ISSN: 3060-4737 Volume 3, Issue 10 IF(Impact Factor) 10.98 / 2026

ATHEROSCLEROSIS: FROM FATTY STREAK TO PLAQUE RUPTURE

Aryan kumar , Medical Student, Samarkand state medical university, Uzbekistan

Ayush raj , Medical Student, Samarkand state medical university, Uzbekistan

Vaishnavi banavath , Medical Student, Samarkand state medical university, Uzbekistan

Dikshant bhanvra, Medical Student, Samarkand state medical university, Uzbekistan

Abstract

Atherosclerosis is a chronic inflammatory disease of medium and large arteries in which lipids, immune cells and connective tissue accumulate within the arterial wall. Its development is gradual, beginning with endothelial dysfunction and retention of cholesterol-containing lipoproteins, followed by the formation of fatty streaks and more complex fibrous plaques. Some plaques remain relatively stable, while others develop a large lipid-rich necrotic core and a weakened fibrous cap. Plaque rupture or surface erosion may expose thrombogenic material to circulating blood, promoting clot formation and potentially causing myocardial infarction or ischemic stroke. This narrative review outlines the biological stages of atherosclerosis, key risk factors, mechanisms of plaque instability, clinical consequences and preventive approaches. It highlights the interaction between lipid metabolism, inflammation, vascular biology and lifestyle-related risks. Early risk assessment, smoking cessation, healthy dietary patterns, physical activity, control of blood pressure and diabetes, and lipid-lowering treatment when indicated can reduce cardiovascular risk. Understanding the pathway from fatty streak to thrombosis helps explain why atherosclerosis can remain silent for years before presenting as an acute medical emergency.

Keywords: Atherosclerosis; fatty streak; endothelial dysfunction; LDL cholesterol; foam cells; inflammation; fibrous cap; plaque rupture; thrombosis; cardiovascular disease.

1. Introduction

Atherosclerosis is one of the major pathological processes underlying coronary artery disease, ischemic stroke and peripheral arterial disease. It is often described as a gradual build-up of fatty material in arteries, but this description captures only part of the story. An atherosclerotic plaque is a living, changing structure containing cholesterol, inflammatory cells, smooth muscle cells, connective tissue and, in

ISSN: 3060-4737 Volume 3, Issue 10 IF(Impact Factor) 10.98 / 2026

advanced lesions, areas of cell debris and calcification [1–3]. The condition may develop over decades without obvious symptoms. Problems arise when a plaque significantly restricts blood flow or when its surface is disrupted and a blood clot forms.

The phrase "from fatty streak to plaque rupture" describes a useful sequence, although real disease is not always linear. Some early lesions do not progress substantially, and advanced plaques differ in their composition and stability. The risk of a clinical event depends not only on how much a plaque narrows an artery, but also on its biological features and the response of the blood-clotting system. This article reviews the major stages of atherosclerosis and explains how prevention can interrupt the process.

2. Initiation: endothelial dysfunction and lipid retention

The inner surface of an artery is lined by endothelial cells. Under healthy conditions, these cells help regulate vessel tone, blood clotting and the movement of immune cells. At arterial bends and branch points, disturbed blood flow can make the endothelium more vulnerable to dysfunction. High levels of low-density lipoprotein (LDL) cholesterol, smoking, hypertension, diabetes and other stresses can further impair its protective functions [2,3].

When apoB-containing lipoproteins such as LDL enter and become retained in the inner arterial layer, they may undergo chemical modification. The altered particles activate local inflammatory signalling. Endothelial cells express adhesion molecules and chemical signals that attract circulating monocytes. Once these immune cells enter the vessel wall, they can develop into macrophages and take up modified lipids. Lipid-loaded macrophages are called foam cells, and their accumulation is a defining feature of early atherosclerotic lesions [2,4].

3. Fatty streak formation

A fatty streak is an early lesion formed largely by collections of foam cells within the intima, the innermost layer of the artery. It may appear as a yellowish area when examined directly. Fatty streaks can occur from a young age and do not necessarily mean that a person will develop a dangerous plaque at that exact site. However, they show that lipid retention and an inflammatory response have begun in the arterial wall [3,4].

