MEDICAL EDUCATION RESOURCE

Cardiovascular Diseases and Hyperlipidemias

? 1. LIPID PROFILE

? Components of a Standard Lipid Profile

- **Total Cholesterol (TC)**
- **Triglycerides (TG)**
- **HDL-cholesterol (HDL-C)**
- **LDL-cholesterol (LDL-C)**
- **VLDL-cholesterol (VLDL-C)**
- **Non-HDL cholesterol** = TC ? HDL
- **Atherogenic Index** = TG / HDL
- **LDL/HDL ratio**

? Normal Reference Ranges

- **Total Cholesterol:** < 200 mg/dL
- **Triglycerides:** < 150 mg/dL
- **LDL:** < 100 mg/dL (optimal)

- **HDL:** > 40 mg/dL (men) ; > 50 mg/dL (women)
- **VLDL:** ~ TG/5 (mg/dL)
- **Non-HDL cholesterol:** < 130 mg/dL

? Interpretation of Lipid Profiles

High LDL-C

- Strongly associated with **atherosclerosis**
- Major target of therapy (statins)

High Triglycerides

- Seen in diabetes, metabolic syndrome, alcoholism, pancreatitis risk
- Treated with fibrates, omega-3

Low HDL-C

- Strong risk factor for coronary artery disease (CAD)

Elevated Non-HDL-C

- Best indicator in diabetics and metabolic syndrome

Atherogenic Dyslipidemia (Typical in Type 2 DM)

- High TG

- Low HDL
- Small dense LDL

? 2. ATHEROSCLEROSIS

? Definition

A chronic inflammatory disease of arteries characterized by formation of **atheromatous plaques** in the intima.

? Pathogenesis (Stepwise)

1. **Endothelial injury**
   – Hypertension, smoking, diabetes, dyslipidemia
2. **LDL infiltration into intima**
   – Oxidation of LDL by ROS
3. **Macrophage uptake ? foam cells**
   – Oxidized LDL taken up by scavenger receptors
4. **Fatty streak formation**
   – Earliest visible lesion
5. **Smooth muscle proliferation & collagen deposition**
   – Formation of fibrous cap
6. **Plaque progression**
   – Narrowing of lumen
7. **Plaque rupture ? thrombosis**
   – Causes MI, stroke

? Why oxidized LDL is dangerous?

- Highly atherogenic
- Attracts monocytes
- Promotes inflammation
- Leads to foam cell formation

? Risk Factors for Atherosclerosis

Modifiable

- Dyslipidemia (high LDL, low HDL)
- Hypertension
- Smoking
- Diabetes mellitus
- Obesity
- Sedentary lifestyle
- Diet high in saturated fats
- Stress, alcohol

Non-modifiable

- Age
- Male sex
- Family history
- Genetic disorders (familial hypercholesterolemia)

? Protective Factors

- High HDL
- Exercise
- Omega-3 fatty acids
- Moderate alcohol
- Antioxidants

? Complications

- Myocardial infarction
- Stroke
- Peripheral vascular disease
- Aneurysm
- Sudden cardiac death

? 3. CORONARY ARTERY DISEASE (CAD)

? Definition

Impairment of blood flow through the coronary arteries ? myocardial ischemia.

? Major Causes

- **Atherosclerosis** (most common)
- Vasospasm
- Thrombosis
- Embolism

? Clinical Presentation

- Chest pain (angina pectoris)
- Radiation to left arm, jaw
- Dyspnea
- Sweating, nausea
- Silent ischemia (common in diabetics)

? Types of Angina

? 1. Stable Angina

- Predictable pain on exertion
- Due to fixed atherosclerotic plaque

? 2. Unstable Angina

- Pain at rest
- Part of acute coronary syndrome
- Plaque rupture + thrombosis (non-occlusive)

? 3. Prinzmetal (Variant) Angina

- Due to coronary vasospasm
- Episodes at rest
- ST elevation during attack

? Acute Coronary Syndrome (ACS)

Includes:

- Unstable angina
- NSTEMI
- STEMI

All involve **plaque rupture + thrombus**.

