Lipid Profile Interpretation

This guide provides a structured approach to **interpreting a lipid profile** and is based on **NICE guidelines** in the United Kingdom. NICE guidance is updated periodically, so it is essential to ensure that you are always following the most recent NICE guidelines or the guidelines in your area.

Lipids

Lipids are fats the body requires for **energy, energy storage** and **cell membrane structure**.

The main groups of lipids are **cholesterol**, **triglycerides** (comprised of glycerol and fatty acids), **free fatty acids** and **phospholipids**. They are obtained through dietary intake and manufactured in the liver. Since lipids are not water-soluble, they are transported in the lymphatic system and bloodstream in combination with proteins, creating structures which are called **lipoproteins**.

When lipids, especially cholesterol, are present in the plasma in high concentrations, they are a risk factor for **cardiovascular disease** due to their role in forming **atheromatous plaques** in the walls of blood vessels.

For more information, see the Geeky Medics guides to **lipids** and **cholesterol metabolism**.

You might also be interested in our premium collection of **1,300+ ready-made OSCE Stations**, including a range of **test result interpretation** stations

Lipid profile

A lipid profile is a blood test quantifying **cholesterol** and **triglyceride** plasma concentrations.

Cholesterol

Cholesterol values are reported as follows:

- **Total cholesterol**
- **High-density lipoprotein cholesterol (HDL-C)**: cholesterol which is being carried in HDL. HDL-C is often described to patients as ‘good’ cholesterol, because it mediates the transport of cholesterol to the liver, where it is excreted from the body, in a process known as **reverse cholesterol transport**. There is an inverse relationship between HDL cholesterol and the risk of cardiovascular disease.
- **Non-high-density lipoprotein cholesterol (non-HDL-C)**: cholesterol carried in lipoproteins other than HDL, including **low-density lipoprotein cholesterol (LDLC)**. Non-HDL-C is calculated by subtracting HDL-C from total cholesterol. Non-HDL cholesterol is described as ‘bad’ cholesterol because of its central role in the pathogenesis of atherosclerosis.
- **Total cholesterol to HDL ratio**: a measure of HDL-C concentration relative to the total cholesterol.

Triglycerides

Elevated concentrations of **triglycerides** are associated with post-prandial hyperlipidaemia and reduced HDL-C, making hypertriglyceridaemia an independent **risk factor** for cardiovascular disease. At concentrations over **10mmol/L**, triglycerides are also associated with **pancreatitis**.

Why perform a lipid profile?

There are three sets of circumstances in which a lipid profile is requested.

Screening

Adults between the ages of **40 and 74 years** without cardiovascular disease may be offered a lipid profile as part of an **NHS health check**. The result can be used as part of the **QRISK3 score**, which estimates an individual’s **risk of cardiovascular disease** in the next ten years.

Additionally, a patient of any age who is noted to have features of hyperlipidaemia on physical examination, such as **tendon xanthoma**, **xanthelasma** or **corneal arcus**, should have a lipid profile performed.

Recent cardiovascular event

Patients who have had a **recent cardiovascular event** (e.g. myocardial infarction, stroke) should have a lipid profile to guide future **risk reduction** strategies.

Monitoring

Patients with **hyperlipidaemia** and those who have had a cardiovascular event should have lipid profile monitoring to assess the effectiveness of lipid-lowering treatment (e.g. statins).

Do patients need to fast?

Since 2014, NICE guidance has stated that most patients **do not need to fast** before a lipid profile blood test.

However, a fasting sample should be obtained in certain circumstances (e.g. initial test shows severe hyperlipidaemia, hypertriglyceridaemia).

Acute illness/injury

A lipid profile should not be performed within six weeks of any acute illness or injury, as there is a **temporary reduction** in cholesterol during this period.

Interpreting a lipid profile

Does the patient have severe hyperlipidaemia?

The first step is to identify patients with **severe hyperlipidaemia**.

Severe hyperlipidaemia is defined as:

- Total cholesterol **>7.5mmol/L (hypercholesterolaemia)** and/or
- Non-HDL-C **>5.9 mmol/L (hypercholesterolaemia)** and/or
- Triglycerides **>4.5mmol/L (hypertriglyceridaemia)**

Some patients will have predominant **hypercholesterolaemia**, some will have predominant **hypertriglyceridemia**, and others will have **mixed hyperlipidaemia**.

Identify any secondary causes

Any **secondary causes** of hypercholesterolaemia or hypertriglyceridemia should be identified, as these may have led to elevated results.

