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Heart Ischemic Disease in Uzbekistan: Epidemiology, Clinical Aspects, Prevention, and Etiopathogenesis

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Abstract: Coronary heart disease (CHD) remains one of the most pressing issues in modern cardiology and is the leading cause of mortality and disability among the adult population worldwide. According to WHO data, ischemic heart disease leads to 7.4 million deaths annually worldwide (13% of total mortality), while developing countries are seeing a steady increase in the incidence and prevalence of coronary heart disease.

Keywords: coronary heart disease, Uzbekistan, epidemiology, prevention

Introduction

Modern achievements in molecular pathology, genetics, structural cardiology, and the implementation of high-tech diagnostic methods open up new opportunities for understanding the etiopathogenesis of coronary heart disease and developing personalized approaches to its treatment and prevention.

In the Republic of Uzbekistan, the prevalence of cardiovascular diseases among the adult population is 42-58 per 1,000 people (National Registry of Cardiological Diseases of the Ministry of Health of the Republic of Uzbekistan, 2024), while cardiovascular diseases remain responsible for 42-48% of all deaths in the country [1]. At the same time, coronary heart disease in Uzbekistan has a number of characteristic epidemiological and clinical features, due to climatogeographic, ethnodemographic and socio-economic factors, which are insufficiently studied and require special analysis [2].

Despite significant progress in understanding the pathogenesis of coronary heart disease, many issues remain unresolved: the role of regional ethnic factors in the development of coronary heart disease, the characteristics of the atherosclerotic process in patients of Central Asian origin, the contribution of various risk factors in extreme climatic conditions, criteria for early diagnosis and risk stratification in the regional population, optimal approaches to primary and secondary prevention [3,4,5]. This has determined the relevance of this study.

Coronary heart disease is a complex, multifactorial disease that develops as a result of an imbalance between the myocardial demand for oxygen and its delivery, caused by atherosclerotic and functional changes in the coronary arteries. According to WHO data, in the structure of causes of death from cardiovascular diseases, coronary heart disease ranks first, accounting for 50-55% of the total cardiovascular mortality. In the Republic of Uzbekistan, the incidence of coronary heart disease has increased by 28-35% over the last 10 years; mortality from acute myocardial infarction accounts for 35-42% of the total mortality from coronary heart disease, with mortality in the first year after a myocardial infarction reaching 22-28% [8]. Many aspects of pathogenesis, natural course, early diagnosis, and optimization of therapeutic and preventive approaches remain insufficiently studied in the context of the regional population [6,7,8].

The aim of the study is to study the etiopathogenetic features of coronary heart disease in the regional population of Uzbekistan, identify the role of various risk factors and molecular genetic markers in the development of atherosclerosis and coronary artery disease, improve the algorithm for early diagnosis and risk stratification, and develop optimal approaches for primary and secondary prevention.

Etiology and pathogenesis

The etiology of coronary heart disease is multifactorial and includes both non-modifiable (age, gender, heredity) and modifiable (hypertension, dyslipidemia, diabetes mellitus, smoking, overweight, hypothyroidism, psycho-emotional stress) risk factors [9].

The pathogenesis of coronary artery disease is associated with the development of atherosclerotic lesions of the coronary arteries, which leads to narrowing of their lumen and insufficient blood supply to the myocardium. The process of atherogenesis begins with damage to the vascular endothelium, resulting in increased vascular wall permeability and the penetration of low-density lipoproteins (LDL) into the intima of the artery [10].

The oxidation of LDL leads to the formation of oxidized lipoproteins (oLDL), which are recognized by macrophages through decay receptors and accumulate in cells, transforming into foamy cells. The accumulation of foamy cells in the intima of the vessel leads to the formation of a lipid band - the first morphological manifestation of atherosclerosis [11].

Under the influence of cytokines (TNF-α, IL-1, IL-6) and growth factors, the migration of smooth muscle cells from the media to the intima, their proliferation, and the synthesis of extracellular matrix occur. A fibrous cap forms over the lipid nucleus—an atherosclerotic plaque[12]. In the early stages of atherogenesis, under appropriate conditions, a regression of atherosclerotic changes and the restoration of endothelial function are possible.

The instability of the atherosclerotic plaque is determined by the thickness of the fibrous cover, the content of the lipid nucleus, the inflammatory activity of macrophages in the plaque, and the state of proteolytic activity. A thin fibrous cover with an extensive lipid core and active inflammatory infiltration characterizes an unstable, "vulnerable" plaque prone to rupture[13].

The rupture of the fibrous cover of the atherosclerotic plaque leads to the exposure of tissue factor and thromboplastin, which activates the coagulation cascade and leads to the formation of a thrombus on the surface of the plaque. Depending on the degree of thrombus formation and the presence of collateral vessels, a clinical event develops - unstable angina pectoris, acute myocardial infarction, or sudden coronary death[14].

At the cellular level, myocardial ischemia causes many pathological processes: depletion of adenosine triphosphate (ATP) reserves, accumulation of lactate and protons (acidosis), imbalance of calcium, potassium, and sodium ions, formation of active forms of oxygen (AFC), activation of apoptosis, and necrosis of cardiomyocytes [15].

Molecular mechanisms for the development of atherosclerosis include impaired endothelial function, activation of NF-κB and MAPK signaling pathways, increased expression of adhesion molecules (ICAM-1, VCAM-1), attraction and transformation of monocytes into macrophages, and their activation via the classical (M1) pathway with the production of pro-inflammatory cytokines.

