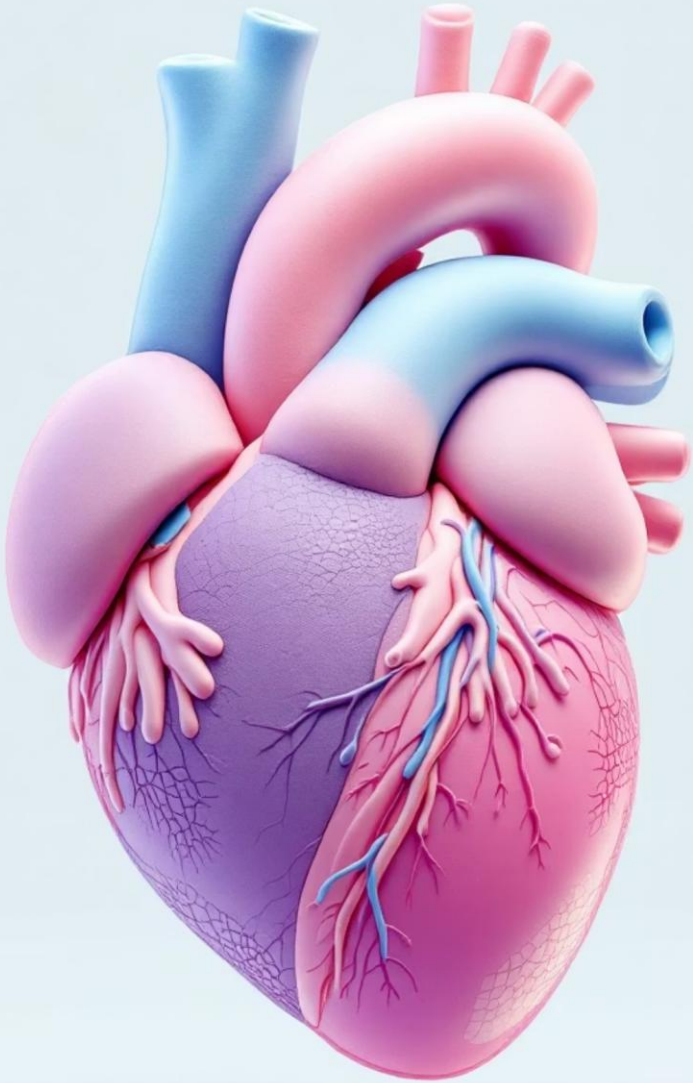
Cardiovascular Diseases

Presented by : Nisha Rahul Acharya
Roll No : 01
Guided By : Dr. Vaishali Dolas



Introduction to CVDs

A broad group of disorders affecting the heart and vascular system.

Leading Global Cause

Morbidity and mortality worldwide.

Complex Pathology

Structural, functional, electrical, vascular abnormalities.

Diverse Categories

CAD, heart failure, arrhythmias, valvular disorders, PVDs.

Global Epidemiology of CVDs

CVDs are a critical public health challenge, accounting for approximately one-third of all deaths globally.

Increasing Prevalence

Driven by aging populations, lifestyle shifts, and improved survival rates from acute cardiac events.

Disproportionate Burden

Significant impact in low- and middle-income countries, exacerbating global health disparities.

Etiology: Multifactorial Origins

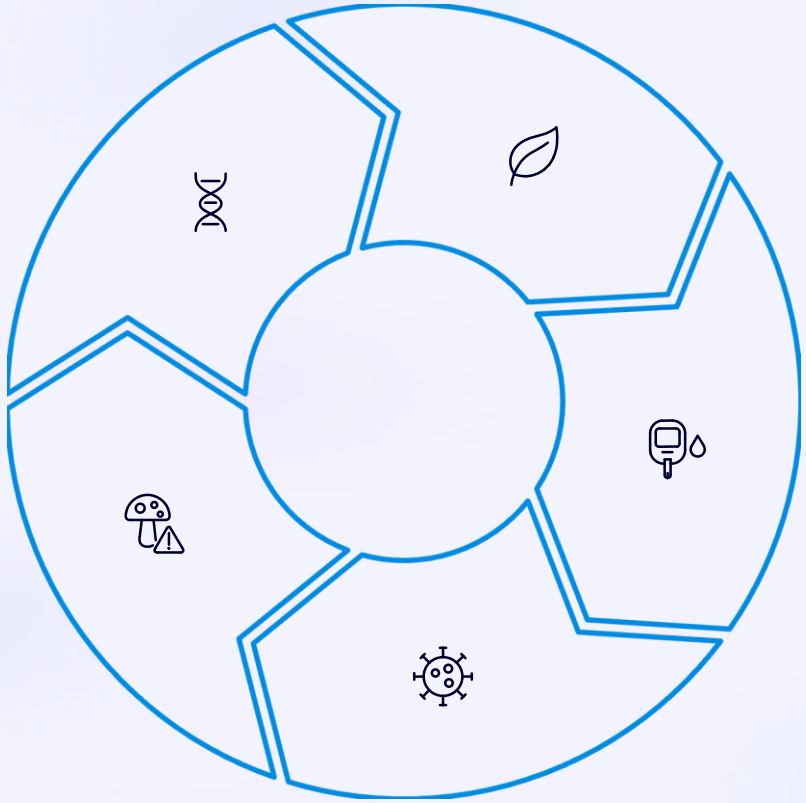
CVDs result from a complex interplay of various factors.

