

Suspicion:

History:

⇔age < 54.

➡ No hx of Rh fever.

General ex:

⇒systemic hypertension.

⇔ Central cyanosis.

Local ex:

⇒ Basal or parasternal thrill in young.

 ⇒ Pure AS.

⇔ Bicuspid aortic valve.

Classification:

Abbott classification					
Cyanotic		A Cyanotic	Potential cyanotic		
$F_3-F_4-F_5$		PS	ASD		
Eisenmenger synd	drome	AS	VSD		
T_A - T_{GA}	2	Coarcitation of aorta	PDA		
Tricuspid atresia		Dextrocardia			
		Clinical classification			
Cyanotic	30 Sept.	A Cyanotic			
As in "Abbott"	2 100	As a Cyanotic + potentia	l cyanotic in "Abbott"		
	ged				
Point	Point A Cyanotic		A Cyanotic		
No	F ₄		Dextrocardia – small VSD		
RVH	F ₃ (PS) -	Eisenmenger syndrome	PS – ASD		
(pulm. HTN)					
LVH	Tricuspid atresia		AS – PDA - Coarcitation of		
			aorta		
Bi. VH	Persistent TA – TGA		Big VSD.		

[A] ACYANOTIC GROUP

Point	① PS	② ASD	③ VSD
Types	Subvalvular	osteum primum (low)	Small muscular type
	Valvular	osteum secondum (at	Big membranous type
	Supravlvular	foramen ovale)	Gerbode type () RA K
	30	Sinus venosus (high)	LV
Heamo –	LOCP	LOCP	LOCP
dynamic	Pulm. Art.Pr.& blood↓	Pulm. Art.Pr.& blood1	Pulm. Art.Pr.& blood↑
	Lung oligaemia	Lung plethora	Lung plethora
92	RVH (pressure	RVH (pressure	Bi. VH (excess blood)
	overload)	overload)	
Complic-	If sever PS + patent	Functional VC of PA	As ASD → reversible
ation	F.O → reversible of	→organic narrowing	of shunt → cyanosis
	shunt \rightarrow cyanosis (F_3)	→ pulm. HTN→	Eisenmenger
		reversible of shunt →	syndrome
2		Eisenmenger s	

Point	① PS	② ASD	③ VSD
Symptoms	Mild case (asympt)	Mild case (asympt)	Mild case (asympt)
	LOCP \$	LOCP\$	LOCP\$
	\$ of complication	(dyspnea –	Dyspnea
		haemoptysis) excess	Haemoptysis
44	*	blood	\$ of complication
	• 1	\$ of complication	
General	Mild case → no signs	Same	Same
Signs	LOCP Sings	Same	Same
	Giant S wave →	No, except in pulm.	No, except in pulm.
	contract ≠ resistance	HTN	HTN
Inspection	RV apex	RV apex	Bi.V apex
Palpation	Thrill on pulm. W may	No thrill (low	
Auscultation	extend up & down	gradient)	at 3 rd , 4 th , spaces
	Dullness in Lt 2 nd pace	Dullness & pulsation	(parasternal)
	→ d.t position	on pulm. Area → d.t	Dullness & pulsation
16. PC	dilatation	pulm. HTN	
	No pulsation		
Auscultation	Weak S ₂	Accentuated S ₂	Accentuated S ₂
Over	Wide splitting W ↑ by	Wide splitting W is	
pulmonary	سكة ضيقة inspiration	fixed (RBBB 80%)+ دم	دم کثیر Inspiration
Area	(inspiration $\rightarrow \uparrow VR \rightarrow$	کثیر	Ejectron click
	1 overload	Inspiration →stop	(relative PS)
	Ejectron click	shunt by \(\tau\) VR	Ejectron soft systolic
	(organic PS)	Ejectron click	murmur
	Ejectron harsh		é no propagation
	systolic murmur	Ejectron soft systolic	
	radiate up & down	murmur	
		é no propagation	

Point		① PS	② ASD	③ VSD
Auscultation	Mitral Pulm. area area	S ₄ († pressure)	S ₃ (augmented RV filling) Mid diastolic murmur (soft) d.t relative T.S by ↑ bl. From RA → RV	
Compl	cation	RVF IE (at pulm. Valve or pulm. Artery) TB (oligaemia) T ₃	RVF No IE (low gradient) Haemoptysis Recurrent chest inf. Eisenmenger syndrome	bI VF IE (at RV) Haemoptysis Recurrent chest inf. Eisenmenger syndrome

Point		① PS	② ASD	③ VSD
	x-ray	Oligeamia (lung) RVH (heart)	(Lung plethora -Lung hilar dance) →	_ `
Investigation	ECG	Post stenotic dil (heart)	vigorous pulsation at ihilum of lung RVH (heart) Dilated pulm. Art. (heart) RVH Rt BBB	· ·
			Lt axis deviation (associated MR) AF	
	ECHO	Diagnostic	Diagnostic	Diagnostic
	Angio.	D type of PS	Show shunt of dye	17 (S 200)
			across I defect	across I defect
	Med.	Prophylactic		Prophylactic ≠ IE
	Surg.	Supra valvular →graft	Closure of defect but	Immediate closure of
ent		Valvular .	wart till	defect once
tt l		→commisurot	→ \$ exaggerated	discovered
Treatment		→ replacement	→ school age	
		Sub valvular →	→ Pulm. Flow	
		myomectomy	becomes at least twice	
			systemic flow.	

(4) COARCITATION OF AURTA (ADULT TYPE)

Def.: Narrowing of segment of aorta distal to subclavian artery.

Haemodynamics:

- ✓ Mechanical obstruction by coarcitation: ↓blood pressure in lower 1/2 of body.
 ↑Blood pressure in upper 1/2 of body.
- Collaterals foimed () Lt Subclavian or its branches & descending aorta.

CP: symptoms:

- ⇒ †bl. Pr. in upper 1/2 (headache epistaxis)
- ⇒ 1bl. Pr. in lower 1/2 (cloudication of LL, Coldness, pallor).

Signs:

- 1. General signs:
 - ⇒ Upper limb → forcible pulse, HTN.
 - ⇒ Lower limb → weak pulse, low BP.
 - Collaterals → Suzmann sign (when pt. lean forward é upper arm hanging down → kinking of subclavian shift of blood to the collaterals → "worm-like" vessels & pulsation in i inter scapular area.

Why HTN UL?

† Blood in upper 1/2

↓bl. Pr. in lower 1/2 → renal ischemia → † rennin

→ → angiotensin II → general VC W† BP mainly in upper 1/2 as lower 1/2 contain empty VS.

2. Cardiac signs:

- ⇒ LV apex: (shifted out & down localized pulging heaving).
- ➡ Over aortic area:
 - Accentuated S_2 ejection click.
 - o Ejection systolic murmur due to: relative AS dilated aorta by 1Pr.
 - Early diastolic murmur due to: (ass. Bicuspid aorta dilatation of thering [2ry to HTN]) AR.

O Systolic murmur W may extend to diastolic, radiate to inter scapular area & heared over infracalviculare area.

- o Continuous murmur in inter scapular area.
- o The murmur disappears by pressure over the feeding vessel.