Secondary causes of **hypercholesterolaemia** include:

- Pregnancy
- Hypothyroidism
- Cholestatic liver disease
- Nephrotic syndrome
- Drugs (e.g. diuretics, ciclosporin, corticosteroids, androgens)

Secondary causes of **hypertriglyceridaemia** include:

- Uncontrolled diabetes mellitus
- Chronic kidney disease
- Hepatocellular liver disease
- Alcohol excess
- Metabolic syndrome (including impaired glucose tolerance, hypertension, central obesity)
- Drugs (e.g. beta-blockers, retinoids and corticosteroids)

These conditions should be identified and, where possible, treated before repeating the lipid profile.

Arrange specialist review

Patients who **do not** have a secondary cause, or if hyperlipidaemia persists following management of the secondary cause, will need further investigation and, often, referral to a **specialist lipid clinic** for consideration of conditions such as **familial hypercholesterolaemia, familial hypertriglyceridaemia**, or **familial combined hyperlipidaemia**.

Patients in whom the secondary cause **cannot be modified** (e.g. where causative drugs are essential for the management of comorbidities) may also require referral to a specialist lipid clinic.

Familial hypercholesterolaemia

Familial hypercholesterolaemia causes elevated levels of **low-density lipoprotein cholesterol (LDL-C)**. Patients often have a **family history** of cardiovascular disease and may develop clinical signs of hyperlipidaemia (e.g. tendon xanthoma).

Familial hypercholesterolaemia is usually caused by mutations in the **low-density lipoprotein receptor (LDLR) gene,** resulting in an **autosomal dominant** inheritance pattern.

Familial hypertriglyceridaemia and familial combined hyperlipidaemia are both also dominantly inherited disorders.

Why was the lipid profile performed?

Further interpretation of results will depend on **why** the lipid profile blood test was performed.

Screening

Where the test was done for **screening**, ask: what do these results tell me about this patient's **risk** of **future cardiovascular disease**?
NICE does not define risk based on specific lipid levels in isolation. Instead, recommendations for lifestyle modification and/or lipid-lowering drug treatments are based upon the **QRISK3 calculator** (so-called because it is the third version of the calculator). QRISK3 combines the lipid profile results with **other information**, including smoking status, blood pressure, height and weight.

There are certain groups for whom QRISK3 is not suitable, as they should all be considered to be at increased risk of CVD:

- Age >85
- Type 1 diabetes
- Chronic kidney disease (eGFT <60)
- Inherited lipid metabolism disorder

The QRISK3 score estimates the likelihood of an individual patient **developing cardiovascular disease** over the next **ten years**. If the risk is **10% or greater**, the patient should be given lifestyle advice regarding modifiable risk factors (e.g. smoking, diet, obesity, and physical activity). The patient should also be offered **lipid-lowering therapy** (atorvastatin), either at the same time as lifestyle advice or later if lifestyle modification alone is ineffective in lowering lipid concentrations. This is referred to as **primary prevention.**

In some groups of patients, for example, those who are **severely obese** (BMI >40kg/m^2), those with **serious mental illness** and those with **autoimmune disorders** (e.g. systemic lupus erythematosus), the QRISK3 score may be an **underestimate** of their cardiovascular risk. This is also the case in patients with **significant hypertriglyceridaemia**. In these patients, it may be necessary to initiate lipid-lowering treatment even when the estimated risk is less than 10%.

Target cholesterol levels

Although NICE does not recommend the use of lipid profile results in isolation to guide management, the NHS advises patients on the following cholesterol treatment target levels:

- **Total cholesterol**: should be 5mmol/L or less
- **HDL-C**: should be >1 mmol/L in men and >1.2 mmol/L in women
- **Total cholesterol to HDL ratio**: should be <6

Primary prevention of cardiovascular disease

NICE recommend **atorvastatin 20mg once daily** as lipid-lowering therapy for the primary prevention of cardiovascular disease.

For more information, see the Geeky Medics guide to **statin counselling**.
Recent cardiovascular event

Where a lipid profile was performed following a **recent cardiovascular event**, ask: what do these results tell me about the patient's **risk** of **further cardiovascular events**?

The QRISK3 tool **cannot be used** in these patients, as it is only validated for patients **without** established cardiovascular disease.

Most patients who have suffered a cardiovascular event will be offered treatment with a statin, irrespective of their baseline lipid values (**secondary prevention**). First-line treatment for secondary prevention is **atorvastatin 80mg once daily**. Baseline lipid values will be used to assess the effectiveness of that treatment (described below).

Monitoring

Where the test was done for **monitoring** purposes, ask: what do these results tell me about the **effectiveness** of the current treatment strategy?

In primary and secondary prevention, the target is to reduce **non-HDL-C ('bad cholesterol') by >40%** from baseline levels after **three months** of treatment with a statin.
