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ATHEROSCLEROTIC PLAQUES IN HUMAN CORONARY ARTERIES

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Abstract. Atherosclerosis, including coronary artery disease (CAD), is one of the most prevalent cardiovascular disorders and is primarily associated with lipid deposition and chronic inflammation within the arterial walls. The progressive development of this condition can lead to serious cardiovascular events, particularly myocardial infarction (MI) and stroke. Although atherosclerosis develops gradually over many years and is more frequently diagnosed in older populations, it continues to represent a major cause of mortality worldwide, despite a reduction in its incidence in certain countries[1-2].The pathological process is characterized by the progressive accumulation and modification of lipids, inflammatory cells, vascular smooth muscle cells, and cellular debris within the arterial intima, beneath the endothelial lining. As atherosclerotic plaques enlarge, they progressively narrow the vascular lumen, potentially reducing blood flow by more than 50%. This impaired circulation may manifest clinically as angina pectoris, especially during physical exertion or emotional stress. In advanced stages, atherosclerotic plaques may become vulnerable and rupture, particularly when they contain substantial lipid deposits and inflammatory components[3]. Plaque rupture in coronary arteries can trigger thrombus formation, resulting in partial or complete arterial occlusion and subsequent myocardial infarction. In some cases, thromboembolic material may enter the systemic circulation and reach cerebral vessels, potentially causing an ischemic stroke.

Key words: coronary artery disease, cardiovascular disease, lipid accumulation, endothelial dysfunction, vascular inflammation

Aim. This article reviews the progression and pathological development of aortic and coronary atherosclerosis in young individuals, drawing on findings from the Pathobiological Determinants of Atherosclerosis in Youth (PDAY) Study. This multicenter investigation examined atherosclerotic changes in individuals aged 15–34 years. Particular attention was given to the age group of 25–45 years, during which fatty streaks are commonly observed and the initial formation of fibrous atherosclerotic plaques becomes increasingly evident.

Methods. The study included individuals between 25 and 45 years of age who died from external causes. Initially, 3,210 cases were collected; however, 334 were excluded because they did not fulfill the established inclusion criteria. Consequently, 2,876 cases were included in the final analysis. Thoracic aortic specimens were obtained from 2,856 individuals, abdominal aortic samples from 2,823, and right coronary artery specimens from 2,788.In addition, biological materials, including blood, liver, kidney tissue, and standardized arterial wall specimens, were collected. The sampling procedure also included perfusion-fixed segments of the left anterior descending coronary artery. These materials were analyzed to examine the relationship between

atherosclerotic changes and established risk factors for coronary heart disease in adults. Furthermore, histopathological investigations were conducted to characterize the structural features of aortic and coronary atherosclerotic lesions, with particular emphasis on their cellular composition, fibrous tissue development, and lipid accumulation.

Results. The findings demonstrated that intimal atherosclerotic changes were present in all aortic specimens and in more than 50% of the right coronary artery specimens among individuals in the youngest age category (25–35 years). Both the frequency and severity of these lesions increased progressively with age, reaching higher levels in the oldest group (35–45 years). Fatty streaks were more extensively distributed among Black participants compared with White participants, whereas no significant differences were observed in the prevalence of raised atherosclerotic lesions between these populations[4]. The extent of raised lesions in the aorta was comparable between men and women; however, women demonstrated a lower burden of raised lesions in the right coronary arteries than men. Although the overall prevalence of atherosclerotic lesions was lower in the right coronary arteries than in the aorta, the proportion of advanced raised lesions relative to total lesions was greater in the coronary arteries.The progression of atherosclerosis is associated with continuous lipid accumulation and the proliferation of vascular smooth muscle cells and connective tissue. These processes contribute to the enlargement and structural transformation of fatty streaks into fibrous plaques. One of the most critical complications is plaque rupture, which exposes circulating blood to highly thrombogenic, lipid-rich material and promotes thrombus formation. Subsequent vascular occlusion may result in myocardial infarction or sudden cardiac death[5]. The PDAY Study investigated the progression of early atherosclerotic changes toward advanced fibrous lesions and sought to identify factors associated with this pathological transformation.Previous investigations within the PDAY project examined the influence of several cardiovascular risk factors, including age, sex, ethnicity, serum lipoprotein concentrations, cigarette smoking, hypertension, glycohemoglobin levels, and obesity, on the extent and histological characteristics of atherosclerotic lesions. The accumulated evidence indicated that risk factors associated with clinically apparent coronary heart disease (CHD) are also closely related to the development and severity of atherosclerosis during early life. The present investigation provides an overview of the morphological progression of macroscopically identifiable atherosclerotic lesions in young American individuals and demonstrates that substantial disease progression may occur even during young adulthood[6-7].For morphological assessment, the left half of each aorta and the right coronary artery were transported to a central laboratory in plastic containers. The arterial specimens were stained with Sudan IV to identify lipid-containing areas, followed by radiographic examination. Three independent pathologists, blinded to the origin of the samples and clinical information, evaluated the stained arterial tissues. Using standardized procedures established by the International Atherosclerosis Project, they estimated the percentage of the intimal surface affected by fatty streaks, fibrous plaques, complicated lesions, and calcification. The combined surface area occupied by fibrous plaques, complicated lesions, and calcified lesions was classified as the extent of raised atherosclerotic lesions. Final lesion scores were determined by calculating the mean of the three independent assessments[9]. Lesion prevalence was evaluated according to the presence of detectable changes and, separately, involvement of at least 5% of the arterial surface. Calcified lesions were assessed using radiographic findings.The anatomical distribution of atherosclerotic lesions varied considerably across different regions of the aorta. In the thoracic aorta, fatty streaks were most frequently identified on the dorsal surface, where their prevalence exceeded 50%, whereas the ventral surface demonstrated considerably fewer lesions, affecting less than 10% of the area. The highest concentration of fatty streaks was observed in the regions located between

