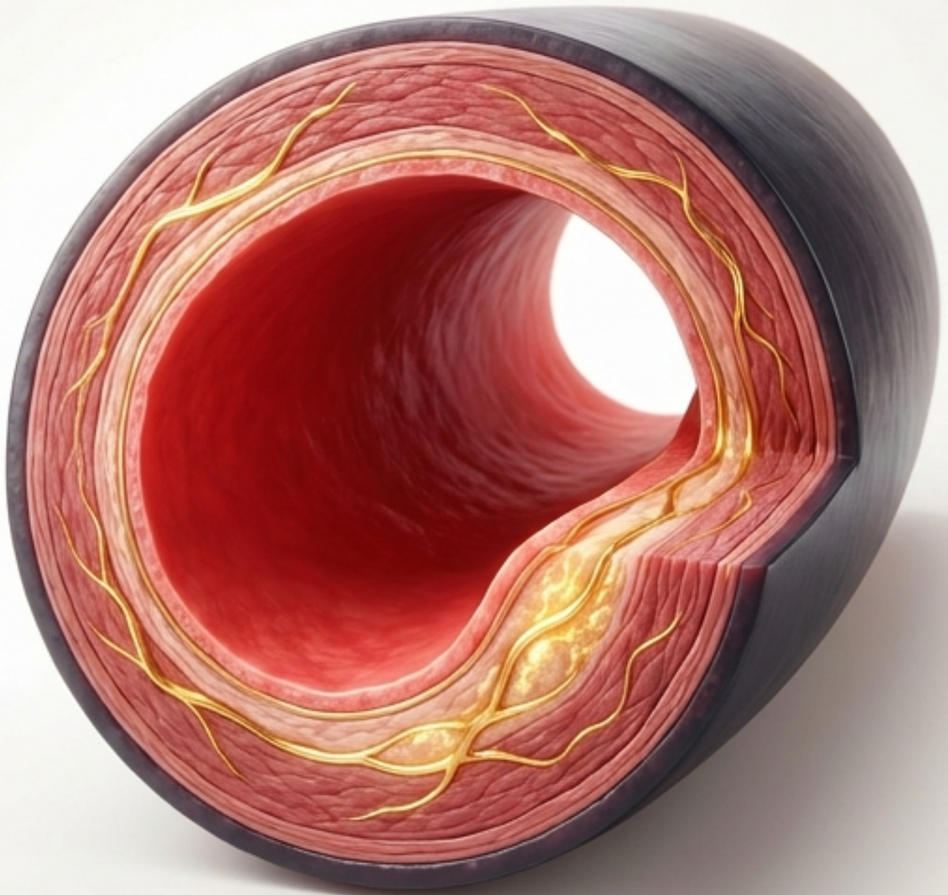
The Illusion of Normal

ApoB, AI Imaging, and the Hidden Truth About Coronary Arteries at Age 60.

Based on peer-reviewed medical research.



THE ILLUSION

What standard imaging often shows at age 60: “Normal” arteries.