? Markers for Myocardial Infarction

- **Troponin I / T** – most specific, rises early
- **CK-MB** – useful for reinfarction
- **LDH-1** – late marker

? Pathophysiology of MI

1. Rupture of vulnerable plaque
2. Platelet activation + thrombus formation
3. Occlusion of coronary artery
4. Myocardial ischemia ? necrosis

? ECG Findings

- ST elevation
- T-wave inversion
- Q waves (late)

? Risk Factors for CAD

Same as atherosclerosis, plus:

- Elevated lipoprotein(a)
- High hs-CRP

- Metabolic syndrome

? Lipoproteins and CAD

- **LDL ? atherogenic**
- **HDL ? protective**
- **Lp(a) ? strong genetic risk factor**

? RELATION OF CHOLESTEROL WITH MYOCARDIAL INFARCTION

? 1. LDL Cholesterol ? the central villain

- **LDL ("bad cholesterol")** is the strongest lipid predictor of MI.
- LDL enters the arterial wall ? becomes **oxidized LDL** ? triggers inflammation.
- Oxidized LDL is taken up by macrophages ? **foam cells** ? **fatty streak** ? **atherosclerotic plaque**.

Higher LDL = Faster plaque formation.

? 2. Small Dense LDL = Highly Atherogenic

- These particles penetrate arterial wall more easily.
- Seen in:
    - Diabetes

- Metabolic syndrome
- Obesity

- Strong association with early CAD and MI.

? 3. HDL Cholesterol ? the protector

- **HDL removes cholesterol from plaques** (reverse cholesterol transport).
- Anti-inflammatory and antioxidant.
- **Low HDL ? high risk of MI** even if LDL is normal.

? 4. Triglycerides and VLDL

- High TG ? ? VLDL ? formation of small dense LDL.
- Increases risk of **pancreatitis** and contributes to CAD.
- Very common in diabetes.

? 5. Lipoprotein(a) — a powerful genetic risk factor

- Lp(a) is similar to LDL but more thrombogenic.
- Promotes:
  - Plaque growth
  - Thrombosis

- Early MI (especially in young individuals)

? 6. Total Cholesterol : HDL Ratio

- A strong predictor of MI.
- Ratio **> 4** significantly increases risk.
- Best used in population screenings.

? 7. Non-HDL Cholesterol

- Non-HDL = Total Cholesterol ? HDL
- Includes all atherogenic particles:
  - LDL
  - VLDL
  - IDL
  - Lp(a)

Best predictor of MI in diabetics and metabolic syndrome.

? 8. Mechanism: How high cholesterol leads to MI

1. LDL enters intima
2. Oxidation ? macrophage uptake

3. Foam cell formation ? fatty streak
4. Smooth muscle proliferation ? fibrous cap
5. Plaque grows ? lumen narrows
6. Plaque rupture
7. **Thrombus forms ? occlusion ? MI**

? 9. Statins reduce MI risk

- Reduce LDL by upregulating **LDL receptors**
- Stabilize plaques
- Reduce inflammation
- Proven to lower mortality after MI

? RISK FACTORS OF ATHEROSCLEROSIS

Atherosclerosis has **modifiable**, **non-modifiable**, and **emerging** risk factors.

? 1. Non-Modifiable Risk Factors

These cannot be changed.

? Age

- Risk increases with age (men >45, women >55).

? Sex

- Males have higher risk
- Women lose protection after menopause

? Genetics / Family History

- Early CAD in first-degree relatives increases risk

? Genetic disorders

- Familial hypercholesterolemia
- Familial combined hyperlipidemia
- Lp(a) excess

? 2. Major Modifiable Risk Factors

These are responsible for majority of cases.

? 1. Hyperlipidemia

- High LDL ? strongest modifiable factor
- Low HDL ? increases risk

? 2. Hypertension

- Damages endothelium

- Accelerates plaque formation

? 3. Smoking

- Free radicals ? endothelial injury
- Increases platelet adhesion
- Lowers HDL
- **Very strong risk factor**

? 4. Diabetes Mellitus

- Glycation of LDL makes it more atherogenic
- Endothelial dysfunction
- Produces **small dense LDL**

? 5. Obesity (especially central obesity)

- Induces insulin resistance
- Raises TG
- Lowers HDL
- Strong link with metabolic syndrome

? 6. Physical Inactivity

- Reduces HDL
- Increases TG and LDL

? 7. Diet high in saturated fats and trans-fats

- Raises LDL sharply
- Promotes plaque formation

? 3. Emerging (Novel) Risk Factors

Increasingly recognized in modern cardiology.

? Inflammatory markers

- **hs-CRP**
- IL-6
- TNF-?