Genetic Factors

Familial hypercholesterolemia, congenital defects.

Other Causes

Toxins (alcohol), autoimmune disorders.



Environmental & Lifestyle

Diet, inactivity, smoking habits.

Metabolic Factors

Dyslipidemia, hypertension, diabetes.

Infectious & Inflammatory

Rheumatic heart disease, myocarditis.

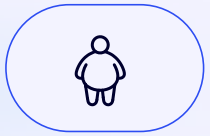
Modifiable Risk Factors

Factors we can change to reduce CVD risk.

Hypertension

Dyslipidemia

Diabetes Mellitus



Obesity & Metabolic Syndrome

Cigarette Smoking

Sedentary Lifestyle

Unhealthy Diet

Excessive Alcohol

Non-Modifiable Risk Factors

Factors beyond our control that influence CVD risk.

Age

Risk increases with advancing years.

Sex

Males > pre-menopausal females;
risk equalizes post-menopause.

Genetic Predisposition

Strong family history indicates higher risk.

Ethnicity

Higher risk in certain populations

Pathogenesis: General Overview

Common mechanisms underlying CVD development.

Endothelial Dysfunction

Initial cellular changes in blood vessel lining.

Inflammatory Activation

Immune response contributing to tissue damage.

Atherosclerotic Plaque Formation

Build-up and progression within arteries.

Thrombosis & Vascular Occlusion

Blood clot formation blocking blood flow.

Structural Heart Remodeling

Changes in heart size and shape.