Investigation:

- X-ray: LVH double aortic knuckle (dilated aortic ring) in 1st space notched lower border of posterior ribs due to collaterals (rosier sign).
- ECG: LVH (HTN).
- ECHO: diagnostic.
- Catheter: pr. Proximal to Coarcitation > pr. Distal to it (gradient inside A)
- Angiography: diagnostic blood is seen shift to collaterals.

Complication:

- LVF.
- IE: at coarcitation at valve in bicuspid aortic valve.
- Rupture aorta.
- Rupture of aneurysm of circle of Willis → subarachnoid Hgc → death.

Treatment:

- Medial: 5 فقاط .
- Surgical: small→ resection & anastomosis.

Large→ resection & graft.

V. large → bypass operation

Common association é coarcitation:

- Bicuspid aortic valve → AR → most important as it nullify deference of blood pr. % pulse volume () VL & LL so I most important sign is carotid femoral delay in D of Coarcitation.
- Aneurysm of circle of Willis, PDA, Turnings syndrome, VSD.

(5) PDA

Def.: persistence of ductus arteriosus, A&B distal to Lt subclavian artery.

Haemodynamics:

- ⇒ Pr. In A&B: bl. Shunt from A&B in both systole & diastole, so both systolic & diastolic Pr&bl↓.
- Shunt of blood \rightarrow lung plethora \rightarrow †LV blood \rightarrow † systolic pr., so systolic \downarrow is compensated while that of diastolic is not, so † S, \downarrow D (big pulse pressure)

- ⇒ Enlarged pulm. Artery & its branches & LAH & LVH.
- Compensatory V.C of pulm. Vessels \rightarrow pulm. HTN W progress till pressure in P>A \rightarrow reversed shunt \rightarrow Eisenmenger syndrome.

Clinical picture:

Symptoms:

- Mild case → no signs.
- ⇒ Throbbing headache, palpitation, cough, dyspean.
- ⇒ Recurrent chest infection, \$ of complication.

Signs:

- 1. General signs:
 - Differential cyanosis in lower limb not upper limb.
 - ⇒ Peripheral phenomena as AR.
- 2. Cardiac signs:
 - ⇒ LV apex (hypertension).
 - Continuous murmur at Lt 1st space machinery murmur W radiate up (neck) & down (apex) & heard over infracalviculare area.
 - Continuous thrill at Lt 1st space:
 - At P: accentuated S_2 ejection systolic murmur (relative PS) d.t excess blood \rightarrow dilate artery é normal ring.
 - At A: ejection systolic murmur (relative AS) d.t excess blood through i valve.
 - o At M: mild diastolic murmur (relative MS) S_3 .

Complication:

- LVF IE.
- Aneurysmal dil. Of shunt rupture of the shunt reversal of the shunt → Eisenmenger syndrome → deferential cyanosis.
- Recurrent chest infection haemoptysis.

Investigation:

- X-ray: LAH LVH pulm. Artery dilatation lung plethora (3/3).
- ECG: LAH LVH.
- ECHO: diagnostic.
- Catheter: pr. Arterialization of pulm. Art. Blood $PAO_2 > RVO_2$.
- Angiography: show the shunt.

Treatment: once diagnosed must be surgically corrected (resection) as operative mortality is lower than I.E (complication).

DD: Coarcitation of aorta & PDA (by femoral pulse → weaker than radial in coarcitation.

N.B: Once pulm. HTN develop in case of PDA:

Eisenmenger syndrome \rightarrow differentiated cyanosis in LL.

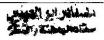
Giant A wave.

Pulsation & dullness over P area.





DEXTROCARDIA



Type l: situs inversus totalis – mirror-like malposition of the heart & other viscera N.B: kartagener's syndrome: (situs inversus totalis – immotile cilia syndrome [bronchiectasis, sinusitis])

Type II: isolated dextrocardia – mirror-like malposition of the heart may be associated with other cardiac anomalies.

Type III: Dextrovarsion: the heart is just displaced to the right side and may be associated with other cardiac anomalies.

Type IV: Dextropostion or acquired dextrocardia: the heart is displaced to the right side by pulmonary, pleural, or diaphragmatic disease.

Differentiation between congenital & acquired dextrocardia:

Items	Congenital	Acquired
1. chest cause	Absent	Present
2. trachea	Central	Shifted to R
3. apex	Localized (LV)	Diffuse
4. situs inversus totalis	May be present	Absent
5. other cardiac anomalies	May be present	Absent

(B) Cyanotic group

Point	1- F ₄	2- F ₃	3- Eisenmenger sy.
Components	PS (Subvalvular) VSD – mild RVH Over riding aorta	PS (valvular) "sever" ASD Marked RVH	Pulm. HTN é reversal of shunt W occurs. Late →ASD Early → VSD – PDA
Haemo – dynamics	PS → lung Oligeamia Bl. Shift across defect to LV → LVH as pressure in RV is high Cyanosis is directly proportional é degree PS	Reversal of the shunt → cyanosis	Reversal of shunt d.t ↑bl. To lung → pulmonary HTN →↑ RV Pr. → cyanosis.
Symptoms	qualitative)	Central cyanosis LCOP (Stenosis – qualitative) Dysnea on exertion (arterial hypoxemia)	Central cyanosis LCOP (hypertensive – qualitative) Dysnea on exertion (arterial hypoxemia)
Signs	Central cyanosis (since birth) Blue clubbing No giant A wave (blue pass via VSD)	Central cyanosis (since birth) Blue clubbing giant A wave (sever stenosis)	Central cyanosis (since birth) Blue clubbing giant A wave (HTN)

Poi	nt	1- F ₄	2- F ₃	3- Eisenmenger sy.
		AMELY 2003 St. 14 Mg	Inspection & palpati	on
		Mild RVH	Marked RVH	Marked RVH
7714.		Thrill over 3 rd & 4 th	Thrill over 3 rd	According to origin of
all 9 min	N. C.	spaces (Subvalvular)	spaces (valvular)	shunt:
معامل المجار المعاملة المجارة المعاملة المحارة		, ,		PDA→thrill(infra.Clav)
				VSD→thrill(parasternal)
				ASD→nothrill(low
				gradient)
				Pulsation & diastolic
		minor mere de envi-	V-100	shock on pulmonary
	8		Percussion	
	9			Dullness in 2 nd Lt space
	1		Auscultation	
	1	$S_2 \rightarrow \text{single}$	$S_2 \rightarrow \text{weak wide}$	$S_2 \rightarrow accent.$ Wide
		accentuate	split	splitted
		No ejection click	Ejection click	Ejection click (may)
			Ejection systolic m	Ejection
		over 3 rd space d.t	over pulm. Area d.t	Systolic m over pulm.
		P.S	P.S.	Area d.t P.S.
			Early diastolic m??	Early diastolic m
			why	"Graham steel"
				Due to dilated valve →
		7 0		2ry PR "surest sigh"
Complication		RVF (rare d.t mild	RVF	RVF
		RVF)	IE	IE
		IE (rare d.t ↓bl) Polycythemia	same	same
		Thrombosis	same	same
ş Ş		Paradoxical emboli	same	same
		(through shunt)		
		TB (Oligeamia)	TB (Oligeamia)	TB (Oligeamia)
		Cyanotic spills	No Cyanotic spills	No Cyanotic spills
8		No dysnea	No dysnea	No dysnea
		No haemoptysis	No haemoptysis	No haemoptysis
	X ray	Oligaemia	Oligaemia	Plethora
		Wooden boot shape	Marked RVH	Marked RVH
		Heart Coeur in	Post-stenotic dil.	Dilated main pulm.
'n		shape (RVH-		Artery and its branches
atic		enlarged aorta –		
tig		exagg. Waist)		
Investigation	ECG	Mild RVH	Marked RVH	Marked RVH
In	ECHO	Diagnostic	Diagnostic	Diagnostic
	catheter	↓Pulm. Artery pr.	↓Pulm. Artery pr.	†Pulm. Artery pr.
		↑RVF=aortic pr.	RVF>aortic pr.	RVF>aortic pr.
	Angio.	Show defect	Show defect	Show defect