? Lipoprotein(a)

- Strong genetic risk factor
- Increases thrombosis tendency

? Homocysteine

- Causes endothelial injury

- Vitamin B6/B12/folate deficiency

? Oxidative stress

- Increases oxidation of LDL

? Chronic inflammatory diseases

- Rheumatoid arthritis
- Psoriasis
- SLE

? 4. Protective Factors

- High HDL
- Regular exercise
- Omega-3 fatty acids
- Antioxidant-rich diet
- Mediterranean diet
- Moderate alcohol intake (not for everyone)

? ULTRA-HIGH YIELD EXAM PEARLS

- **High LDL = strongest modifiable risk factor for MI.**
- **Low HDL increases CAD risk even if LDL is normal.**
- **Lp(a) is a major genetic predictor of early MI.**
- Smoking + diabetes + high LDL ? explosive risk combination.
- Diabetics have **small dense LDL**, the most atherogenic form.
- Atherosclerosis begins with **endothelial injury**.
- Fatty streak is the **earliest lesion** of atherosclerosis.

? PREVENTION OF ATHEROSCLEROSIS

Atherosclerosis prevention = **reduce risk factors + stabilize plaques + improve vascular function**.

? 1. Lifestyle Modifications (Most Important)

? Dietary changes

- Reduce **saturated fats** and **trans-fats**
- Increase **omega-3 fatty acids**
- Increase **soluble fiber** (oats, legumes)
- Increase **fruits & vegetables**

- Use **unsaturated fats** (olive, mustard oil)
- Reduce red meat, processed foods, high-sugar meals

? Weight control

- Target **BMI < 25**
- Reduce central obesity (waist circumference: men < 90 cm, women < 80 cm)

? Exercise

- 150 min/week moderate aerobic exercise
- Increases HDL
- Reduces TG and BP

? Smoking cessation

- Eliminates a major inflammatory trigger
- Improves HDL
- Reduces MI risk dramatically

? Alcohol (if used)

- Small amounts may increase HDL
- Avoid binge drinking

? 2. Control of Medical Conditions

? Diabetes control

- Maintain HbA1c < 7%
- Prevents small dense LDL formation

? Hypertension control

- BP target <130/80
- ACE inhibitors / ARBs beneficial

? Dyslipidemia treatment

- Aim LDL < 100 mg/dL
- In diabetics or CAD: LDL < 70 mg/dL

? Homocysteine control

- Vitamin B6, B12, folate

? 3. Pharmacological Prevention

? Statins (first-line)

- Lower LDL
- Stabilize plaques

- Anti-inflammatory

? Fibrates

- Lower triglycerides
- Increase HDL

? Niacin

- Lowers Lp(a)
- Increases HDL

? PCSK9 inhibitors (powerful LDL lowering)

? Aspirin

- Used in secondary prevention
- Prevents thrombosis on ruptured plaques

? 4. Screening & Early Detection

- Lipid profile every 5 years
- More frequent in diabetics, obese individuals, smokers
- hs-CRP may help in borderline cases

? HYPO-LIPOPROTEINEMIAS

These are **rare genetic disorders resulting in abnormally low lipoprotein levels**.

? 1. Abetalipoproteinemia

Pathology:

- Absence of **Apo B** containing lipoproteins (chylomicrons, VLDL, LDL)

Features:

- Fat malabsorption
- Steatorrhea
- Failure to thrive
- Acanthocytosis
- Peripheral neuropathy
- Retinitis pigmentosa

Labs:

- Very low cholesterol
- Almost no LDL/HDL/VLDL

? 2. Hypoalphalipoproteinemia (Low HDL)

Causes:

- Tangier disease
- Apo A-I mutations
- LCAT deficiency
- Secondary (diabetes, smoking, obesity)

Features:

- Very low HDL
- Cholesterol deposits in tonsils (Tangier)

? 3. Familial Lecithin–Cholesterol Acyltransferase (LCAT) Deficiency

Features:

- Low HDL
- Corneal clouding
- Hemolysis
- Proteinuria / CKD

? HYPER-LIPOPROTEINEMIAS (FREDRICKSON CLASSIFICATION — TYPE I TO V)

This classification is extremely high-yield.