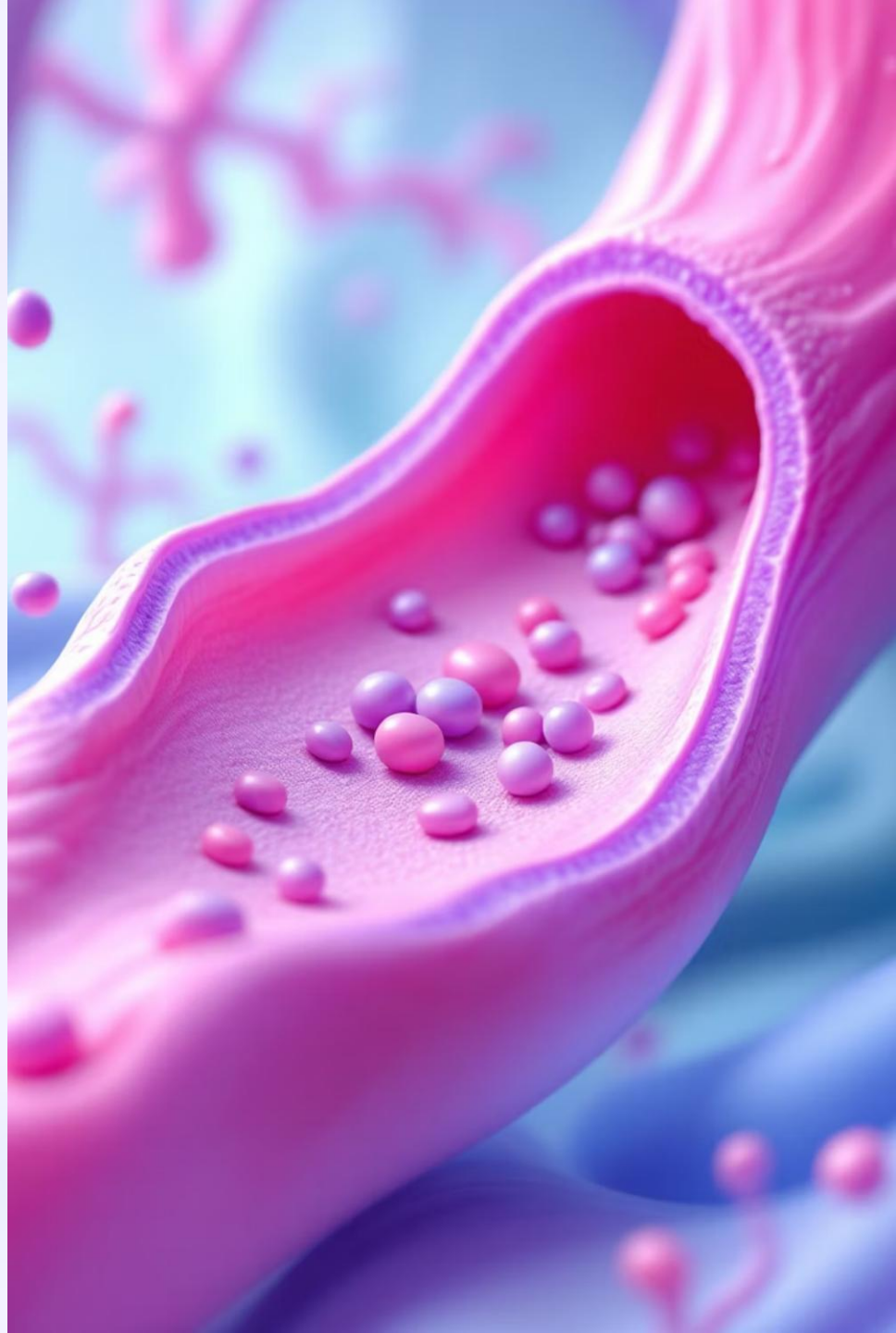
Electrical Conduction Abnormalities

Disruptions in heart's electrical rhythm.

Pathogenesis of Atherosclerosis

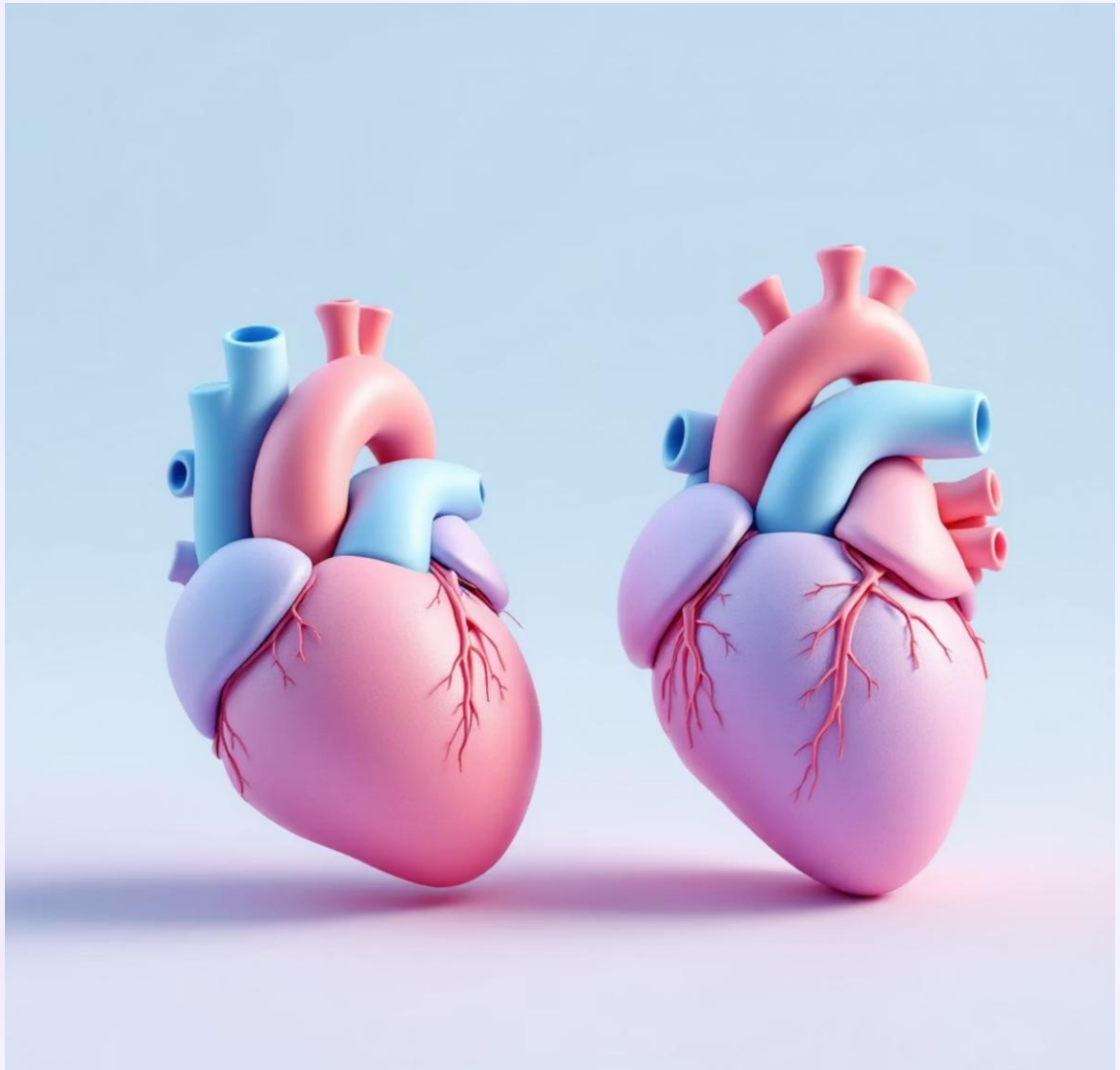
A step-by-step breakdown.

