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IMPACT OF DYSLIPIDAEMIA ON THE CLINICAL COURSE OF CORONARY HEART DISEASE IN YOUNG MEN

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Abstract:

This study investigated the role of dyslipidemia in the development of coronary heart disease (CHD) in young men in the study objects were 230 patients with CHD hospitalized in the somatic intensive care units, emergency therapy units 1 and 2 of Samarkand Branch of Republican Scientific Center for Emergency Medical Care (SF RSCEMC) during the period 2018-2021. The patients were divided into 2 groups according to their age. The 1st main group consisted of 126 patients of young age (18 to 44 years). The 2nd comparative group included 104 elderly patients (60 to 74 years). According to the results, it was found that in patients of young and old age there was a disorder of lipid metabolism, but in patients of young age among lipid profile indicators there was an increase in low density lipoprotein (LDL) and triglycerides (TG), in elderly patients there was an increase in total cholesterol (TG) indicators.

Keywords: CHD, dyslipidaemia, young age, male

INTRODUCTION. Every year, more than seventeen million people around the world die from CHD, with mortality of more than half of cases due to CHD and, despite significant advances in addressing the prognosis, therapy and prevention of this disease, is still one of the pressing problems of modern cardiology [1]. Recently, the peculiarities of development and course of CHD, in particular its acute forms, in different groups of patients depending on sex, age, comorbidity and other features have been actively studied. Young people constitute the main active labour and production resources of society, determining the socio-economic perspective of any state [9]. With the observed rejuvenation of the age of onset of CHD, with a high proportion of fatal outcomes, the priority of public health is to determine the features of the clinical course, as well as the search and elimination of risk factors (FR) of CHD development in young patients [7, 11, 21].The most important FR in the development of atherosclerosis and its complications are lipid metabolism disorders/dyslipidemia (LDL).

In the pathogenesis of CHD, the most important prognostic factor is LDL. DLD is an imbalance between atherogenic and non-atherogenic lipoproteins, in which blood lipid/lipoprotein concentrations exceed the normal range [4, 16]. Asymptomatic atherosclerotic changes in the coronary arteries (CA) associated with DLDD are already detected at a young age and progress steadily over decades, already in middle age the rate of detection of atherosclerotic changes in the CA approaches 100% before leading to the development of clinical manifestations of NHS [8, 17]. With the observed rejuvenation of the age of onset of CHD, with a high proportion of fatal outcomes, the priority of public health is to determine the features of the clinical course, as well as the search and elimination of risk factors (FR) of CHD development in young patients [7, 11, 21].The most important FR in the development of atherosclerosis and its complications are lipid metabolism disorders/dyslipidemia (LDL).

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cardiovascular disease (CVD) [5, 13]. A sufficient number of studies have documented a high prevalence of lipid abnormalities in younger individuals with CHD compared with the older age group [19, 22]. Among the 7 leading FRs, elevated levels of COX make a major contribution to the development of premature death in the population and account for 23%. One in five men is found to have lower HDL levels, and one in three men is found to be hypertriglyceridemic [6]. Lower HDL levels and higher triglyceride (TG) levels have been reported in younger patients with CHD, further indicating that VLDL in young adults is an important FR in the development of CHD [18]. In LDL, smooth myocytes may be able to capture modified LDL and turn into foam cells [15, 24]. It has been noted that obese patients (BMI 30 kg/m2 or more) often develop atherogenic LDL [20, 25] and have increased TG concentrations in the blood and decreased HDL levels, while free fatty acid release from adipocytes into the bloodstream is increased and accompanied by increased liver LDL synthesis [3]. In this process, there is low activity of peripheral lipoprotein lipase, which cannot fully break down TG-rich particles [19, 22, 23].

The study of problems associated with subclinical atherosclerosis is considered to be important, because the detection and treatment of DLD in the early stages of the pathological process, can be potentially reversible or significantly slow its progression. In this regard, the development of optimal diagnostic and treatment algorithms will help to effectively address the problems associated with the atherosclerotic process.

PURPOSE OF THE STUDY: To investigate the effect of dyslipidaemia on the clinical course of coronary heart disease in young men.

