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COMPREHENSIVE REVIEW OF COMMON CARDIOVASCULAR PATHOLOGIES: FROM THE PATHOPHYSIOLOGY OF MYOCARDIAL INFRACTION TO STATIN PHARMACOLOGY

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Abstract: Cardiovascular diseases (CVDs) represent a formidable global health challenge, encompassing a range of conditions affecting the heart and blood vessels. This review synthesises current understanding of several key CVDs, including coronary artery disease (CAD), myocardial infarction (MI), hypercholesterolemia, atherosclerosis, and heart failure. We delve into their respective introductions, risk factors, causes, signs and symptoms, and diagnostic approaches. A particular focus is given to the pathophysiology of MI, highlighting the intricate cascade from endothelial injury to irreversible myocardial necrosis. Furthermore, the roles and mechanisms of action of statins—a cornerstone in the pharmacological management of hypercholesterolemia and ASCVD—are discussed in detail, including their pharmacokinetics, pharmacodynamics, therapeutic uses, adverse effects, and significant drug-drug interactions. The aim is to provide a concise yet comprehensive overview of these critical cardiovascular conditions, underscoring the multidisciplinary approach required for their diagnosis and treatment.
This study has been undertaken to investigate the determinants of stock returns in Karachi Stock Exchange (KSE) using two assets pricing models the classical Capital Asset Pricing Model and Arbitrage Pricing Theory model. To test the CAPM market return is used and macroeconomic variables are used to test the APT. The macroeconomic variables include inflation, oil prices, interest rate and exchange rate. For the very purpose monthly time series data has been arranged from Jan 2010 to Dec 2014. The analytical framework contains**.**

IndexTerms - CARDIOVASCULAR DISEASE ,CORONARY ARTERY DISEASE,CORONARY HEART DISEASE,HEART DAMAGE, CHOLESTROL,LIPOPROTEIN.

INTRODUCTION:

Cardiovascular disease (CVD) encompasses all forms of illnesses affecting the blood circulation system, comprising the heart and vessels, which respectively displace and carry the blood. In other words, it is a complex disease comprising many conditions acquired and present at birth [1]. Cardiovascular disease, also known as heart disease, includes the following 4 entities: coronary artery disease (CAD, also known as coronary heart disease, CHD), cerebrovascular disease, peripheral artery disease (PAD), and aortic atherosclerosis. CAD is a result of reduced perfusion of the myocardium that leads to angina pectoris from ischemia and can finally lead to myocardial infarction MI and heart failure. Aortic atherosclerosis is the entity responsible for the aneurysms of the aorta, the thoracic and abdominal aortas. This presentation will examine the diagnosis and treatment of cardiovascular diseases and the medical team's responsibilities in these processes [2]. These factors directly contribute to poor quality of life and serious health complications, including heart attacks, strokes, heart failure, and arrhythmias, among many complications that lead to lifelong disability. The effects of CVD transcend physical health to mental well-being and pose immense economic strain on families and health systems globally [3]. The mechanism of risk factors differs based on the disease. Dietary risk factors are thought to account for 53% of cardiovascular deaths. Atherosclerosis is found in coronary artery disease, cerebral vessel disease, and peripheral artery disease. Inactivity, obesity, high blood pressure, smoking, high blood cholesterol levels, a poor diet, excessive alcohol intake, and inadequate sleep are risk factors that can contribute to cardiovascular diseases. Hypertension is thought to account for 13% of cardiovascular deaths, tobacco for 9%, diabetes for 6%, lack of physical activity for 6%, and obesity for 5%. Undiagnosed diseases can sometimes cause rheumatic heart disease. Approximately 90% of cardiovascular diseases can be prevented. [4]

INTRODUCTION OF CORONARY ARTERY DISEASE:

Coronary artery disease (CAD) is identified based on the development of atherosclerosis in the coronaries. In some instances, this can be symptom-free. However, coronary heart disease (CHD), commonly referred to as ischemic heart disease (IHD), can be associated with stable angina, acute coronary syndrome (ACS), and silent myocardial ischemia, among other disorders. Deaths caused by CHD are essentially brought about by CAD. In most instances, ACS is associated with signs and symptoms, such as

those experienced in unstable angina and myocardial infarction. In this discussion, we can refer to CHD as "CAD" for ease of clarification.[5].

