PULMONARY HEART DISEASE

1-PRIMARY PULMONARY - HTN.

2-SECONDARY PULMONARY- HTN.

3-CHRONIC COR PULMONALE.

4-PULMONARY EMBOLISM.

PULMONARY HYPERTENSION

Normal – pulmonary artery has low resistance to blood flow-

because it has thin media muscle layer of

pre-capillary blood arterioles

as compared with systemic vessels.

mPAP- at rest is 14+ 3

up to 20 mmHg

<u>Definition</u>

Increase in mean pulmonary artery pressure mPAP of greater than 25 mmHg at rest or of greater than 30 mmHg during exercise

primary pulmonary hypertension- PPH
secondary-PH- in association with other disease

PULMONARY HYPERTENSION- Causes

1- Pulmonary vascular disorder

- Primary pulmonary hypertension
- Acute massive pulmonary thromboembolism
- Multiple recurrent pulmonary thromboembolism-
-Pulmonary veno-occlusive disease -
- Parasitic infection e.g. schistosomiasis -

2- Disease of the lungs and HYPOXIA-

COPD

Interstitial lung disease- -

PULMONARY FIBROSIS- -

3--Musculoskeletal disorders

Kyphoscoliosis – -

HYPOVENTILATION -

-polyneuropathy-

Poliomyelitis- Myositis- Mysthenia gravis.

5- Disturbance of respiratory control

HYPOVENTILATION -

Obstructive or non- obstructive-sleep apnea -

- Morbid obesity (Pickwickian syndrome)

-brain stem -CVA-

respiratory center damage

6- Cardiac disorders

- Mitral valve stenosis

- Left ventricular failure

-cardiac tumor-- Left atrial myxoma

-CARDIAC--LT- RT-SHUNT-

VSD- ASD- PDA- Congenital heart disease

7- Miscellaneous

- Appetite suppresant drugs, e.g. dexfenfluramine

- Type I glycogen storage disease

- Lipid storage disease e.g. Gausher`s disease

VASCULITIS- -

e.g. SLE- SCLERODERMA- -

-THYROID DISESAE -

-- Liver cirrhosis- portal hypertension -

HIV- SARCOIDOSIS

Sickle cell disease

Primary pulmonary hypertension

Is a condition of unknown cause characterized by

clinical, radiological and ECG evidence of pulmonary hypertension

increase PAP and PVR

with normal PCWP

PVR= mpAP- mPCWP /CO =

normal value -1.5 wood unit

- primary PHH is less common
  than the secondary type
- There is
  a female predominance of 3/1
- It presents commonly in the 3rd. Decade
- There is about 6-12% of
  familial origin
  with autosomal dominant inheritance

Clinical feature

Insidious onset

dyspnea-fatigue-weakness-angina- syncope-

late with symptoms of

R. Ventricular failure and physical finding of

Pulmonary hypertension i.e.

JVP- large a and v wave ,

L. parasternal heave ,

loud P2, Pulmonary flow murmur ,

RV- 4th- HS, 3rd-HS

tricuspid & pulmonary regurgitation murmur,)

signs of R ventricular failure –

ANASRCA

(hepatomegaly, ascits, and peripheral oedema)

Investigation

Chest X-ray :-

Enlarged MAIN pulmonary arteries

Marked tapering of peripheral arteries

OLIGAEMIC LUNG –

pruning of peripheral arteries-

lucent lung fields

Right atrial and ventricular enlargement

ECG:-

-RVH- right ventricular hypertrophy

right atrial enlargement

Echocardiography :-

enlarged and dilated right side of the heart

Pulmonary function tests :- are usually normal

Pulmonary angiography :- to confirm diagnosis

<u>Differential diagnosis</u> :-

exclusion of secondary causes – MVS, congenital heart disease with Eisenmenger`s

-VASCULITIS- sickle cell

<u>Treatment</u> -

Avoid heavy physical activity-

Avoid pregnancy carry high risk of mortality

air plane travel should be with O2 supplement-

patient should be vaccinated for influenza-

Surgery- avoid GA-under spinal epidural anesthesia -

-ANTI-COAGULATION – warfarin –VASODILATORS- calcium channel blockers- endothelin-receptor antagonist (bosentan)- prostanoids -prostacycline- prostglandine- analogues – EPOPROSTENOL-short acting- IV-infusion. phosphodiesterase type 5

Chronic cor pulmonale

Definition :-

RV- enlargement and may be failure secondary to increase in afterload duo to diseases the thorax wall - air way – lung-parenchymal and pulmonary circulation .

