



THE INCIDENCE OF MYOCARDIAL INFARCTION IN YOUNG PEOPLE

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Article history:	Abstract:
<p>Received: April 10th 2021 Accepted: April 22th 2021 Published: May 20th 2021</p>	<p>Due to the unfavorable tendency towards the "rejuvenation" of ischemic heart disease (IHD), the etiological aspects of the development of acute myocardial infarction (AMI) in 211 patients under the age of 45 have been studied. A retrospective analysis of 132 case histories of patients with an assessment of the epidemiological components of coronary artery disease was carried out. Among the patients, men prevailed (88%), among the risk factors for coronary artery disease, atherogenic dyslipidemia was observed in 100% of cases, 75% of patients smoked, arterial hypertension was noted in 68% of patients, hypodynamia - in 56%, body mass index above normal - in 55%. A burdened family history of coronary artery disease was observed in 42% of patients. According to coronary angiography, 98.8% of patients had stenosis of the coronary arteries over 50%, while 65.0% of them had multivessel disease. There was a statistically significant relationship $F = 10.339$, ($p = 0.002$) between the burdened hereditary history and the development of AMI in young patients. Molecular genetic research revealed prothrombotic polymorphisms in the genes of the hemostasis system in 67% of patients.</p>

Keywords: Cardiovascular diseases, risk factors, acute myocardial infarction

Cardiovascular diseases (CVD) are a major socially significant problem, as they occupy a leading place in the structure of mortality, disability and disability of the working population [1]. Methods The study design was retrospective. A continuous sample of case histories of patients with AMI admitted to the cardiac intensive care unit of the City Clinical Hospital No. 1 of Arkhangelsk at the age of up to 45 years from 1999 to 2008 was carried out. AMI diagnosis (with Q wave and without Q wave) - inclusion criterion was confirmed by electrocardiogram data and positive markers of myocardial damage. The criteria for exclusion from the analysis of epidemiological data were autoimmune diseases, oncopathology, diabetes mellitus, the presence of valvular pathology of rheumatic genesis, hemoblastosis. Based on the inclusion and exclusion criteria, patients under 45 years of age were grouped into 211 people. Of the 211 patients, 132 people were included in the study - residents of the city of Arkhangelsk. We carried out a retrospective analysis of case histories ($n = 132$) with an assessment of the epidemiological components of the development of coronary artery disease, such as age, gender, body mass index, the presence of smoking, hypertension, the form and severity of coronary artery disease, the presence of concomitant pathology and features of a hereditary prothrombotic history (the presence of close relatives of the patient for indications of AMI, extensive cerebrovascular accident - stroke, thromboembolic complications).

Amplification of genomic DNA regions containing the indicated polymorphisms was carried out on the basis of polymerase chain reaction (PCR) technology. The identification of allelic variants caused by point nucleotide substitutions was carried out using the analysis of the polymorphism of the lengths of the restriction fragments of the PCR product (PCR-RFLP method). After 35 PCR cycles, the resulting amplificate was incubated with 10 units. specific restriction endonuclease under conditions recommended by the supplier (Sibenzyme LLC, Russia, or MBI Fermentas, Lithuania). DNA fragments were visualized in transmitted ultraviolet light after staining the gel with ethidium bromide