- Endothelial Injury
- LDL Infiltration
- LDL Oxidation
- Monocytes → Foam Cells



Pathogenesis of Heart Failure

Brief overview of the mechanisms leading to impaired cardiac function.



Impaired Cardiac Function

Systolic or diastolic dysfunction leading to inefficient pumping.

Neurohormonal Activation

SNS, RAAS, ADH activation results in vasoconstriction and sodium retention, further stressing the heart.

Cardiac Remodeling

Hypertrophy and fibrosis alter heart structure, reducing its ability to function optimally.

Progressive Decline

Ultimately leads to a continuous decrease in cardiac output.

Classification & Types of CVDs

Diverse categories of cardiovascular diseases.

Coronary Artery Diseases

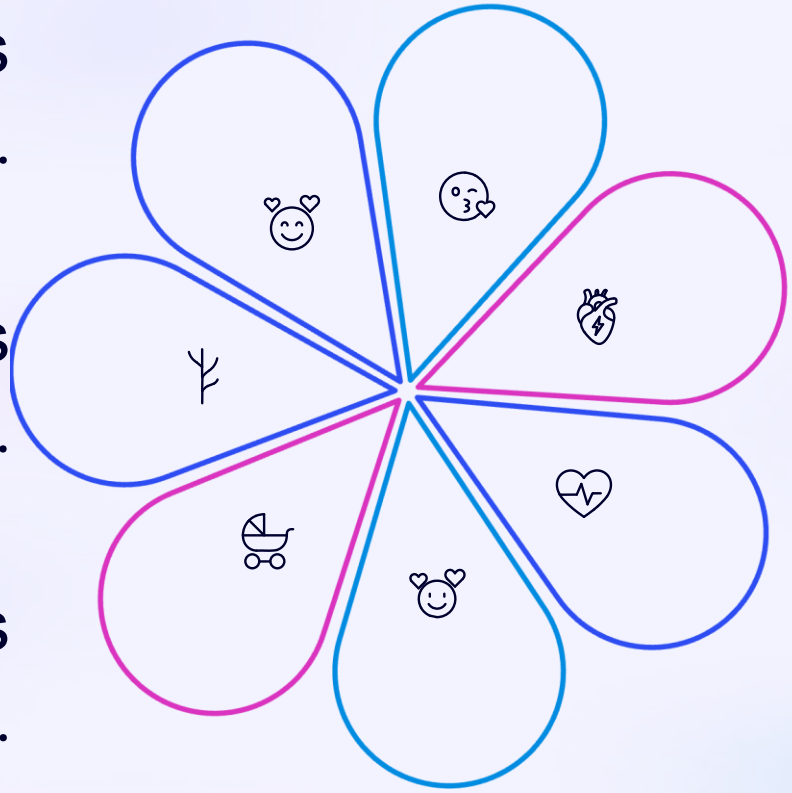
Angina, ACS(UA, NSTEMI, STEMI).

Vascular Diseases

PAD, aortic aneurysm, DVT/PE.

Congenital Heart Diseases

ASD, VSD, Tetralogy of Fallot.



Heart Failure

HFrEF, HFpEF.

Cardiomyopathies

Dilated, hypertrophic, restrictive.

Arrhythmias

AFib, SVT, VT/VF, conduction disorders.