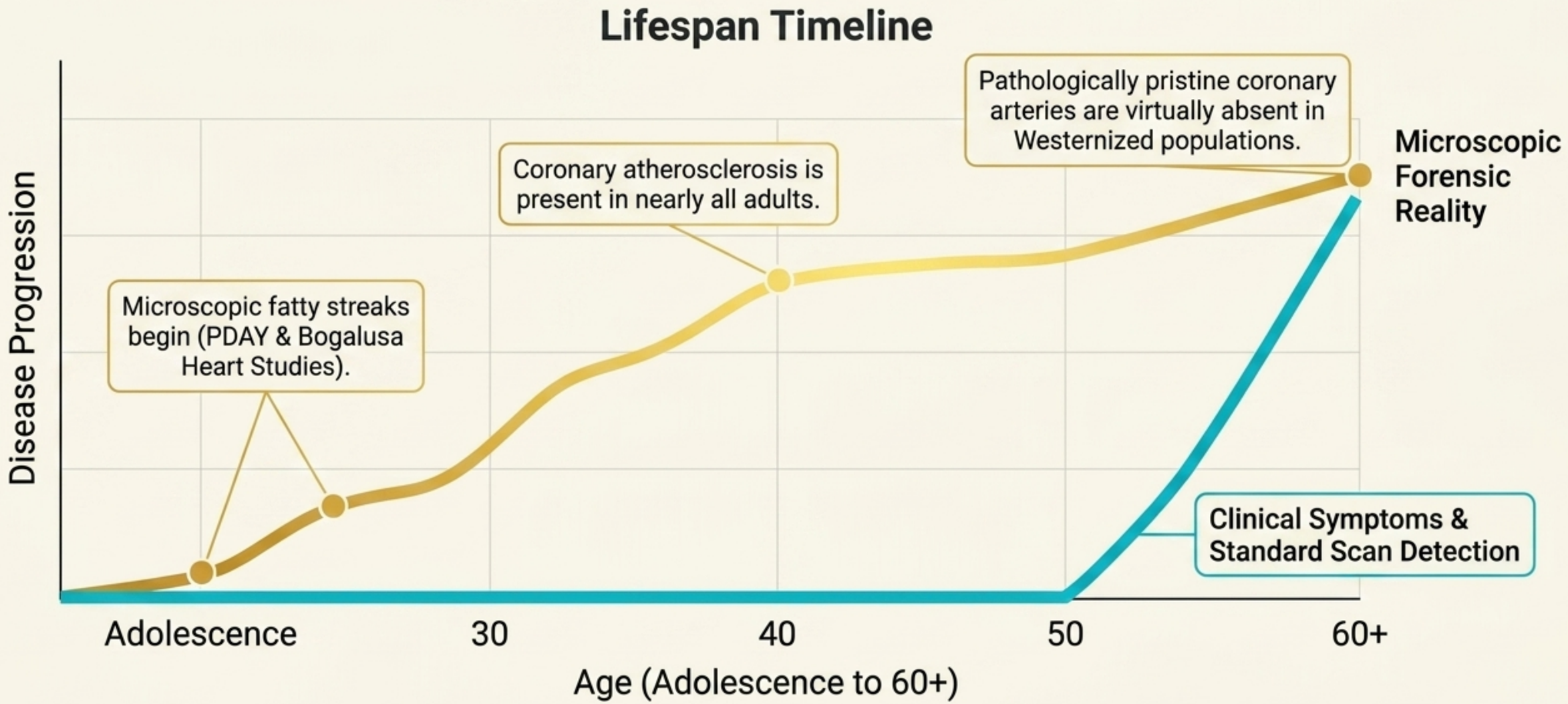
THE REALITY

A clinical assertion at odds with physiological reality

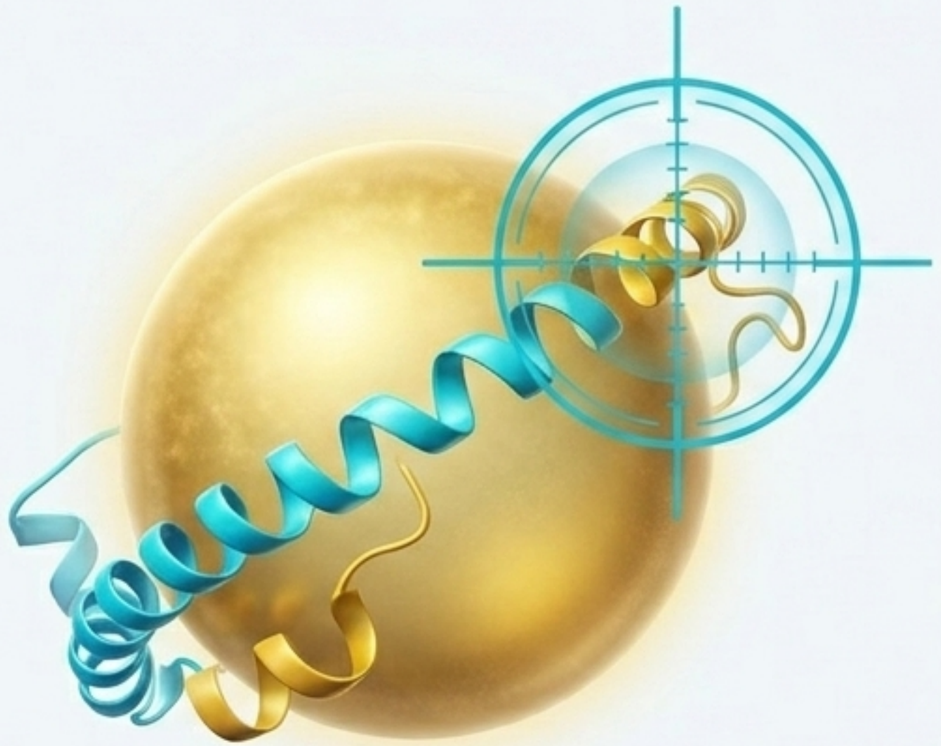
Heart attacks frequently occur in patients with 0% visible anatomical blockages

How can a 60-year-old with elevated cholesterol possess pristine arteries? The answer lies in the resolution of our tools.

The forensic reality contradicts the clinical illusion



Prolonged exposure to ApoB is the primary driver of atherosclerotic plaque



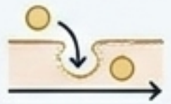
Exploded View: ApoB Lipoprotein

(The Rule)

1 Particle = 1 ApoB molecule.
Every atherogenic particle (LDL, VLDL, IDL) carries exactly one ApoB protein.

(The Mechanism)

The probability of a particle becoming trapped in the subendothelial space is driven by concentration and time.