MATERIAL AND METHODS. The object of the study was 230 patients with CHD hospitalized in the departments of somatic intensive care, emergency therapy №1 and 2 of Samarkand branch of RRCEMP during the period 2018-2021. The patients were divided into 2 groups according to their age. Group 1 included 126 patients of young age (18 to 44 years). Group 2 included 104 elderly patients (60 to 74 years of age). The control group consisted of 110 healthy people.

Inclusion criteria: young men aged 18 to 44 years and elderly men aged 60 to 74 years with confirmed diagnosis of CHD, who signed a consent to participate. Exclusion criteria: men aged 18 to 44 and 60 to 74 years with excluded diagnosis of CHD, patients with severe comorbidities**.** General clinical, instrumental, biochemical and statistical investigations were used.

In the examination of patients, height and weight were assessed by calculating BMI according to the Broca formula, recommended for evaluation by the WHO committee (1995). BMI was defined as the ratio of body weight in kilograms to height in meters, squared:

BMI=weight in kg/height in m2

Normal BMI is 20-25 kg/m2. Overweight is set at a BMI of 25.1 to 30 kg/m2. In I degree of obesity BMI is 30 to 34.9 kg/m2, II degree 35-39.9 kg/m2, III degree BMI is 40 kg/m2.

Blood lipid spectrum indices were determined for the content of: LDL, LDL, TG, HDL, and atherogenicity coefficient. Blood lipids were determined by homogeneous enzymatic colorimetric method on a Hitachi-902 biochemical analyzer. HDL was determined in the supernatant after precipitation of lipoproteins of other classes with dextran sulphate, LDL concentration was calculated by Friedwald formula: LDL=OCHS-LDL-TG/5 or LDL (in mmol/l)=OCHS-LDL-TG/2.2 The distribution of CHC between atherogenic and antiatherogenic lipoproteins was studied using the atherogenicity coefficient (CoefficientA) and was determined by the following formula:

Coefficient A= (GC - HDL)/(HDL),

Where coefficient A is the coefficient of atherogenicity (in relative units). Normally, the coefficient of atherogenicity is within 2-3 units.

RESULTS OF THE STUDY. The results of anthropometry revealed the following changes. Patients in the 1st group had an average height of 1,77±0,06 m, and in the 2nd group 1,74±0,05 m ($p<0,001$*), in the control group 1,77±0,08 m ($p>0,05$). The average body weight of patients in Group 2 was 6,5 kg higher than in Group 1 and was respectively 83,2±7,18 kg and 76,7±7,51 kg ($p<0,001$*), in the control group the average body weight was 75,9±10,2 kg ($p>0,05$). BMI averaged 24.6±3.44 kg/m2 in group 1, 27.7±2.46 kg/m2 in group 2, ($p=0.04$*) and 23.6±3.07 kg/m2 in control group,($p>0.05$). Among the patients in Group 1, 68 (53,9%) patients had normal body weight, in Group 2 only 12 (11,5%) patients ($p<0,001$*), in the control group 76 (69,1%),($p2<0,01$*) were found.
Overweight was diagnosed in 50 (39,7%) patients in Group 1, 69 (66,3%) in Group 2 ($p1<0,001$*), 32 (29,1%) males in the control group ($p2<0,05$*). Grade I obesity was found in 4 (3.2%) patients in Group 1, 15 (14.4%) in Group 2 ($p<0.001$*), 2 (1.8%) in the

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control group (p2>0.05*). Grade II obesity in group 1 was detected in 3 (2,4%) patients, in group 2 in 5 (4,8%), (p1>0,05).Grade III obesity in group 1 was observed in only 1 (0,8%) patients, in group 2 and in 3 (2,9%) patients, (p1>0,05) (Table 1).