? TYPE I — Hyperchylomicronemia

Defect:

- **Lipoprotein lipase (LPL) deficiency**
  OR
- **Apo C-II deficiency**

Lipid elevated:

- **Chylomicrons**

Features:

- Pancreatitis
- Eruptive xanthomas
- Lipemia retinalis
- No ? risk of atherosclerosis

? TYPE IIa — Familial Hypercholesterolemia

Defect:

- **LDL receptor defect**
  OR
- Apo B-100 defect

Lipid elevated:

- **LDL**

Features:

- Tendon xanthomas
- Premature CAD (young MI)
- Very high LDL

? TYPE IIb — Combined Hyperlipidemia

Defect:

- Overproduction of VLDL + LDL

Lipid elevated:

- **VLDL + LDL**

Features:

- Very common
- High CAD risk
- Seen in metabolic syndrome

? TYPE III — Dysbeta-lipoproteinemia

Defect:

- Apo E2/E2 homozygosity (Apo E defect)

Lipid elevated:

- **IDL + remnants**

Features:

- Palmar xanthomas (orange creases)
- Tuberoeruptive xanthomas
- High risk of atherosclerosis

? TYPE IV — Hypertriglyceridemia

Defect:

- Overproduction of VLDL

Lipid elevated:

- **VLDL**

Features:

- High triglycerides

- Pancreatitis risk
- Common in diabetes

? TYPE V — Mixed Hyperlipidemia

Defect:

- Increased VLDL + decreased LPL activity

Lipid elevated:

- **VLDL + Chylomicrons**

Features:

- Pancreatitis
- Eruptive xanthomas
- Lipemia
- Alcohol worsens it

? ULTRA-HIGH YIELD COMPARISON TABLE

TYPE	ELEVATED LIPOPROTEIN	MAIN DEFECT	KEY FEATURE
I	Chylomicrons	LPL / Apo C-II	Pancreatitis

TYPE	ELEVATED LIPOPROTEIN	MAIN DEFECT	KEY FEATURE
IIa	LDL	LDL receptor	Tendon xanthomas
IIb	LDL + VLDL	Overproduction	Common CAD
III	IDL / remnants	Apo E defect	Palmar xanthomas
IV	VLDL	Overproduction	High TG
V	VLDL + Chylomicrons	LPL impaired	Pancreatitis

? IMPORTANT POINTS TO REMEMBER — Cardiovascular Diseases & Hyperlipidemias

? LIPID PROFILE (High-Yield Takeaways)

- **LDL is the most atherogenic lipoprotein** and the primary target for therapy.
- **HDL is protective** because it performs reverse cholesterol transport.
- **Triglycerides correlate with pancreatitis risk**, not directly with MI risk.
- **Non-HDL cholesterol** (TC – HDL) is the best measure in diabetics and metabolic syndrome.
- **Small dense LDL particles** (seen in diabetes) are the most dangerous for plaque formation.
- **Lipoprotein(a)** is a strong genetic risk factor for premature MI.

? ATHEROSCLEROSIS (Pathogenesis Essentials)

- **Endothelial injury is the initiating event** (smoking, hypertension, diabetes).
- **Oxidized LDL** is central to plaque development ? taken up by macrophages ? foam cells.
- **Fatty streak** is the earliest visible lesion of atherosclerosis.
- **Plaque rupture**, not gradual narrowing, is the most common cause of MI.
- Atherosclerosis is an **inflammatory disease**, not just lipid accumulation.
- Diabetes produces **small dense LDL**, greatly accelerating atherosclerosis.

? CORONARY ARTERY DISEASE (Key Concepts)

- MI occurs due to **rupture of a vulnerable plaque**, not necessarily the largest plaque.
- **Troponins** are the most sensitive and specific biomarkers for MI.
- **CK-MB** helps detect reinfarction.
- **Stable angina** occurs with exertion; **unstable angina** occurs even at rest.
- **Prinzmetal angina** is due to coronary vasospasm (transient ST elevation).
- **Silent ischemia** is common in long-standing diabetics.

? CHOLESTEROL & MYOCARDIAL INFARCTION

- High LDL = strong predictor of MI.

- High HDL = protective (removes cholesterol from plaques).
- Cholesterol-rich diets increase LDL and accelerate plaque growth.
- **Total cholesterol/HDL ratio > 4** increases MI risk sharply.
- **Lp(a)** promotes thrombosis and early MI (familial risk).

? PREVENTION OF ATHEROSCLEROSIS

- **Statins** reduce MI risk, stabilize plaques, and lower inflammation.
- **Smoking cessation** dramatically reduces risk within 2–3 years.
- **Exercise raises HDL**, lowers TG, improves endothelial function.
- **Mediterranean diet** lowers cardiovascular risk.
- Hypertension, diabetes, and dyslipidemia must be tightly controlled.