Risk is quantified as “ApoB-years” — the total lifetime burden of arterial wall penetrations.

Differentiating the true drivers of atherogenesis

Lipoprotein Metric	Definition & Pathological Role	Predictive Power
Low-Density Lipoprotein Cholesterol (LDL-C)	The total mass of cholesterol contained within LDL particles.	Causal, but frequently discordant with actual particle count.
Apolipoprotein B (ApoB)	The structural protein reflecting true particle burden.	The strongest predictor of clinical risk; out-predicts LDL-C in cases of discordance.
Lipoprotein(a) [Lp(a)]	An LDL-like particle with an additional [apo(a)] protein.	Independent risk factor promoting both calcification and localized thrombosis.

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Particle count dictates collision risk, not total mass

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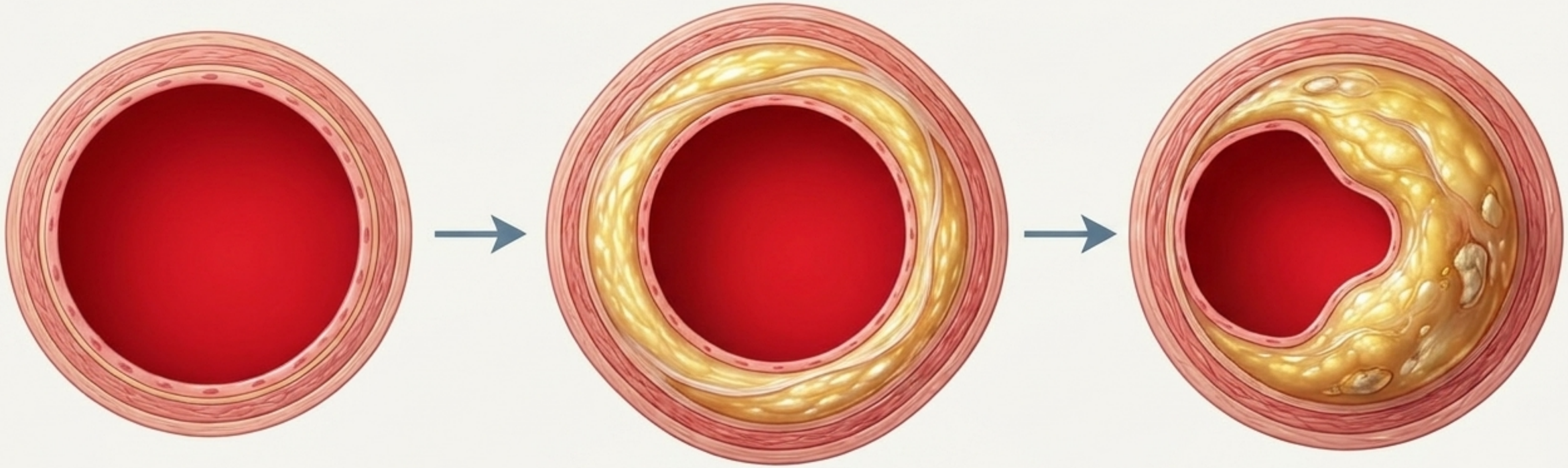
The Glagov Effect: Compensatory outward remodeling



Healthy Vessel

Plaque accumulates, but the vessel expands externally to preserve blood flow. The lumen remains untouched.

Plaque exceeds ~40% of the internal elastic lamina area. Hemodynamic narrowing finally begins.