Valvular Heart Diseases

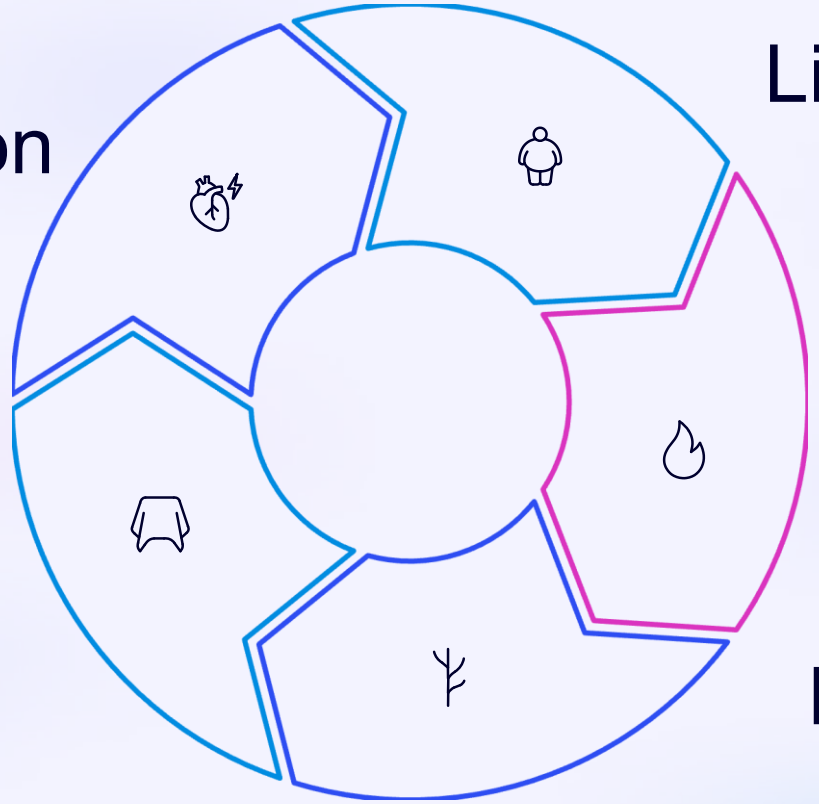
Mitral/aortic stenosis/ regurgitation.

Pathogenesis

CAD develops due to atherosclerotic obstruction of coronary arteries.

Endothelial Dysfunction

Lipid Accumulation



Inflammation

Plaque Rupture & Thrombosis

Plaque Formation

This cascade leads to ischemia, unstable angina, or myocardial infarction.

Step 1: Endothelial Injury

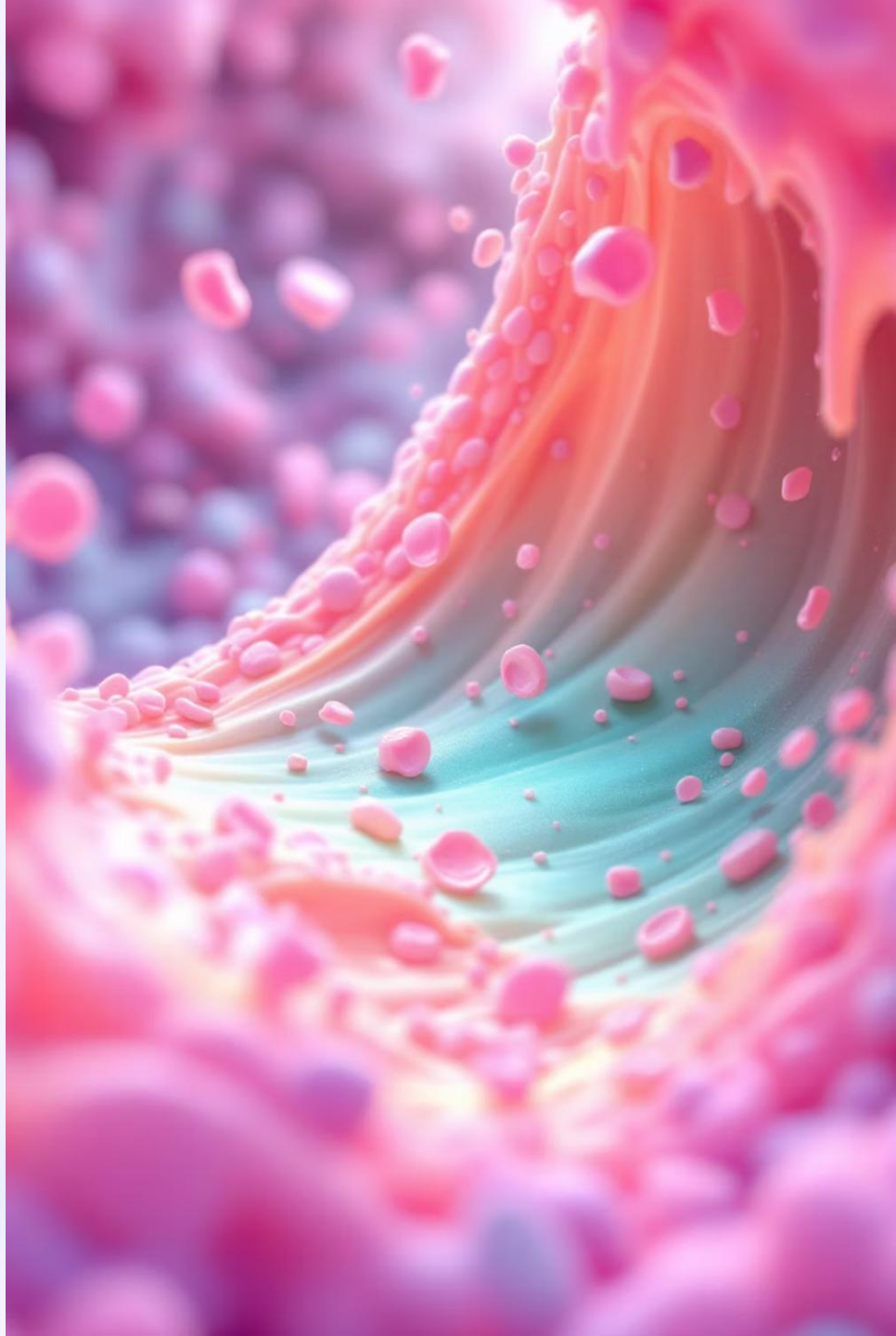
Triggers:

