES

NEUROLOGY

HEADACHE

Migraine	Tension type	Cluster
Females > Males	Females > Males	Males > Females
Unilateral	Bilateral	Unilateral
Throbbing	Band like compressive	Deep piercing Peri orbital
One day		60 Mins → Episodic ↓ Multiple
Migraine associated features	**Feature less headache**	**Features**
• Nausea, vomiting	X	+
• Photophobia	±	+
• Phonophobia	±	+
• Osmophobia	X	+
• Movement → Headache	X	Restless
• Disabling		Autonomic → lacrimation → Red eye → Nasal congestion

Treatment-Migraine

- NSAIDS/Triptans
- Gepants (small-molecule CGRP receptor antagonists)
- Ubrogepant, rimegepant are approved for acute migraine. headache
- Ditans
- Lasmiditan (a selective 5-HTIF agonist) – If vascular contraindications for triptans.
- Intranasal third-generation agents
- Zavegepant (nasal spray) is a CGRP receptor antagonist approved for acute migraine.

Prevention

- Beta blockers
- Topiramate
- Valproate
- Monoclonal antibodies
  - Erenumab (S.C) → GRP-Rs antibody (Galacanezumab)
  - Galcanezumab (S.C)
  - Fremenezumab (S.C)
  - Eptinezumab (I.V)

  } CGRP antagonists
- Botulinum toxin
- TCA-Amitriptyline
- CCB-Flunarizine

PARKINSONS DISEASE

- T : Tremor
- R : Rigidity
- A : Akinesia / Hypokinesia
- P : Postural reflex abnormalities
- Micrographia
- Masked facies
- Reduced eye blinking
- Drooling
- Soft voice (hypophonia)
- Dysphagia
- Freezing
- Anosmia
- Sensory disturbances (e.g., pain)
- Mood disorders (e.g., depression)
- Sleep disturbances (e.g., RBD)
- Autonomic disturbances
  - Orthostatic hypotension
  - Gastrointestinal disturbances
  - Genitourinal disturbances
  - Sexual dysfunction
- Cognitive impairment / Dementia

Treatment

- Levodopa + Carbidopa
- Foslevodopa-Foscarbidopa- SC inj to reduce off time
- Dopamine agonists
  - Oral Pramipexole

Ropinirole

- Skin patch: Rotigotine
- Subcutaneous injection: Apomorphine (used in off phenomenon)

Catechol-O-Methyl Transferase Inhibitors

- Entacapone
- Opicapone (newer drug)

Monomine Oxidase B Inhibitors

May have a protective effect on neurons.

- Selegiline
- Rasageline
- Safinamide

Anticholinergic Drugs

MOA : Re-establishes balance between acetyl choline & dopamine (therefore reduces tremor).

- Trihexyphenidyl
- Benztropine

Rx of Neuropsychiatric Complications of Dopamine

Rx- parkinson disease psychosis

- Pimavanserin (inverse serotonin antagonist)
- Quetiapine
- Clozapine

Istradefylline : A2A antagonist

- Deep Brain stimulation
- Areas targeted for PD
  - Subthalamic nuclei
  - Globus pallidus interna

Levadopa induced dyskinesia → Rx- amantadine

ALZHEIMERS DISEASE

Clinical Features

- 4 A's:
  - Amnesia : Episodic memory loss (earliest symptom). Mainly anterograde amnesia, can be retrograde
  - Apraxia : Loss of learned skills
  - Agnosia : (Object agnosia, Visual agnosia, Direction agnosia)
- Aphasia : naming affected. Logopenic aphasia : Unable to find appropriate words, progressive, word-finding pauses, anomia

Investigations

- CSF : ↑ Tau, ↓ amyloid, ↑ neurofilaments.
- Blood : Early sign → ↑ phosphorylated tau assay, ↑ neurofilaments (P-Tav-217)
- Imaging :

a. MRI :

- Early → Hippocampal (medial-temporal lobe) atrophy,
- Later → Diffuse cortical atrophy, enlarged ventricles.

b. PET : Tau-labelled and amyloid-labelled. Can detect early changes.