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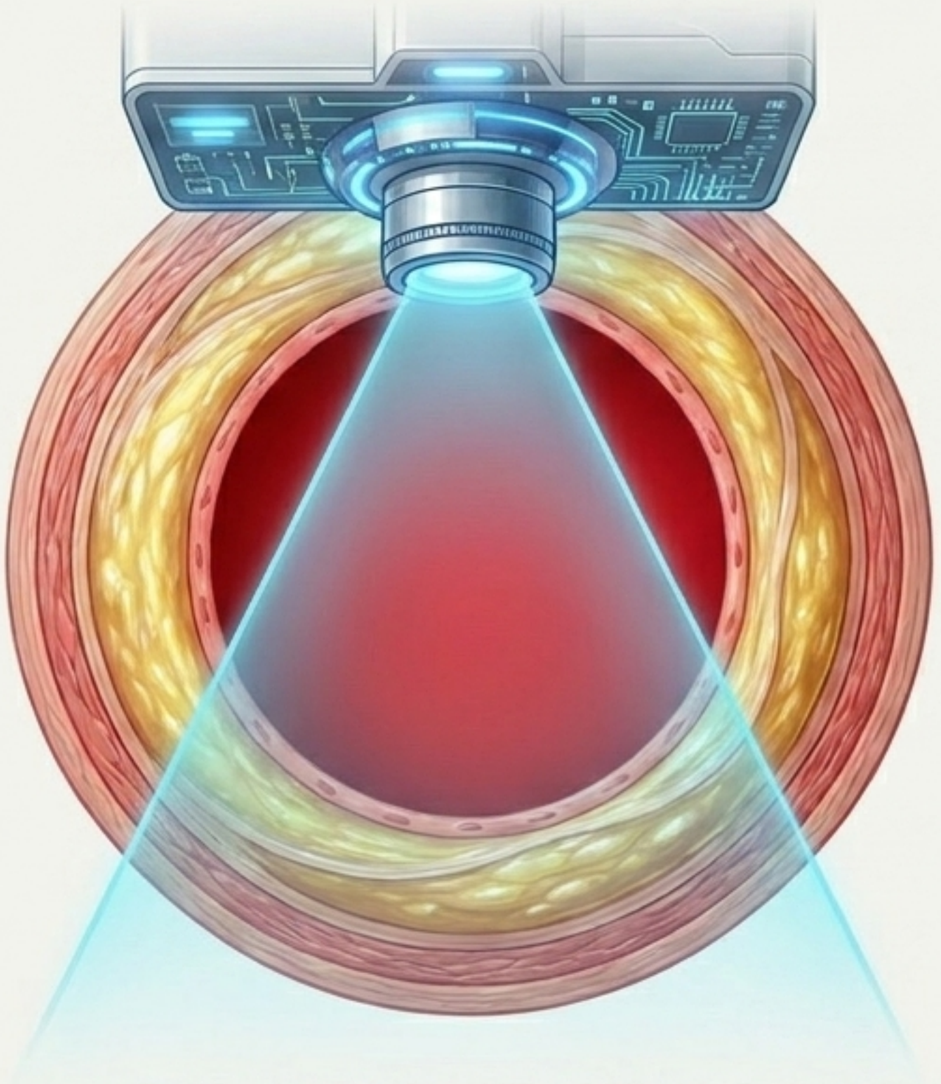
Standard angiography only sees the silhouette of the lumen



Because standard invasive angiography and conventional visual CCTA focus entirely on the hollow center, **massive lipid-rich plaque burdens can exist in the expanded wall while remaining angiographically invisible.**

The danger is hidden in the walls, entirely missed by the camera.

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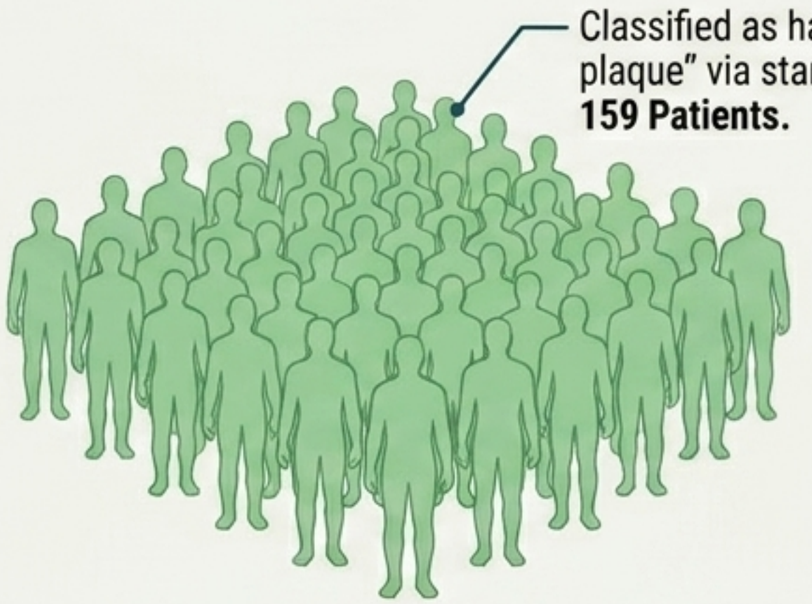
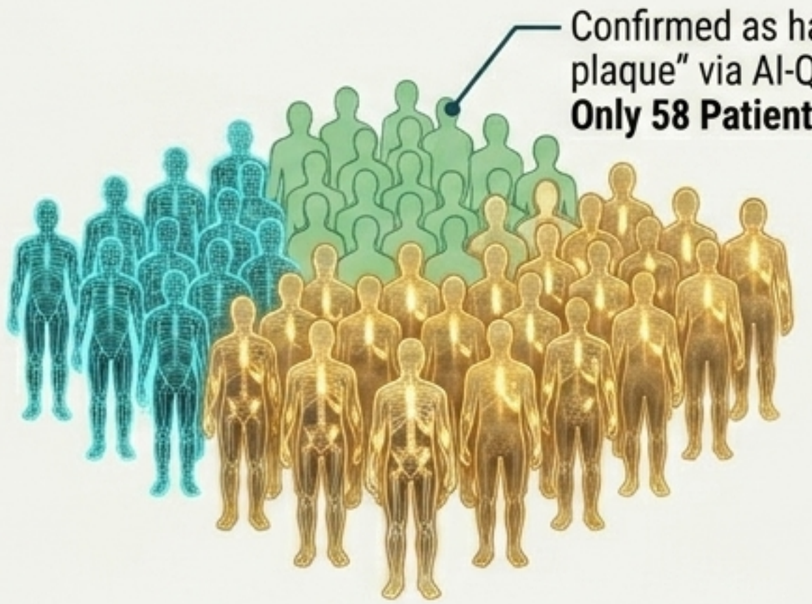
Artificial Intelligence reveals the concealed pathology