Compared with the general population, the risk/benefit profile associated with revascularisation differs among patients with ESRD. For patients with stable ESRD but multiple-vessel coronary disease, bypass grafting despite the risks associated with stroke, coronary infection, or MI appears beneficial. For patients with acute coronary disease associated with ESRD, procedures associated with the target vessel remain the most beneficial. [6]

This paper aims at discussing the issues associated with patients presenting with coronary artery disease but with concomitant ESRD.

Coronary Artery Disease (CAD) is one of the most frequent heart conditions, involving the accumulation of atherosclerotic plaque on the inner lining of the arterial wall. This slows the flow of blood, thereby compromising the oxygen supply to the heart muscles. This condition is the major source of morbidity and mortality in the US and the whole world.[5]

INTRODUCTION TO MYOCARDIAL INFARCTION:

A heart attack, sometimes referred to as a myocardial infarction (MI), is a potentially fatal medical illness that happens when blood flow to a portion of the heart muscle is cut off. Heart tissue is damaged or dies as a result of this lack of blood flow, which might have serious repercussions for the person. Heart attacks are one of the world's top causes of death because they can result in long-term heart damage as well as other consequences. A myocardial infarction is categorised according to the type of blockage and the degree of heart damage:A more serious kind of heart attack brought on by a total blockage of a coronary artery is known as an ST-Elevation Myocardial Infarction (STEMI): Changes in the heart's electrocardiogram (ECG) are typically used to diagnose it.Non-ST-Elevation Myocardial Infarction (NSTEMI): A less serious form of heart attack in which there may be less damage to the heart muscle and a partial or transient occlusion.To restore blood flow and stop additional heart damage, both types of myocardial infarction require immediate medical attention. [7]

INTRODUCTION TO HYPERCHOLESTEROLMIA:

Lipoprotein abnormalities are relevant to practice because of the impact of lipoproteins on atherogenesis and, by association, the risk of atheromatous cardiovascular disease (ASCVD). In patients with established ASCVD (secondary prevention), reducing cholesterol has been found to reliably decrease cardiovascular mortality and cardiovascular events in both men and women and in middle-aged and older individuals. In individuals without cardiovascular disease (primary prevention), there is also extensive evidence with statins.

Lipoprotein disorders were, in the past, the concern of specialists in the field of lipids. With the benefit of the use of statins, particularly through the reduction of cardiovascular events, the treatment for hypercholesterolemia has become possible for family and internal medicine practitioners. Yet, the concern for a considerable number of patients who require adequate treatment for lipid-lowering medications continues to exist.[8]

Familial hypercholesterolemia (FH) is an autosomal dominant inherited condition with early-onset atherosclerosis. The condition was first recognised in the latter part of the 1930s by Carl Müller, a Norwegian clinician, in what remains to this day as a classic paper, proposing a concept for hypercholesterolemia and tendinous xanthomas and their possible monogenic inheritance with cardiovascular (CV) disease [1]. The phenotype was later elucidated in a seminal report in 1963 with strict criteria for both heterozygous and homozygous forms with dominant inheritance, and this condition has been divided into two forms: HeFH for the milder heterozygous form and HoFH for both homozygous forms. Although Dr Müller first hypothesized both causal and interventive treatment might be successful, there has been almost 50 years for this to be possible.[9]

INTRODUCTION ATHEROSCLEROSIS:

Atherosclerosis is a disease characterised by the accumulation of lipids, fibrous elements, and calcification within large arteries. This process is initiated by the activation of the endothelium, followed by a sequence of events that imply vessel narrowing and activation of the inflammatory pathways to result in atherosclerotic plaque. The cardinal outcome of all these processes leading to cardiovascular complications remains the leading cause of death worldwide.[10]

Atherosclerosis, an entity where lipids accumulate and is a major reason for the occurrence of cardiovascular events, has been a point of interest for several years. Nonetheless, there has been a recent surge in interest surrounding the initiation of atherosclerosis, wherein there has been a shift from the classic mechanism related to the vascular involvement by oxidised lipids to the newly found role of chronic inflammation. The concentration of pro-inflammatory cytokines, together with the role of the involvement of transcription factors, results in a self-feeding mechanism wherein atherosclerotic plaques can be formed and developed. Ranging from the expression of the nod-like receptor protein 3 (NLRP3) inflammasomes and Notch/Wnt signalling mechanisms to the enhancement of the VEGF-A expression and the reduction in the expression of connexins Cx32, Cx37, and Cx40, all further elevate the mechanisms related to the dysfunction of the endothelium and atherosclerotic plaque development. Within this manuscript, the discussion shall provide a glimpse into the molecular mechanisms related to the initiation and progression of atherosclerotic plaques, and shall further provide a look at the risk factors related to atherosclerosis [11].