adjacent intercostal artery openings. In contrast, advanced raised lesions were uncommon in the thoracic aorta, even among older participants, occupying less than 4% of the arterial surface in the reported age group.In the abdominal aorta, fatty streaks were also more frequently detected on the dorsal than on the ventral surface, although the distribution of lesion-prone and lesion-resistant regions was less clearly differentiated than in the thoracic segment. Areas with a higher prevalence of atherosclerotic changes were mainly located between adjacent lumbar artery openings and along the blood-flow pathways leading to the celiac, superior mesenteric, renal, and inferior mesenteric arteries[10]. Conversely, regions situated downstream from arterial flow dividers demonstrated relatively fewer lesions. The left dorsal region of the abdominal aorta, extending from the inferior mesenteric artery opening toward the aortic bifurcation, showed the greatest susceptibility to the development of advanced raised atherosclerotic lesions.

Conclusion. The findings of the PDAY Study demonstrate that atherosclerosis is a progressive pathological process that begins during early adulthood and becomes increasingly pronounced with advancing age. Early atherosclerotic changes, particularly fatty streak formation, were frequently observed in the aorta and coronary arteries, while advanced fibrous and raised lesions showed differences in their prevalence according to age, sex, and anatomical location. The distribution of lesions was not uniform throughout the arterial system, suggesting an important contribution of local vascular structural and hemodynamic factors to atherosclerotic development.The study also highlights the close relationship between conventional cardiovascular risk factors and the progression of atherosclerotic lesions in young individuals. Continuous lipid accumulation, inflammatory processes, and vascular smooth muscle cell proliferation contribute to plaque development and structural instability. Plaque rupture and subsequent thrombus formation represent critical mechanisms leading to acute cardiovascular complications, including myocardial infarction and sudden cardiac death.Overall, these findings emphasize the importance of early identification of cardiovascular risk factors, preventive interventions, and regular assessment of vascular health in young adults. Understanding the initial stages of atherosclerosis may contribute to improved strategies for preventing advanced coronary artery disease and reducing cardiovascular mortality.

References

1. Ajoolabady A, Pratico D, Lin L, Mantzoros CS, Bahijri S, Tuomilehto J, et al. Inflammation in atherosclerosis: pathophysiology and mechanisms. Cell Death Dis. 2024;15:817. doi: 10.1038/s41419-024-07166-8
2. Bentzon JF, Otsuka F, Virmani R, Falk E. Mechanisms of plaque formation and rupture. Circ Res. 2014;114(12):1852–1866.
3. Borén J, Chapman MJ, Krauss RM, Packard CJ, Bentzon JF, Binder CJ, et al. Low-density lipoproteins cause atherosclerotic cardiovascular disease: pathophysiological, genetic, and therapeutic insights. Eur Heart J. 2020;41(24):2313–2330.
4. Gimbrone MA Jr, García-Cardeña G. Endothelial cell dysfunction and the pathobiology of atherosclerosis. Circ Res. 2016;118(4):620–636.
5. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. N Engl J Med. 2005;352:1685–1695.
6. Libby P. Inflammation and the pathogenesis of atherosclerosis. Vasc Pharmacol. 2024;157:107255. doi: 10.1016/j.vph.2023.107255.
7. Libby P. Inflammation in atherosclerosis. Nature. 2002;420:868–874.

8. Libby P. The changing landscape of atherosclerosis. Nature. 2021;592:524–533.
9. Luca AC, David SG, David AG, Țarcă V, Pădureț IA, Mîndru DE, et al. Atherosclerosis from newborn to adult—epidemiology, pathological aspects, and risk factors. Life (Basel). 2023;13(10):2056. doi: 10.3390/life13102056.
10. Tabas I, Bornfeldt KE. Intracellular and intercellular aspects of macrophage immunometabolism in atherosclerosis. Circ Res. 2020;126(9):1209–1227.