CHOLESTEROL & PREVENTION

My cholesterol is normal. Can I still be at risk of a heart attack?

The short answer

Yes. A cholesterol result within the laboratory reference range can be reassuring, but it does not prove that your cardiovascular risk is low or that your coronary arteries are free of atherosclerosis. LDL cholesterol matters, but risk is shaped by the combination of what drives disease, your individual susceptibility and whether disease is already present.

Risk comes from more than one number

1. Forces driving disease

LDL and other atherogenic particles, blood pressure, smoking, diabetes, kidney disease, weight, inflammation, diet and activity.

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2. Your susceptibility

Age, genetics, family history, Lp(a), inflammatory conditions and other factors influence how strongly those exposures translate into disease.

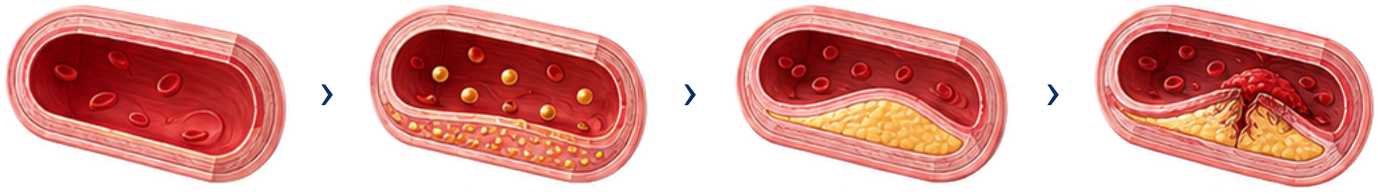
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3. Disease already present

Coronary plaque is the accumulated result of years of exposure and susceptibility. Imaging can sometimes show this disease directly.

How a heart attack develops

Atherosclerosis often begins silently decades before symptoms. ApoB-containing particles become retained within susceptible areas of the artery wall, promoting inflammation and plaque formation. A heart attack usually occurs when a plaque is disrupted and a clot suddenly blocks blood flow.



A wide channel for blood.

Atherogenic particles enter and remain in the arterial wall.

Plaque accumulates and the wall thickens over time.

A clot can form rapidly and obstruct the artery.

The key point

A blood test measures one of the forces that drives atherosclerosis. It does not directly show whether plaque is already present. That is why two people with the same LDL cholesterol can have very different cardiovascular risk.

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What does 'normal cholesterol' actually mean?

Patients often use the word *normal* to mean several different things. A laboratory reference range, a calculated risk estimate and a treatment target answer different questions.

Laboratory reference range

Describes how your result compares with a reference population. It is not a personalised cardiovascular target.

Risk estimate

Tools such as QRISK3 estimate the chance of a future cardiovascular event from several clinical factors.

Treatment target

The cholesterol level we aim for after considering your overall risk and whether atherosclerosis is already present.

What is in a standard lipid profile?

Total cholesterol

A broad overview

The total cholesterol carried in all circulating lipoproteins.

HDL cholesterol

Associated with risk

Often called 'good cholesterol', although raising HDL-C is not itself a treatment target.

LDL cholesterol

A major causal driver

LDL-C reflects the mass of cholesterol carried inside LDL particles. Greater lifelong exposure means greater atherosclerotic risk.

Triglycerides

Metabolic context

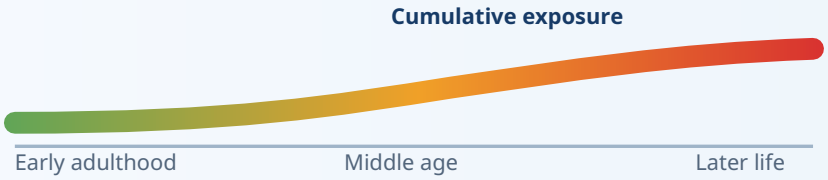
Higher levels often accompany insulin resistance, obesity, diabetes and triglyceride-rich atherogenic particles.

Non-HDL cholesterol: all atherogenic cholesterol together

Calculated as total cholesterol minus HDL-C. It captures cholesterol carried by LDL and other ApoB-containing particles and is especially useful when triglycerides are raised.