At this stage, the lesion is often relatively flat and may not significantly obstruct blood flow. The process can nevertheless continue if exposure to atherogenic lipoproteins and other risk factors persists. Some lesions may remain limited, while others develop extracellular lipid pools and recruit smooth muscle cells. This variation is important:

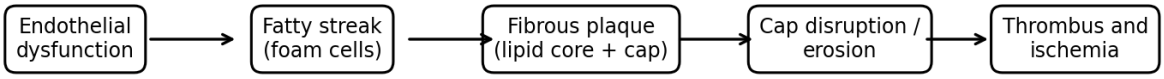
ISSN: 3060-4737 Volume 3, Issue 10 IF(Impact Factor) 10.98 / 2026

atherosclerosis is a dynamic process shaped by inherited susceptibility, lifetime exposures and local conditions within the vessel.

4. From fatty streak to fibrous plaque

As a lesion advances, smooth muscle cells migrate from the middle layer of the artery into the intima. They proliferate and produce extracellular matrix, including collagen, which contributes to a fibrous cap over the lipid-rich region. Meanwhile, ongoing inflammation and cell death can create a necrotic core containing cholesterol, cellular debris and other material. Advanced plaques may also contain calcium deposits, small blood vessels and evidence of previous bleeding [2–4].

The fibrous cap matters because it separates the thrombogenic contents of the plaque from circulating blood. A thick, collagen-rich cap with sufficient smooth muscle cells is generally more stable. A plaque with a large necrotic core, intense inflammation and a thin cap may be more vulnerable to disruption. Still, plaque behaviour is complex; not every plaque with a high-risk feature will rupture, and some acute coronary events occur through superficial erosion rather than classic cap rupture.



Simplified pathway; progression is variable and not every plaque ruptures.

Figure 1. Simplified progression from endothelial dysfunction to thrombosis. The stages are conceptual and do not imply that every lesion follows the same course.

Stage	Main biological changes	Possible significance
Endothelial dysfunction	Reduced protective signalling and increased permeability/adhesion signalling.	Creates conditions that favour lipoprotein retention and inflammation.

ISSN: 3060-4737 Volume 3, Issue 10 IF(Impact Factor) 10.98 / 2026

Fatty streak	Macrophages take up modified lipids and become foam cells.	Early lesion; may remain limited or progress.
Fibroatheroma	Lipid-rich necrotic core develops beneath a fibrous cap.	May narrow the artery and can develop features of instability.
Plaque rupture or erosion	Plaque surface barrier is disrupted or endothelial lining is lost.	Blood contacts thrombogenic material; clot formation may begin.
Thrombosis	Platelets and clotting proteins form a thrombus at the lesion.	Can suddenly reduce or block blood flow, causing ischemia.

5. Plaque rupture, thrombosis and clinical events

Plaque rupture occurs when the fibrous cap tears, allowing blood to contact highly thrombogenic material within the plaque. Platelets adhere and activate, and the coagulation system generates fibrin, producing a thrombus. If the clot rapidly blocks a coronary artery, the result may be an acute myocardial infarction. In a cerebral artery or an artery supplying the brain, a thrombotic event can contribute to ischemic stroke. Plaque-related disease in leg arteries can cause pain during walking, poor wound healing or, in severe cases, tissue loss [1,2].

Plaque erosion is another route to thrombosis. In erosion, the endothelial surface is lost or damaged without the same type of fibrous-cap tear. Both rupture and erosion can produce acute events, so plaque instability should not be reduced to one mechanism alone [5]. A plaque may also gradually narrow an artery and limit blood flow during increased demand, causing exertional chest discomfort or leg pain even without sudden rupture.

