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SHORT COMMUNICATION

Early-Onset Myocardial Infarction in Young Adults (18-30 Years): A Multisystem Pathophysiological Disorder beyond Premature Atherosclerosis

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Abstract

Myocardial Infarction (MI) in young adults aged 18–30 years is an emerging clinical concern with a steadily increasing global incidence. Although traditionally attributed to premature atherosclerosis, growing evidence suggests that early-onset MI results from a complex interplay of endothelial dysfunction, coronary vasospasm, thrombophilia, genetic susceptibility, metabolic syndrome, and substance exposure. These mechanisms may precipitate acute coronary events even in the absence of advanced atherosclerotic plaque burden. Unlike previous reviews that primarily emphasize premature coronary artery disease, this short communication provides an integrated multisystem pathophysiological perspective on early-onset MI. Understanding these interacting mechanisms is essential for early cardiovascular risk stratification, targeted preventive strategies, and timely intervention beginning in adolescence and early adulthood.

Introduction

Myocardial Infarction (MI) is increasingly being diagnosed in individuals aged 18-30 years, representing a significant shift in cardiovascular epidemiology and global disease burden. Recent data suggest a rising incidence of premature acute coronary syndromes in young adults, particularly in South Asian populations, driven by rapid lifestyle transitions and early exposure to cardiometabolic risk factors [1,2]. Unlike traditional coronary artery disease in older adults, young-onset MI is often multifactorial and may occur even in the absence of advanced atherosclerotic burden [3,4]. This changing pattern reflects increasing prevalence of obesity,

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dyslipidemia, smoking, and psychosocial stress beginning in adolescence and early adulthood [5,6]. Understanding these mechanisms is essential for early prevention, targeted screening, and long-term cardiovascular risk stratification [7].

Etiological spectrum of early-onset myocardial infarction

Premature atherosclerosis: Early atherosclerotic changes begin in childhood and progress silently over decades under the influence of dyslipidemia, obesity, insulin resistance, and sedentary behavior [8,9]. Autopsy studies have demonstrated early fatty streak formation in adolescents, which may progress to fibrous plaques in young adulthood [10]. In younger individuals, plaques are typically lipid-rich, inflamed, and rupture-prone, predisposing to acute thrombosis and sudden coronary occlusion [11].

Endothelial dysfunction: Endothelial dysfunction represents an early and reversible stage of vascular disease and is strongly associated with smoking, vaping, oxidative stress, unhealthy diet, and chronic psychological stress [12]. These factors impair nitric oxide bioavailability, leading to vasoconstriction, platelet activation, and reduced fibrinolytic activity [13]. The resulting pro-thrombotic vascular environment significantly increases susceptibility to acute coronary events even in the absence of significant obstructive coronary disease [14].

Thrombophilia and hypercoagulable states: Inherited and acquired hypercoagulable conditions contribute to early coronary thrombosis in young adults. These include Factor V Leiden mutation, antiphospholipid antibody syndrome, protein C and S deficiencies, elevated homocysteine levels, and systemic inflammatory states [15]. These conditions promote intravascular thrombosis and may lead to complete coronary occlusion without significant underlying atherosclerotic plaque burden [16].

Coronary vasospasm and substance exposure: Coronary vasospasm is an important non-atherosclerotic cause of MI in young individuals. Substances such as cocaine, amphetamines, nicotine, and vaping products induce intense sympathetic stimulation, leading to transient but severe coronary artery constriction [17]. This vasospastic response reduces myocardial oxygen supply while simultaneously increasing demand, thereby precipitating ischemia and infarction even in structurally normal coronary arteries [18].

Genetic and familial disorders: Genetic predisposition plays a critical role in premature coronary artery disease. Familial hypercholesterolemia and other inherited lipid metabolism disorders result in lifelong exposure to elevated LDL cholesterol levels, significantly accelerating atherosclerotic progression [19]. A strong family history of premature cardiovascular disease further increases susceptibility, highlighting the importance of early lipid screening and genetic evaluation in high-risk individuals [20].

Metabolic syndrome: Metabolic syndrome, characterized by central obesity, insulin resistance, hypertension, and dyslipidemia, is increasingly prevalent among adolescents and young adults [5,7]. This cluster of metabolic abnormalities promotes chronic systemic inflammation, endothelial dysfunction, and accelerated vascular aging, thereby increasing long-term cardiovascular risk [6,8].

Pathophysiological mechanism: Early myocardial infarction in young adults results from the interaction of four principal mechanisms:

- Structural vascular changes (early atherosclerosis) [9,10]
- Functional vascular instability (endothelial

dysfunction and vasospasm) [12,17]
• Hemostatic imbalance (thrombophilia and hypercoagulability) [15,16]
• External triggers (stress, smoking, drug exposure) [13,18]

These synergistic pathways culminate in acute coronary artery occlusion and myocardial ischemia, often presenting as sudden cardiac events in otherwise apparently healthy individuals [11,14].

Clinical implications

Early-onset MI often presents atypically with non-classical symptoms, leading to delayed recognition and treatment. Routine cardiovascular screening is not universally implemented in this age group, contributing to underdiagnosis of high-risk individuals [7]. Identification of modifiable risk factors during adolescence, including obesity, smoking, and dyslipidemia, is critical for prevention strategies [5,8].

Conclusion

Early-onset myocardial infarction should no longer be considered solely a consequence of premature atherosclerosis but rather a complex multisystem disorder involving vascular dysfunction, metabolic abnormalities, thrombogenic predisposition, genetic susceptibility, and environmental exposures. Appreciating this integrated pathophysiological framework may facilitate earlier diagnosis, improve risk stratification, and guide more effective preventive strategies in young adults. Future research should focus on identifying novel biomarkers, incorporating genetic and polygenic risk assessment, applying artificial intelligence for personalized cardiovascular risk prediction, and conducting large prospective multicenter studies to optimize prevention and management strategies in this vulnerable population.

Declaration

Ethical approval and consent to participate

Not applicable, as this study is a short communication based on previously published literature and does not involve human participants or animal experiments.

Consent for publication

Not applicable.

Availability of data and materials

All data supporting the findings of this study are available within the cited published literature and PubMed-indexed sources.

Competing interests

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Author contributions

The author solely conceptualized, designed, wrote, and revised the manuscript.

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