Cumulative exposure matters

Atherosclerosis reflects exposure over years, not just today's blood test. The combination of **how high** a risk factor has been and **how long** it has been present matters.



A reference range is not a personal target

The LDL level that is reasonable for a low-risk person may be too high for someone with established plaque or markedly increased risk. Treatment intensity should reflect the person, not simply whether a laboratory has flagged the result.

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Looking beyond the standard lipid profile

Most decisions can be made from clinical history, blood pressure, standard blood tests and an appropriate risk estimate. When the picture remains uncertain, selected biomarkers can reveal information that conventional cholesterol measurements may miss.

Apolipoprotein B - ApoB

How many atherogenic particles are circulating?

Each major atherogenic lipoprotein particle carries one ApoB molecule. ApoB therefore gives an estimate of the total number of LDL, remnant and Lp(a) particles capable of entering the artery wall.

LDL-C



How much cholesterol 'cargo' is being carried

ApoB



Approximately how many atherogenic 'vehicles' are carrying it

LDL-C and ApoB usually track together, but they can be discordant. ApoB can be particularly informative when triglycerides are high, or in diabetes, obesity, metabolic syndrome or very low LDL-C.

Lipoprotein(a) - Lp(a)

Have I inherited an additional cardiovascular risk factor?



Lp(a) is an LDL-like particle whose concentration is determined predominantly by genetics and is usually stable through adult life. Higher levels are associated with higher risk of atherosclerotic cardiovascular disease and aortic valve disease, independently of LDL-C.

Contemporary European guidance recommends considering an Lp(a) measurement at least once in adulthood. It is especially useful when disease is premature, family history is strong or a treatment decision sits close to a risk threshold.

Risk modifier in ESC/EAS: >50 mg/dL (>105 nmol/L)

Where does QRISK fit?

A very useful starting estimate - but not a scan of your arteries

NICE recommends QRISK3 to estimate 10-year cardiovascular risk in adults aged 25 to 84 without established CVD. It combines age, blood pressure, cholesterol and multiple clinical factors to support shared decision-making.

Lifetime risk also matters. NICE suggests considering lifetime-risk discussion particularly when QRISK3 is below 10% or in people under 40 who already have cardiovascular risk factors.

More testing is not automatically better

Advanced tests add most value when the result is likely to change a decision: whether to start treatment, how intensively to lower cholesterol, whether relatives should be assessed, or whether imaging would genuinely add useful information.

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Imaging the coronary arteries: what can we see?

Blood tests estimate the forces that drive atherosclerosis. Imaging can sometimes answer a different question: **is coronary plaque already present, and how much?** Calcium scoring and CT coronary angiography provide different information and should not be treated as interchangeable screening tests.

01 CT calcium score

How much calcified coronary plaque is present?

A calcium score is a quick, non-contrast CT scan with a relatively low radiation dose. It quantifies calcium embedded within atherosclerotic plaque in the coronary artery walls and reports an Agatston score.

Calcium develops as part of atherosclerosis. The score is therefore a **surrogate - an indirect marker - of overall coronary disease burden.** Higher burdens are associated with greater future cardiovascular risk.

CALCIUM WITHIN ATHEROSCLEROTIC PLAQUE

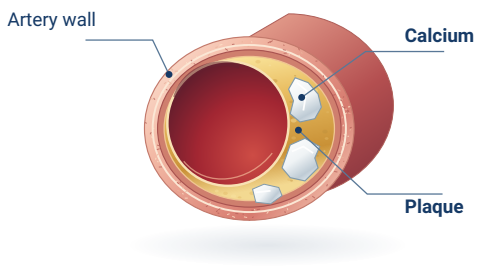


Illustration of the vessel wall; not an actual CT image.

AGATSTON SCORE

0	1-99	100-399	400+
No detectable calcium	Mild burden	Moderate burden	High burden

A score of zero is reassuring, but it does not exclude non-calcified plaque. The score does not directly measure how narrowed an artery is.