INTRODUCTION TO HEART FAILURE:

Heart failure means your heart is unable to pump enough oxygen-filled blood to your body to support your life, but heart failure does not mean your heart has stopped or is going to stop beating, and without enough oxygen, your other body organs will not function well, which creates a big problem for the body, etc.

Heart failure may involve one or both sides of your heart:

In right-sided heart failure, your heart is not strong enough to move enough blood through to get oxygen from the lungs.

In the case of heart failure on the left side, there is not enough oxygenated blood being pumped out to the body. This is because the left side of the heart fails to perform its functions due to:

Lacking strength, even for pumping blood.

Too thick or stiff to relax and fill with blood properly. Left-sided heart failure is more common than right-sided heart failure.[12]

CORONARY ARTERY DISEASE RISK FACTORS:

- Age, Environmental risk factors, Family history and genetics, Heart and blood vessel diseases [13].

MYOCARDIAL INFARCTION RISK FACTORS:

Your age, family history, lifestyle, and several medical issues can all raise your risk of heart disease and heart attacks. We refer to these as risk factors. Furthermore, approximately 50% of Americans have at least one of these three major heart disease risk factors: smoking, high blood pressure, and high blood cholesterol. [14].

HYPERCHOLESTEROLMIA RISK FACTORS:

The following are risk factors for elevated cholesterol:

eating patterns. High cholesterol can result from consuming excessive amounts of trans or saturated fats. Alcohol. When a blood clot obstructs blood flow to a portion of the brain, a stroke occurs. Additionally, it is an emergency that requires immediate medical attention [15].

ATHEROSCLEROSIS RISK FACTORS:

The complicated process of atherosclerosis frequently begins in childhood and advances with age. High blood pressure, High cholesterol, and obesity. Diabetes or insulin resistance [16].

HEARTFAILURE RISK FACTORS:

Characteristics and lifestyle choices that raise your risk of heart failure are known as risk factors. Your chance of heart failure is significantly increased if you have one or more of these risk factors: coronary artery disease or heart failure (HF). It can cause chest pain. Elevated triglycerides during fasting Low levels of HDL (good) cholesterol, high blood pressure, and elevated blood sugar while fasting [17].

CORONARY ARTERY DISEASE CAUSES:

The accumulation of lipids, cholesterol, and other materials in and on the heart artery walls is what causes coronary artery disease. We refer to this condition as atherosclerosis. Plaque is the term for the accumulation. Blood flow can be obstructed by artery narrowing brought on by plaque. Insulin resistance or diabetes. Elevated blood pressure. Not exercising. Smoking or using tobacco products [18].

MYOCARDIAL INFARCTION CAUSES:

Infections: Although they don't directly cause myocardial infarction, several bacterial or viral infections can promote inflammation in the coronary arteries, which raises the chance of plaque rupture. Air Pollution, Genetic Factors, Autoimmune Conditions, Excessive Alcohol Use [19].

HYPERCHOLESTEROLEMIA CAUSES:

Among the causes of hypercholesterolemia are:

Your genes (hypercholesterolemia, either familial or pure) Obstructive liver disease. Anorexia nervosa with hypothyroidism. Persistent renal failure. Nephrotic syndrome. Amiodarone [20].

ATHEROSCLEROSIS CAUSES:

Endothelium injury is frequently caused by: High cholesterol, High blood pressure, inflammation, such as that caused by lupus or arthritis Diabetes or obesity, Smoking [21].

HEART FAILURE CAUSES:

Heart attack, inherited heart disease, such as cardiomyopathy, a disorder that weakens the heart muscle Heart inflammation, such as myocarditis; high blood pressure (hypertension); arrhythmia (an irregular heartbeat); Certain behaviours, such as smoking, drinking alcohol, consuming foods high in cholesterol or fat, and not exercising enough [22].

SIGNS AND SYMPTOMS OF CORONARY ARTERY DISEASE:

Insufficient oxygen-rich blood flow to the heart causes symptoms of coronary artery disease. Symptoms of coronary artery disease can include:

Angina is a pain in the chest. You might have chest pain, tightness, heaviness, pressure, or squeezing. chills. exhaustion. heartburn. feeling queasy. breathlessness. acute dizziness or light-headedness [23].