Pathophysiology :-

HYPOXIA causing -pulmonary vasoconstriction and increase in pulmonary vascular resistance and pulmonary pressure there is progressive deterioration of R .ventricular function, with further hypoxia more dysfunction of R. and L. ventricles

Clinical features

-- chest pain ,exertional dyspnea,
syncope , fatigue and sudden death
-- on examination,
signs of pulmonary hypertension
and right sided - HF

-large `a` wave, prominent `v` wave
-right ventricular parasternal heave
-pulmonary ejection sound and flow murmur
-loud P2
-right. ventricular 4^{th}. and 3^{rd}. Heart sounds
-tricuspid and pulmonary regurgitation murmur
-hepatomegaly , ascitis and peripheral oedema

Investigations

Chest X-ray

- **right ventricular enlargment**
- **right atrial dilatation**
- **prominent pulmonary artery**
- **tapering of pulmonary artery**
- **oligaemic peripheral lung fields**

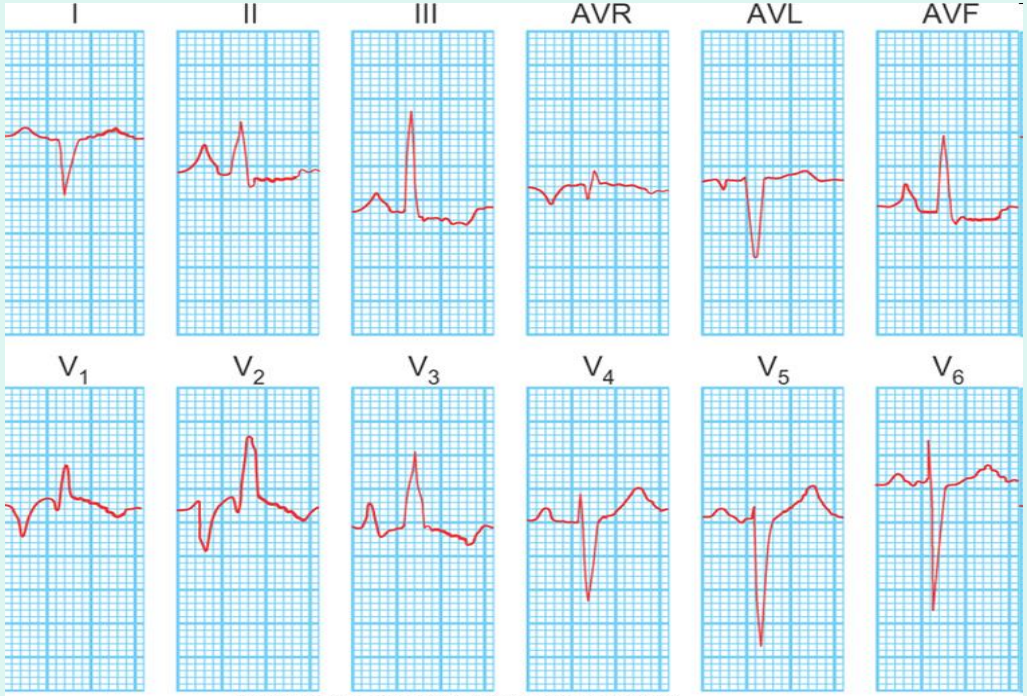
ECG

right ventricular hypertrophy

(right axis deviation, dominant R wave in lead V1, and inverted T wave in right precordial leads) and

right atrial hypertrophy

(tall peaked P wave in lead II)



Echocardiography

- right ventricular dilatation and/or

- right ventricular hypertrophy

Other investigations like

- cardiac catheterization

- pulmonary angiography

Treatment

- Treat underlying condition

- Diueretics for R. sided heart failure

- O2 therapy for hypoxia

Pulmonary embolism

--Thrombus from venous system- DVT-

, may dislodge and embolize

into the pulmonary arterial system.