Table 1
Characteristics of patients with NHS according to anthropometric data

Anthropometric indicators	Group 1 (n=126)	Group 2 (n=104)	) Control group (n=110)	Mann-Whitney-Wilcoxon tester-value
Height (m)	1,77±0,06	1,74±0,05	1,77±0,08	1vs2: p<0,001* 1vs3: p>0,05
Body weight (kg)	76,7±7,51	83,2±7,18	75,9±10,2	1vs2: p<0,001* 1vs3: p>0,05
BMI (kg/m2)	24,6±3,44	27,7±2,46	23,6±3,07	1vs2: p=0,04* 1vs3: p>0,05
Normal body weight	68 (53,9%)	12 (11,5%)	76 (69,1%)	1vs2: p<0,001* 1vs3: p<0,01*
Overweight body weight	50 (39,7%)	69 (66,3%)	32 (29,1%)	1vs2: p<0,001* 1vs3: p<0,05*
First degree obesity	4 (3,2%)	15 (14,1%)	2 (1,8%)	1vs2: p<0,001* 1vs3: p>0,05
Second degree obesity	3 (2,4%)	5 (4,8%)	0 (0%)	1vs2: p>0,05 1vs3: NA
Grade III obesity	1 (0,8%)	3 (2,9%)	0 (0%)	1vs2: p>0,05 1vs3: NA

In our study, the influence of LDL on the clinical course of CHD was assessed to determine predictors of the prognosis of adverse outcomes.One of the objectives of the present study is the assessment of lipid status in patients with NHS, as a result, we studied the lipid spectrum among young and elderly patients. As the results of investigation showed that level of LDL, LDL, TG were increased in both groups. The level of LDL in group 2 was increased by 0,33 mmol/l in comparison with group 1 and was 7,13±0,75 mmol/l and 6,8±0,86 mmol/l, respectively (p<0,001*), in control group mean LDL was 3,32±0,60 (p<0,001*). There were no statistically significant differences between the groups in HDL level of 1.0±0.15 mmol/l in group 1 and 0.97±0.16 mmol/l in group 2 (p=0.034*), although this index was lower than normal in elderly group, in control group this index was 1.2±0.18 mmol/l (p<0.001*). The LDL in group 1 was 4.5±0.83mmol/l, in group 2 - 4.32±0.62mmol/l respectively (p=0.038*), which indicates impaired lipid metabolism in patients with CHD, while in control group mean LDL was 2.96±0.83mmol/l (p<0.001*).

In group 1 patients Tg was significantly higher and amounted 3.11±0.92 mmol/l, in group 2 it was 2.87±0.81 mmol/l, (p<0.0001*), in control group Tg was 2.21±0.74 mmol/l (p<0.001*). CA was elevated in both groups, which was 5.92±1.26 in group 1, 6.52±1.2 in group 2 in control group 1.83±0.8 (p=0.03), (Figure 1).

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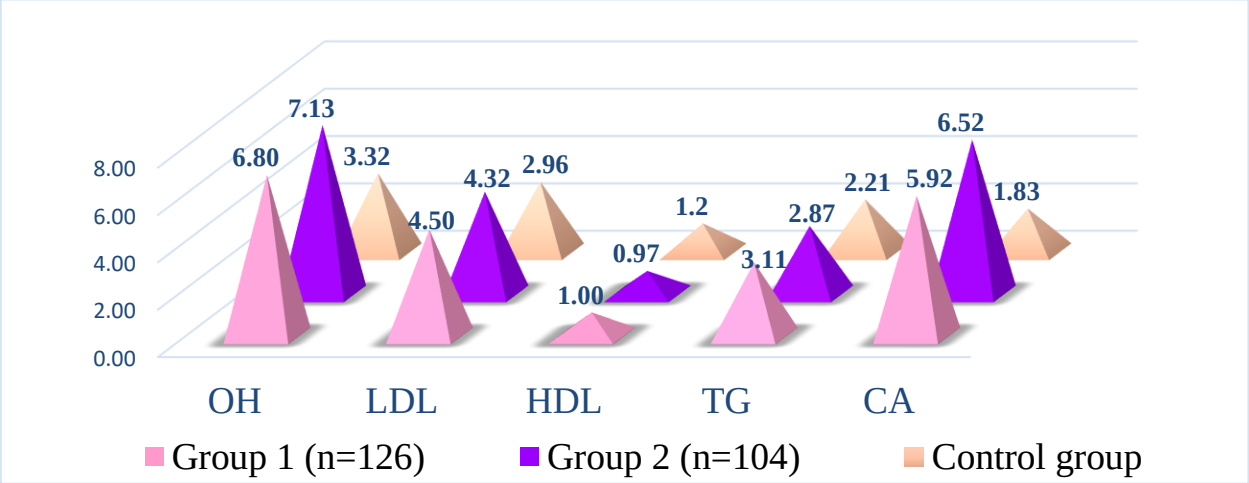