? PRIMARY HYPERLIPOPROTEINEMIAS (Type I–V)

(One-line memory)

- **Type I** ? Chylomicrons ? ? pancreatitis, no atherosclerosis
- **Type IIa** ? LDL ? ? tendon xanthomas, early MI
- **Type IIb** ? LDL + VLDL ? ? common, high CAD risk
- **Type III** ? IDL ? ? palmar xanthomas

- **Type IV** ? VLDL ? ? hypertriglyceridemia
- **Type V** ? VLDL + chylomicrons ? ? pancreatitis

? HYPO-LIPOPROTEINEMIAS

- **Abetalipoproteinemia** ? no Apo B ? absent chylomicrons/VLDL/LDL ? fat malabsorption, acanthocytosis.
- **Tangier disease** ? HDL extremely low ? orange tonsils.
- **LCAT deficiency** ? low HDL ? corneal clouding and proteinuria.

? RISK FACTORS FOR ATHEROSCLEROSIS (Top-Scoring List)

? Modifiable

- High LDL
- Low HDL
- Hypertension
- Smoking
- Diabetes
- Obesity (especially central)
- High saturated fat intake
- Sedentary lifestyle

- Stress, alcohol excess

? Non-modifiable

- Age
- Male sex
- Family history
- Genetic disorders

? Emerging

- Elevated hs-CRP
- High Lp(a)
- Homocysteinemia
- Chronic inflammatory diseases

? ULTRA-SHORT EXAM PEARLS

- LDL lowering (statins) reduces MI risk more than any other drug class.
- HDL < 40 mg/dL is a strong, independent predictor of CAD.
- Diabetes = **atherosclerosis equivalent**; treat LDL aggressively.
- Small dense LDL = most atherogenic particle.

- Type IIa hyperlipoproteinemia ? very high LDL ? premature MI.
- Type I hyperlipoproteinemia ? pancreatitis but **no risk** of atherosclerosis.
- Palmar xanthomas = Type III dysbetalipoproteinemia.

? MCQs — Cardiovascular Diseases & Hyperlipidemias

? 1. Which lipoprotein is most strongly associated with atherosclerosis?

A. HDL
B. Chylomicron
C. LDL
D. VLDL

Answer: C. LDL

? 2. Which lipid fraction is most protective against coronary artery disease?

A. LDL
B. VLDL
C. HDL
D. Lp(a)

Answer: C. HDL

? 3. Atherogenic dyslipidemia (common in Type 2 diabetes) includes:

A. High HDL, low LDL
B. Low HDL, high TG, small dense LDL

C. High HDL and high TG

D. High LDL only

Answer: B. Low HDL, high TG, small dense LDL

? 4. The earliest visible lesion in atherosclerosis is:

A. Fibrous cap

B. Fatty streak

C. Thrombus

D. Calcified plaque

Answer: B. Fatty streak

? 5. Which form of LDL is most dangerous?

A. Large buoyant LDL

B. Small dense LDL

C. LDL-2

D. LDL-4

Answer: B. Small dense LDL

? 6. Which enzyme modifies LDL into oxidized LDL?

A. Lipoprotein lipase

B. Lecithin cholesterol acyltransferase

C. Myeloperoxidase / free radicals

D. Apo C-II

Answer: C. Myeloperoxidase / free radicals

? 7. Which lipoprotein is most associated with premature myocardial infarction?

A. HDL
B. Lp(a)
C. IDL
D. Chylomicron remnants

Answer: B. Lp(a)

? 8. Which of the following increases HDL?

A. Smoking
B. Obesity
C. Aerobic exercise
D. High saturated fat intake

Answer: C. Aerobic exercise

? 9. Which condition presents with markedly elevated triglycerides and eruptive xanthomas?

A. Type IIa
B. Type III
C. Type IV
D. Type V

Answer: D. Type V
(chylomicrons + VLDL)

? 10. Which disorder has palmar xanthomas?

A. Type I
B. Type IIa
C. Type III
D. Type V

Answer: C. Type III (Dysbetalipoproteinemia)

? 11. Which disorder shows tendon xanthomas?

A. Type IIa
B. Type I
C. Type IV
D. Type V

Answer: A. Type IIa (Familial Hypercholesterolemia)

? 12. Pancreatitis is MOST associated with which lipid abnormality?

A. High LDL
B. High HDL
C. Very high triglycerides
D. High Lp(a)

Answer: C. Very high triglycerides

? 13. Which lipoprotein is lowest in abetalipoproteinemia?

A. HDL
B. LDL
C. VLDL

D. All Apo B–containing lipoproteins

Answer: D. All Apo B–containing lipoproteins

? 14. Tangier disease is characterized by:

A. High HDL
B. Low HDL
C. High LDL
D. High triglycerides

Answer: B. Low HDL

? 15. LCAT deficiency primarily affects:

A. LDL
B. Chylomicrons
C. HDL
D. IDL

Answer: C. HDL

? 16. What is the major initiating event in atherosclerosis?