Standard Visual CCTA



Standard visual interpretation of CCTA systematically underestimates plaque burden.

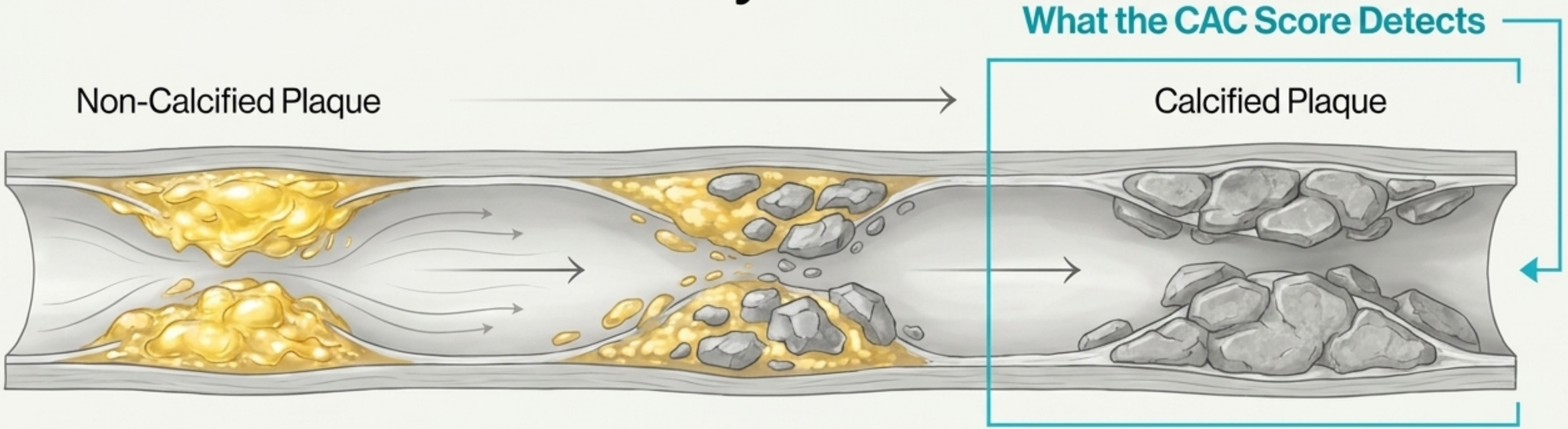
AI-QCT Volumetric Analysis



AI-QCT volumetric analysis resulted in diagnostic modification for 39% of patients.

The number of patients confidently classified as having 'no plaque' plummeted from 159 down to 58.

Calcification is a marker of plaque healing, not active vulnerability



Non-Calcified Plaque:
Biologically active, prone to erosion/rupture, highly correlated with near-term MI risk. (Invisible to CAC).

Calcified Plaque:
Chronic, healed lesions. High scores (≥300 in MESA study) indicate massive cumulative lifetime burden and long-term risk.

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A Calcium Score of zero does not confer immunity

Calcium scanning looks for the ash of old fires. It cannot see the active spark of lipid-rich plaque.

