Multisociety Jordan National Dyslipidemia Management

Clinical Protocols Initiative

A Collaborative Program of Nine Jordanian Medical Societies

Coordinated by the Jordan Cardiac Society (JCS)

Participating Societies

* Jordanian Cardiac Society (JCS)

* Jordan Atherosclerosis Society (JAS)

* Jordan Society of General Practitioners (JSGP)

* Jordan Society of Internal Medicine (JSIM)

* Jordan Society of Nephrology (JSN)

* Jordan Society of Family Medicine (JSFM)

* Jordan Emergency Medicine Society (JEMS)

* Jordan Endocrine and Diabetes Society (JEDS)

* Jordan Nutrition Society (JNS)

Core Principle

Understanding ApoB in Clinical Practice

Cholesterol and triglycerides are fatty substances that do not dissolve in the blood, which is water-based. To circulate in the bloodstream they must be transported inside protein particles known as lipoproteins.

Apolipoprotein B (ApoB) is the structural protein present on all atherogenic lipoprotein particles, including:

* LDL
* VLDL
* IDL
* Remnant particles
* Lipoprotein(a)

Each atherogenic particle contains one ApoB molecule. Therefore, measuring ApoB reflects the total number of atherogenic particles circulating in the blood.

These particles penetrate the arterial wall and promote the development of atherosclerosis.

Atherosclerosis is driven by ApoB-containing lipoproteins, particularly LDL particles.

Clinical evidence consistently shows:

“The lower the LDL, the lower the cardiovascular risk.”

LDL cholesterol therefore remains the primary therapeutic target.

1. Lipid Screening

Screening should begin:

* From age ≥5 years if there is a family history of premature ASCVD

* From age ≥2 years when severe homozygous familial hypercholesterolemia is suspected

Minimum Tests for Cardiovascular Risk Assessment

Routine baseline evaluation should include:

* Blood pressure measurement

* Lipid panel (Total cholesterol, LDL, HDL, triglycerides, non-HDL cholesterol)

* HbA1c or fasting glucose

* Body mass index and waist circumference

* Smoking status

Optional Tests (Risk-Based)

* Lp(a) – once in a lifetime, particularly with family history of premature cardiovascular disease

* ApoB – useful when triglycerides are elevated or when cardiovascular risk appears higher than expected

Fasting is not routinely required.

Fasting is recommended only when triglycerides exceed 400 mg/dL.

2. Cardiovascular Risk Stratification

Accurate cardiovascular risk assessment is the cornerstone of preventive cardiology.

Risk stratification determines:

* The intensity of lipid-lowering therapy

* Blood pressure targets

* Timing of combination therapy

* Frequency of follow-up

The higher the vascular risk, the earlier and more intensive treatment should be initiated.

Two Approaches to Risk Estimation

1. Risk Calculators

Risk calculators estimate 10-year cardiovascular risk.

European models:

* SCORE2

* SCORE2-OP

American models:

* PREVENT

* ASCVD Risk Estimator Plus

2. Clinical Risk Classification

In high-burden regions, a clinical risk-based classification may be more practical.

Low Risk

* No major cardiovascular risk factors

* No diabetes

* No CKD

* No vascular disease

LDL-C Target: <116 mg/dL

Lifestyle measures are usually sufficient.

Moderate Risk

* One major risk factor

(hypertension, smoking, dyslipidemia, obesity, strong family history)

LDL-C Target: <100 mg/dL

Pharmacologic therapy should be considered if lifestyle measures fail.

High Risk

* ≥2 major risk factors

* Diabetes without organ damage (<10 years)

* LDL ≥190 mg/dL

* CKD (eGFR 30–59)

LDL-C Target: <70 mg/dL

Combination lipid-lowering therapy is often required.