-- 10% of PE- may be fatal

-- lung tissue is ventilated but not

perfused resulting –

intrapulmonary dead space- alveolar collapse– ventilation- perfusion- mismatch -

- HYPOXIA- PULMONARY VASOCONSTRICTION

-- There is reduction in the cross-sectional area

of the pulmonary arterial bed ,

this lead to pulmonary hypertension

and reduced cardiac output

Clinical feature

-- HIGH INDEX OF SUSPESION

Unexplained sudden onset of

- DROP IN O2 sat.

tachycardia and dyspnea

- Pluretic chest pain
- Hemoptysis – -

pulmonary infarction-30% -

-There are three typical clinical presentations

* Small /medium pulmonary embolism
* Massive pulmonary embolism
* Multiple recurrent pulmonary embolism

Investigations

Chest X-ray :-

linear atelectasis, blunting of costophrenic angle raised hemi- diaphragm , wedge shaped pulmonary infarct, \ abrupt cut-off of a pulmonary artery or translucency of an under-perfused distal lung zone

ECG :- is usually normal but may show

sinus tachycardia, atrial fibrillation ,or right ventricular strain (S1,Q3,T3) pattern is rare

Blood tests :-

polymorphonuclear leucocytosis, elevated ESR, and increased LDH level-D-DIMER

Investigation cont.