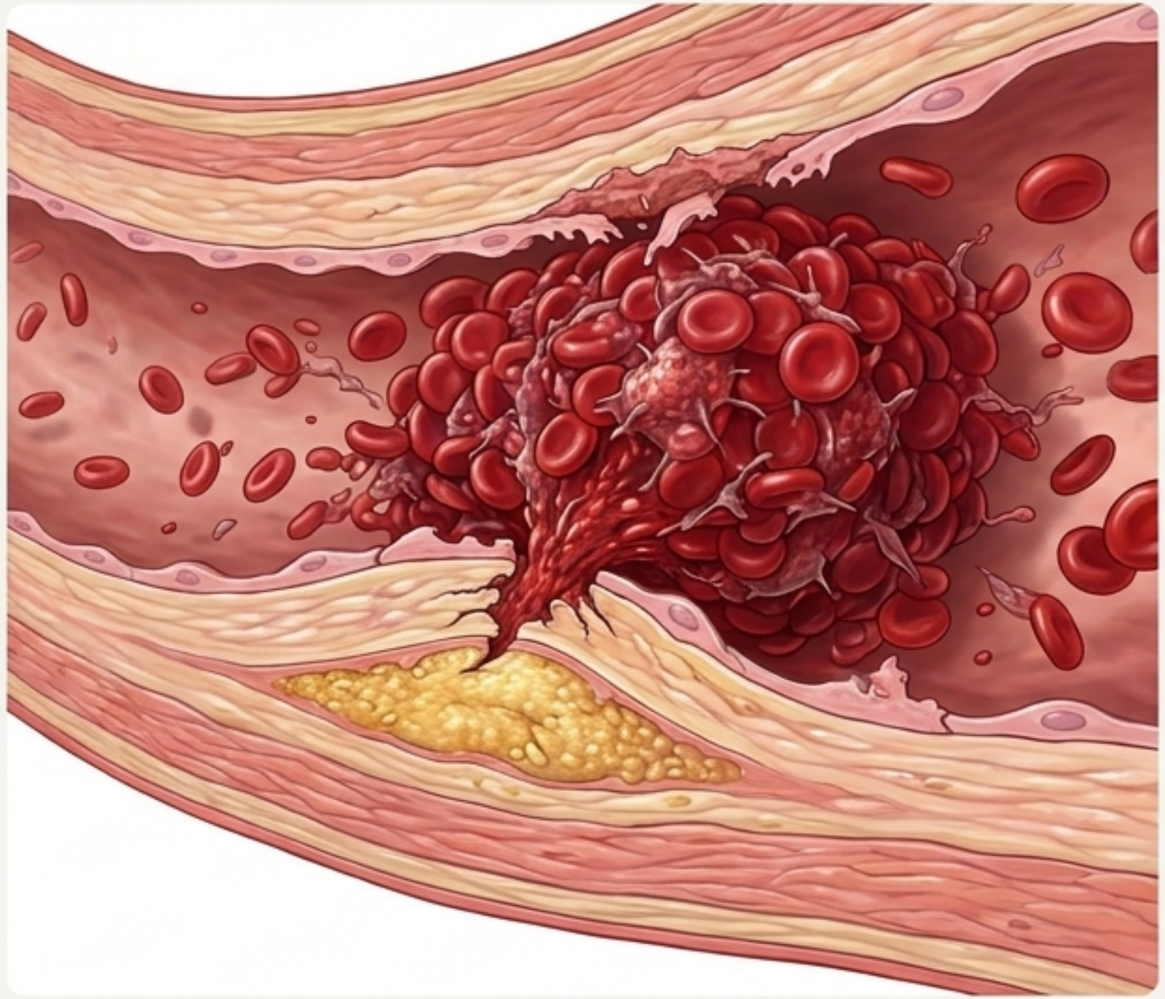
High ApoB & Non-Calcified Plaque
(The Spark)

Calcified Plaque / CAC Score
(The Ash)

25–30% of acute coronary syndromes occur in individuals with absent or minimal coronary calcification at baseline.

In high-ApoB individuals, a CAC score of zero reflects a delay in disease expression, not the absence of disease.

MINOCA: Myocardial Infarction with Non-Obstructive Coronary Arteries



Even microscopic, non-obstructive plaques can undergo sudden endothelial erosion. This triggers a localized thrombosis (clot) that blocks the artery, causing a heart attack without any pre-existing visible narrowing.

Accounts for approximately **5–15%** of all acute myocardial infarctions.

ApoB remains a strong independent predictor of this risk, even with zero visible obstructive disease.