A. Plaque rupture
B. LDL deposition
C. Endothelial injury
D. Thrombosis

Answer: C. Endothelial injury

? 17. Which dietary fat most increases LDL levels?

A. Monounsaturated fats
B. Omega-3 fatty acids
C. Saturated fats
D. Polyunsaturated fats

Answer: C. Saturated fats

? 18. The primary target of lipid-lowering therapy is:

A. HDL
B. LDL
C. Triglycerides
D. VLDL

Answer: B. LDL

? 19. Primary prevention of atherosclerosis includes all EXCEPT:

A. Smoking cessation
B. Weight control
C. Tight glycemic control
D. Long-term anticoagulation

Answer: D. Long-term anticoagulation
(Only for selected high-risk cases, not general prevention.)

? 20. Which class of drugs is first-line for high LDL?

A. Fibrates
B. Statins

C. Niacin

D. Bile acid resins

Answer: B. Statins

? 21. Which drug primarily lowers triglycerides?

A. Statins

B. Fibrates

C. PCSK9 inhibitors

D. Ezetimibe

Answer: B. Fibrates

? 22. Which lipoprotein travels from intestine to liver?

A. Chylomicron

B. HDL

C. LDL

D. VLDL

Answer: A. Chylomicron

? 23. Foam cells are formed by macrophages ingesting:

A. HDL

B. Oxidized LDL

C. VLDL

D. Apo E

Answer: B. Oxidized LDL

? 24. Which condition is NOT a risk factor for atherosclerosis?

A. Hypothyroidism
B. Low HDL
C. High homocysteine
D. High bilirubin

Answer: D. High bilirubin *(actually protective)*

? 25. Atherosclerosis is MOST strongly accelerated in which condition?

A. Hypoglycemia
B. Diabetes mellitus
C. Hypotension
D. Low protein diet

Answer: B. Diabetes mellitus

? CLINICAL CASE–BASED QUESTIONS (WHOLE CHAPTER)

? 1. Young Patient With Early MI

A 28-year-old man with no hypertension or diabetes presents with sudden chest pain. ECG shows anterior MI.
Lipid profile:

- LDL = 240 mg/dL
- HDL = 32 mg/dL

- Triglycerides = 120 mg/dL
  His father had MI at age 42.

Diagnosis:
?? **Familial Hypercholesterolemia (Type IIa)**

Reason: Very high LDL + early MI + family history.

? 2. Chest Pain on Climbing Stairs

A 56-year-old man experiences chest tightness while climbing stairs. Pain settles with rest.
Lipid profile:

- LDL: 180 mg/dL
- HDL: 38 mg/dL

Diagnosis:
?? **Stable Angina (due to fixed atherosclerotic plaque)**

? 3. Elderly Woman With Unstable Angina

A 68-year-old woman develops chest pain at rest. ECG shows ST depression.
Troponins are negative. LDL is 170 mg/dL.

Diagnosis:
?? **Unstable Angina (ACS without enzyme rise)**

? 4. Sudden-Onset Severe Chest Pain

A 60-year-old diabetic man presents with severe crushing chest pain at rest.
ECG: ST elevation in inferior leads.

Troponin-I positive.

Diagnosis:
?? **STEMI (plaque rupture + thrombosis)**

? 5. Atypical Chest Pain in Diabetic

A 54-year-old man with Type 2 DM has no chest pain but complains of fatigue and breathlessness. ECG shows ST depression.

Diagnosis:
?? **Silent Myocardial Ischemia**

Reason: Neuropathy blunts pain sensation.

? 6. Lipid Profile Suggesting Metabolic Syndrome

A 48-year-old obese man:

- TG = 280 mg/dL
- HDL = 29 mg/dL
- Fasting glucose = 120 mg/dL
- Waist circumference: 102 cm

Diagnosis:
?? **Atherogenic Dyslipidemia associated with Metabolic Syndrome**

? 7. Severe Hypertriglyceridemia With Abdominal Pain

A 35-year-old alcoholic presents with acute epigastric pain.
TG = 950 mg/dL
Amylase high.

Diagnosis:
?? **Hypertriglyceridemia-induced pancreatitis (Type IV or V)**

? 8. Eruptive Xanthomas Everywhere

A 22-year-old man has multiple yellow papules over back and arms.
TG = 1250 mg/dL
Chylomicrons present.

