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Perspective

Development and Progression of Iliac Artery Atherosclerosis Due to Hyperlipidemia

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DESCRIPTION

Atherosclerosis is a progressive vascular disease characterized by the accumulation of lipids, inflammatory cells and fibrous elements within the arterial wall, leading to vessel narrowing and impaired blood flow. One of the major systemic risk factors contributing to atherosclerosis is hyperlipidemia, a condition marked by elevated levels of cholesterol and triglycerides in the blood. While coronary and carotid arteries are commonly discussed in relation to atherosclerosis, the iliac arteries are also significantly affected, particularly as part of peripheral arterial disease. The iliac arteries, which supply blood to the pelvis and lower limbs, are vulnerable to lipid-induced vascular damage due to their size, flow dynamics and exposure to systemic metabolic abnormalities. Understanding how hyperlipidemia contributes to atherosclerotic changes in the iliac artery is essential for early detection, prevention and management of lower limb ischemic conditions.

Hyperlipidemia plays a central role in the initiation of atherosclerosis by increasing circulating Low-Density Lipoprotein (LDL) cholesterol, which infiltrates the endothelial lining of arteries. In the iliac arteries, persistent exposure to elevated LDL leads to endothelial dysfunction, an early and critical step in atherogenesis. The damaged endothelium becomes more permeable to lipoproteins and expresses adhesion molecules that attract circulating monocytes. These monocytes migrate into the arterial wall and differentiate into macrophages, which engulf oxidized LDL particles to form foam cells. The accumulation of foam cells creates fatty streaks, the earliest visible lesions of atherosclerosis, which can be observed in the iliac arteries even before clinical symptoms appear.

As hyperlipidemia persists, the inflammatory process within the iliac artery intensifies. Smooth muscle cells from the arterial media migrate into the intima and proliferate, contributing to the formation of a fibrous cap over the lipid-rich core. This process results in the development of atherosclerotic plaques that progressively thicken the arterial wall and narrow the lumen. In the iliac arteries, such narrowing can significantly reduce blood flow to the lower extremities, particularly during physical activity when oxygen demand increases. Elevated triglycerides and reduced high-density lipoprotein (HDL) cholesterol further exacerbate plaque progression by promoting inflammation and reducing protective lipid transport mechanisms.

Atherosclerotic changes in the iliac artery are not only structural but also functional. Hyperlipidemia impairs nitric oxide production, a molecule essential for vasodilation and vascular health. Reduced nitric oxide availability leads to increased arterial stiffness and diminished adaptive responses to changes in blood flow. Over time, plaques within the iliac artery may become unstable, especially in individuals with poorly controlled lipid levels. Plaque rupture can trigger thrombosis, potentially resulting in acute arterial occlusion and severe ischemia of the lower limbs. Although complete occlusion is less common than gradual stenosis, both conditions significantly affect mobility and quality of life.

Clinically, atherosclerosis of the iliac artery associated with hyperlipidemia often presents as part of peripheral arterial disease. Patients may experience symptoms such as intermittent claudication, characterized by pain or cramping in the hips, thighs, or calves during walking that resolves with rest. In advanced cases, chronic ischemia can lead to rest pain, non-healing ulcers, or tissue damage. However, many individuals remain asymptomatic in the early stages, making hyperlipidemia-driven iliac artery disease underdiagnosed. Imaging studies such as Doppler ultrasound, computed tomography angiography and magnetic resonance angiography are commonly used to identify atherosclerotic changes and assess the severity of arterial involvement.

The management of atherosclerotic changes in the iliac artery focuses heavily on controlling hyperlipidemia to slow disease progression and reduce complications. Lifestyle modifications, including a balanced diet low in saturated fats, regular physical activity and weight management, are foundational strategies. Pharmacological therapy, particularly statins, plays a major role by lowering LDL cholesterol, stabilizing plaques and reducing vascular inflammation. Effective lipid control has been shown to improve endothelial function and decrease the risk of plaque

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progression in peripheral arteries, including the iliac arteries. In severe cases where blood flow is significantly compromised, interventional procedures such as angioplasty or stenting may be required.

CONCLUSION

In conclusion, hyperlipidemia is a major contributor to atherosclerotic changes in the iliac artery through its effects on endothelial dysfunction, lipid accumulation, inflammation and plaque formation. These changes can lead to significant impairment of blood flow to the lower extremities and contribute to the development of peripheral arterial disease. Early identification and management of hyperlipidemia are essential to prevent or delay atherosclerosis in the iliac arteries. By addressing lipid abnormalities and associated risk factors, it is possible to reduce disease burden, improve vascular health and enhance long-term outcomes for affected individuals.