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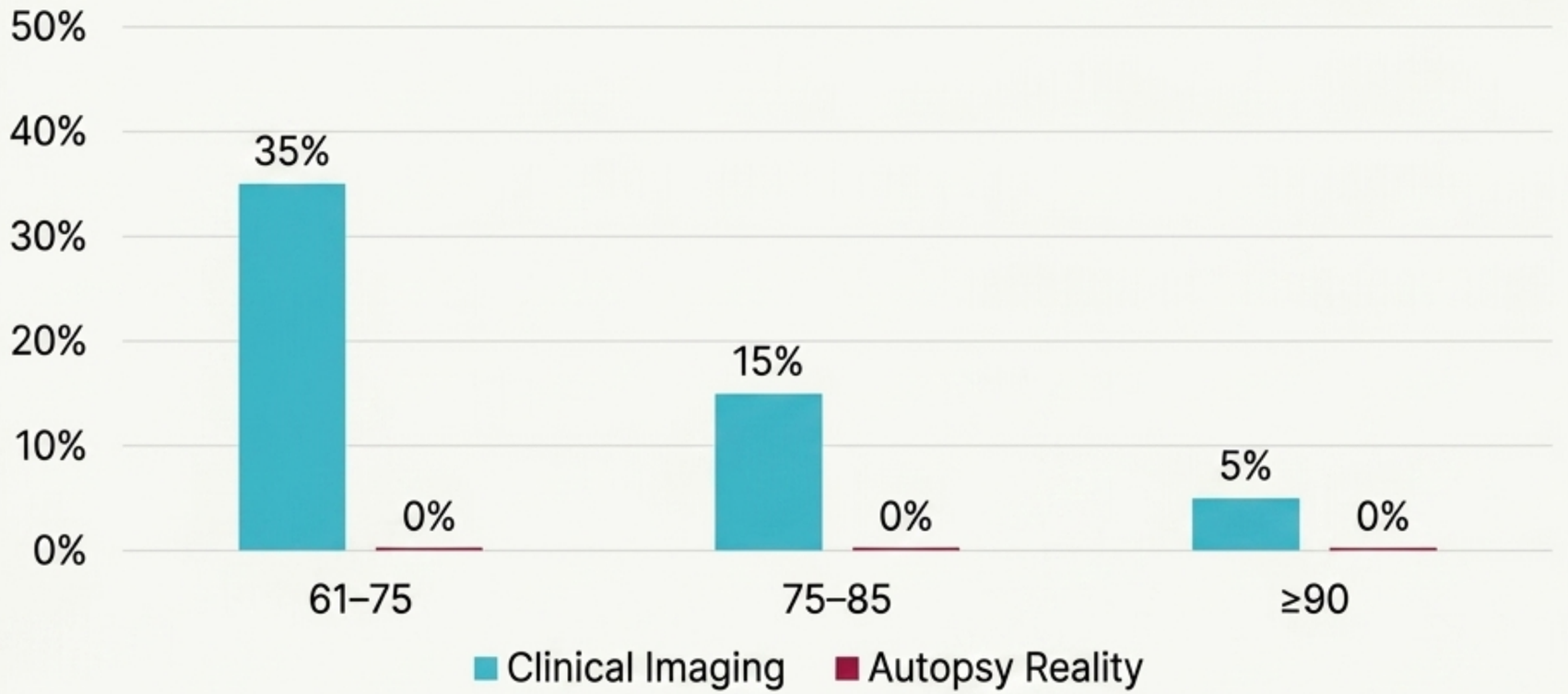
The gap between clinical scanning and physical reality across a lifespan



Clinical imaging reports 28%–40% have a CAC score of zero.

Autopsies confirm true pristine arteries are ~0%.

Clinical imaging reports ~5% have a CAC score of zero.

Autopsies confirm true pristine arteries are ~0%.

A subset of the oldest old exhibit a **cholesterol paradox** where higher LDL-C implies longer survival—likely reflecting age-dependent immune functions and survival bias, not an absence of atherosclerosis.

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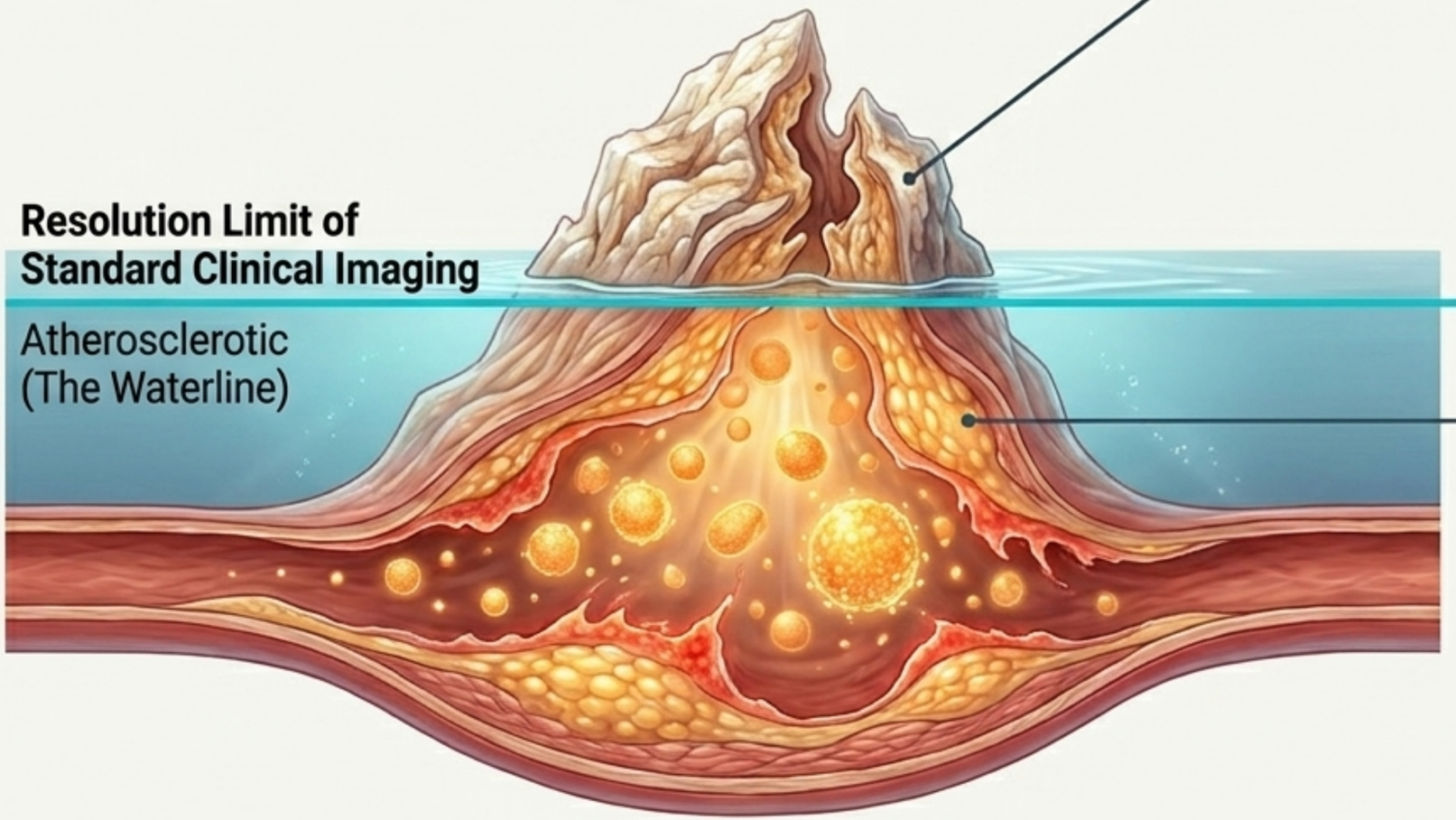
The atherosclerotic iceberg: Standard imaging only sees the surface