Point	1- F ₄	2- F ₃	3- Eisenmenger sy.
Treatment	Rarecyanotic spills Squatting – inderal – morphine.	Prophylaxis ≠ IE	Prophylaxis ≠ IE
Service Control of the Control of th	Complete surgery correction "broeck op" Shunt op. till suitable age: Blalock op. waterson op.	Complete surgery correction: Commisurotomy for PS. Closure of ASO	No role of surgery. Recently: heart-lung transplantation

RHEUMATIC FEVER

Def: connective tissue disease characterized by (vasculitis – fibrinoid degeneration – immune disturbance)

AE: group A-beta hemolytic streptococci – infection after period (4W)

Pathogenesis:

- 1. Cross reactive immunity (shared antigen): protein of streptococci is similar to cardiac ms protein immunology so body form antibodies against streptococci which cross react with cardiac protein.
- 2. Autoimmunity: streptococci damage CT \rightarrow proteins which is already altered so act as antigen \rightarrow stimulate AB \neq it.
- 3. Hapten (incomplete).

Evidence:

- Epidemiology: as in army camps.
- Immunology: all cases of RH fever are ass é gp A-B- haemolytic strain.
- Prophylactic: prevention of store throat by penicillin → prevention of Rh. Fever.

Predisposing factors:

- Age: 5-15 y common, > 15 arthritis recurrent attack.
- Sex: equal in both but chorea > in \subsetneq .
- Race: white races.
- Familial: heridotifamilial.
- Season: autumn & winter.
- Area: temperate zone.
- Recurrent streptococcal infection (overcrowding &bad hygienic condition)

Pathology:

1. Reaction:

- Exudative reaction: exudative formation affect serous membranous (synovial – pleural – pericardium – peritoneum) – resolve completely.
- Preoperative reaction (Ashoff's nodule): paravasculare nodules that heal by fibrosis consist of (from in to out): fibrinoid degeneration (center) lymphocytes plasma cues Ashoff giant cells fibroblasts layer of fibrosis.

ين يادر هو الهيدم

2. Site:

- General:
 - o non-specific: inflammatory edema inflammatory cell infiltration vascular phenomena –
 - o Specific: Ashoff's nodule.
- Extra cardiac:
 - Pleurisy meningo encephalitis pneumonia vasculitis.
- Cardiac: pancarditis in all layers as follow:
 - Pericarditis: dry pericarditis: i 2 layers attached by fibrinous adhesion. Wet pericarditis: with adhesion.
 - o Myocarditis.
 - o Endocarditis:
 - Mural endocardium: necrosis of posterior wall of Lt Atrium → fibrosis (mac-callum patch) → predispose to thrombus formation.
 - Valvular endocardium: mild attack → edema (long course)
 → Stenosis, sever attack → regurge at once.

NB: frequency of valve affection: M > A > T > P due to closing pressure \hat{W} is (120, 80, 30, 10).

Clinical picture:

A- Major:

- [1] Arthritis: most common "sp in adult ":
 - Polyarthritis affect big joints as knee elbow joints is hot, red, painful, swollen, tender, é limited movement migratory.
 - Leave joint free.
 - Respond dramatically to asprine (salycilates).
- [II] Carditis: most serious "sp in children"
 - Pericarditis: dry (pericardial rub chest pain), wet (dullness outside i apex).
 - Myocarditis: tachycardia out of proportion to fever (N: $1C^{\circ} = 10$ beat /min.) Tick tack rhythm ($S_1 = S_2$) due to loss of i muscular component of S_1 dilating heart (continous) HF summation gallop \uparrow (P-R) interval (A V) incompetence (TR₁ MR) due dilated ring.
 - Endocarditis: early (edema Stenosis).

Late (damage → short period → regurge), (Fibrosis → long time → Stenosis)

- For mitral valve: mid diastolic murmur (carry comb murmur) due to edema Pan systolic murmur due to MR.
- For aortic valve: ejection systolic murmur due to edema of valve or fever Early diastolic murmur due to AR.
- [III] Erythema marginatum: on trunk proximal parts of limbs occur on crops → coalesce → patch → clear center & red margin evanescent.

[IV] S.C nodules: accumulation of Ashoff's nodules:

- Round or oval symmetrical from few mm to few cm.
- On pressure area & extensol surfaces not tender.
- Not attached to overlaying skin but attached to underlying tendon.
- Firm in consistency indicate sever Carditis.

[V] Chorea: common in \mathcal{P}

- Involuntary movement (shoulder, face, tongue, extremities)
- Marked hypotonia emotional instability.

B- minor:

[1] Clinical:

- Arthralagia acute abdomen vasculitis pleurisy peritonitis pallor
- Pneumonia epistaxis Erythema nodosum.
- Fever sweating weatness loss of weight tachycardia.
- [2] Lab.: †acute phase reactants (ESR, CRP) & leucoytosis prolonged P R interval † ASO titre.

Investigation:

[1] Cardiac:

- X-ray chest: to evaluate the heart.
- ECG: Myocarditis (long > 0.22 sec. = 1st degree HB), evidence of pericarditis.
- ECHO: to evaluate the heart.

[2] Laboratory:

- ESR: \uparrow ed \geq 100 / 1st h (for follow up).
- CRP: +ve (recent infection), -ve in (pure chorea isolated EM).
 - Abnormal ptn that agglutinate muco polysaccharide of pneumococci.
 - Non-specific: present in other disease.
- Leucocytosis: é esenophillia, if é esenophillia →septic arthritis.
- ASO (250 Todd's unit) +ve if recent infection.

Diagnosis: revised Joune's criteria

- ① Major + ② Minors (clinical + INV)
- Or ② Majors.
- Evidence of recent strept. Infection.
- HX of Rh disease or presence of Rh valve disease.
- but take i following precautions into consolidation:
 - Arthritis, fever, Leucocytosis, (not specific).
 - > If arthritis is taken as major not to take arthralagia as minor.
 - ➤ If Carditis is taken as major not to take (tachycardia long P-R) as minor.

DD: (Causes of arthritis) (causes of fever in cardiac pt) (PUO)

(Causes of chorea) (Causes of pericarditis)

(Causes of acute abdomen) (Leukemia & hemolytic anemia).

Treatment:

[1] Prophylactic:

a. Eradication of infection:

Treatment of sore throat by P:

Old protocol (Procaine P 600.000 U/day for 10 days IM).

Recent protocol (ampicillin 500 mg / 8h / day for 10 days).

Removal of septic foci: tonsillectomy of huge infected tonsils.

b. Avoid activity (if recurrent)

Long acting penicillin (benzathine) 1200.000 U IM:

(12w in summer – 14w in winter) for (fever – 54 from last attack – till age of 25 y) W is longer.