SIGNS AND SYMPTOMS OF MYOCARDIAL INFARCTION:

Although shortness of breath and chest discomfort are the typical signs of a heart attack, there are many more symptoms as well. The following are among the most typical signs of a heart attack: stiffness or pressure in the chestpain in the jaw, chest, back, and other upper body parts that either disappear and then return or last longer than a few minutes. Shortness of breath, upper back pain, nausea, vomiting [24].

SIGNS AND SYMPTOMS OF HYPERCHOLESTEROLEMIA:

Signs and Symptoms of elevated cholesterol can include:

Angina (chest discomfort) Breathlessness Fatigue, Extremity numbness, Tightness or pain in the chest, yellowish fat deposits beneath the skin, or slurred speech (a probable stroke indication), Light-headedness or dizziness, and unexpected and sudden heart attack [25].

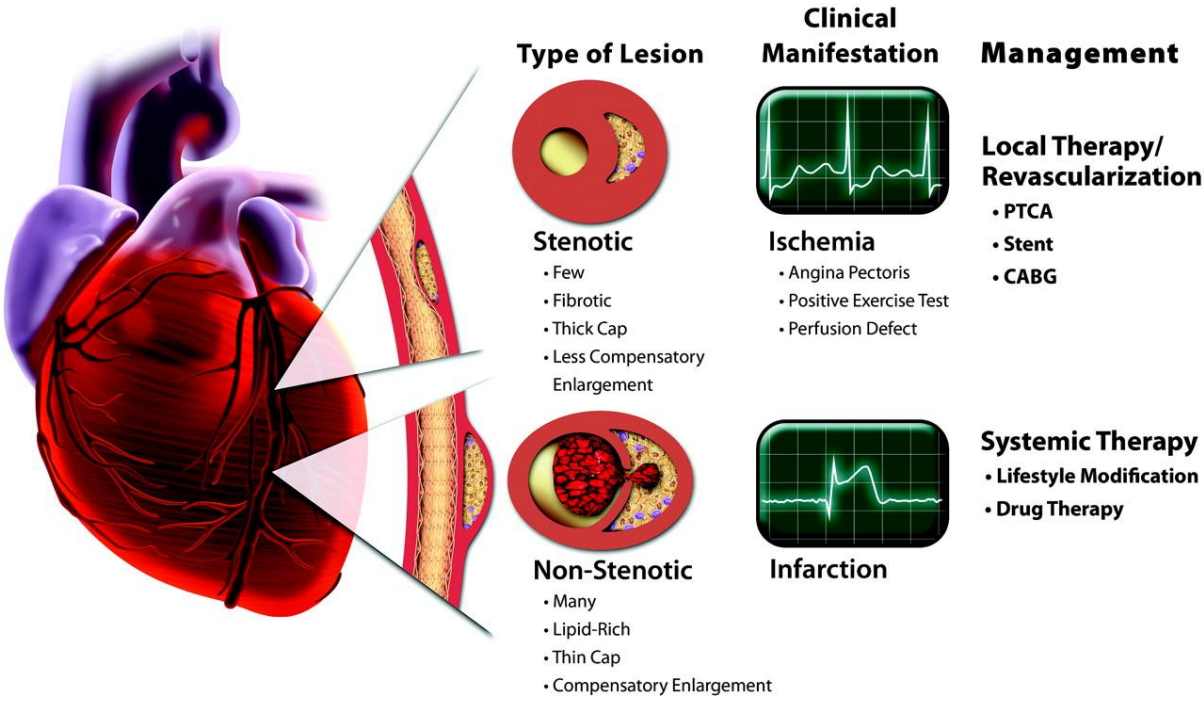
SIGNS AND SYMPTOMS OF ATHEROSCLEROSIS:

Cramping in the buttocks when walking, pain in your arm, leg, and other areas supplied by the blocked artery, and chest pain (stable angina) palpitations in the heart, Breathlessness exhaustion, mental disorientation if the obstruction impairs brain circulation, weakness and hemiparesis (loss of feeling on one side of the body), muscle weakness, and leg cramps as a result of poor circulation [26].

SIGNS AND SYMPTOMS OF HEART FAILURE:

Pedal edema, Increased Nighttime Urination Needs Fast or Unusual Heartbeats, enlargement of the abdomen, breathlessness, Weakness and Fatigue, Absence of Nausea or Appetite, and having trouble focusing [27].