Treatment

Acetylcholine is needed in the nucleus of Meynert.

1. Acetylcholinesterase inhibitors : Eg: Donepezil, Rivastigmine, Galantamine.
2. NMDA antagonist : Memantine - Used in late stages
3. Monoclonal antibodies act on Aβ42 : Eg: Lecanemab, Donanemab.

LEWY BODY DEMENTIA

Clinical Features

Core Features

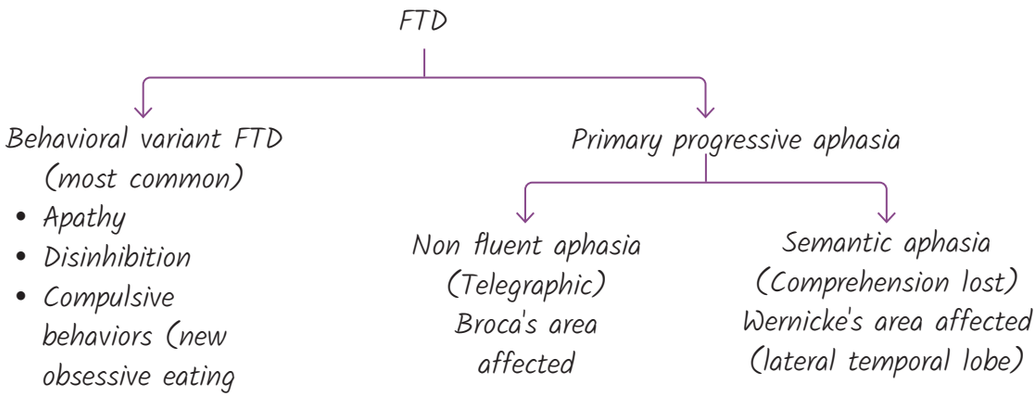
- Fluctuating cognition (Episodic delirium)
- Parkinsonism : Rigidity + Hypokinesia
- Visual hallucinations
- REM Behavioural disorder (Sometimes considered a core feature)

Supportive/suggestive

- *Capgras syndrome*
- *↑ Sensitivity to Anti-psychotics (especially Ist gen)*
- *↑ Risk of neuroleptic malignant syndrome*
- *↓ Dopamine activity in basal ganglia seen on SPECT/PET*
- *Autonomic dysfunction*
- *Depression*
- *Cingulate island sign - on PET scan occipital lobe shows hypometabolic activity*
- *Hypo-osmia/Excessive Daytime Sleepiness (EDS)*

Rx- rivastigmine, clonazepam (RBD), memory - Ⓝ

FTD

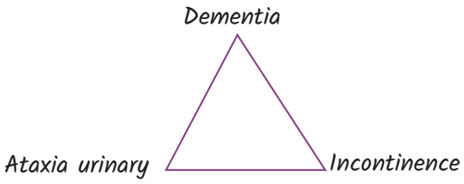


Drawing : Spared

NORMAL PRESSURE HYDROCEPHALUS (NPH)

- No papilledema
- Imaging → Large ventricles seen
- Reversible

Clinical Features (Triad)



Treatment

- V-P shunt

MOTOR NEURON DISEASE

Spinomuscular atrophy/ Progressive muscular atrophy

- B/L - symmetric weakness (hallmark)
- Wasting, fasciculations (particularly tongue, due to 12th CN involvement)
- Extraocular muscles spared.
- Face muscles spared.
- Intellectual development - Normal
- Bulbar involvement +
- Nasal regurgitation
- Frog like appearance
- Jug handle position of hands (due to inability to lift hands).
- Bell-shaped chest (due to muscle atrophy).
- Deep tendon reflexes (DTR) : Absent.