If sensitive to $P \rightarrow \text{sulphadiazine 1 gm oral/day}$.

c. Guard against infective endocarditis.

[2] Curative: (in active stage):

- 1. Rest till ESR drop to normal value, ↓ signs of inflammation.
- 2. diet (light & nutritious)
- 3. Treatment of infection (crystalline P 1 million U / 6h for 10 days.
- 4. for arthritis: salycilates:

Dose: (6 gm /day) Or (100 mg / kg/ day till clinical response → ↓ dose to 2/3 till laboratory response appear → gradual é drawal to avoid rebound.

Action: analgesic - antipyretic - anti-inflammatory - St. Steroid secretion.

- 5. Carditis: prednisone 60 gm /day ↑gradually till response occur → then continue effective dose for 4W → then ↓ to 40 mg / day for 4W → then gradual withdrawal by 2.5 g/day to avoid addisonian crises.
- **6.** Carditis + arthritis:

Prednisone 60 mg /day for 1 W then stop & give Salycilates 60 mg / kg/day for 1 W.

Recently steroids not superior to Salycilates & both not prevent cardiac damage.

- 7. Chorea: reserpine, its SIE is hypertension so used in ttt of chorea.
- 8. heart failure: steroids + anti failure measure :
 - ➤ Steroids in cardiomegally HF.
 - Salycilates in mild Carditis.
 - > 50% pt. gives no history of Rt fever.
 - > Steroids not prevent chronic Valvular affection.
 - ➤ But in 1 inflammatory signs (1 fatality in a cute stage).

MITRAL STENOSIS

⇒ Degree (Grads) of MS :-

 $rac{1}{2} \ge 2.5 \text{ cm}^2 \rightarrow \text{Asymptomatic}$.

 $= 1.6 - 2.5 \text{ cm}^2 \rightarrow \text{Mild}$

 $rac{1}{2} - 1.5 \text{ cm}^2 \rightarrow \text{Moderate}$

Burk St. January

⇒ Aetiology of MS:-

A) Organic:-

Rheumatic endocarditis: - commonest, More in female & occur years after acute stage Congenital: - Lutenabacher's syndrome (MS + ASD) & Parachute mitral valve.

- ★ Obstruction of mitral orifice by → Tumour (Lt atrial myxoma), Thrombus & Nodules (fused libman sac nodules of SLE)
- * Infective endocarditis.

B) Functional:-

Conditions of 1 blood flow through the valve :- MR, PDA, VSD & Hyperdynamic circulation

Conditions of dilated LV with intact M.valve (relative stenosis) - AR & HTN

★ Carry Comb's murmur: It occurs in acute stage of Rh. fever due to edema of the cusps → relative narrowing of mitral valve → mid diastolic low pitched murmur.

⇒ Haemodynamics:- [Long course]

- Gradual progressive narrowing of MV $\rightarrow \uparrow$ Lt.A pressure \rightarrow Lt A dilation to overcome the resistance of M.valve \rightarrow No symptoms occur.
- With more narrowing → LA can't push all the blood through MV → blood stagnates in pulmonary veins & capillaries → ↑ pulmonary venous pressure (pulmonary congestion)
- # to protect against ↑ in pulmonary capillary pressure → V.C. of pulmonary arterioles
 (functional narrowing) → later organic narrowing due to intimal proliferation →
 thrombosis of the vessels may occur.
- The above mentioned events → ↑ pulmonary arterial pressure → pulmonary HTN
 → Rt.V. hypertrophy → right ventricular failure later on .

⇒ Stages of MS:- There are 4 stages;

- 1- Stage of Complete compensation (Asymptomatic stage):-
 - ★ There is ↑ in Lt. Atrial pressure due to blood stagnation.
- 2- Stage of Pulmonary congestion (Congestive MS):-
 - ★ There is ↑ in pulmonary venous pressure.
- 3- Stage of Pulmonary arterial hypertension (hypertensive MS):-
 - \star there is \uparrow in pulmonary arterial pressure & Rt. ventricular hypertrophy \to LCOP.
- 4- Stage of RVF (MS with RVF):- It shows systemic congestion.

 Mechanism of pulmonary HTN in MS:-

- <u>1- Passive pulmonary HTN:</u> ↑ arterial pressure → ↑ pulmonary V. pressure & to maintain passage of blood from arteries to veins, there must be equal \(\frac{1}{2}\) in pulmonary venous pressure (constant AV gradient).
- 2- Constriction pulmonary HTN:- Lung congestion → reflex protective V.C. (functional) of pulmonary arterioles → pulmonary hypertension.
- 3- Obliterative pulmonary HTN:- Prolonged VC → irreversible organic narrowing of arterioles.
- 4- Acute obstructive pulmonary HTN: Prolonged recumbency → may cause venous thrombosis \rightarrow massive pulmonary embolism & acute pulmonary hypertension.

⇒ Clinical picture:-

) SYMPTOMS

- * Stage 1:- No or mild symptoms.
- * Stage 2:- Symptoms of lung congestion (pulmonary edema not common).
- ★ Stage 3:- Symptoms of low COP with gradual relief of lung congestive symptoms.
- ★ Stage 4:- Symptoms of systemic congestion (heart failure).
- ★ Symptoms of complications: It may be the first presentation.

B) SIGNS

I | General signs :- It varies with the stage;

- * Stage I:- no or mild signs.
- ★ Stage II: Bilateral fine basal crepitation.
- ★ Stage III :- Signs of Low COP :- weak pulse, Low Bp, Pallor, Peripheral cyanosis, Cold extremities & Malar flush (cyanotic flush over cheeks).
 - ⇒ Giant (a) wave due to pulmonary hypertension.
- * Stage IV: peripheral signs of RSHF (systemic congestion)
- * Signs of complications maybe present.

II] Local signs on Cardiac examination :-

⇒ Stage I & II:

- $\ensuremath{\textit{G}}$ Apex :- normal site , slapping , diastolic thrill & palpable S_1 .
- Auscultation: There are 3 main findings;

☆ Accentuated S1, due to:-

- → Closure of rigid mitral cusps.
- → Opening of mitral cusps as wide as possible in diastole at high atrial pressure followed by violent closure of cusps .
- \rightarrow Sudden tension of pliable part of MV by tension of chordae tendinae .
- → Low position of mitral cusps in LV at end of diastolic due to small amount of blood as result of stenosis .

☆ Opening snap :- (heard [] Lt. sternal border & cardiac apex)

- \rightarrow It is a sharp snapping sound due to opening of rigid stenotic mitral cusps (organic stenosis).
- → It occurs after S2 (separated from it by isometric relaxation phase) & just before the murmur .
- \rightarrow \uparrow degree of MS \rightarrow \downarrow diastolic between opening snap & S2 .
- \rightarrow <u>Value :- [TRP]</u> \Rightarrow T:- tight MS, R:- Rheumatic, P:- Pure (no MR), Pliable (no calcification).

$\stackrel{*}{\cancel{\triangle}}$ Murmur :- (' ↑ Degree of MS \rightarrow ↓ murmur).