Radionuclide ventilation/ perfusion scanning

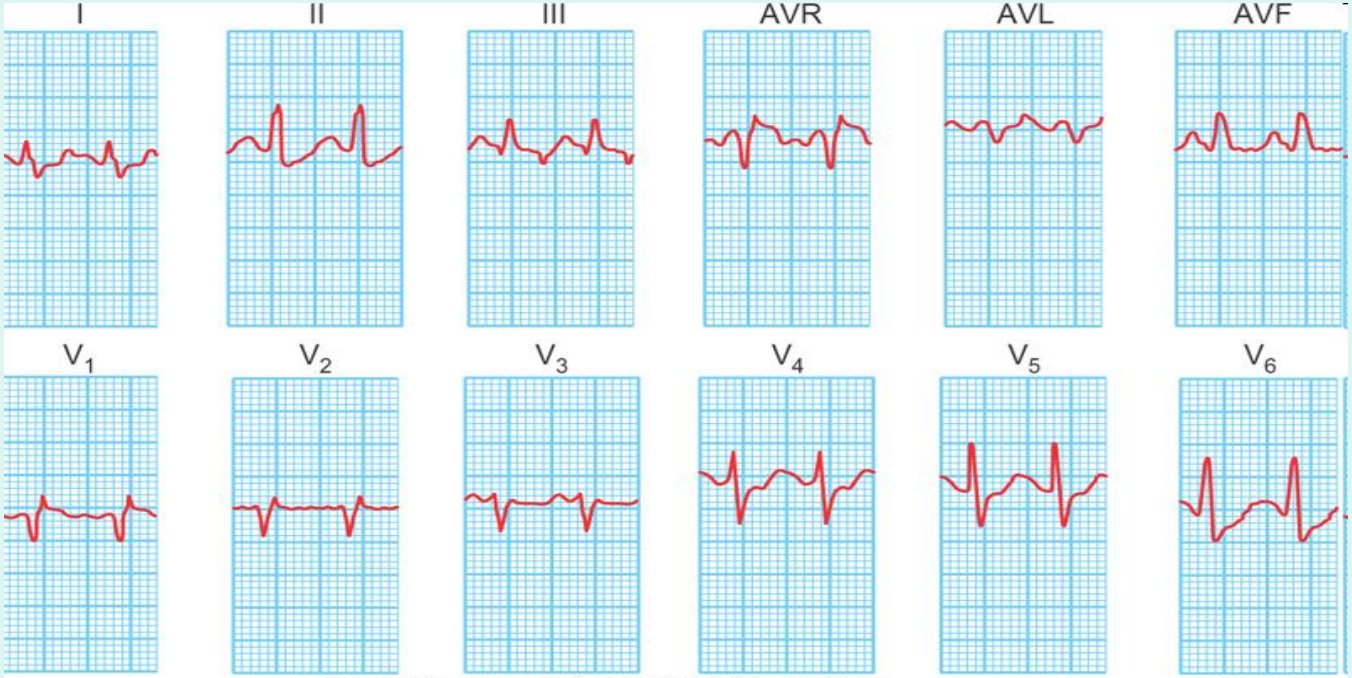
Ultrasound scanning –

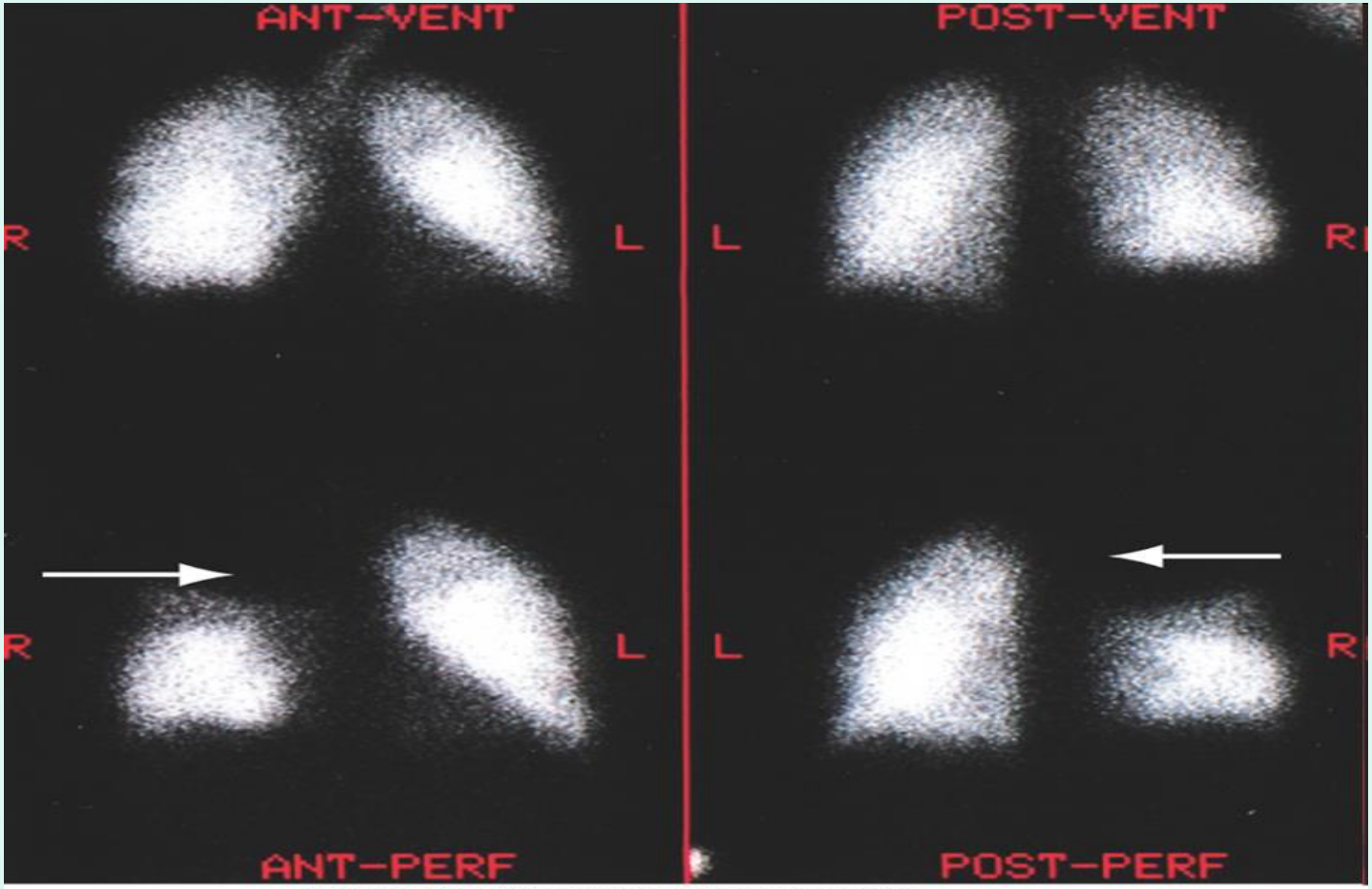
DOPPLEX-

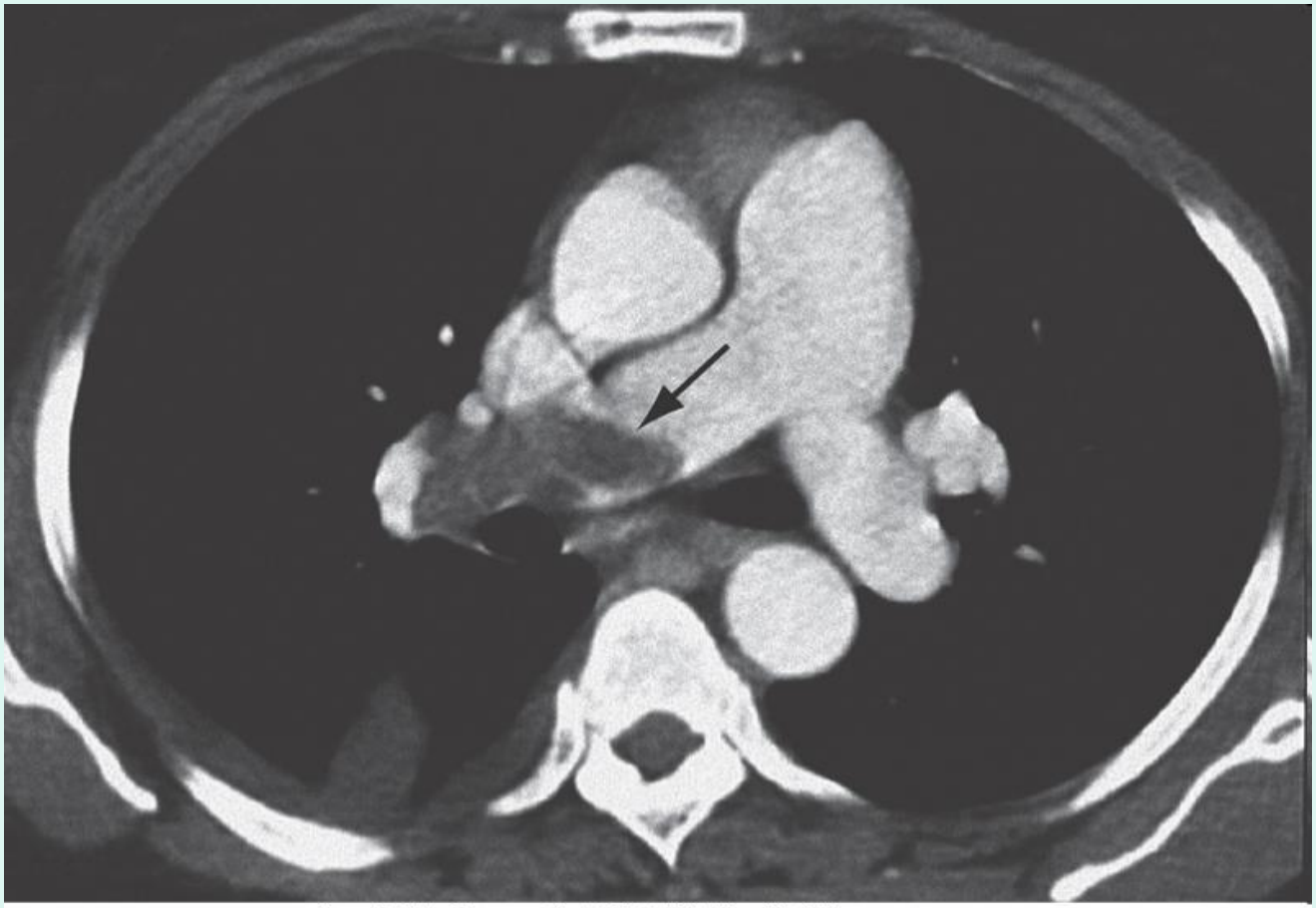
of pelvic or ilio-femoral - popliteal veins

HIGH RESOLUTION- CT ANGIO- scan

MRV- imaging







Treatment

Acute management :-

- High flow O2 therapy
- Bed rest
- Analgesia
- I .V. fluids
- Inotropics
- Admit to i.c.u

Dissolution of the thrombus :-

Fibrinolytic therapy like streptokinase (250 000 u.) by i.v. infusion over 30 minutes ,fallowed by streptokinase 100 000 units i.v. hourly for up to

12-72 hours).

Surgery :- Pulmonary embolectomy is only indicated in massive pulmonary embolism

Prevention of further emboli :-

- LMWH or conventional heparin

Oral anticoagulants- -

WARFARIN- -

DABIGATRAN – -

APIXBAN - REVORXIBAN -

Inferior vena cava filter