Very High Risk

* Established ASCVD

* Complicated diabetes

* Severe CKD (eGFR <30)

* Polyvascular disease

LDL-C Target: <55 mg/dL

Extreme Risk

* Recurrent ASCVD events

* Progressive vascular disease despite optimal therapy

LDL-C Target: <40 mg/dL

Advanced therapies such as PCSK9 inhibitors should be considered early.

Regional Considerations

Cardiovascular disease in our region often occurs earlier and more aggressively due to:

* Low public awareness of preventive cardiology

* Delayed clinical presentation

* Limited structured screening programs

* High prevalence of diabetes, obesity and smoking

* Restricted access to advanced therapies

* Fragmented follow-up systems

Therefore, earlier treatment intensification may be appropriate.

3. Treatment Strategy

Step 1 – Lifestyle Measures

Recommended for all patients:

* Mediterranean-style diet

* Reduced saturated fats

* Increased fiber intake

* Regular physical activity

* Weight control

* Smoking cessation

* Avoid excessive alcohol intake

Step 2 – Risk-Based Lipid Management

Step 0 – Define LDL Target

High Risk → <70 mg/dL

Very High Risk → <55 mg/dL

Extreme Risk → <40 mg/dL

Stepwise Treatment

1. Start High-Intensity Statin

* Atorvastatin 40–80 mg

* Rosuvastatin 20–40 mg

Reassess LDL after 4 weeks.

2. If LDL remains above target

Add Ezetimibe

Reassess after 4 weeks.

3. If LDL still above goal

Consider advanced therapies.

Advanced Therapies

PCSK9 inhibitors

Indicated particularly in:

* Extreme risk

* Recurrent ASCVD events

* Multivessel coronary disease

* Diabetes with ASCVD

Major outcome trials:

* FOURIER

* ODYSSEY

Alternative Therapy

Inclisiran

Dosing schedule:

* Day 0

* Month 3

* Every 6 months thereafter

LDL reduction ≈ 50%

Statin Intolerance

Consider:

* Bempedoic acid

(≈20% LDL reduction)

4. Triglycerides

If TG >150 mg/dL

* Intensify lifestyle

* Optimize metabolic control

If TG ≥500 mg/dL

* Prevent pancreatitis

* Consider fibrates

5. Residual Risk

If LDL targets are achieved but cardiovascular risk persists, evaluate:

* ApoB

* Triglycerides

* Lipoprotein(a)

* Metabolic risk factors

LDL reduction should always be addressed before additional lipid abnormalities.

Omega-3 Therapy (Icosapent Ethyl)

ACC recommends icosapent ethyl (EPA) in statin-treated patients with:

* Triglycerides 135–499 mg/dL

* High ASCVD risk

Clinical trials showed a small increase in atrial fibrillation:

* 5.3% vs 3.9%

Absolute increase ≈ 1.4%

Use caution in patients with:

* Prior AF

* Enlarged left atrium

* Multiple AF risk factors

Statin Safety

A large Lancet meta-analysis (2026) involving more than 123,000 patients found:

* No strong evidence that statins cause most commonly reported side effects.

The only consistently confirmed adverse effect was:

* Mild elevation of liver enzymes.

Many muscle symptoms reported in practice are not directly caused by statins.

Statin-Associated Muscle Symptoms

True statin myopathy is rare and usually mild.

If symptoms occur:

* Check creatine kinase (CK)

* Exclude secondary causes such as hypothyroidism or drug interactions

CK Interpretation

CK <4× ULN

→ Continue therapy or briefly pause and reassess

CK ≥10× ULN

→ Consistent with myopathy; discontinue statin

Clinical Principle

The cardiovascular benefits of statins greatly outweigh the small risk of muscle toxicity.

References

ESC / EAS Dyslipidaemia Guidelines

https://www.escardio.org/Guidelines/Clinical-Practice-Guidelines/Dyslipidaemias

ACC Clinical Guidance

The Lancet (2026)

Statins Beyond the Myths: Benefits Stand Strong, Risks Limited

https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(25)01578-8/fulltext