- \rightarrow Site: at or inside the apex.
- → Timing: Early ⇒ pre-systolic, Late ⇒ mid-diastolic with pre-systolic accentuation.
- -- Character :- Rumbling , low pitched .
- \rightarrow Intensity:- Loud with pre-systolic accentuation (due to atrial contraction).
- \rightarrow Propagation:- localized.
- \rightarrow Position of patient:- Lt. lateral position.
- \rightarrow Respiration: \uparrow with expiration (-ve Carvallo's sign).
- \rightarrow Method:- best heard by cone.
- (N.B) ≥ S₂ is followed by Isometric relaxation phase → opening of MV, blood pass either by gravity (70%) causing "mid-diastolic murmur" or by atrial contraction (30%) producing "pre-systolic accentuation", So; in AF with MS, NO pre-systolic accentuation as there is NO atrial contraction.

⊃ Stage III :- (Stage of pulmonary HTN); there are 2 groups of signs:

Signs of pulmonary hypertension:-

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- ★ Inspection: pulsation in the Lt. 2nd space.
- * Palpation: pulsation in the Lt. 2^{nd} space & diastolic shock (palpable S_2).
- ★ Percussion: Dullness in the Lt. 2nd space.
- * Auscultation :- as stage I & II; plus;

→ At pulmonary area :-

- Accentuated S₂:- early ⇒ close splitting & late ⇒ wide splitting.
- Ejection systolic murmur (relative PS).
- ◆ Early diastolic murmur (Graham steel) ⇒ surest sign of pulmonary HTN.
- + Ejection click maybe heard.

→ At tricuspid area :-

* S4; due to strong Rt. atrial contraction as a result of pulmonary HTN (pressure over load).

Signs of Rt. ventricular hypertrophy:-

- * At apex :- shifted outwards, diffuse, retracting & heaving.
- ★ Pulsation: 3rd, 4th parasternal & epigastric pulsations.
- * Dullness:- broad bare area. & dullness at lower end of the sternum.

⊃ Stage IV :- It shows each of the following:--

- Pulmonary hypertension & Marked RV hypertrophy (as stage III).
- Proto-diastolic gallop (S3) of heart failure on tricuspid area.
- ightharpoonup Functional TR due to Rt.V. dilatation ightharpoonup Systolic thrill & pansystolic murmur.

☐ Investigations: MOST of diseases in cardiology, should be investigated by;

- 1- X-ray
- 2- Electrocardiogram (ECG).
- 3- Echocardiography.
- 4- Cardiac catheterization.
- 5- Angiography.
- 6- Certain specific investigation, according to the disease to be investigated.

1-X-ray:-(Comment on heart, blood vessel & both lung fields)

- Stage I:- Normal & may show mild Lt. Atrial dilatation .
- ⊃ Stage II :- There are 2 views ;
 - * Postero-anterior view :- It shows 3 +ve data;
 - > Left atrial dilatation :- It is detected by
 - Obliteration of cardiac waist (Mitralization).
 - Prominent left atrial appendage ...
 - Double contour of right border of heart (it means huge Lt. atrium).
 - Wide carina of trachea.
 - > Pulmonary venous congestion :- It is detected by these signs;
 - ◆ Cephalization of upper lobe vessels (filled with blood) ⇒ Mostash sign.
 - ◆ Hilar opacity (if associated with pulmonary oedema ⇒ bat wing appearance).
 - Kerley's B lines; due to interstitial edema in base of lung.
 - Pulmonary haemosiderosis; due to rupture capillaries.
 - ◆ Reticulosis → It means interstitial oedema & fibrosis.
 - Fluffy cotton exudates → pulmonary oedema.
 - > Calcified mitral valve may appear.
 - N.B.: Kerley's (A) lines means interstitial oedema NOT at the base of lung.
 - * Lateral view with barium: Enlarged LA displaces the esophagus posteriorly.
- ⇒ Stage III & IV :- It shows the following ;
 - ★ ↓ Pulmonary congestion.
 - * Radiological signs of pulmonary HTN :- (Dumbbell appearance)
 - Dilatation of Pulmonary artery & its Left branch.
 - ◆ Peripheral lung oligaemia ⇒ Pruned lung appearance.
 - * Right ventricular (RV) enlargement .
 - * Right atrium (RA) & SVC enlargement.
- $\it N.B.:-Dumbbell\ appearance\ occur\ in\ ASD$, $\it Pulmonary\ HTN\ \&\ bilharzial\ cor\ pulmonale$.



Complication:

A. Local:

- > Pancreatic phlegmon: local mass due to digestion of the pancreas.
- Pancreatic ascites, pancreatic cyst.
- > Pancreatic abscess.

B. General:

- > ARDS, RF.
- ➤ Pleural effusion, pericardial effusion.

Investigation:

1. Serum amylase is elevated.

2. US & CT.

Treatment:

- 1. Nothing per mouth.
- 3. Aspiration of gastric contents.
- 5. Analgesics for pain.

2. IV fluids.

4. Peritoneal lavage in sever cases.

Chronic pancreatitis

Retiology: sever attack of acute pancreatitis.

Clinical picture: pain – Malabsorption – DM.

Investigation: as acute pancreatitis.

Treatment:

- 1. As acute pancreatitis.
- 2. Treatment of malabsorption & DM.
- 3. Pancreatic enzymes orally.

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<u> 2- ECG :-</u>

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- ⊃ Stage I :- no abnormality .
- ⇒ Stage II :- broad & bifid P wave (P-Mitral) due to Lt. Atrial enlargement .
- → Stage III & IV: RVH, tall & peaked P wave (P-pulmonale) due to RAH & Pulmonary HTN.

3- Echocardiography: - (It is diagnostic); It has a general scheme;

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- * Anatomical diagnosis of MS & any associated lesions...
- * Functional measurement of ejection fraction & COP.
- * Chamber enlargement; either Rt or Lt.
- * Complications: calcification, pliability & LA thrombosis.
- * Preoperative assessment: to select MV valve prosthesis " depend on size of LV out flow tract"
- * Postoperative follow up & diagnosis of re-stenosis & para-prosthetic mitral leak.

4- Catheterization: (Assess pressure, degree & function)

- * Wedge pressure & pulmonary artery pressure are elevated (in MS & LSHF) .
- ★ Presence of diastolic gradient across MV (when it is opened → surest sign) due to
 ↑ LA pressure (normally there is NO pressure gradient) .
- * ↑ RV end diastolic volume in RV Failure (RVF).
- ★ Assessment of degree of stenosis; via MS index = COP / LA pressure which is normally = $5/5 \Rightarrow 100 \%$ but; in MS \rightarrow there is \downarrow COP & \uparrow LAP \rightarrow < 100 % (<1).
- \star Preoperative assessment \rightarrow it is the main indication .
- * Diagnosis of other valve lesion & assessment of myocardial function.

5- Angiography.

⇒ Complications of MS:-

- Pulmonary: Haemoptysis, Chest infection, Pulmonary HTN, Pulmonary infarction, Pulmonary edema & cardiac asthma.
- Fight ventricular: TR, RVF, RV enlargement.

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Left atrial :-

- * Thrombosis, Embolization & aneurysmal dilatation.
- * Pressure manifestations; on
 - ⇒ Lt bronchus → cough, dyspnea,
 - ⇒ Lt recurrent laryngeal nerve → horsiness of voice.
 - ⇒ Oesophagus → dysphagia (Ortner's syndrome)
- ★ Dysarrhysthmia → AF & Extrasystole.
- George Complication: see treatment of MS (therapeutic complications).

