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Opinion Article

Cellular Mechanisms of Foam Cell Formation in Iliac Artery Atherosclerosis

Isabella Rossi*

Department of Cardiology, University of Milan Medical School, Milan, Italy

DESCRIPTION

Foam cell formation and plaque development are central events in the pathogenesis of atherosclerosis, particularly in hyperlipidemic iliac arteries. Hyperlipidemia, characterized by elevated levels of Low-Density Lipoprotein (LDL) cholesterol, triglycerides, or reduced High-Density Lipoprotein (HDL) cholesterol, is a major risk factor for vascular disease. The iliac arteries, responsible for supplying blood to the pelvis and lower limbs, are particularly susceptible to atherosclerotic changes due to their large size, high blood flow and exposure to systemic risk factors such as hypertension, smoking and diabetes. The pathological cascade begins when excess LDL cholesterol circulates in the bloodstream and penetrates the endothelial lining of the iliac arteries. Within the arterial wall, LDL particles undergo oxidative modification, transforming into Oxidized Ldl (OXLDL), which is highly atherogenic. OxLDL triggers an inflammatory response by stimulating endothelial cells to express adhesion molecules and chemokines, attracting circulating monocytes into the arterial intima. Once within the intima, these monocytes differentiate into macrophages, which attempt to engulf oxLDL through scavenger receptor-mediated endocytosis. However, macrophages cannot efficiently process the excess lipids, leading to the formation of lipid-laden foam cells. Foam cells are the hallmark of early atherosclerotic lesions, also known as fatty streaks and their accumulation within the arterial wall marks the beginning of plaque development.

As foam cells accumulate, they release pro-inflammatory cytokines and growth factors that perpetuate local inflammation and recruit additional immune cells. This chronic inflammatory state promotes the proliferation of smooth muscle cells from the media into the intima. These smooth muscle cells synthesize extracellular matrix components such as collagen and elastin, forming a fibrous cap over the lipid core. The interplay between foam cells, inflammatory mediators and smooth muscle cells leads to the progressive thickening of the arterial wall and narrowing of the lumen, a process known as stenosis. In the iliac arteries, this narrowing can significantly reduce blood flow to the lower extremities, resulting in symptoms such as intermittent claudication, leg fatigue and in severe cases, critical limb ischemia. Triglyceride-rich lipoproteins and remnants also contribute to foam cell formation by promoting endothelial dysfunction, oxidative stress and inflammatory signaling, further accelerating plaque development. Conversely, HDL cholesterol plays a protective role by facilitating reverse cholesterol transport, removing excess cholesterol from foam cells and inhibiting inflammation, highlighting the importance of a balanced lipid profile in maintaining vascular health.

The progression of foam cell accumulation and plaque formation in the iliac arteries is influenced not only by lipid levels but also by the duration of hyperlipidemia, genetic predisposition and coexisting cardiovascular risk factors. Patients with long-standing, untreated hyperlipidemia are at higher risk of developing complex plaques, which may contain necrotic cores, calcifications and areas of intraplaque hemorrhage. These features increase the vulnerability of plaques to rupture, which can precipitate acute vascular events such as thrombosis or distal embolization, potentially leading to sudden limb ischemia. Clinical studies have shown that foam cell-rich plaques in the iliac arteries are commonly associated with multi-level peripheral arterial disease, reflecting the systemic impact of dyslipidemia on the vasculature. Diagnostic evaluation of plaque burden typically involves imaging techniques such as Doppler ultrasound, computed tomography angiography, or magnetic resonance angiography, which allow clinicians to visualize the degree of stenosis and assess plaque composition.

Management of foam cell accumulation and plaque progression in hyperlipidemic patients focuses primarily on controlling lipid levels and reducing systemic inflammation. Lifestyle modifications, including a diet low in saturated fats and cholesterol, regular physical activity, smoking cessation and weight management, are essential strategies for improving lipid profiles and vascular health. Pharmacological therapies, particularly statins, are highly effective in lowering LDL cholesterol, stabilizing atherosclerotic plaques, reducing inflammation and promoting plaque regression in some cases. Emerging treatments targeting inflammatory pathways and triglyceride-rich lipoproteins also show promise in mitigating foam cell formation and slowing plaque development. In severe cases of iliac artery stenosis, endovascular interventions such as

Correspondence to: Isabella Rossi, Department of Cardiology, University of Milan Medical School, Milan, Italy, E-mail: isabella.rossi@unimi.it

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angioplasty or stenting may be necessary to restore blood flow and prevent complications.

CONCLUSION

In conclusion, foam cell formation and plaque development are central mechanisms by which hyperlipidemia contributes to iliac artery disease. The accumulation of lipid-laden macrophages, chronic inflammation and smooth muscle cell proliferation collectively drive atherosclerotic plaque growth, leading to arterial stenosis and impaired blood flow. Understanding these processes emphasizes the critical importance of early lipid management, lifestyle modification and targeted pharmacological therapy in preventing plaque progression, preserving vascular function and reducing the risk of complications in patients with hyperlipidemic iliac arteries.