Its strongest preventive role is as a risk modifier when the treatment decision is genuinely uncertain or close to a threshold.

02 CT coronary angiography

What do the coronary arteries actually look like?

CTCA uses intravenous contrast to show the coronary artery lumen and vessel wall. It can detect both calcified and non-calcified plaque and define whether a narrowing is present.

- Is coronary plaque present, and how extensive is it?
- Is there a narrowing that could explain symptoms?
- Are there plaque features associated with higher future risk?

PLAQUE COMPOSITION AND THE ARTERY LUMEN

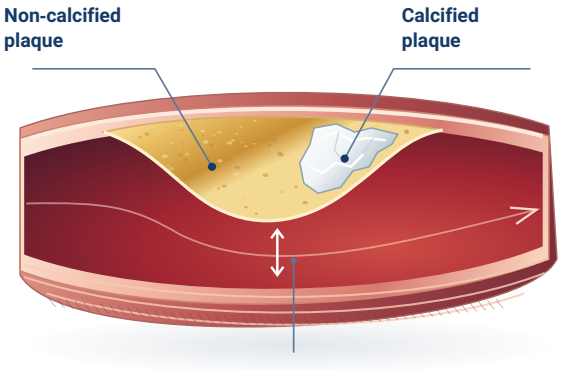


Illustration of anatomy; not an actual CT image.

CTCA is particularly valuable when there is a clinical indication to define coronary anatomy; it is not a routine population screening test for everyone who feels well.

What CT cannot tell us

CT can identify plaque features associated with increased risk, but it cannot predict with certainty which individual plaque will rupture or whether a particular person will have a heart attack. In asymptomatic people, finding coronary calcium alone does not automatically mean that further diagnostic imaging is needed.

Further reading: ACR/RSNA - Calcium scoring | CT coronary angiography

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From a risk estimate to a personal prevention plan

The aim is not to collect more numbers. It is to understand cardiovascular risk well enough to change its trajectory - over the next 10 years and, where relevant, over a lifetime.

1

Start with conventional risk

History, symptoms, blood pressure, standard lipids, smoking, diabetes, kidney disease, weight and QRISK3.

2

Look for underestimated risk

Family history, metabolic disease, inflammatory conditions, premature disease and cumulative exposure.

3

Use biomarkers selectively

ApoB and Lp(a) can refine risk when conventional lipids do not tell the whole story.

4

Establish disease burden

When appropriate, imaging can show whether plaque is present and how much disease has accumulated.

5

Agree personal targets

Set cholesterol, blood-pressure and metabolic goals according to actual risk and disease burden.

6

Measure response over time

Review treatment, repeat relevant measurements and adjust the plan as life and risk change.

What a personal prevention plan may include

Medical treatment and lifestyle work together

- ✓ Know your blood pressure and lipid profile.
- ✓ Do not smoke.
- ✓ Build regular aerobic and resistance exercise.
- ✓ Use a heart-healthy dietary pattern and manage weight.
- ✓ Treat diabetes, hypertension and other medical risks appropriately.
- ✓ Review progress rather than treating prevention as a one-off test.

Questions worth asking at your consultation

- Is my cholesterol simply within the laboratory range, or is it appropriate for *my* cardiovascular risk?
- What are my 10-year and, where useful, lifetime cardiovascular risks?
- Could family history, ApoB or lipoprotein(a) change the picture?
- Would a calcium score or another cardiac test genuinely change what we do?
- If plaque is present, how should that alter the intensity of prevention?

In summary

A normal cholesterol result does not guarantee low cardiovascular risk. The most useful assessment combines conventional risk factors, cumulative exposure, selected biomarkers and - when clinically appropriate - evidence of atherosclerosis itself. **We are not trying to find a single 'normal' cholesterol number; we are trying to understand your risk well enough to change its trajectory.**

Clinical framework informed by NICE NG238, the 2025 ESC/EAS dyslipidaemia update, the 2019 ESC/EAS lipid guidance for ApoB, and the 2024 ESC chronic coronary syndrome guideline. Clinical decisions should always be individualised.