Chapter

2

Cardiovascular System

A. HYPERTENSION

Q 1. What is hypertension? Give etiopathogenesis, clinical manifestations, pharmacological and nonpharmacological management of hypertension.

Hypertension

Hypertension is defined as systolic blood pressure greater than 140 mmHg and diastolic pressure greater than 90 mmHg.

Types of Hypertension

1. **Primary (essential) hypertension:** Its exact cause is not known. But there is generally narrowing of blood vessels. It may be benign hypertension or malignant hypertension.
2. **Secondary hypertension:** Its cause is known. It may be caused due to secondary disorders like renal disorders, endocrine disorders or toxemia of pregnancy.

Etiopathogenesis

- The etiology of primary hypertension is not known.
- Secondary hypertension can be caused due to:
  a. Renal diseases, e.g. chronic diffuse glomerulonephritis, pyelonephritis, polycystic kidneys.
  b. Endocrine diseases, e.g. Cushing's syndrome, pheochromocytoma, primary aldosteronism.
  c. Vascular lesion, e.g. renal artery disease, contraction of aorta.
  d. Other causes: Obesity, thyroid problems, stress, high alcohol intake, high salt and fatty diet, less physical activity.

Clinical Manifestations

- Severe headache
- Fatigue
- Vision problems
- Chest pains
- Nose bleed
- Difficulty in breathing
- Irregular heart beats
- Feeling of pulsations in the neck or head
- Giddiness, palpitation
- Anxiety, nervousness, dizziness
- Sweating, facial flushing
- Epistasis, vomiting.

Nonpharmacological Management of Hypertension

- Lifestyle modification as the first line of treatment
- Adopt DASH eating plan (a diet rich in fruits and vegetables)
- Reduce intake of sodium
- Exercise regularly
- Reduce weight
- Reduce alcohol consumption
- Stop smoking
- Stress management
- Yoga, meditation
- Reduce exposure to air pollution and cold temperature.

Pharmacological Management of Hypertension

Pharmacological management means management of hypertension with drugs.

1. **Diuretics:** Diuretics are the drugs which increase the urine output by helping the kidneys to inhibit the sodium reabsorption in the distal convoluted tubules, ascending limb and loop of Henle.
   Examples: Hydrochlorothiazide, chlorthalidone, furosemide.
2. **β-blockers:** These drugs reduce the workload of the heart and blood vessels, causing the heart to beat slowly and with less force.
   Example: Propranolol, atenolol.
3. **α-blockers:** These drugs cause the peripheral vasodilation of blood vessels.
   Examples: Prazosin.

4. **Ca^{2+} channel blockers:** These drugs block the movement of extracellular calcium into the cells, causing vasodilation and decreased heart rate.
   Examples: Nifedipine, amlodipine.
5. **ACE inhibitors:** These drugs inhibit the angiotensin converting enzyme and reduce the conversion of angiotensin-I to angiotensin-II and prevents vasoconstriction.
   Examples: Captopril, ramipril, enalapril.
6. **Vasodilators:** These medicines directly act on the muscles of arterial wall and prevent the muscles from tightening and arteries from narrowing.
   Examples: Hydralazine, nitroglycerin, sodium nitroprusside.

B. ANGINA (ANGINA PECTORIS)

Q 1. What is angina pectoris? Give etiopathogenesis, clinical manifestations, nonpharmacological and pharmacological management of angina pectoris.

Angina (Angina Pectoris)

- Angina pectoris involves pains in the chest.
- Angina pectoris is a heart disease in which there is a paroxysmal pain in the chest, usually due to interference with the supply of oxygen to the heart muscles and precipitated by excitement.
- Angina pectoris refers to the chest pain towards left arm shoulder due to less supply or more demand of oxygen.
- Angina is due to imbalance between myocardial oxygen requirement and oxygen supply.

Types of Angina

a. Stable angina
b. Unstable angina
c. Variant angina
d. Microvascular angina.

Etiopathogenesis of Angina

- The pathogenesis of angina involves basic underlying mechanism of an imbalance in myocardial oxygen supply and demand.
- Angina is related to limited blood flow in coronary artery, hence also called coronary artery disease (CAD).

- The blood supply to myocardium may be decreased due to four main mechanisms:
  a. Atherosclerosis
  b. Coronary vasospasm
  c. Embolism
  d. Congenital coronary abnormality.
- Angina is caused by reduced blood flow to heart muscle. Blood carries oxygen, which heart muscle needs to survive. When heart muscle is not getting sufficient oxygen, it causes a condition called ischaemia.