Diagnosis:
?? **Type V Hyperlipoproteinemia**
(VLDL + Chylomicrons)

? 9. Palmar Xanthomas

A 50-year-old man with orange-colored creases on palm.
Lipid profile:

- TC = 350 mg/dL
- TG = 400 mg/dL
- IDL elevated

Diagnosis:
?? **Type III Hyperlipoproteinemia (Dysbetalipoproteinemia)**

? 10. Tendon Xanthomas

A 40-year-old patient shows nodular swellings over Achilles tendon.
LDL = 280 mg/dL.

Diagnosis:
?? **Familial Hypercholesterolemia (Type IIa)**

? 11. Orange Tonsils

A 6-year-old boy has extremely low HDL (3 mg/dL) and enlarged yellowish/orange tonsils.

Diagnosis:
?? **Tangier disease (Hypoalphalipoproteinemia)**

? 12. Malabsorption + ACANTHOCYTES

A young child has fat malabsorption, failure to thrive, ataxia, and RBC acanthocytes.

Diagnosis:
?? **Abetalipoproteinemia**
(Apo B absent ? no chylomicrons/VLDL/LDL)

? 13. Post-Menopausal Woman With High LDL

A 56-year-old woman after menopause has increased LDL despite normal body weight and diet.

Reason:
?? Loss of estrogen ? **estrogen protects HDL and LDL metabolism**.

? 14. Early Atherosclerosis in Diabetic

A 52-year-old diabetic woman has normal LDL (110 mg/dL) but still develops CAD.

Reason:
?? Presence of **small dense LDL** (highly atherogenic) even when LDL seems normal.

? 15. High hs-CRP in a Patient With Borderline Lipids

A 45-year-old man:

- LDL = 118 mg/dL
- HDL = 38 mg/dL
- hs-CRP = significantly high

Interpretation:
?? **High inflammatory state ? unstable plaques ? high CAD risk** even if LDL borderline.

? 16. Coronary Artery Spasm

A 40-year-old woman presents with severe chest pain at rest, early morning.
ECG: transient ST elevation during pain.

Diagnosis:
?? **Prinzmetal Angina** (coronary vasospasm)

? 17. Sudden Cardiac Death

A 62-year-old non-diabetic smoker collapses suddenly. Autopsy: ruptured plaque with thrombus in LAD.

Mechanism:

?? **Plaque rupture** is the most common cause of MI.

? 18. Statin-Induced Muscle Pain

A 58-year-old man on statins develops muscle aches.

CK-MB normal, CK (creatine kinase) elevated.

Diagnosis:

?? **Statin-induced myopathy**

? 19. LDL Very High in Hypothyroidism

A 40-year-old woman with fatigue and weight gain has:

LDL = 210 mg/dL

TSH = high.

Reason:

?? **Hypothyroidism decreases LDL receptor expression ? ? LDL**.

? 20. Aspirin for Prevention

A 50-year-old man with CAD is prescribed low-dose aspirin.

Why?

?? **Prevents thrombosis over ruptured atherosclerotic plaques** (secondary prevention).

? 21. High-Carbohydrate Diet Worsens Lipids

A man switches to high-carb, low-fat diet.

After 3 months:

TG ?, HDL ?.

Reason:

?? Excess carbs ? ? VLDL ? ? TG ? ? HDL.

? 22. High Lp(a) in a Young Man With MI

A 32-year-old non-smoker with no diabetes presents with MI.
Lipid profile near-normal except:
Lp(a) = very high.

Diagnosis:

?? **Genetic predisposition to premature CAD** due to high Lp(a).

? 23. Patient With Normal Lipids but MI Family History

A 40-year-old man with normal LDL still gets MI. His father and brother had MI in their 40s.

Explanation:

?? **Genetic risk overrides lipid levels** (Lp(a), inflammation, endothelial function).

? 24. Patient With CAD + High Homocysteine

A 55-year-old man with CAD has homocysteine level elevated.

Mechanism:

?? Homocysteine damages endothelium ? accelerates atherosclerosis.
(Linked with low B6/B12/folate)

? 25. Atherosclerosis in Chronic Inflammatory Disease

A 42-year-old woman with rheumatoid arthritis develops CAD early.

Reason:
?? Chronic inflammation accelerates endothelial injury.

? VIVA VOCE — Cardiovascular Diseases & Hyperlipidemias (Whole Chapter)

? LIPID PROFILE

1. Which lipoprotein is most atherogenic?

LDL.

2. Which lipoprotein is most protective?

HDL.

3. What does non-HDL cholesterol represent?

All atherogenic particles (LDL + VLDL + IDL + Lp(a)).