Materials and Methods

The study was conducted at the Republican Specialized Scientific and Practical Medical Center of Cardiology and clinics of the Department of Cardiology of medical universities in Tashkent during the period from 2023 to 2026. The research type is prospective controlled with elements of comparative analysis.

The study included 520 patients aged 40–75 years, divided into three groups: the main group consisted of 180 patients with verified coronary artery disease (stress angina, post-infarction state); the second group consisted of 180 patients with risk factors without clinical signs of coronary artery disease; the control group consisted of 160 healthy individuals without cardiovascular pathology. Inclusion criteria: age 40-75, consent to participate in the study, ability to complete the full scope of the examination. Exclusion criteria: acute myocardial infarction with a history of less than 6 months, severe heart failure (HR < 30%), significant kidney disorders (HR < 30 ml/min), oncological diseases, and mental disorders.

Clinical methods included a thorough collection of life and disease history, assessment of risk factors (heredity, smoking, hypertension, dyslipidemia, diabetes mellitus, obesity), clinical assessment using CCS (Canadian Cardiovascular Society) scales to classify angina pectoris, and SCORE for risk stratification.

Instrumental methods included electrocardiography in 12 branches, transtorakal echocardiography, veloergometry or treadmill test with ST segment and heart rate analysis, coronary angiography (for patients in the main group), and coronary artery computed tomography with calcium index assessment.

Laboratory methods included determining lipid profile levels (total cholesterol, LDL, HDL, triglycerides), fasting glucose, glycated hemoglobin, inflammatory markers (CRO, TNF-α, IL-6), coagulation indicators (MNO, ACHTV, fibrinogen), and enzyme activity (AST, ALT, KFC).

Results

Molecular genetic methods included genomic DNA isolation, genotyping of functionally significant polymorphisms of genes associated with atherosclerosis and coronary heart disease: APOE (ε2/ε3/ε4), APOB (rs676210), ACE (I/D), AGT (M235T), eNOS (T786C), IL-6 (rs1800795), CRP (rs1130864), TNF-α (G308A). Genotyping was performed using the real-time PCR method.

Statistical processing of the data was performed using SPSS 26.0 and R 4.0. For quantitative variables, parametric (Student's t-test) and non-parametric (Mann-Whitney's U-test) methods were applied. Qualitative indicators were compared using the $\chi 2$ criterion. Correlation analysis was conducted using the Pearson and Spearman method. Differences were considered significant at $p < 0.05$.

Research results. During the study, 520 patients were examined: 312 men (60.0%) and 208 women (40.0%). The mean age in the primary group was 58.3±8.5 years, in the second group it was 56.2±7.8 years, and in the control group it was 54.6±7.2 years. Clinical and anamnesis analysis revealed that 146 patients (81.1%) in the main group had a complicated inheritance of coronary heart disease and myocardial infarction.

The prevalence of the main risk factors in the main group was as follows: arterial hypertension was identified in 158 patients (87.8%), hypercholesterolemia in 142 (78.9%), type 2 diabetes mellitus in 68 (37.8%), active smoking in 94 (52.2%), obesity (BMI ≥ 30 kg/m2) in 106 (58.9%), while in the second

group, the prevalence of these factors was significantly lower ($p < 0.05$).

Analysis of the lipid profile revealed that the average level of total cholesterol in the primary group was 6.8±1.2 mmol/L, compared to 5.2±0.9 mmol/L in the control group ($p < 0.001$). LDL levels significantly exceeded the norm: 4.6 ± 1.1 mmol/l vs 2.8 ± 0.7 mmol/l ($p < 0.001$), while HDL decreased: 0.9 ± 0.3 mmol/l vs 1.4 ± 0.4 mmol/l ($p < 0.001$).

Inflammation markers were significantly elevated in the main group: CRO 6.2 ± 2.4 mg/l vs 1.8 ± 0.5 mg/l in the control group ($p < 0.001$), TNF-α 4.8 ± 1.6 pg/ml vs 2.1 ± 0.8 pg/ml ($p < 0.01$), IL-6 3.2 ± 1.3 pg/ml vs 1.4 ± 0.6 pg/ml ($p < 0.01$).

In molecular genetic analysis, the APOE ε4 polymorphism was identified in 108 patients (60.0%) of the main group, which was significantly associated with an increase in cholesterol levels ($r = 0.52$; $p < 0.001$). ACE I/D polymorphism was found in 132 patients (73.3%), especially the DD genotype, which correlated with the presence of arterial hypertension ($r = 0.38$; $p < 0.01$).

Discussion

The results obtained indicate the complex nature of coronary heart disease in the regional population of Uzbekistan, due to the interaction of modifiable and non-modifiable risk factors, genetic predisposition, and inflammatory mechanisms. The high prevalence of arterial hypertension (87.8%) and dyslipidemia (78.9%) in the main group confirms their key role in atherogenesis and aligns with data from other developing countries.

The identified increase in inflammatory markers indicates a significant contribution of the inflammatory component to the development and progression of coronary heart disease, which aligns with modern concepts of the inflammatory nature of atherosclerosis[14,15].

The identified association of genetic polymorphisms (especially APOE ε4 and ACE DD) with risk factors and clinical manifestations of CAD confirms the importance of genetic predisposition and justifies the use of genotyping for a personalized approach to prevention.

Conclusion

A comprehensive analysis of the etiopathogenetic factors of coronary heart disease in the regional population of Uzbekistan allows for a better understanding of the mechanisms of development and progression of atherosclerosis, the identification of individual risk markers, and the development of personalized approaches to primary and secondary prevention. The introduction of genotyping and inflammation markers into clinical practice will facilitate early diagnosis and optimize therapeutic and preventive measures in high-risk patients.

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