The Illusion: Late-stage, heavily calcified, inward-bulging obstructions.

- Obstructive Plaque (>40%)
- High LDL-C Mass
- Coronary Calcium (Ash)

The Reality: Decades of silent ApoB particle penetration, hidden by outward arterial remodeling.

- Non-calcified Plaque (The Glagov Effect)
- High ApoB Particle Burden
- Endothelial Erosion / MINOCA risk (The Spark)

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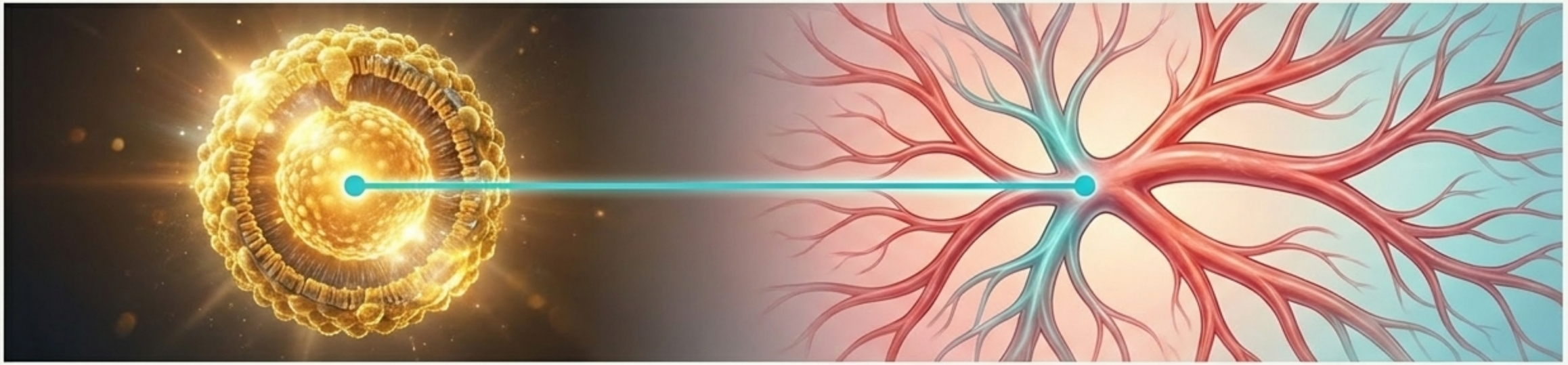
Reversing heart disease requires treating the depths, not just the surface



Waiting for standard imaging to reveal an obstructive stenosis is waiting for end-stage disease.

The absence of calcium (CAC=0) or visible narrowing does not equate to the absence of danger. The risk is hidden in the walls.

True prevention demands quantifying the exact particle burden (ApoB) and utilizing high-resolution, volumetric analysis (AI-QCT) to map the microsc.

Curing Heart Disease, LLC — Utilizing the best available science to reverse the timeline of atherosclerosis.