at a concentration of 1 µg / ml. The study design for molecular genetic analysis was approved by the local ethics committee of the Northern State Medical University (2007). Table 1 Studied DNA polymorphisms and methods of their identification Gene Localization Polymorphism Method Factor I, β-subunit (FI) 4q28 —455 G / A PCR — RFLP Factor II (FII) 11p11 — q12 20 210 G / A PCR — RFLP Factor V (FV) 1q23 1691 G / A PCR — RFLP Inhibitor of plasminogen activator type I (PAI-1) 7q21.3 - q22 - 675 4G / 5G PCR — RFLP Glycoprotein IIIa (GpIIIa) 17q21.32 1565 T / C PCR — RFLP Methylenetetrahydrofolate reductase (mthfr) 1p36.3 677 C / T PCR — RFLP For mathematical processing of the research results, the SPSS for Windows software package (version 15) was used. The Kolmogorov - Smirnov test was used to test the variational series for normal distribution. Quantitative data are presented as the arithmetic mean (M) ± standard deviation (SD) for a normal distribution and as a median (Me) and quartiles (Q) for a distribution other than normal. The significance of the differences was determined using the Mann-Whitney test. Statistical significance was assigned at p <0.05. To describe the relationship of categorical variables, the chi-square test of independence, Fisher's exact test, was used. Results Based on the purpose of our study, the main epidemiological characteristics of young patients with AMI were analyzed. Thus, the average age of the patients was (40.1 ± 4.6) years. Gender 47 Medical ecology Human ecology 2012.09 analysis showed the prevalence of males among patients - 88%, women accounted for 12%. Analysis of AMI by localization showed that anterior infarction was diagnosed in 72 cases, posterior basal - in 60. Large-focal Q-AMI developed in 107 patients, œ-Q-AMI - in 25 (Table 2). Table 2 Clinical characteristics of patients with acute myocardial infarction Sign Patient age, years p 25-35 (n = 17) 36-44 (n = 115) Gender male, abs. (%) 14 (82.4) 102 (88.7) 0.738 Age, years Me (Q1; Q3) 33 (27.5; 34.5) 42 (40; 43) 0.044 Angina, abs. (%) 1 FC 2 FC 3 FC 4 FC 2 (11.8) 1 (5.9) 1 (5.9) 0 0 31 (27.4) 13 (11.3) 15 (13.1) 2 (1.7) 1 (0.9) 0.089 Smoking, abs. (%) 14 (82.4) 85 (73.9) 0.455 Presence of initial continuous drug therapy CVD 3 (17.6) 45 (40) 0.033 CHF, abs. (%) 1 FC 2 FC 3 FC 4 (23.5) 3 (17.6) 1 (5.9) 0 66 (57.9) 35 (30.4) 30 (26) 1 (0.9) 0.008 Obesity, abs. (%) Grade 1 Grade 2 Grade 3 8 (47.1) 7 (41.2) 1 (5.9) 0 23 (20) 15 (13) 5 (4.3) 3 (2.6) 0.026 Increased body mass index, abs. (%) 4 (23.5) 30 (33) 0.534 Cardiac arrhythmias,% 29.4 21.4 0.823 Arterial hypertension, abs. (%) Grade 1 Grade 2 Grade 3 11 (64.7) 3 (17.6) 5 (29.4) 3 (17.6) 76 (66.1) 4 (3.5) 50 (43.5) 22 (19.1) 0.914 History of cerebrovascular accident, abs. (%) 0 5 (4.4) 0.383 AMI in history, abs. (%) 1 (5.9) 26 (31.6) 0.024 Complicated history of coronary artery disease, abs. (%) 3 (76.5) 42 (36.5) 0.002 Analysis of risk factors for coronary artery disease showed that 100% of patients had laboratory markers of atherogenic dyslipidemia in various combined variants (Table 3). Thus, an increase in the level of total cholesterol was observed in 62.1% of patients in the sample, hypertriglyceridemia in 9.8%, an increase in the level of low-density lipid cholesterol in 26.5%, and a decrease in high-density lipid cholesterol in 72.7%. The next significant risk factor for AMI was arterial hypertension. Thus, 68% of patients had indications of hypertension in the anamnesis. More than half (55%) of patients had a body mass index higher than the