- Hypertension
- Smoking
- Hyperglycemia
- Dyslipidemia

Endothelial Changes:

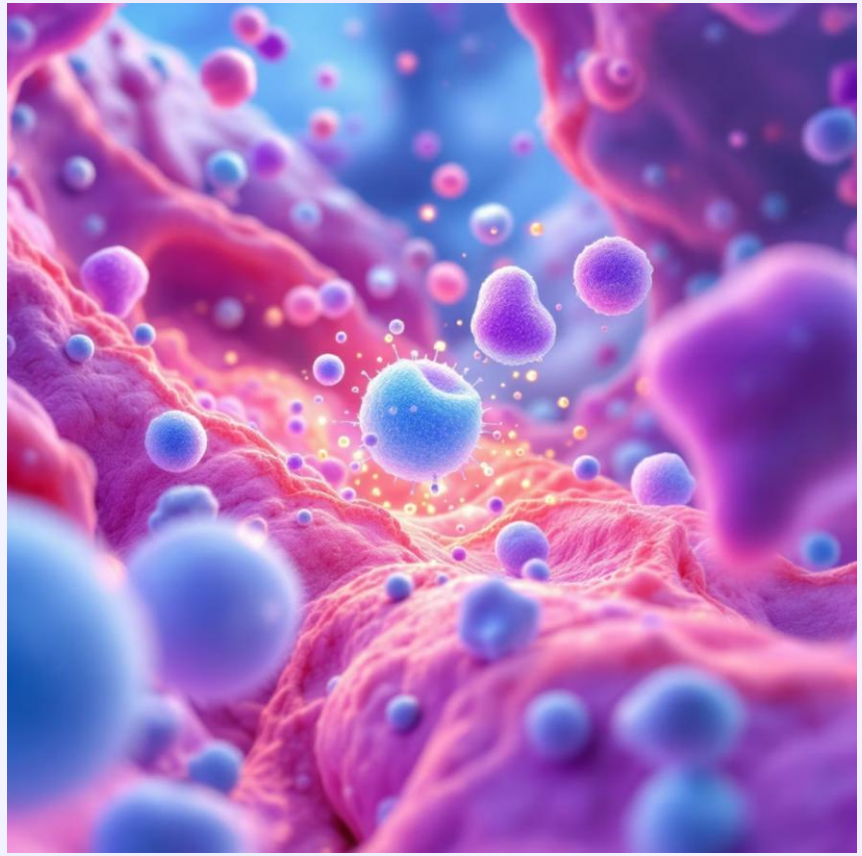
- Reduced Nitric Oxide production
- Loss of Antithrombotic properties
- Impaired Vasodilatory capacity

Result: Increased permeability to lipoproteins and inflammatory cells.



Step 2: Lipid Entry & Oxidation

Low-density lipoprotein (LDL) particles infiltrate the subendothelial space.



Within this space, LDL undergoes **oxidation (ox-LDL)**, transforming into a highly inflammatory molecule.

Ox-LDL attracts monocytes and stimulates the release of cytokines, key signals in the inflammatory response.

This process is a key event in initiating **atheroma formation**.