CORONARY ARTERY DISEASE PATHOPHYSIOLOGY:



[28]

MYOCARDIAL INFARCTION PATHOPHYSIOLOGY:

Myocardial Infarction – Pathophysiology Flow Chart Risk Factors, Atherosclerosis, Genetic predisposition

⬇

Endothelial Injury or Dysfunction Caused by: Shear stress, Oxidative stress

⬇

Lipid Accumulation in Intima, LDL cholesterol enters the arterial wall, Oxidation of LDL, Formation of fatty streaks

⬇

Inflammatory Response, Monocytes migrate into the intima and convert into macrophages. Macrophages ingest oxidised LDL, forming foam cells.

⬇

Formation of Atherosclerotic Plaque, Smooth muscle cell migration and proliferation, Lipid-rich necrotic core develops.

⬇

Plaque Instability, Inflammation weakens the fibrous cap, Thinning and rupture of plaque.

⬇

Plaque Rupture or Erosion exposure of: Collagen, Tissue factor, Lipid core

⬇

Platelet Activation and Aggregation, Platelets adhere to exposed collagen

Release: ADP, Thromboxane A_2, Platelet aggregation occurs

⬇

Thrombus Formation

Development of occlusive thrombus

⬇

Coronary Artery Occlusion, Partial occlusion → NSTEMI or unstable angina

Complete occlusion → STEMI

⬇

Reduced Coronary Blood Flow

Oxygen supply to the myocardium decreases.

⬇

Myocardial Ischemia, Lactic acid accumulation, ATP depletion

⬇

Loss of Myocardial Contractility Occurs within 1–2 minutes, Reduced cardiac output

⬇

Irreversible Myocardial Cell Injury, After 20–40 minutes of severe ischemia

Mitochondrial dysfunction

⬇

Myocardial Necrosis (Infarction), Death of cardiac muscle cells, Release of biomarkers: Troponin I/T, CK-MB, Myoglobin

⬇

Inflammatory Healing Response, Neutrophil infiltration (1–3 days), Macrophage cleanup (3–7 days), Granulation tissue formation (1–2 weeks)

⬇

Scar Formation, Fibrous tissue replaces necrotic myocardium, Permanent loss of contractile function [29].

STEMI

Atherosclerotic plaque rupture

⬇

Exposure of the thrombogenic core

⬇

Massive platelet aggregation

⬇

Complete coronary artery occlusion.

⬇

Severe, sustained ischemia

⬇

Transmural myocardial necrosis

⬇

ST-segment elevation on ECG [30].