standard values. At the same time, grade 1 obesity was noted in 22 patients, grade 2 obesity - in six, grade 3 - in three. A burdened family history of coronary artery disease was noted in 42% of patients, the presence of the fact of smoking - 79% of patients. More than half of the patients (56%) noted a decrease in physical activity. A personal history of AMI was indicated in 14% of patients. By the time of AMI, clinical manifestations of exertional angina were noted in 25% of patients. Table 3 Indicators of lipid spectrum in patients with acute myocardial infarction (M ± SD) Patient age, years Sign 5 7) 3 17 2 (n 36-44 (n = 115) p Cholesterol, mmol / L 5.40 ± 1.09 5.50 ± 1.02 0.738 Low density lipoproteins, mmol / L 3, .4 ± 1.1 2.8 ± 1.3 0.543 Triglycerides, mmol / L 1.7 ± 1.1 1.5 ± 1.3 0.914 Analysis of prehospital drug therapy before admission to the hospital revealed that only 26 (19%) hospitalized patients received continuous drug therapy for hypertension, coronary artery disease, chronic heart failure (CHF), with nitrates being the most frequently prescribed group of drugs - 20 cases of prescription, and the most rarely prescribed group - statins (3 cases). In 58% of cases, when the patient was admitted to the hospital for urgent indications, a coronary angiographic study (CAG) was performed: in 98.8% of patients, signs of coronary artery stenosis were revealed by more than 50%. At the same time, a single-vessel lesion of the coronary basin was observed in 25 patients, two-vessel disease in 25, three-vessel disease in 12 and more than four vessels - in 12 patients. According to the CAG data of the trunk lesion, the left coronary artery was damaged in 7 patients, the right coronary artery - 44, the circumflex coronary artery - 36, the anterior interventricular coronary artery - 54 patients (Table 4). Table 4 Damage to coronary arteries in patients with acute myocardial infarction, abs. (%) Coronary artery disease Patient age, years p 25-35 (n = 17) 36-44 (n = 115) 1-vascular 9 (52.9) 16 (13.9) 2-vascular 2 (11.8) 23 (20.0) 3- vascular 2 (11.8) 10 (8.7) 4-vascular 0 8 (7.0) 5-vascular 0 3 (2.6) 6-vascular 0 1 (0.9) Total (M ± SD) 1, 12 ± 0.90 1.34 ± 0.80 0.988 For a more detailed study of anamnestic data, depending on age, the studied sample 48 Human ecology 2012.09 Medical ecology of patients was ranged by age groups: 1st group 25-35 years old and 2- I am a 36-44 year old group. There were no statistically significant differences in the incidence of hypertension (p = 0.914), angina pectoris (p = 0.089), manifestations of arrhythmia (p = 0.823). However, in the older age group, clinical manifestations of angina pectoris of a higher functional class were noted (3, 4 FC). Past myocardial infarctions in the anamnesis occurred significantly more often in the 2nd group of patients (p = 0.024), the degree of manifestation of CHF was also significant in the older age group (p = 0.008), which justified the constancy of drug therapy prescriptions in the 2nd group of patients (p = 0.033). There were no statistically significant differences in the manifestation of atherogenic dyslipidemia, the presence of the fact of smoking, depending on age. Conversely, the burdened heredity for coronary artery disease in younger patients was observed much more often (p = 0.002, Table 5). Table 5 Dependence of clinical and anamnestic data of patients with acute myocardial infarction on age Sign Fisher's criterion 25-35 years old (n = 17) / 36-44 years old (n = 115) p Affected coronary arteries 0.357 0.534 Left