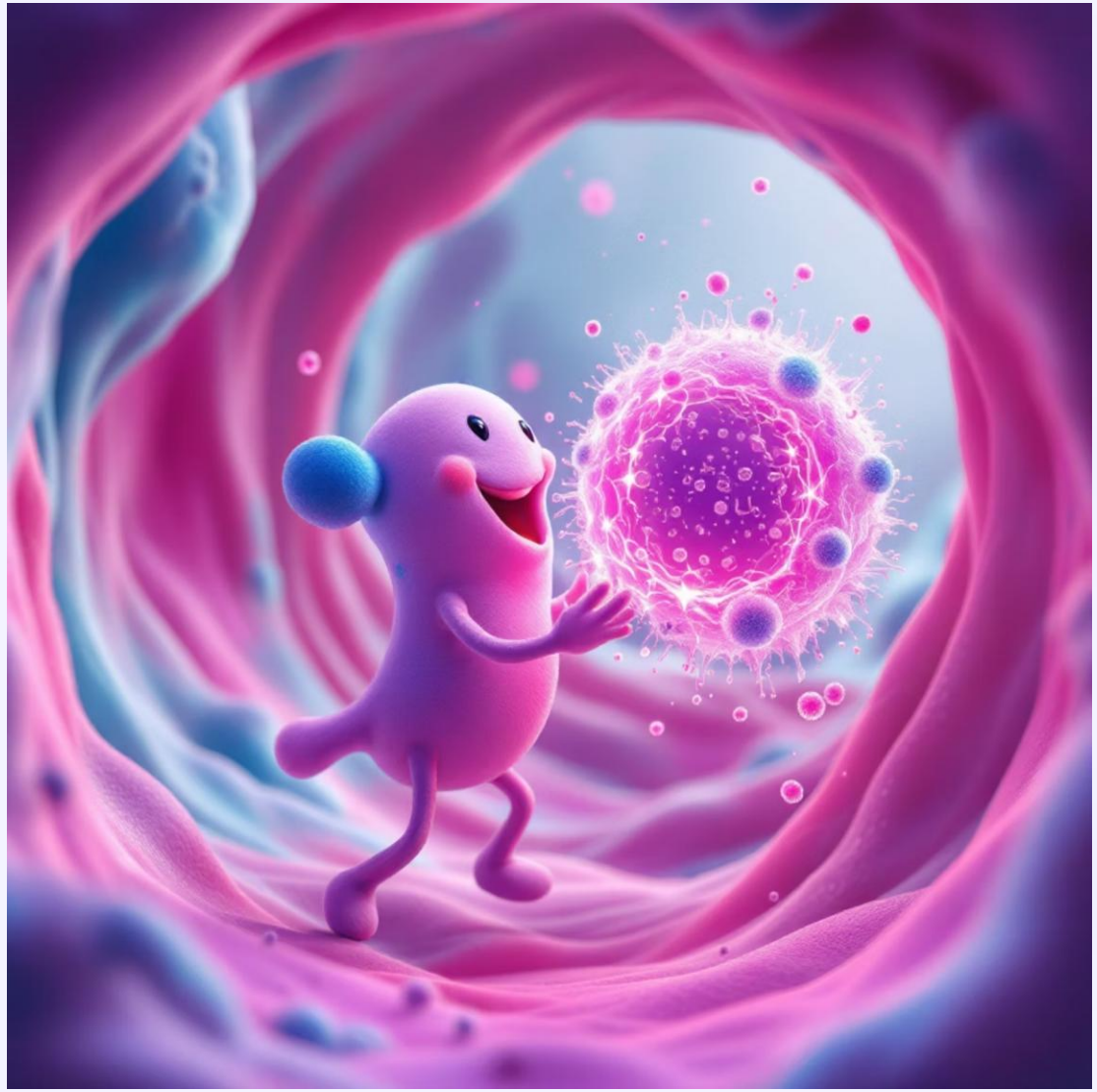
Step 3: Inflammation & Foam Cell Format

Monocytes, attracted by ox-LDL, migrate into the subendothelial space and differentiate into macrophages.