NSTEMI

Atherosclerotic plaque rupture or erosion

↓

Partial thrombus formation

↓

Incomplete coronary artery occlusion

↓

Reduced blood flow to the myocardium

↓

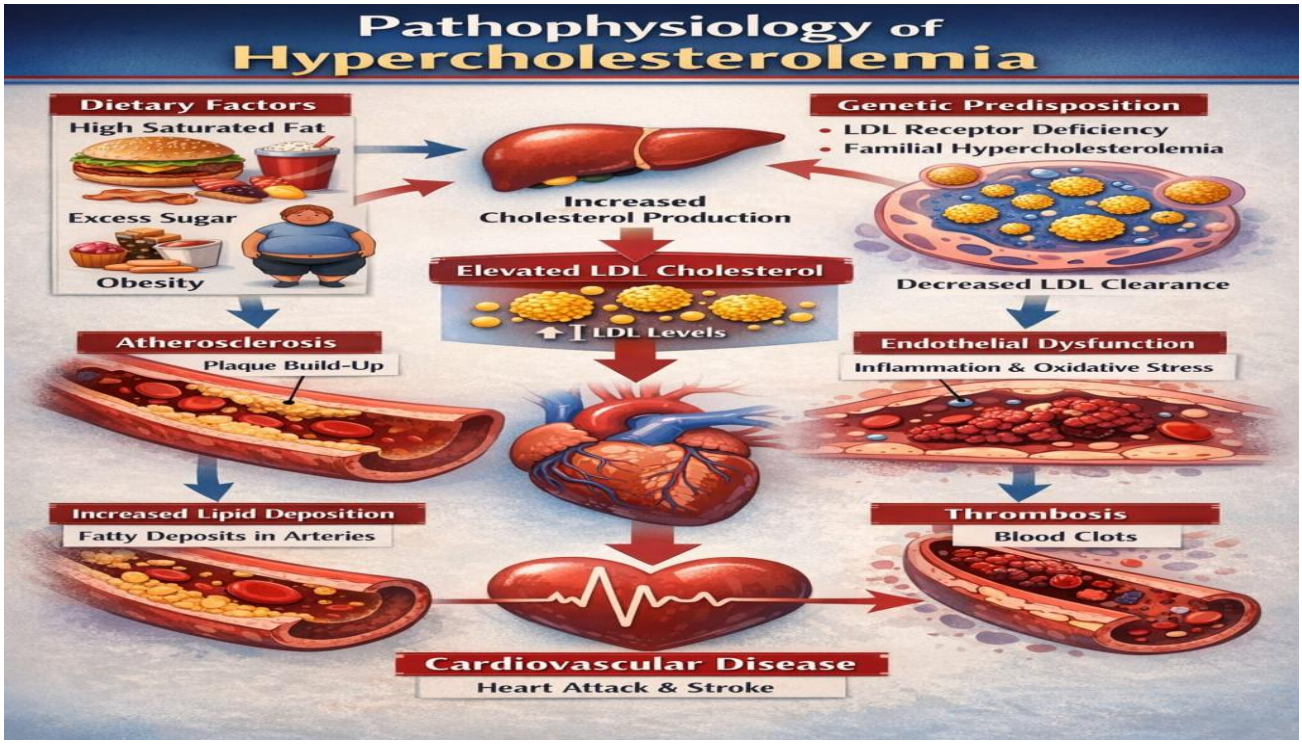
Subendocardial ischemia

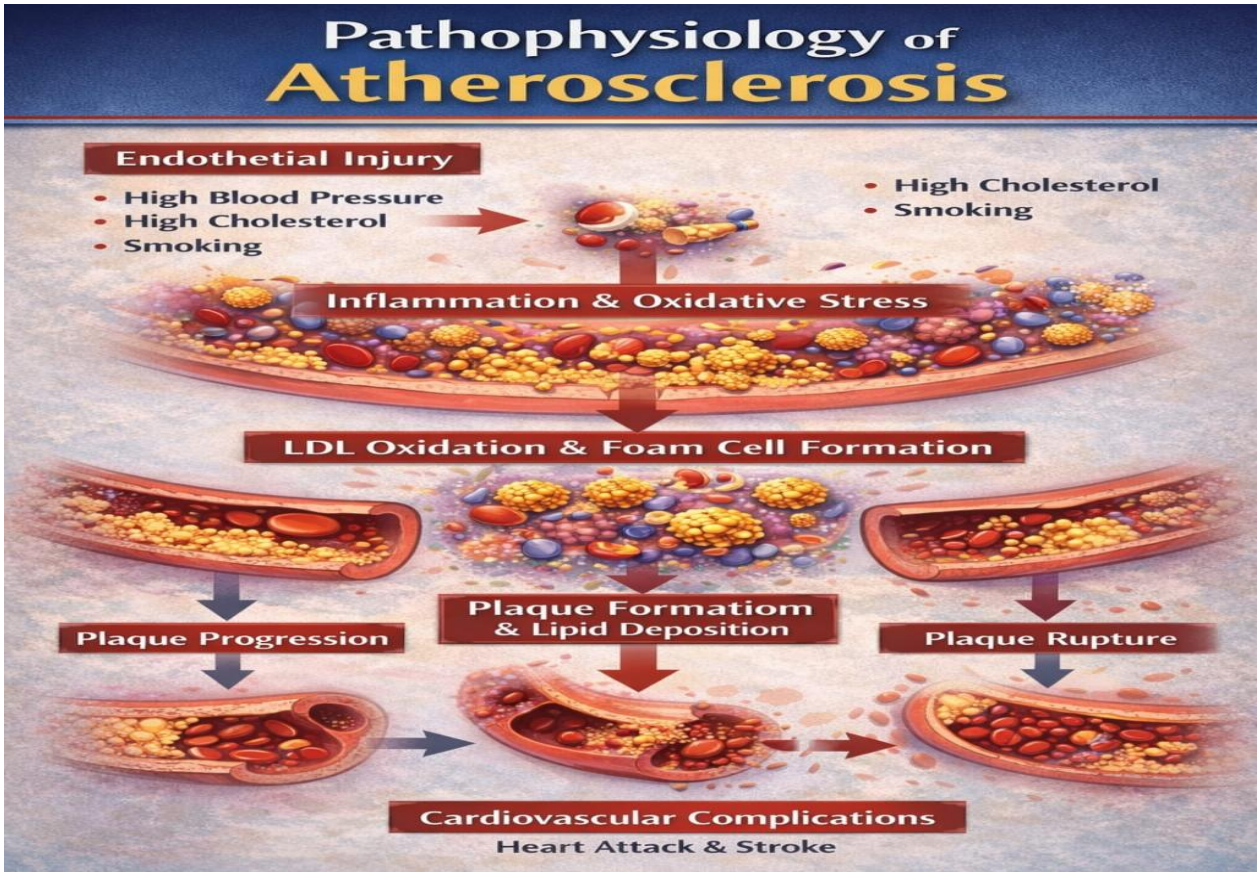
↓

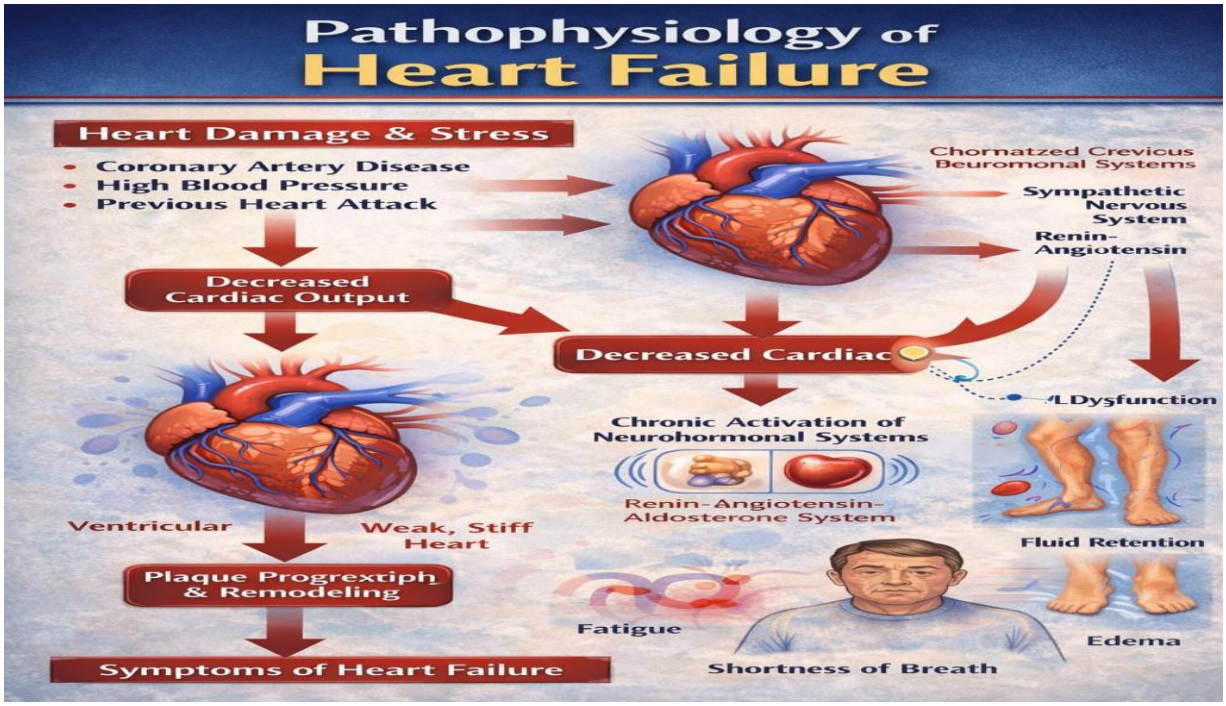
Limited myocardial necrosis

↓

ST-segment depression or T-wave inversion [31].







Coronary Artery Disease Includes:

Blood tests
(a) Blood sugar
(b) Blood cholesterol
(c) C-reactive protein (CRP)
Electrocardiogram (ECG or EKG).
Echocardiogram.
Nuclear stress test.
Heart CT scan. [32].

HEART DIAGNOSIS:

Computed tomography (CT)
Magnetic resonance imaging (MRI)
Digital subtraction angiography (DSA)
Positron emission tomography (PET)
Blood test
1. Blood sugar
2. Complete Blood picture
3. Electrocardiogram
Lumbar puncture (also called a spinal tap) .

