KEEP IN MIND:
Every Point in the Handout is important….

HYPERTENSION

In your exam, we expect you to know :

- ***Definiton*** **of Hypertension**

  Diastolic more than 90mmHg. Or Systolic more than 140mmHg.

- ***Predisposing Factors*** **of Hypertension**

  People who are more predisposed to it, such as:

  Africans – Smokers – Obese – Alcoholic – Type A personalities (stressful) – Having family history of HTN ….

- **Essential hypertension Is the most common**

  The reason is unknown (multifactorial : developed because of lots of factors)

- **Benign And Malignant *Associations* (Hemorrhage)**

- ***Complications*** **of Hypertension**

  MORE severity →MORE complications

  Complications happen in:

  - ***Malignant HTN*** **-- all the time**

    Retinal Hemorrhage

    People more predisposed to developing it → have more complications (get it earlier)

  - ***Benign HTN*** **– if not treated**

ISCHEMIC HEART IDSEASE

- Common types of IHD
  - Angina Pectoris
  - Acute MI
  - Sudden Cardiac Death
  - Chronic IHD with Congestive Heart Failure

- Contributing Factors (the list of things that cause it)

- Types of Angina Pectoris

  Stable: occurs when there's an increase in O_2 supply - e.g. while exercising – relieved by rest

  Variant (Prinzmetal): occurs even at rest – due to coronary artery spasm – responds to Vasodilators.

  Unstable: *OFTEN* occurs at rest – due to disruption of an atherosclerotic plaque

  → partial thrombosis – embolism - Vasospasm

  →subsequent MI (right before it)

  That's why we call it Pre-infarction Angina

- MI → need to know that its COAGULATIVE NECORSIS

  Common Cause: A blockage in coronary arteries - it can be associated with Athersclerosis or Thrombosis

  > Atherosclerosis:
  > Abnormal Thickness of arteries
  > Thrombosis:
  > Formation of a blood clot due to hemorrhage in the atherosclerosis or other causes.

- MUST KNOW – Which vessel supplies which part of the heart (and vice versa)
- Factors Affecting the size of the infarct
- Blood vessels involved commonly
- Pathogenesis – they might not ask (but u never know)
- EVENTS OF ISCHEMIC CARDIAC MYOCYTES, u should know:
  - When does the Infarct reach its full size? (within 2-3hrs.) and what happens during this period?
  - Irreversible cellular injury (myocardial necrosis) within 20-40minutes.

- Complications of MI

  (One of the complications: if heart will rupture – muscle ruptures → blood in pericardium → hemopericardium)
- The enzymes elevated in MI (CK-MB and Troponin)
- Chronic Ischemic Heart disease (some questions about it)
- Sudden Cardiac Death, is due to atherosclerosis (most common)

Atherosclerosis

- Causes (Risk Factors) the modifiable and non-modifiable ones
- Morphology of the block (how does the atherosclerotic plaque look like)
- Complications (a very important MCQ)
- HDLD → Good Cholesterol
  VLDL , LDL ,Chylomicron → Bad Cholesterol

Rheumatic Fever

- Must know the causative organism (gp. A streptococci)
- It is Pancarditis (affects all the heart)
- Aschoff nodules - morphology is important

  aschoff nodules , aschoff bodies has fibrinoid necrosis of vessels
  because blood vessels supplying the heart are the one that undergoes necrosis
  The myocardial is inflamed and not necrosed
  (in MI it is the heart the is necrozed → coagulative)

- Complications – we should know abt them
- The side of the heart, more likely to have Rheumatic Fever (left side)
- Valves commonly involved (Mitral Stenosis →most common)
- Clinical features
- Critireia of diagnosis (2 criteria major and minor → 2 major or 1 major and 2 minor)

If there's a Good evidence of infection → they'll ask us about the criteria

P.S. Rhuematic Fever is not included in the MID-TERM

Questions By the Students:

- **What is the difference between Rheumatic Fever and Rheumatic Carditis?**
  Rheumatic fever has a clinical feature of carditis (the same thing)

- **Why is – younger people develop more complications?**
  Because they are less responsive to medications

- **Why is – the Left Ventricle is the one having Hypertrophy in MI??**
  a person with systemic HTN usually have ↑P.R
  → the heart has to contract more to fight it - it keeps contracting faster and stronger
  → it will be more hypertrophic (like a muscle exercising → it will become big) →
  we have hypertrophy (muscle fibers are the same but they r bigger)

there's no dilatation (theory)
→ heart doesn't have a place to dilate when it is a "left Ventricular Hypertrophy" because the lung is next to it
→ hypertrophy is to the lumen's side (it becomes big inside)

But when it is the right atrium that is having Hypertrophy (in other cases)
If there's Pulmonary Hypertension (for instance)
right side of Heart has to contract more → there's dilatation becuase there's more space than the left side.
(it dilate to the outside – unlike the left ventricle).

- **It is normal if a person is "obese" and is having "normal Cholesterol levels" because they may be taking Anti-Hypercholesterolemic drugs (and vice versa with thin people)**

- **TYPES OF ANIGINA**
  - Angina Pectoris is what we see – when we exercise- the heart realizes it can go on –
  perhaps there's a block → we give them drug to dilate it)
  - Crescendo Angina → getting worst (with less effort → more pain) blood vessel is developing a thrombus/rupture/clot
  → it start developing ATH (atherosclerosis)
  - Variant Angina →Spasm = blood flow reduced → no O_2 to muscle (variant Angina)

- **Prevention of ATH**
  (primary: we prevent it when we are not having it – no heart problems – prevent any cardiac symptom before it happens)
  (secondary: already developed it - will do things to stop it).

Written By: Pathology Team Leader – Dona Baraka