Treatment

- Curative treatment
  - Onasemnogene (SMN-1 gene therapy)
- Nusinersen
  - Mechanism of action - I SMN-2 activity and blocks splicing
- Risdiplam
  - Mechanism of action - modify splicing → increase protein production.
- Reldesemtiv
  - Mechanism of action - increase muscle function

Amyotrophic Lateral Sclerosis

Clinical Features

- Incidence - Males > Females
- Onset - 6th decade (typical)
- Mixed pattern of disease : UMN + LMN
- Asymmetric progressive weakness

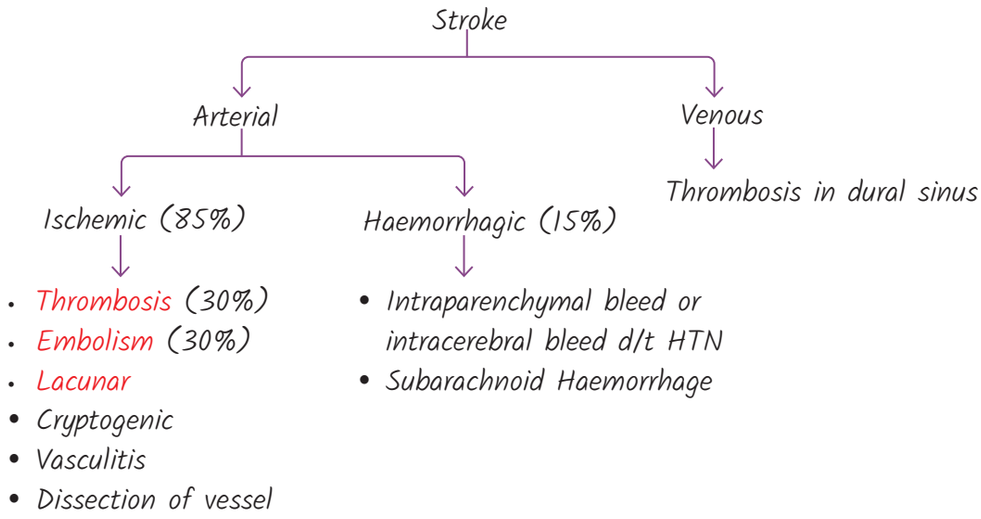
  - Spastic gait (tend to fall on uneven floor)
  - Muscle wasting in upper limb
- Sensory system - Normal
- Bladder - normal
- Bulbar manifestations + (hoarseness, dysphagia, dysarthria, regurgitation)
- No involvement of extra-ocular muscles.
- CSF findings - Normal
- Split hands
  - Thenar muscles atrophied.
  - Hypothenar muscles preserved / minimally atrophied.
- Higher mental functions - Normal
  - Except in 20% - they develop FTD (Fronto-Temporal Dementia)
- HPE
  - Bunina bodies
  - TDP-43 inclusions accumulate (toxic to neurons).
- Sporadic > Familial

Treatment

- Riluzole → deceases glutamate
- Edaravone → antioxidant, TOFERSEN [ASO- for SOD-1 ALS] → ↓ neurofilament light chains

Myasthenia Gravis	**Lambert Eaton syndrome**
Post-synaptic involvement	Pre-synaptic involvement
Antibodies formed are : • Anti-Ach receptor • Anti-MUSK • Anti-LRP-4	Antibodies against Ca2+ channel
Earliest symptoms : • Ptosis • Diplopia	Earliest symptoms : • Proximal muscle weakness • Dysphagia, Autonomic features +
Deep tendon reflexes present	Deep tendon reflexes (DTR) ↓/absent ↓ Exertion ↓ DTR becomes normal
No autonomic dysfunction	Autonomic dysfunction is a classical sign • Dry mouth • Sweating • Bladder abnormalities • Sexual dysfunction
EMG repetitive nerve stimulation shows decremental response	EMG repetitive nerve stimulation shows incremental response

STROKE



Stroke Management

MULTIPLE SCLEROSIS

- Autoimmune disease
- Demyelinating disease
  - Environmental factor (EBV, Vitamin D deficiency)
- Females > Males (3:1)
- Age group affected - 15-50 years (MC in 15-30 years)

Disseminated Disease in Space (DIS)

2 out of 4 Zones must be involved:

1. Juxta cortical / cortical zone
2. Periventricular Zone - Dawson's finger seen in MRI.
3. Infratentorial area
   a. Brain Stem
   b. Cerebellum
4. Spinal cord
5. Optic nerve

Disseminated in Time (DIT)

1. MRI must show old and new lesions (or)
2. CSF - Oligoclonal bands (+)

Clinically Isolated Syndrome

1. Earliest Feature in Multiple Sclerosis
2. Optic neuritis
3. B/L Medial Longitudinal Fasciculus (MLF) lesion (MLF connects nucleus of CN 6 and CN 3 - aids in binocular vision).