4. What is the formula for LDL (Friedewald)?

LDL = TC – HDL – (TG/5).
(Not valid if TG > 400 mg/dL.)

5. What increases HDL?

Exercise, estrogen, moderate alcohol.

6. What decreases HDL?

Smoking, obesity, poorly controlled diabetes.

7. Which marker strongly predicts MI risk?

Total cholesterol / HDL ratio.

8. What is atherogenic dyslipidemia?

Low HDL + high TG + small dense LDL (common in Type 2 DM).

? ATHEROSCLEROSIS

9. What is the initiating event in atherosclerosis?

Endothelial injury.

10. What is the earliest visible lesion?

Fatty streak.

11. What forms foam cells?

Macrophages ingesting oxidized LDL.

12. What causes plaque rupture?

Weak fibrous cap + inflammation.

13. Why is diabetes a major risk factor?

Small dense LDL + endothelial dysfunction + chronic inflammation.

14. What is the role of oxidized LDL?

Highly atherogenic; promotes foam cell formation.

15. Which arteries are commonly affected?

Coronary ? carotid ? femoral.

16. What is the most dangerous plaque?

Inflamed, lipid-rich plaque with thin fibrous cap (“vulnerable plaque”).

? CORONARY ARTERY DISEASE

17. What causes angina pectoris?

Myocardial ischemia due to reduced coronary blood flow.

18. What is stable angina?

Predictable chest pain on exertion; relieved by rest.

19. What is unstable angina?

Chest pain at rest; part of ACS; plaque rupture without biomarkers.

20. What is Prinzmetal angina?

Coronary vasospasm causing transient ST elevation.

21. What is the most common cause of MI?

Plaque rupture with thrombosis.

22. Which biomarker is most specific for MI?

Troponin I or T.

23. Which biomarker helps detect reinfarction?

CK-MB.

24. What is silent ischemia?

Ischemia without pain (common in diabetics due to neuropathy).

? CHOLESTEROL & MI

25. Which lipid abnormality most strongly predicts MI?

High LDL.

26. Which lipoprotein predisposes to early MI?

Lipoprotein(a).

27. Why is HDL protective?

Reverse cholesterol transport + anti-inflammatory effect.

28. What is small dense LDL?

Highly atherogenic LDL seen in diabetes and metabolic syndrome.

29. Why does high triglyceride increase MI risk indirectly?

Raises VLDL ? increases small dense LDL.

? PREVENTION OF ATHEROSCLEROSIS

30. First-line drug class for LDL lowering?

Statins.

31. Which lifestyle change raises HDL the most?

Aerobic exercise.

32. Which diet is best for cardiovascular health?

Mediterranean diet.

33. What is the effect of smoking on lipids?

? HDL, ? oxidation of LDL.

34. Why is hypertension a risk factor?

Causes endothelial injury and plaque formation.

35. Which vitamin deficiencies increase homocysteine?

Vitamin B6, B12, folate deficiency.

? HYPOLIPOPROTEINEMIAS

36. Key feature of abetalipoproteinemia?

Absence of Apo B lipoproteins (chylomicrons, VLDL, LDL).

37. What is seen in Tangier disease?

Extremely low HDL + orange tonsils.

38. LCAT deficiency causes what?

Low HDL + corneal clouding + renal disease.

? HYPERLIPOPROTEINEMIAS (Type I–V)

(One-liners)

39. Type I hyperlipoproteinemia—main finding?

? Chylomicrons ? pancreatitis (no CAD).

40. Type IIa hyperlipoproteinemia—main feature?

Very high LDL ? tendon xanthomas + early CAD.

41. Type IIb hyperlipoproteinemia—lipids raised?

LDL + VLDL.

42. Type III hyperlipoproteinemia—classical sign?

Palmar xanthomas (Apo E defect).

43. Type IV hyperlipoproteinemia—main finding?

? VLDL + high triglycerides.

44. Type V hyperlipoproteinemia—lipids raised?

Chylomicrons + VLDL ? pancreatitis.

? CLINICAL PEARLS

45. Which lipid abnormality causes pancreatitis?

Very high triglycerides (usually > 1000 mg/dL).

46. Which lipoprotein travels from intestine to liver?

Chylomicrons.

47. What does HDL do to cholesterol?

Takes it back to liver (reverse transport).

48. What increases Lp(a)?

Genetic factors — not diet.

49. Which lipid lowering drug causes flushing?

Niacin.

50. Which drug class increases LDL receptor expression?

Statins.