Clinical Manifestation of Angina

- Chest pain
- Dyspnoea
- Increased sweating
- Fatigue, weakness
- Breathlessness
- Reduced cardiac output
- Indigestion
- Pulse rate and blood pressure increases
- Heart burn
- Palpitation
- Abdominal pain
- Back discomfort.

Nonpharmacological Management of Angina

- Smoking cessation
- Avoid alcohol consumption
- Weight loss
- Reduction in cholesterol intake
- Balanced diet and nutrition
- Salt restriction
- Lifestyle modifications.

Pharmacological Management of Angina

1. **Nitrates:** Nitrates work as vasodilators and arterial dilators. By dilating the blood vessels of the heart, nitrates can reduce the stress on the heart by improving blood flow to the heart muscle. These drugs relieve angina symptoms.
   Examples: Nitroglycerines.

2. **Antiplatelet drugs:** Antiplatelet drugs work by decreasing platelet aggregation and inhibiting thrombus formation.
   Examples: Aspirin.
3. **Ca^{2+} channel blockers:** These drugs cause vasodilation and decreased heart rate by blocking the movement of extracellular calcium into the cells.
   Examples: Verapamil, diltiazem.
4. **β-blockers:** These drugs reduce the workload of the heart and blood vessels, causing the heart to beat slowly and with less force.
   Examples: Propranolol.
5. **Surgery:** Surgery may be recommended if medicines are not helping to control angina. Two main types of surgeries for angina pectoris are:
   a. Coronary angioplasty.
   b. Coronary artery bypass surgery.

C. MYOCARDIAL INFARCTION (HEART ATTACK)

Q 1. What is myocardial infarction? Give etiopathogenesis, clinical manifestations, nonpharmacological and pharmacological management of myocardial infarction.

Myocardial Infarction (MI)

Myocardial infarction (MI) or acute myocardial infarction (AMI) is generally known as heart attack and it refers to tissue death (infarction) of the heart muscle (myocardium).

- Myocardial infarction is the irreversible death (necrosis) of heart muscle secondary to prolonged lack of oxygen supply (ischaemia). Hence, it is also called ischemic heart disease (IHD).
- Commonly myocardial infarction is known as heart attack, occurs when blood flow decreases and stop to a part of heart (myocardium), it leads to damage of the heart.

Causes of Myocardial Infarction

- Loss of blood supply to the myocardium of heart.
- Blockage in coronary artery.
- Deposition of fat in coronary artery.
- Coronary artery vasospasm.
- Ventricular hypertrophy.
- Hypoxia.

Types of Myocardial Infarction

1. ST elevation myocardial infarction.
2. Non-ST elevation myocardial infarction (NSTEMI).

Clinical Manifestations of Myocardial Infarction

- Chest pains/chest discomfort
- Dyspnoea
- Fatigue, weakness
- Anxiety
- Restlessness
- Heart burn
- Shortness of breath
- Excessive sweating
- Nausea, vomiting, light headedness
- Palpitation
- Sleepiness
- Hypertension.

Etiopathogenesis of Myocardial Infarction

- The most common cause of myocardial infarction is the rupture of an atherosclerotic plaque on an artery supplying heart muscle.
- Plaques become unstable, rupture and additionally promote the formation of blood clot, that blocks artery.
- The blockage of an artery can lead to tissue death in tissue being supplied by that artery. This may lead to heart attack.
- There are many factors which are causative agents for blockage of coronary arteries.
  i. Bad cholesterol
  ii. Saturated fats
  iii. Transfats
- A myocardial infarction may result from a heart with a limited blood supply subject to increased demands, such as in fever, a fast heart rate, hyperthyroidism, too few red blood cells in the bloodsteam, or low blood pressure.
- Damage or failure of procedures such as percutaneous coronary intervention or coronary artery bypass grafts may cause a myocardial infarction.
- Spasm of coronary arteries, such as Prinzmetal's angima may cause blockage.

Nonpharmacological Management

- Avoid alcohol consumption
- Avoid smoking
- Avoid cholesterol intake
- Bed-rest
- Reduce sodium intake
- Regular medical checkup
- Psychotherapy to relieve nervousness
- Increase intake of high fibre rich diet
- Proper counselling and educating to the people concerned

Pharmacological Management

The following pharmacological agents are used to treat myocardial infarction:

1. Vasodilators, e.g. nifedipine, glyceryl trinitrate.
2. β-blockers, e.g. propranolol, metoprolol, atenolol, esmolol.
3. ACE inhibitors, e.g. captopril, enalepril, ramipril.
4. Thrombolytic agents, e.g. streptokinase, urokinase.
5. Analgesics (to reduce pain), e.g. morphine.
6. Surgery: Angioplasty or coronary bypass.