Figure 1: Lipid spectrum in patients with CHD and controls

The analysis of lipid spectrum parameters depending on the clinical manifestation of CHD in young and elderly men revealed that the highest values of atherogenic lipoproteins were observed in patients with acute myocardial infarction (AMI) compared to patients who were hospitalized with the diagnosis of first-time angina and progressive angina pectoris. This suggests that patients with high LDL, CHC, TG and low HDL contributed to an earlier and more severe course of CHD, which were detected in patients with AMI and acute coronary syndrome (ACS)(Table 2). For this reason, these patients should be closely monitored for body weight and LDL, OSH and TG levels, as dyslipidaemia can lead to the most formidable complications and may be the cause of early disability in the young population.

Table 2

Lipid spectrum indexes according to clinical variant of CHD in groups 1 and 2

Lipid spectrum (mmol/l)	Patients with an ASD	Patients with PSN		Patients with ACS pST		Patients with OCSDST		Patients with AMIsQ		Patients with AMI without Q	
	Group 1	Group 1	Group 2	Group 1	Group 2	Group 1	Group 2	Group 1	Group 2	Group 1	Grou p 2
OXC (3.6-7.8 mmol/l)	6.35± 1.04	6.83± 1.12	7.15± 1.09	7.04± 1.07	7.18± 1.14	7.27± 0.641	6.27± 1.40	7.99± 0.645	7.64± 0.897	6,16± 2,49	-
P-value	-	0,13		>0.65		>0.05		>0.40		-	
LDL (2.02-4.79 mmol/l)	4.38± 0.793	4.30± 0.9	4.6± 0.7	4.66± 0.771	4.46± 0.739	5.06± 0.741	3.88± 0.703	4.98± 0.086	4.86± 0.806	4.92± 0.021	-
P-value	-	0,06		0,32		<0.001*		>0.64		-	
HDL (0.72-1.63 mmol/l)	1.07± 0.168	1,0± 0,1	1,0± 0.2	0.982 ± 0.161	0.938 ± 0.116	0.957 ± 0.132	0.966 ± 0.087	1.10± 0.187	0.908 ± 0.106	1.10± 0.148	-
P-value	-	0,57		0,25		>0.85		>0.08		-	
TG (0,5-3,61 mmol/l)	3.06± 0.995	2.98± 0.983	3.34± 0.94	3.32± 0.914	3.28± 0.852	3.38± 0.948	2.43± 0.986	3.38± 0.724	3.36± 0.917	2.72± 1.45	-
P-value	-	0,056		0,87		<0.04*		>0.96		-	
CA no more than 3	6.1± 1.1	6.6± 1.16	6.93± 1.08	6.82± 1.07	6.96± 1.16	7.08± 0.652	6.02± 1.42	7.77± 0.631	7.44± 0.925	8.44± 1.01	-
P-value	-	>0.05		>0.64		>0.06		>0.44		-	

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CONCLUSIONS: Thus, the obtained analyses of lipid profile among patients with NHS showed that LDL in group 1 and group 2 were almost equally elevated compared to the control group and were 6.8±0.86 and 7.13±0.75 mmol/l,TG in group 1 patients were significantly higher and were 3.11±0.92mmol/l, while in group 2 patients this index was 2.87±0.81mmol/l.
Lipid profile indices by clinical variants of unstable angina and AMI were statistically significant, so that in patients with first-onset and progressive angina the atherogenic lipoproteins were lower compared to those in young and elderly patients with AMI. High values of atherogenic lipoproteins and atherogenicity coefficient values contributed to the early development of ACS and AMI, which is important for the correction of these disorders.

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