ISSN: 3060-4737 Volume 3, Issue 10 IF(Impact Factor) 10.98 / 2026

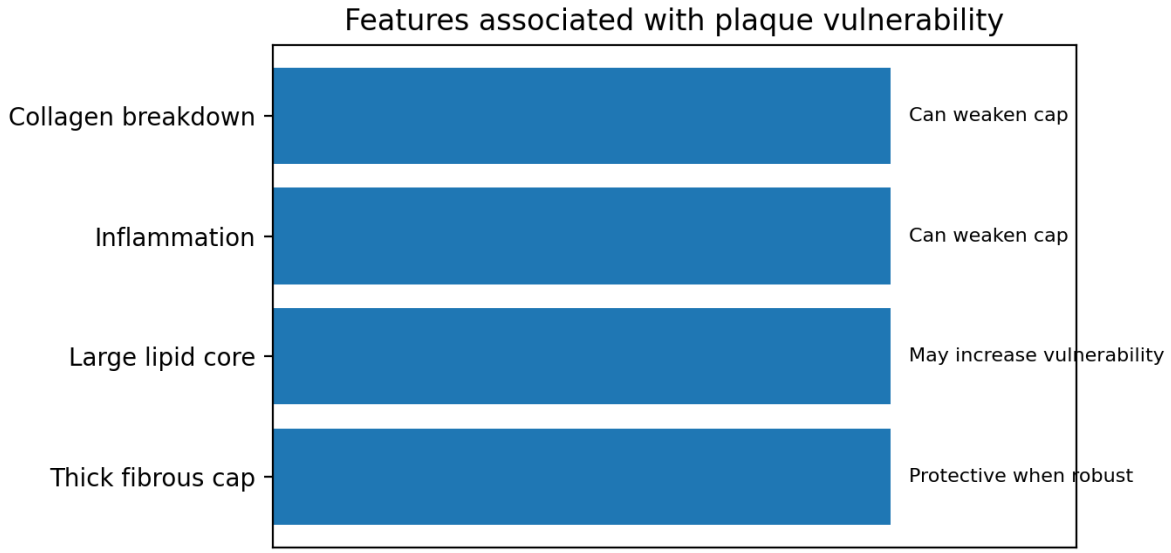


Figure 2. Qualitative illustration of factors relevant to plaque stability. This is explanatory, not a measured risk score.

6. Risk factors

Atherosclerosis develops through interactions among modifiable and non-modifiable risk factors. Age and family history influence baseline risk, while high LDL cholesterol, high blood pressure, diabetes, tobacco exposure, physical inactivity and unhealthy dietary patterns can increase risk. Chronic kidney disease and some inflammatory conditions are also associated with cardiovascular risk. Risk factors often cluster: for example, insulin resistance, elevated blood pressure and abnormal blood lipids may occur together and amplify overall risk [1,6].

LDL cholesterol is particularly important because apoB-containing lipoproteins can enter and become retained in the arterial wall. Lowering LDL exposure over time reduces the opportunity for plaque development and progression. Smoking promotes endothelial dysfunction and inflammation, while high blood pressure increases mechanical stress on arteries. Diabetes can affect lipoproteins, inflammation and vascular function. The contribution of each factor varies between individuals, so prevention is most effective when tailored to overall cardiovascular risk rather than a single measurement.

Risk factor	**Relationship to atherosclerosis**	**Prevention or management approach**
High LDL cholesterol	Increases the pool of atherogenic lipoproteins	Heart-healthy diet; lipid testing; medication when indicated.

ISSN: 3060-4737 Volume 3, Issue 10 IF(Impact Factor) 10.98 / 2026

	that may be retained in the arterial wall.	
Tobacco use	Damages vascular function and promotes inflammatory and thrombotic effects.	Avoid smoking and second-hand smoke; seek cessation support.
Hypertension	Adds vascular stress and increases cardiovascular event risk.	Measure blood pressure and follow a clinician-guided plan.
Diabetes	Contributes to vascular injury and often coexists with other risk factors.	Glucose management and comprehensive risk reduction.
Physical inactivity and diet quality	May influence weight, blood pressure, lipid levels and metabolic health.	Regular activity and a balanced dietary pattern.
Age and family history	Non-modifiable factors that affect baseline risk.	Use them to guide earlier assessment and prevention.

7. Prevention and management

Prevention begins before symptoms appear. A dietary pattern rich in vegetables, fruit, legumes, whole grains, nuts and suitable sources of unsaturated fats can support cardiovascular health. Limiting trans fats, excess saturated fat, highly processed foods and excess sodium can help improve risk factors. Regular physical activity, maintaining a healthy weight, sufficient sleep and avoiding tobacco also contribute to risk reduction. These measures are valuable even when medication is required; they complement rather than replace treatment [1,6].