D. HYPERLIPIDAEMIA

Q 1. Define hyperlipidaemia. Give etiopathogenesis, clinical manifestations, nonpharmacological and pharmacological management of hyperlipidaemia.

Hyperlipidaemia

Hyperlipidaemia is a disease in which blood contains a high concentration of lipids such as cholesterol and triglycerides.

- Hyperlipidaemia is also known as hypercholesterolemia in which non-HDL cholesterol and LDL (bad) cholesterol levels in the blood are elevated.
- Cholesterol is a molecular fat which is produced by liver and is essential for the health of cell membranes, brain functioning, hormone production and vitamin storage.

Types of Hyperlipidaemia

1. **Primary hyperlipidaemia:** It is also called familial hyperlipidaemia because it is caused by specific genetic abnormalities.

2. **Secondary hyperlipidaemia:** It is also called acquired because it is caused by another disorders like diabetes, nephrotic syndrome, hypothyroidism and with drugs like corticosteroids, β-blockers and oral contraceptives.

Etiopathogenesis of Hyperlipidaemia

- Hyperlipidaemia can be due to primary or secondary causes.
- The primary causes include familial hypercholesterolemia which is a genetic disorder.
- Secondary causes: Diabetes mellitus, hypothyroidism, renal disease, high carbohydrate diet, alcohol consumption, hepatic disease, obesity, cholestatic liver disease, Cushing's disease.
- Drugs like corticosteroids, β-blockers estrogens may cause hypercholesterolemia.
- Other factors: Smoking, drinking a lot of alcohol, eating food having high saturated fat or trans fats, being stressed, being overweight, etc.

Clinical Manifestation of Hyperlipidaemia

- Chest pain/pressure (angina)
- Blockage of blood vessels in brain and heart
- High blood pressure
- Stroke
- Myocardial infarction
- Coronary heart disease
- Carotid artery stenosis.

Nonpharmacological Management of Hyperlipidaemia

- Reduce alcohol intake
- Reduce smoking
- Weight loss
- Reduce intake of fat and saturated fat
- Modification of lifestyle
- Eat more fruits and vegetables
- Increase fibre intake in diet.

Pharmacological Management of Hyperlipidaemia

It includes the use of pharmacological agents in the treatment of hyperlipidaemia.

1. **HMG-CoA reductase inhibitors**, e.g. lovastatin, simvastatin, metastatin, pravastatin, fluvastatin, atorvastatin, pitavastatin, rosuvastatin.

2. **Fibric acid derivatives**, e.g. clofibrate, fenofibrate, gemfibrozil, ciprofibrate, bezafibrate, fluvastatin.
3. **Bile acid sequestrants**, e.g. cholestyramine, colestipol.
4. **LDL oxidation inhibitor**, e.g. probucol.
5. **Pyridine derivatives**, e.g. nicotinic acid, nicotinamide.
6. Cholesterol absorption inhibitors, e.g. ezetimibe.
7. Miscellaneous agents, e.g. β-sitosterol.

E. CONGESTIVE HEART FAILURE (CHF)

Q 1. What is congestive heart failure (CHF)? Give the etiopathogenesis, manifestations, nonpharmacological management and pharmacological management of congestive heart failure (CHF).

Congestive Heart Failure (CHF)

Congestive heart failure is a chronic progressive condition in which the heart cannot pump enough blood to meet the metabolic needs of the body because of pathological changes in myocardium.

- CHF is a serious disease characterized by a reduction in heart's pumping capacity.
- CHF is also known as 'heart failures'.
- Heart failure means inability of heart to supply oxygenated blood for the body needs.

Types of CHF (CCF)

1. Left-sided heart failure: It is caused by:
   a. Ischemic heart disease
   b. Myocardial diseases
   c. Systemic hypertension
2. Right-sided heart failure: It is caused by:
   a. Lung's disease
   b. Pulmonary hypertension
   c. Myocardial diseases
   d. Tricuspid valve rupture

Causes of CHF

- Coronary artery disease
- Congenital heart disease
- Tachyarrythmia
- Valvular heart disease

- Hypertension
- Rheumatic fever
- Pneumonia
- Diabetes, hypo/hyperthyroidism, pheochromocytoma
- Deficiency of thiamine, selenium, calcium, etc.
- Alcohol, cocaine, cannabis
- Beat-blockers, calcium antagonists
- Severe anaemia, obesity

Etiopathogenesis of Congestive Heart Failure

- Pressure and volume overload, muscle loss, primary muscle disease or excessive peripheral demands, such as high output failure, are all causes of CHF.
- The heart muscle becomes less contractile as a result of CHF.
- Common causes of heart failure include coronary artery disease, including myocardial infarction (heart attack), high blood pressure, atrial fibrillation, valvular heart disease, alcohol use, infection and cardiomyopathy.