Radiologically Isolated Syndrome

- Radiological signs of multiple sclerosis present but clinical features are not present.
  - Central Vein sign - Vein surrounded by demyelinated sclerosed zone.
  - Dawson's finger - Periventricular bands of sclerosis.

CLINICAL FEATURES

Sensory > motor, cerebellar and autonomic dysfunction

1. Pain.
2. Fatigue.
3. Ocular manifestations
   Blurring of vision (due to optic neuritis).
   Relative Afferent Pupillary Defect (RAPD)
   Fundus - Usually normal, but may show papillitis, pale disc (due to optic atrophy)
   Internuclear ophthalmoplegia - Due to MLF lesion (Adduction paralysis).
4. Sensory Symptoms :
   Pins and needle sensation.
   Paresthesias - complaints of vibration sense, numbness, raw/swollen sensation, warm water running over skin.
5. MS-Hug sign (band like squeezing sensation around trunk).
6. Spastic gait (weakness)
7. Autonomic dysfunction - Bladder dysfunction, sexual dysfunction

8. Lhermitte's Sign - Flexion of neck cause shock-like sensation and its moves down the spine to legs.
9. Cerebellar symptoms - Incoordination, Ataxia.
10. Brain stem symptoms
    B/L Internuclear ophthalmoplegia
    Trigeminal neuralgia (maxillary and mandibular division)
    Glossopharyngeal neuralgia
11. Cognitive deficits - Memory, speech, visuo-spatial problems (mild, occur late).
12. MS in pregnancy
    During Pregnancy - Suppression of Symptoms.
    Post Pregnancy - Flare up of symptoms.

Treatment

- Steroids
- Disease modifying drugs
- For clinically/Radiologically isolated sign- Teriflunomide and dimethyl fumarate
- For mild disease- Glatiremer. Moderate disease-DMF plus one more drug
- For severe disease- Mab- Natalizumab, Alemtuzumab, Ocrelizumab
- PPMS → Ocrelizumab
- SPMS → Siponimod

GBS

1. Acute Inflammatory Demyelinating Polyneuropathy (AIDP) - 70% - M/c
2. Acute Motor Axonal Neuropathy (AMAN)
3. Acute Motor Sensory Axonal Neuropathy (AMSAN)
4. Miller Fischer type/descending GBS- Ophthalmoplegia, Ataxia, Areflexia

Triggers

1. Infections
    GI : Campylobacter jejuni (diarrhea occurs first, then GBS)
    Respiratory
    Viral: CMV, EBV, Zika, HIV
    Mycoplasma
2. Vaccines: H1N1, Covid (Pfizer > Moderna)
3. Pregnancy
4. Surgery
5. Epidural anesthesia

Antibodies Present in

1. AIDP → Anti-GM_1 Ab, Tcell + macrophages → Demyelination
2. AMAN → Anti-GD_{1a} Ab, Anti-GM1
3. Miller Fischer → Anti-Gq_{1b}

CLINICAL FEATURES

- Parasthesis, pain
- Deep tendon reflexes absent (areflexia).
- Weakness usually begins in LL, then UL is involved (B/L symmetric ascending flaccid paralysis).
- Fever absent at onset of weakness.
- Respiratory muscle weakness present.
- Residual weakness occurs.
- B/L cranial nerves involved :
  - LMN palsy of 7th nerve (poor smile, unable to blow cheeks)
  - 9, 10 cranial nerves
  - 3, 4, 6 (in Miller Fischer type)
- Bladder dysfunction does not occur in early stages.
- Respiratory failure - in 30% untreated patients.
- Mortality - 5-10%
- Autonomic dysfunction :
  - Persistent tachycardia
  - Labile BP (use only short acting drugs to treat)
  - Sweating
  - Bladder dysfunction
- Joint position sense, vibration sense are affected.
- CSF :
  - Protein ↑
  - Cell count normal

