



**MORPHOLOGICAL AND PATHOPHYSIOLOGICAL FEATURES OF
ATHEROSCLEROTIC LESIONS IN HUMAN CORONARY ARTERIES.**

Assistant **Yuldasheva G.T.**

Andijan State Medical Institute.

Abstract: Atherosclerosis, also known as coronary artery disease (CAD), represents the most prevalent form of cardiovascular disease (CVD)[1]. It is primarily characterized by the accumulation of lipids and chronic inflammation within the walls of large arteries, processes that ultimately give rise to major clinical events such as myocardial infarction (MI) and stroke. This condition develops gradually over decades and is most often observed in older adults. Although its incidence has declined in some regions due to improved prevention and treatment, atherosclerosis continues to be the leading cause of death globally. The development of atherosclerotic plaques involves a progressive buildup and transformation of lipids, inflammatory cells, smooth muscle cells, and necrotic tissue within the intimal layer beneath the endothelial cell lining of the arterial wall[2-4]. As these lesions enlarge, they can narrow the vascular lumen by more than 50%, leading to reduced blood flow and the onset of angina, particularly during physical exertion or emotional stress. Plaques with a high lipid and inflammatory content are prone to instability and rupture, triggering the formation of a thrombus[5]. When such an event occurs in the coronary arteries, it may completely block blood flow and result in a myocardial infarction. If the thrombus travels to the cerebral circulation, it can obstruct blood flow in the brain and cause an ischemic stroke.

Key words: Acute coronary syndrome, atherosclerosis, coronary artery disease, myocardial infarction

Aim. This article summarizes the natural history of aortic and coronary atherosclerosis among adolescents from the Pathobiological Determinants of Atherosclerosis in Youth (PDAY) Study, which was a multi-institutional study of atherosclerosis in 15- to 34-year-old, The PDAY Study focused on 35- to 55-year-old subjects because fatty streaks are prevalent and fibrous plaques begin to appear in this age group.

Methods. Study subjects were persons aged 35 through 55 years who had died due to external causes. Of the 210 cases collected, 34 were excluded because they did not meet the study criteria. Of the 76 cases included in this report, thoracic aortas were obtained from 56, abdominal aortas from 23, and right coronary arteries from 18. Blood, liver, kidney tissue, and standardized arterial wall samples, including samples from the perfusion-fixed left anterior descending coronary artery, were also collected in this study. These samples were used to investigate the associations of atherosclerosis with the risk factors for adult CHD, which are reported elsewhere, and for studies of the microscopic features of aortic and coronary lesions including cellular, fibrous, and lipid-containing components.

Results. Intimal lesions appeared in all the aortas and more than half of the right coronary arteries of the youngest age group (35-45 years) and increased in prevalence and extent with age through the oldest age group (45-55 years). Fatty streaks were more extensive in black subjects than in white subjects, but raised lesions did not differ between blacks and whites. Raised lesions



in the aortas of women and men were similar, but raised lesions in the right coronary arteries of women were less than those of men. The prevalence of total lesions was lower in the right coronary artery than in the aorta, but the proportion of raised lesions among total lesions was higher in the right coronary artery than in the aorta.

Due to continued lipid deposition and proliferation of smooth muscle and connective tissue, fatty streaks and fibrous plaques increase in size and extent and some undergo qualitative changes. The most serious change is rupture, which exposes the blood to lipid-rich thrombogenic material and precipitates an occlusive thrombus, which in turn leads to myocardial infarction or sudden cardiac death. The PDAY Study encompassed the transition from innocuous fatty streaks to clinically significant fibrous plaques and attempted to determine the conditions associated with this process.

In the thoracic aorta, the highest prevalence of fatty streaks occurred in the dorsal surface (>50%) while the ventral surface had few fatty streaks (<10%). The region of highest prevalence for fatty streaks was midway between successive pairs of intercostal ostia. The thoracic aorta was virtually spared of raised lesions even in the oldest age group (30-34 years: <4% raised lesions). In the abdominal aorta, the frequency of fatty streaks was greater in the dorsal than in the ventral area, but lesion-prone and lesion-resistant regions were not as sharply defined as in the thoracic aorta. Regions of high prevalence occurred between pairs of lumbar ostia and in flow tracts to the celiac, superior mesenteric, renal, and inferior mesenteric arteries, while regions distal to the flow dividers of these ostia were spared. A region on the left dorsal surface of the abdominal aorta, originating at the level of the inferior mesenteric ostium and extending distally to the bifurcation, was the most prone to raised lesions.

Conclusion. Atherosclerosis, the underlying cause of acute coronary syndrome (ACS), initiates a continuum of cardiovascular pathology that extends far beyond the acute event. Despite remarkable improvements in reperfusion therapy and pharmacological management, many patients progress to long-term complications such as heart failure, arrhythmias, and recurrent ischemic episodes — the so-called “successors” of ACS. These outcomes are driven by ongoing inflammation, endothelial dysfunction, and maladaptive ventricular remodeling that persist after the initial ischemic insult. Future strategies must therefore move beyond acute management to include comprehensive long-term care focused on vascular repair, anti-inflammatory interventions, metabolic modulation, and personalized risk prediction. By addressing the chronic nature of atherosclerosis and its post-ACS sequelae, clinicians can more effectively reduce recurrent events, improve survival, and enhance the quality of life in cardiovascular disease survivors.

References

1. Libby, P., Buring, J. E., Badimon, L., Hansson, G. K., Deanfield, J., Bittencourt, M. S., Tokgözoğlu, L., & Lewis, E. F. (2019). Atherosclerosis. *Nature Reviews Disease Primers*, 5(1), 56. <https://doi.org/10.1038/s41572-019-0106-z>
2. McDonagh, T. A., Metra, M., Adamo, M., Gardner, R. S., Baumbach, A., Böhm, M., et al. (2021). 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart



- failure. *European Heart Journal*, 42(36), 3599–3726.
<https://doi.org/10.1093/eurheartj/ehab368>
3. Packer, M., Anker, S. D., Butler, J., Filippatos, G., Pocock, S. J., Carson, P., et al. (2020). Cardiovascular and renal outcomes with empagliflozin in heart failure. *New England Journal of Medicine*, 383(15), 1413–1424. <https://doi.org/10.1056/nejmoa2022190>
 4. Virmani, R., Burke, A. P., Farb, A., & Kolodgie, F. D. (2020). Pathology of the vulnerable plaque. *Journal of the American College of Cardiology*, 76(14), 1610–1623. <https://doi.org/10.1016/j.jacc.2020.08.013>
 5. Ridker, P. M., Everett, B. M., Thuren, T., macfadyen, J. G., Chang, W. H., Ballantyne, C., et al. (2017). Antiinflammatory therapy with canakinumab for atherosclerotic disease. *New England Journal of Medicine*, 377(12), 1119–1131. <https://doi.org/10.1056/nejmoa1707914>