⇒ Differntial diagnosis of MS:-

- On clinical level; MS is differentiated from;
 - ★ Other lesions causing similar murmur (Diastolic murmur → Mention from clinical)
 - * Other causes of lung congestion.
 - * Other causes of hart failure (mention by name).
- DD of aetiology of MS:- differentiate [] organic & functional causes.
- DD of MS from similar conditions:-
 - ★ Other causes of left sided hypertrophy (lung congestion).
 - * Other causes of obstruction of blood flow from LA to LV, as; LA myxoma, ball & valve thrombus, veno-occlusive pulmonary diseases & cor triatriatum.
- Silent MS:- MS without murmur; due to ↓ blood flow across MV, causes are;
 - \star \downarrow LA pressure \to \downarrow blood flow as in ; Pulmonary embolism , Sever pulmonary HTN , associated PS , RVF , TR & sever tachycardia .
 - \star 1 LV pressure as in ; LVF , AS & systemic HTN .
 - * Associated ASD as in Lutembacher syndrome.
- DD of MS from MR.
- $\ensuremath{\mathscr{G}}\xspace DD$ of MS from tricuspid valve lesions .



⇒ Treatment of MS:-

- A) Medical treatment: (General scheme for all valvular heart diseases)
 - ☆ Prophylaxis against Rheumatic fever ⇒ Long acting penicillin.
 - ☆ Prophylaxis against Infective endocarditis (IE).
 - ❖ Symptomatic treatment ➡ Diuretic (for lung congestion symptoms), Digitalis (for AF), Antibiotic (for chest infections) & Anti coagulants (for thrombo-embolization).
 - ☆ Treatment of complications ⇒ HF, AF, infection, embolism.
 - ☆ Moderation of life ⇒ No smoking, No excess effort & Good hygenic habits.

B) Surgical treatment:-

- ☆ Indications:- [best age 20-25 y]
 - \triangleright Tight MS (valve area < 1 cm² & pressure > 20 mmHg).
 - > Serious diagnosis as haemoptysis.
 - ➤ Embolism with NO capacitation → Paralysis.
 - > Progressive intolerable symptoms due to mechanical narrowing of MV.

☆ Contraindications :-

- ▶ Rheumatic activity.
- ➤ I.E.
- \triangleright H.F.
- > Associated significance aortic lesion; unless surgically corrected.
- N.B. :- In pregnant females \rightarrow postpone the op. after delivery except in tight MS .
- ☆ Types of operations:- (Valvotomy, Valvoplasty & Valve replacement)

Closed operation :-

- ✓ In cases without significant MR (pure MS), much fibrosis & cusp calcification .
- ✓ Methods:- transatrial & transventricular commissurotomy.

Open operation :-

- ✓ It is done in cases not suitable for closed surgery .
- ✓ Methods:- open commissurotomy, valve replacement & valvoplasty.

A Post operative compositions:-

- > Embolism.
- > Dysarrhysthmias (due to sudden relieve of obstruction).
- \triangleright Tearing of mitral cusps \rightarrow MR.
- > Post commissurotomy syndrome :- (fever + 3 P)
 - \checkmark It is post commissurotomy autoimmune pericarditis .
 - \checkmark It is due to allergic reaction to injured pericardium .
 - ✓ C/P:- fever, pleural & pericardial rub occurring 2-6 weeks after operation
 - ✓ Treatment :- Corticosteroids .
- > Re-stenosis of mitral valve.
- > Complications of valve replacement (artificial valve) :-
 - ✓ Thrombo-embolism in case of Infective Endocarditis .
 - ✓ Haemlytic anaemia ; in HF (technique fault)
 - ✓ Fatty infiltration of the ball of the valve .
 - ✓ Calcification of tissue valves .

C) Ballon Dilatation :- It can replace surgery in patients indicated for valvotomy.

Certain IMPORTANT N.B.s:-

Results of association [] MS & AF :-

- ◆ Loss of pre-systolic accentuation .
- ◆ ↑ incidence of LA thrombosis.
- ◆ No (a) wave in neck veins.
- Paroxysmal $AF \rightarrow precipitate pulmonary oedema$.

Assessment of MV mobility & pliability :-

ullet It is very important as , mitral valvotomy is only possible if valve is pliable &non calcified; other wise $DO \rightarrow valve\ replacement$.

Variable S₁.

No S₄.

No opening snap.

- ◆ Methods of assessment :-
 - ♦ Auscultation \Rightarrow S_l is not accentuated & no O.S in non pliable valve .
 - $\triangle X$ -ray \Rightarrow may show calcification.
 - \triangle ECHO \Rightarrow It is the most reliable method.

> Value of measurement of capillary wedge pressure: It either \(\) or \(\);

- If $\uparrow \Rightarrow$ it means post capillary obstruction \rightarrow LSHF & MS.
- If $\downarrow \Rightarrow$ it means pre-capillary obstruction \rightarrow Cor-pulmonale & embolism

Causes of Haemolysis in MS:

- Bronchitis.
- Pulmonary congestion .
- ◆ Pulmonary edema & PND .
- Pulmonary infraction :- $MS \rightarrow RVF \rightarrow DVT \rightarrow Pulmonary$ embolism $\rightarrow P$.infarction.
- Pulmonary apoplexy: due to rupture of dilated collaterals [] bronchial & pulmonary veins.

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Emergencies in MS:-

- Fever of Carditis (Rh.fever or IE).
- ◆ Palpitation (AF).
- ◆ Thrombo-embolic complications ⇒ blindness, deafness, hemiplegia & sudden death.
- * Cardiac asthma.
- ◆ Pulmońary oedema .
- ◆ Haemoptysis :- due to pulmonary apoplexy .
- Death :- sudden death due to thrombosis \rightarrow closed MV.

A Criteria of Tight MS:-

- ◆ Congestive MS with dyspnea > grade II .
- Hypertensive MS (pulmonary HTN).
- * MS with RVF.
- ◆ Mitral valve area < 1cm2.
- ◆ LA pressure > 20 mmHg.
- O.S. very near to S2 ; i.e. , < 0.06 second , heard by phonocardiogram .
- ◆ ↑ length of murmur unless pulmonary HTN develops .
- MS index < 20 %.

Criteria of Cortriatriatum :-

- ◆ It is a congenital anomaly of the heart in which there is accessory chamber which receive blood from pulmonary veins & connected to LA by an orifice <1cm in diameter (this orifice plays the role of Mitral valve after stenosis).
- ◆ It results in ⇒ Pulmonary congestion & HTN.
- It is differentiated from MS by; NO characteristic murmur & NO left atrial enlargement by X-ray & ECG.

> Types & Criteria of Artificial valve :-

A) Tissue valves (Bio-prosthesis):-

- ◆ Subtypes ⇒ Cadaveric or Xenograft (from animals)
- ◆ Advantages ⇒ No heamolysis , No anticoagulants & No thrombosis .
- Disadvantages ⇒ less durable .

B) Mechanical valves :-

- ◆ Types ⇒ Ball & socket , Tilting disc & Starr Edward valve .
- Advantages ⇒ Durable .
- Disadvantages
 ⇒ Heamolysis , Anticoagulant , Thrombosis & NOT suitable for old & pregnants .