HYPERCHOLESTEROLMIA:

Total cholesterol.
Low-density lipoprotein (LDL) cholesterol.
High-density lipoprotein (HDL) cholesterol.
Triglycerides. [34].

ATHEROSCLEROSIS:

Medical history and physical exam
Blood test
Total cholesterol
Low-density lipoprotein (LDL) cholesterol.
High-density lipoprotein (HDL) cholesterol
Triglycerides
C- reactive protein
Electrocardiogram (ECG or EKG)
Echocardiogram (echo)

Angiography
Coronary CT scan (computed tomographic)
Cardiac MRI (magnetic resonance imaging)
Cardiac PET (positron emission tomography) scan
Carotid ultrasound
Abdominal ultrasound [35].
Quit Smoking/Avoid Tobacco
Manage Stress
Limit Alcohol Consumption
Get Quality Sleep [36].
1. Generic names (examples)
Atorvastatin, Simvastatin, Rosuvastatin, Pravastatin, Lovastatin, Fluvastatin,Pitavastatin
2. Pharmacological class
HMG-CoA reductase inhibitors
3. Therapeutic class
Antihyperlipidemic drugs / Lipid-lowering agents
4. Mechanism of action (MOA)
Statins competitively inhibit HMG-CoA reductase, the rate-limiting enzyme in hepatic cholesterol synthesis.
This leads to:
↓ Intracellular cholesterol in hepatocytes
↑ Up-regulation of LDL receptors on liver cells
↑ clearance of LDL-cholesterol from plasma
↓ LDL, ↓ triglycerides, and mild ↑ HDL
5. Pharmacokinetic parameters (PK)
Parameter :
(a)Absorption: Well absorbed orally
Bioavailability Variable (e.g., atorvastatin ~14%)
(b) Distribution: High plasma protein binding (>95%)
(c) Metabolism: Mainly hepatic (CYP3A4: atorvastatin, simvastatin; CYP2C9: fluvastatin)
Half-life Short (1–4 h) except atorvastatin (~14 h), rosuvastatin (~19 h)
(d) Excretion: Mainly biliary/faecal; minimal renal
6. **Pharmacodynamic parameters (PD):**
(a): Primary effect: Reduction of LDL-cholesterol (20–60%)
(b) Secondary effects:
↓ triglycerides
↑ HDL (5–10%)
Plaque stabilization
Anti-inflammatory and endothelial protective effects [37,38].
7. **Therapeutic uses**
Hypercholesterolemia (primary & secondary)
(a)Mixed dyslipidemia
(b) prevention of atherosclerotic cardiovascular disease (ASCVD)
(c) secondary prevention after myocardial infarction or stroke
(d) familial hypercholesterolemia
8. **Adverse effects (side effects)**
(a) Myalgia
(b) Myopathy
(c) Rhabdomyolysis (rare but serious)
(d) Elevated liver enzymes (AST, ALT)
(e) Gastrointestinal upset
(f) New-onset diabetes mellitus (small risk)
9. **Contraindications**
(a) Active liver disease
(b) Unexplained persistent elevation of transaminases
(c) Pregnancy
(d) Lactation
(e) known hypersensitivity to statins
10. Drug–drug interactions

(a). CYP3A4 inhibitors (erythromycin, clarithromycin, azole antifungals) → ↑ statin toxicity
(b) Fibrates (especially gemfibrozil) → ↑ risk of myopathy
(c) Grapefruit juice → ↑ plasma levels (especially simvastatin, atorvastatin)
(d) warfarin → ↑ anticoagulant effect
(e) Cyclosporine → ↑ statin concentration [39,40].

CONCLUSION:

In conclusion, cardiovascular diseases, driven by a complex interplay of genetic predispositions, lifestyle choices, and environmental factors, continue to be a leading cause of global morbidity and mortality. Effective management necessitates early diagnosis, comprehensive risk factor modification, and targeted therapeutic interventions. Statins, as evidenced by their extensive clinical use and well-understood mechanisms, remain pivotal in lipid-lowering strategies and the primary and secondary prevention of atherosclerotic cardiovascular disease. Continued research into the molecular mechanisms underpinning these diseases, coupled with advancements in personalised medicine, holds promise for further improving patient outcomes and alleviating the significant burden of CVDs worldwide.

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