  } Albumino cytological dissociation

Treatment

- IV Ig
- Plasmapharesis
- Supportive care

NOTES

NOTES

ENDOCRINOLOGY

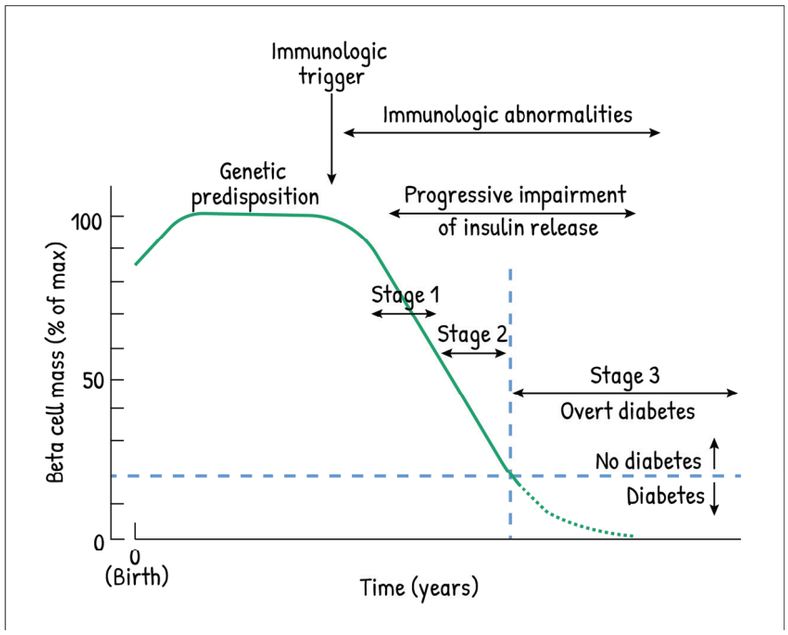
DIABETES MELLITUS

Pre-Diabetes	Diabetes
100-125 mg/dl - IFG	≥ 126 mg/dl
140-199 mg/dl - IGT	≥200 mg/dl
5.7-6.4 %	≥6.5 %

METABOLIC SYNDROME

Criteria for diagnosis are (positive if any 3 present) :

1. Abdominal girth ≥90 cm in males & ≥80 cm in females.
2. Triglycerides ≥150 mg/dL.
3. HDL ≤ 50 mg/dL in women & ≤ 40 mg/dL in men.
4. BP > 130/85 mm Hg (or) known hypertensive.
5. Fasting glucose ≥100 mg/dL (or) known diabetic.



Stage 1 :
Stage 2 :
Stage 3 :

- Teplizumab : anti CD3 monoclonal antibody delays progression from stage 1 → stage 2 → stage 3 in patients with :
  - HLA DR4 +
  - HLA DR3 -
  - Zn-T8 Ab -

Treatment of type 2 DM

Efficacy for glucose lowering	Efficacy for weight loss
Very high : Dulaglutide (high dose), Semaglutide, Tirzepatide Insulin Combination oral, combination injectable (GLP-1 RA/Insulin)	Very high : Semaglutide, Tirzepatide
	High : Dulaglutide, Liraglutide

1. Metformin
2. Sulfonylureas
3. Meglitinides
4. SGLT-2 inhibitors
5. Incretins
6. Thiazolidinediones
7. α-glucosidase inhibitors
8. Amylin analogues
9. Bromocriptine
10. Insulin

Basal Insulins	Duration of Action
• NPH	12 hrs
• Glargine = Acidic	24 hrs
• Detemir	
• Degludec	42 hrs

Peakless

Prandial Insulins

1. Regular insulin = 30-45 mins to act

2. Rapidly acting Insulins
   Faster action (within 0-15mins)
   Aspart
   Glu-lisine
   Lys – Pro

NOTES

NOTES