🗷 LSHF is a failure of symptoms (i.e. show many symptoms & less signs) .

- RSHF is a failure of signs (i.e. show many signs & less symptoms).
- Mesocardium means ⇒ Persistnt mediastinal heart .

Causes of haemolysis in cardiac patient :-

- ◆ the same 5 causes of haemolysis in MS +
- ◆ Sever HTN .
- Rupture aortic aneurysm in a bronchus.,
- * Associated T.B. .

LEFT ATRIAL MYXOMA

- → Definition: It is a rare benign tumour of LA usually arising from inter-atrial septum.
- ⇒ Clinical picture: (3F, 3H, 3 NO & 3 sentences)
 - ☆ Familial, Females > Males & Forty years old.
 - ☆ High temperature (Fever), High ESR & Hypergammaglobulinaemia.
 - ☆ NO past history of Rh. fever, NO opening snap & Normal first heart sound.
 - ☆ Intermittent murmur of MS (Postural).
 - ☆ Syncobal attack related to position (Postural).
 - ☆ Embolization with out AF incase of MS (friable).

□ Investigations:-

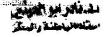
- ☆ Angiocardiography:- reveals filling defect.
- ☆ Echocardiograhys: Tumour Plob ⇒ Diagnostic.
- ⇒ Treatment :- Excision.

MITRAL INCOMPTENCE

Aetiology of Mitral regurgitation (MR):-

A) Organic MR:-

- ☆ Rheumatic MR ⇒ (the commonest)
- ☆ Congenital:- It is associated with ASD.
- ☆ Infective endocarditis.
- ☆ Post commissurotomy & Atherosclerosis.
- ☆ Mitral valve prolapse & Myocardial diseases
- ☆ Papillary muscle dysfunction due to; Myocardial ischaemia, Hypertrophic obstructive cardiomyopathy, associated with LV dilatation & Infiltration by sarcoidosis & haemochromatosis.
- B) Functional MR: due to Dilatation of mitral valve secondary to LV Dilatation as in;
 - ☆ Systemic hypertension, and;
 - ☆ Aortic valve lesion (AS).



A) Acute MR (low compliance MR):- It occurs in the following cases:-

- ☆ Infective endocarditis
- ☆ Acute Rheumatic fever.
- ☆ Post commissurotomy.
- A Papillary Muscle rupture (acute MI) especially that of anterior wall.

N.B.; In acute MR; LA has no time to dilate and accommodate the regurgitant blood \rightarrow Great \uparrow in its mean pressure \rightarrow pulmonary congestion & acute pulmonary oedema \Rightarrow medical emergency that need treatment of pulmonary edema, valve replacement & vasodilators

B) Chronic MR (high compliance MR):-

- ☆ It occurs in other gradual causes.
- ☆ During systole ⇒ part of the blood in LV regurge in LA → ↓ COP & LA enlargement
- ☆ During diastole ⇒ larger amount of blood reach LV → LVH → LVF → lung congestion
- \leftrightarrows Finally ; LA accomedate excess blood \to so , mean LA pressure & pulmonary venous pressure are normal .
- ☆ Because of all these data ⇒ Pulmonary HTN not occur or mildly present.

Clinical picture :-

A) SYMPTOMS

- ☆ Asymptomatic; in mild cases.
- ☆ Symptoms of low cardiac out put.
- ☆ Symptoms of lung congestion.
- ☆ Palpitation .
- ☆ Symptoms of complication.

B) SIGNS

1) General signs :- either;

- ☆ No signs
- ☆ Signs of Low COP.
- ☆ Signs of lung congestion due to LVF.

2) Local signs (cardiac signs):- It involves;

- I) Inspection & Palpation.
- II) percussion.
- III) Auscultation.

I) Inspection & Palpation :-

- ☆ LV apex :- Localized , Pulging , Shifted downword & outword & hyperdynamic .
- ☆ Systolic thrill over apex.
- II) Percussion: Signs of pulmonary HTN (may occur late).

III) Auscultation :- either ;

☆ Heart sounds:-

- \checkmark S₁ \rightarrow weak & muffled (due to weak closure of mitral valve).
- \checkmark S₂ \rightarrow within normal.
- \checkmark S₃ \rightarrow due to rapid rush of blood from LA \rightarrow LV during rapid filling phase with vibration of the ventricular wall (more in functional MR).

☆ Murmur: There are 2 types;

Murmur due to MR :-

- Site:- maximum over apex.
- Timing:- pansystolic murmur (start with S₁)
- Character: harsh in organic murmur & soft in functional one.
- Propagation: propagated to axilla except in posterior leaflet regurge which radiate to base of the heart.
- Position: ↑ by left lateral position.
- ◆ Respiration: ↑ with expiration.

☐ Investigations:-

- 1) X-ray: Enlarged LA & LV, Pulmonary congestion (LVF) & Calcified MV.
- 2) ECG:-Enlarged L.A (P-mitral) & enlarged L.V..
- 3) Echocardiography: MR, mitral valve prolapse & chamber enlargement.
- 4) Angiocardiography:-A dye injected in LV → regurgitate to LA (diagnostic).
- 5) Cardiac catheterization: It will show; \(\tau\) LA pressure & MR.

- S LVF.
- Rheumatic activity (more common)
- Infective endocarditis (more common).

Differential diagnosis of MR:-

Differntial diagnosis from similar conditions:-

- ☆ Other causes of systolic murmur over the apex.
- ☆ In Acute MR: from other causes of acute pulmonary edema (see sheet).

Differntial diagnosis of aetiology: i.e. from;

- ☆ Mitral valve prolapse.
- ☆ Papillary MS dysfunction :- its murmur is ;
 - Mid-systolic or late systolic (crescendo-decreascendo) as ⇒ AS
 - Propagated to base of the heart.

➡ Treatment:-

A) Medical treatment :-

- General schema for any valvular heart disease (5).
- - They will ↑ blood flow to the aorta → ↓ blood pass to atrium, because blood
 is shifter to the dilated aorta.
- Treatment of complication :- as ; H.F , infection ,.... etc .

B) Surgical treatment:-

- Valvoplasty .
- Valve replacement: by;
 - ☆ Prosthesis.
 - ☆ Homograft:-in;
 - Old people > 65 years.
 - Female in fertile period to avoid risk of anti-coagulant if she gets pregnant.

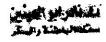
N.B.:- MS is better than MR; because

- ☑ MS has ⇒ long period of free symptoms (late pulmonary oedema), rare IE (due to fibrosis)
 & 2 types of operations can be done.
- MR has

 progressive course (it performance)

 progressive course (it performance)

 progressive course (it performance)



MITRAL VALVE PROLAPSE (BARLOW'S SYNDROME)

- ⇒ Other name: Click murmur syndrome.
- Definition: It is myxomatous degeneration of the posterior cusp of MV & chorda tendinae → redundant cusp → bulge in the LA during systole
- ⇒ Incidence: More common in young female, Familial tendency & Maybe considered as normal variant.

Aetiology: - Un know but;

- It maybe associated with hereditary syndromes, as; Marfan, Ehler danlos syndrome & Pseudo myxoma elasticum.
- It maybe associated with congenital anomalies

 PDA.
- It maybe idiopathic.

⇒ Pathophysiology :-

During ventricular systole, posterior leaflet (common) of MV, prolapse into LA, leading to; Abnormal ventricular contraction, Papillary muscle strain & Some degree of MR.