coronary artery trunk 0.091 0.764 Right coronary artery 0.078 0.780 The circumflex artery 3.298 0.072 Posterior interventricular artery 0.956 0.330 Diagonal branch 2.104 0.144 Intermediate branch 0.839 0.361 Obtuse edge branch 1.954 0.361 Obesity 1.8 0.181 Increased body mass index 6.04 0.015 Arterial hypertension 0.579 0.579 hypertension 10.39 0.0012 Degree of arterial hypertension 0.012 0.912 Smoking 0.557 0.452 Hypodynamia 1.148 0.225 Chronic heart failure 6.971 0.009 Ejection fraction 0.165 0.687 Cholesterol level 0.318 0.574 There were no significant differences in the number of coronary artery lesions in the groups: (1.12 ± 0.90) vessels in group 1 versus (1.34 ± 0.80) in the 2nd (p = 0.988). There were also no statistically significant differences in the degree of coronary artery disease. To establish the degree of correspondence between the observed clinical and anamnestic data and the development of AMI in different age groups, the algorithm for calculating the exact value of significance by the Fisher method was used (see Table 5). As a result, a relationship was established between the presence of a hereditary predisposition and the development of AMI in younger patients (Fisher's exact test 10.339, p = 0.002). The next stage of our study was the analysis of individual genetic polymorphisms of the hemostasis system (F1 455 G / A, FII 20210 G / A, FV1691 G / A, MTHFR 677 C / T, PAI-1 675 4G / 5G, GpIIIa PIA1 / A2). Molecular genetic analysis by PCR was performed in 75 patients who agreed to this study at various times of the postinfarction period in 2007-2008. Table 6 shows the frequency of distribution of genotypes of the analyzed polymorphisms of the hemostasis system in the examined group of patients. Thus, in 67% of cases, various prothrombotic combinations of genetic polymorphisms of the hemostatic system were identified. According to the presented results, in the examined group of patients, there were no cases of known prothrombotic homozygous mutations in the FV 1691 G / A and FII 20210 G / A genes and no homozygous polymorphisms in the GpIIIa PIA1 / A2 gene. On the contrary, there was a significant frequency of detection of polymorphisms in the genes MTHFR 677C / T, GpIIIa PIA1 / A2, F1 455G / A, PAI-1 6754G / 5G. The calculation of the frequency of detecting several mutations simultaneously in one patient showed that in 32% of cases there was a combination of two polymorphisms, while in 14 patients they were represented by polymorphisms in the MTHFR 677C / T and PAI-1 6754G / 5G genes, in 5 patients in the GpIIIa genes PIA1 / A2 and PAI-1 6754G / 5G, in 4 in the GpIIIa PIA1 / A2 ^ F1 455G / A genes and in one patient in the F1 455G / A and MTHFR 677C / T genes (see Table 6). Table 6 Distribution of genotypes of polymorphisms of the hemostasis system in the studied patients Polymorphism Genotype Frequency of genotype occurrence, abs. Frequency of occurrence of genotype,% Factor I -455 G / A O << 42 1 1 1 44 26 5 58.7 34.7 6.7 Factor II 20 210 G / A 0 10 21 21 21 000 222 73 2 0 97.3 2.7 0 Factor V 1691 G / A * 0 91 91 9 666 73 2 0 97.3 2.7 0 MTHFR 677 C / T 0 ^ ü b Γ-- [> - h- t ' r fQ fp CD 41 32 2 54.7 42.7 2.7 PAI-1 -675 4G / 5G -675 4G / 4G -675 4G / 5G -675 5G / 5G 12 29 34 CD t ^ e ° --n O O Y 34 GpIIIa PI A1 / A2 A1 / A1 A1 / A2 A2 / A2 60 15 0 80 20 0 Note. In our study, we noted a significant frequency of detection of genetic polymorphisms, which, according to some data, are associated with the metabolic component of the development of IHD, endothelial dysfunction, unfavorable course of IHD and arterial thrombosis (MTHFR 677 C / T, GpIIIa PIA1 / A2, F1 455 G / A, PAI- 1,675 4G / 5G). According to the current theory, atherosclerosis is an inflammatory process involving a network of vascular wall cells, monocytes, T-lymphocytes, pro-inflammatory cytokines, chemokines and growth factors [4]. In this case, inflammation is accompanied by an increase in the activity of blood coagulation with the development of hypercoagulable syndrome, which means that the connection between inflammation and coagulation is assessed as bilateral. It should be borne in mind that, along with the classic CVD risk factors, thrombophilic disorders are latent and life-long risk factors for the development of thrombotic conditions [9]. The priority group for specific preventive measures to prevent vascular events are not only patients with established CVDs, but also those with a high risk of their development, taking into account the hereditary history [5]. This dictates the need for early detection of genetically determined disorders in the hemostasis system, determination of their relationship with already known risk factors for coronary heart disease, as well as the development of methods for the prevention and control of such disorders. The data obtained will help to identify groups of increased risk of occlusive-thrombotic conditions, to determine the tactics of managing patients with 50 Human Ecology 2012.09 Medical ecology of coronary heart disease against the background of a thrombophilic state, and therefore, to reduce the risk of thrombotic complications

CONCLUSIONS:

1. As a result of the study, a relationship was established between the presence of a hereditary predisposition and the development of acute myocardial infarction in young patients (Fisher's exact test 10.339, p = 0.002)
2. Along with the well-known modifiable and non-modifiable risk factors, genetically determined thrombophilic conditions were revealed in 67% of the examined patients, which aggravate the course of IHD in the form of thrombotic occlusion of the coronary bed. This dictates the need for further research in this direction, since the initial task on the path of early initiation of CVD prevention is to create a prognostic model that could identify young people at high risk of thrombotic events.

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