Clinical Manifestations of CHF

- Shortness of breath
- Excessive tiredness
- Swelling in legs, ankles and feet
- Swelling in abdomen
- Chest pain, dyspnoea
- Palpitation and irregular heart beat
- Nausea and loss of appetite
- Concentration problems or lack of alertness
- Feeling of fatigue and weakness
- Nocturia
- Fluid retention
- Slow weight gain

Nonpharmacological Management of CHF

- Cessation of smoking, alcohol consumption
- Regular exercise

- Diet and fluid restrictions
- Controlling conditions like hypertension, diabetes
- Eating healthy food
- Maintaining healthy weight
- Reduce and manage stress

Pharmacological Management of CHF

1. **Beta-blockers**, e.g. metoprolol, atenolol, bisoprolol, celiprolol, etc.
2. **Angiotensin converting enzyme inhibitors**, captopril, enalapril, ramipril.
3. **Angiotensin receptor blockers**, e.g. losartan, candesartan, telmisartan, valsartan (diovan), irbesartan, olmesartan.
4. **Glycosides**, e.g. digoxin, digitoxin.
5. **Diuretics**, e.g. furosemide, hydrochlorothiazide, bumetanide, spironolactone.

SHORT ANSWER TYPE QUESTIONS **3 Marks**

1. Define hypertension. Explain etiopathogenesis of hypertension.
2. What are clinical manifestations of hypertension? Mention nonpharmacological measures to control hypertension.
3. What is hypertension? Give pharmacological treatments for hypertension.
4. Define angina pectoris. Mention types of angina pectoris. State clinical symptoms of angina pectoris.
5. Write a brief note on etiopathogenesis of angina pectoris.
6. What do you mean by myocardial infarction? Give the causes, types and clinical manifestations of myocardial infarction.
7. Describe etiopathogenesis of myocardial infarction.
8. Write about nonpharmacological and pharmacological treatments/ management of myocardial infarction.
9. Define hyperlipidaemia? Give its types. Mention clinical manifestations of hyperlipidaemia.
10. Write a note on etiopathogenesis of hyperlipidaemia.
11. What are pharmacological and nonpharmacological management of hyperlipidaemia?

12. What is congestive heart failure (CHF)? Give types of CHF. What are the causes of CHF?
13. Explain the following in case of CHF:
    a. Etiopathogenesis
    b. Clinical manifestations
14. Mention nonpharmacological and pharmacological management of CHF.

OBJECTIVE QUESTIONS WITH ANSWERS IN BOLD LETTERS

1. Angina pectoris is defined as chest pain caused by **coronary heart disease**.
2. The angina which is provoked by physical exertion or emotional stress is **stable angina**.
3. The condition in which there is an inadequate supply of oxygen to a portion of myocardium is known as **ischaemia**.
4. Hypertension is a condition in which both systolic and diastolic pressures are **greater than 140 mmHg and 90 mmHg**, respectively.
5. The measure of myocardial oxygen consumption is **anginal index**.
6. Hyperlipidaemia is a condition of having serum concentration of total cholesterol **more than 200 mg/dl**.
7. Angina where there is no coronary artery blockage is **microvascular angina**.
8. **Atorvastatin** is a first-line lipid lowering drug.
9. Anginal index is calculated as:
   Angina index = heart rate × systolic BP
10. The most common type of angina is **stable angina**.
11. Full form of NSTEMI is **non-ST segment elevation myocardial infarction**.
12. The total cholesterol of higher than **240 mg/dl** is abnormal.
13. The anginal attacks are unpredictable and almost always occurs at rest or during sleep is called **Prinzmetal's angina**.
14. HDL should be less than **50 mg/dl**.
15. Acquired hyperlipidaemia is also called **secondary dyslipoproteinemia**.
16. Crescendo angina is a **stable angina**.

17. In **NSTEMI** condition, biochemical markers of ischaemia appears in the blood.
18. Heart failure is a condition in which **the heart cannot pump enough blood to meet body's needs**.
19. Cardiovascular disease (CVD) is a general term used for conditions affecting the **heart and blood vessels**.
20. Blood clot that lodges the cerebral artery, causes **stroke**.
21. Blood pressure refers to the force of blood pushing against **artery walls**.
22. The medical term of chest pain is **angina**.
23. The normal range of blood pressure is **120/80 mmHg**.
24. The device used to measure blood pressure is **sphygmomanometer**.
25. Hypertension is commonly called the **silent killer**.