⇒ Clinical presentation :-

A) SYMPTOMS

- ☆ Asymptomatic :- in most cases .
- A Chest pain :- due to stretch of chordae tendinae, stabbing pain in left infra-mammary.
- A Palpitation: due to abnormal ventricular contraction & associated Dysarrhysthmia.
- ☆ Sudden death due to :- associated Dysarrhysthmia.

B) SIGNS

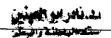
- ☆ Murmur: Mid or late systolic murmur due to some regurgitation, which ↑ with standing & it radiate to the base of the heart.
- ★ Added sound: Mid systolic click due to tension of chordae tendinae & sudden prolapse of the valve.

- 1)X-ray:- Normal; except if associated with marked MR.
- 2) ECG: Slight depression of ST segment.
- 3) Echocardiography: shows posterior movement of MV cusps into LA during systole

⇒ Treatment :-

A) Medical treatment :-

- ☆ Prophylaxis against IE.
- Avoid heavy exercise.
- $^{\bigstar}$ $\beta\text{-blockers}$ (Indral) for treatment of chest pain , palpitation & psychological treatment .
- Anti-coagulants:- to prevent thrombo-embolism if marked MR with AF is present.
- B) Surgical treatment: Valvoplasty (if sever MR is present) or Valve replacement.



AORTIC VALVE

AORTIC STENOSIS

⇒ Aetiology:- [Normal Aortic valve area = 3 cm²]

A) Organic AS:-

* Congenital heart diseases :- they are either;

- Valvular heart diseases.
- Supra-valvular heart diseases: Hourglass narrowing above AV, e.g. William's syndrome;
 - ✓ Low seated ear, Protruded upper jaw.
 - ✓ Hypertelorism , Collagen defect .
 - ✓ Congenital hyperparathyroidism
 → Infantile hypercalcaemia.
- Sub-valvular heart diseases :- Obstruction in LV out flow tract which may be;
 - \checkmark Idiopathic hypertrophic sub-aortic stenosis \rightarrow Dynamic obstruction .
 - ✓ Idiopathic membranous sub aortic stenosis → fixed obstruction .

* Rheumatic heart diseases :- shows;

- History of Rheumatic fever.
- Double aortic lesion.
- Presence of atrial fibrillation (AF).

☆ Calcification: It is either;

- Primary: In old people & associated with coronary atherosclerosis.
- Secondary:- to other aortic lesion as Rheumatic, congenital,

☆ Infective endocarditis.

B) Functional AS:- due to;

- ☆ ↑ blood flow across the valve as in AR & hyperdynamic circulation .
- ☆ Dilatation of aorta but without dilatation of aortic ring as in; systemic HTN, Aortic aneurysm & atherosclerosis.

\Rightarrow Haemodynamics:- [In this case Aortic valve area = 0.8 cm²]

- In Systole; there is obstruction of LV out flow results in;
 - $\Leftrightarrow \downarrow LV$ stroke volume $\rightarrow \downarrow COP \rightarrow \downarrow$ peripheral & coronary blood flow.
 - $\updownarrow\uparrow \text{Pressure load on LV} \to \text{LVH} \to \text{LVF}$ & pulmonary congestive symptoms .

⇒ Clinical Picture :-

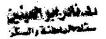
A) SYMPTOMS

- Mild cases: Asymptomatic.
- Symptoms of Low COP.
- Symptoms of LVF → lung congestion.
- Symptoms of complications.
- Angina pectoris due to :-
 - Low COP $\rightarrow \downarrow$ coronary blood flow.
 - LVH $\rightarrow \uparrow O_2$ demand.
 - ullet Shortening of diastole (due to long systolic with AS) \longrightarrow impaired coronary filling .
 - Concomitant coronary atherosclerosis .
 - \uparrow tension inside the cardiac MS \rightarrow squeezing of coronaries.
- Syncopal attacks: they are of 2 types;
 - Exertional :- due to Low COP.
 - At rest:-(rare); occur in calcific AS, due to extension of calcification to AV
 bundle → Adam's stock attacks & / or Complete heart block.

B) SIGNS

1) General signs :-

- ☆ Signs of Low COP.
- ☆ Signs of LVF (lung congestion).
- ☆ Pulse :- either;
 - ✓ Plateau pulse [pulsus tardus et parvus] ⇒ Isolated pulse .
 - ✓ Pulsus bisferance [bifid pulse , systolic double peak] ⇒ combined AS & AR .
- ☆ Blood pressure :- ↓ Systolic BP & ↓ pulse pressure.
- ☆ Systolic thrill over carotid vessels.



2) Cardiac signs :-

- ☆ LV apex :- Shifted out & down, localized, Pulging & Heaving (pressure over load).
- $\stackrel{*}{\simeq}$ Systolic thrill over $A_1 \rightarrow$ propagated to neck & apex.

☆ Auscultation:-

I] Over aortic area:-

- > S1:- weak (dt ↓ aortic pressure) & delayed (paradoxical splitting).
- > Systolic ejection click:- due to opening of rigid Aortic cusps.
- > Murmur :-
 - \rightarrow Site: maximum over A₁.
 - → Timing :- Mid (ejection) systolic .
 - → Character: harsh (organic) or soft (functional).
 - \rightarrow Position: \uparrow by leaning forward.
 - \rightarrow Respiration :- \uparrow by expiration .
 - → Propagation: selective propagation to neck arteries & apex.

II | Over Mitral area :-

- > Ejection systolic murmur (propagated), due to AS.
- > Pan-systolic murmur :- due to secondary MR.
- \triangleright Pre-systolic gallop (S4):- In sever cases (AS \rightarrow † LV pressure \rightarrow † LA contraction).

⇒ complications of AS :-

- \$ Sudden death :- due to;
 - > Ventricular fibrillation, with continuous contraction.
 - > Cardiac stand still (cardiac arrest).
 - > Myocardial infarction.
 - > Formation of thrombus over the stenotic orifice.
- ☆ Infective endocarditis .
- $\triangle LVF$.
- ☆ Rheumatic activity .

The KING in Medicine

□ Investigations:-

1) X- ray:- It will show;

- LV enlargement.
- Pulmonary congestion (if there is LVF)
- · Calcific Aortic valve.
- Small aortic knuckle.
- Post-stenotic dilatation.

2) ECG:-LV enlargement.

3) Echocardiography: - for;

- Diagnosis & localization of AS.
- Diagnosis of IHSS.

4) Cardiac Catheterization: It shows systolic gradient across site of obstruction;

- ⊃ In Valvular type:-
 - $◆ ≤ 50 \text{ mmHg} \rightarrow \text{mild AS}$
 - 50-100 mmHg \rightarrow Moderate AS.
 - > 100 mmHg \rightarrow Sever AS.
- ⊃ Sub-Valvular type: Gradient difference in LV cavity.
- Supra-valvular type: Gradient difference in ascending aorta.

5) Angiography: - It shows type of stenosis;

- ⊃ Sub-valvular :- Slit like LV cavity.
- ⇒ Valvular: ↓ mobility of aortic valve.
- Supra-valvular :- Hour glass narrowing of ascending aorta .

Assessment of severity of AS:-

- History taking (Symptoms).
- Clinical examination (Signs).
- Investigations.