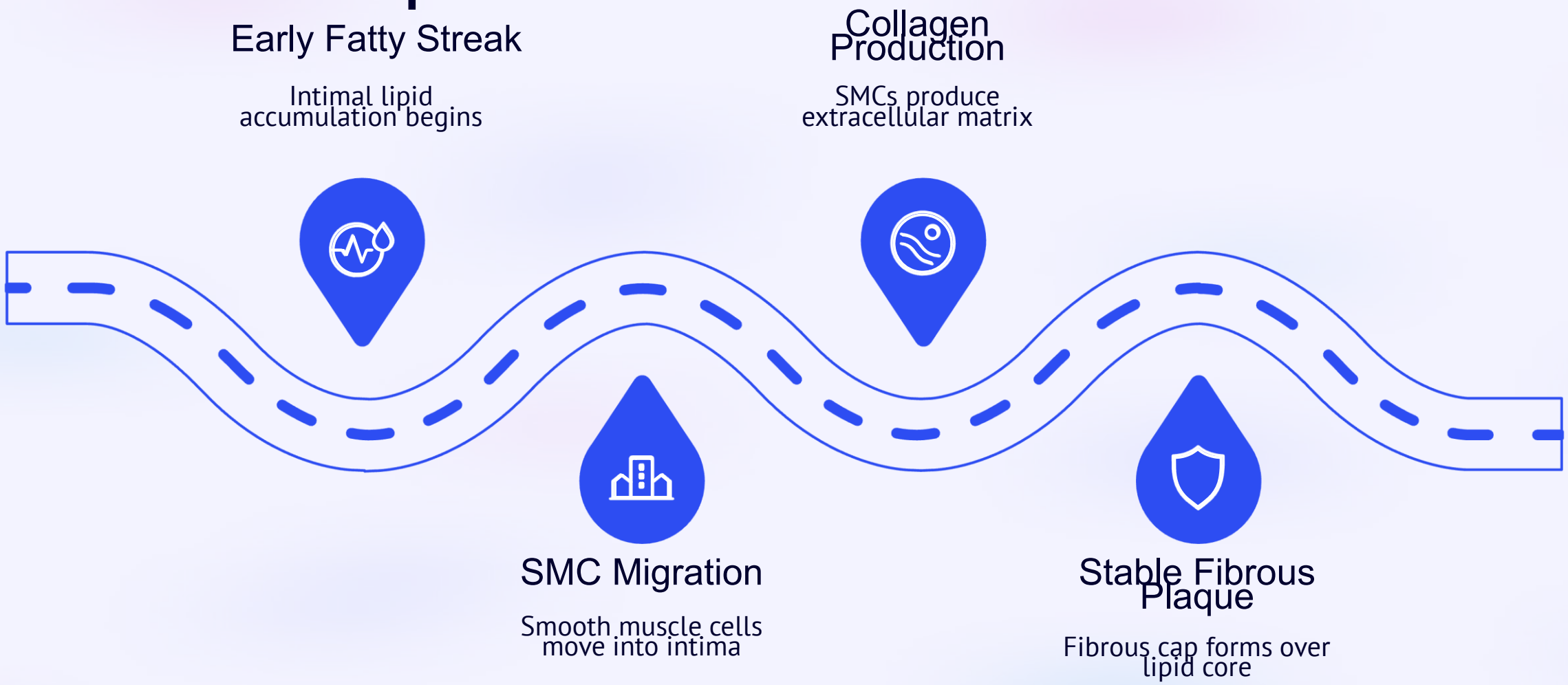
These macrophages avidly ingest oxidized LDL, becoming engorged **foam cells**.

The accumulation of these foam cells forms the **fatty streak**, the earliest visible lesion of atherosclerosis.

T-lymphocytes further contribute to **chronic vascular inflammation** within the lesion.



Step 4: Smooth Muscle Migration & Fibrous Cap Formation



Smooth muscle cells migrate from the media to the intima, where they proliferate and produce extracellular matrix components, primarily collagen.

Pathogenesis of Plaque Rupture

Vulnerable Plaques:

- Thin fibrous cap
- Large lipid core
- High macrophage density

Mechanical stress leads to **cap rupture or erosion**.

Exposed lipid core triggers **platelet adhesion and clot formation**.

Clinical Outcomes:

- Unstable Angina
- NSTEMI (partial occlusion)
- STEMI (complete occlusion)

Thrombosis & Myocardial Ischemia

Activated platelets aggregate, leading to fibrin deposition and the formation of a **coronary thrombus**.

The degree of obstruction by this thrombus dictates the clinical presentation.

Severe or complete occlusion results in **transmural ischemia**, characteristic of STEMI.

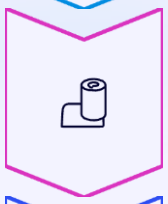
Persistent ischemia can lead to myocardial **necrosis**, arrhythmias, and heart failure.

Management Overview

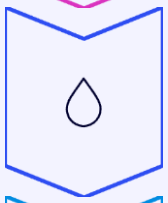
Aims to restore flow, reduce demand, stabilize plaque, prevent thrombosis.

Restore Coronary Blood Flow

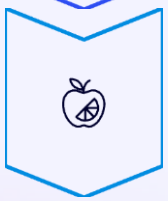
Reduce Myocardial Oxygen Demand



Stabilize Plaque



Prevent Thrombosis



Modify Risk Factors

Management strategies are divided into Acute Management (for ACS) and Chronic/Long-term Management.

Acute Management: Unstable Angina & NSTEMI

1. Antiplatelet Therapy

- Aspirin (loading dose)
- P2Y12 inhibitors (Clopidogrel/Ticagrelor)

2. Anticoagulation

- Unfractionated heparin or LMWH

3. Anti-ischemic Therapy

- Nitrates
- Beta-blockers
- High-intensity statin

4. Risk Stratification

- ECG + Troponins
- Early invasive strategy (angiography) for high-risk patients