Clinical prevention may include measurement of blood pressure, lipid profile and blood glucose, together with assessment of family history and other conditions. Statins and other lipid-lowering medicines may be recommended according to the person's cardiovascular risk and clinical history. Antiplatelet medicines are appropriate for some patients but are not recommended for everyone because they can cause bleeding. Treatment decisions should be made with a qualified healthcare professional, who can weigh expected benefit against risks and review progress over time.

Urgent symptoms should never be ignored. New chest pressure, breathlessness, cold sweating, sudden weakness or numbness on one side, facial drooping, difficulty speaking or sudden loss of vision may indicate a heart attack or stroke. Emergency

ISSN: 3060-4737 Volume 3, Issue 10 IF(Impact Factor) 10.98 / 2026

medical services should be contacted immediately. Atherosclerosis can be silent for years, which is why prevention and early management remain important even when a person feels well.

8. Discussion

The journey from fatty streak to plaque rupture demonstrates that atherosclerosis is both a lipid disorder and a chronic inflammatory disease of the arterial wall. Lipid retention initiates the process, immune activity sustains it, and the balance between tissue repair and breakdown helps determine whether a plaque remains stable. A large plaque is not automatically the most dangerous; composition, cap integrity, inflammation and the local clotting response also matter [2,5].

Public-health strategies should therefore focus on reducing risk across the lifespan. Affordable access to healthy food, opportunities for physical activity, tobacco control, routine screening and effective treatment of hypertension, diabetes and high cholesterol can reduce the burden of cardiovascular disease. Explaining the underlying biology helps people understand why prevention should start early and continue consistently, rather than waiting for symptoms.

Conclusion

Atherosclerosis begins with changes in the arterial lining and retention of cholesterol-containing lipoproteins. The resulting inflammatory response creates fatty streaks, which may develop into complex plaques containing a lipid-rich necrotic core and a fibrous cap. When a plaque ruptures or undergoes erosion, a blood clot may form and abruptly restrict blood flow, leading to serious outcomes such as myocardial infarction or ischemic stroke. Progression is variable, and not every fatty streak becomes a dangerous lesion. Reducing LDL cholesterol exposure, avoiding tobacco, controlling blood pressure and diabetes, maintaining physical activity and following a balanced diet can lower cardiovascular risk. Timely assessment and evidence-based treatment are essential for people at increased risk. Understanding the process from early lesion to thrombosis reinforces the importance of prevention long before symptoms develop.

References

1. American Heart Association. What Is Atherosclerosis? Reviewed March 13, 2026. https://www.heart.org/en/health-topics/cholesterol/about-cholesterol/atherosclerosis

2. Gisterå A, Hansson GK. The immunology of atherosclerosis. Nature Reviews Nephrology. 2017;13:368–380. https://doi.org/10.1038/nrneph.2017.51

ISSN: 3060-4737 Volume 3, Issue 10 IF(Impact Factor) 10.98 / 2026

3. Bennett MR, Sinha S, Owens GK. Vascular smooth muscle cells in atherosclerosis. Circulation Research. 2016;118(4):692–702. https://doi.org/10.1161/CIRCRESAHA.115.306361

4. The Role of Lipids and Lipoproteins in Atherosclerosis. Endotext. National Center for Biotechnology Information. https://www.ncbi.nlm.nih.gov/books/NBK343489/

5. Libby P. Inflammation in atherosclerosis. Nature. 2002;420:868–874. https://doi.org/10.1038/nature01323

6. Arnett DK, Blumenthal RS, Albert MA, et al. 2019 ACC/AHA Guideline on the Primary Prevention of Cardiovascular Disease. Circulation. 2019;140:e596–e646. https://doi.org/10.1161/CIR.0000000000000678

7. Rosenfeld ME. An overview of the evolution of the atherosclerotic plaque: from fatty streak to plaque rupture and thrombosis. Zeitschrift für Kardiologie. 2000;89(Suppl 7):VII2–VII6. https://pubmed.ncbi.nlm.nih.gov/11098552/