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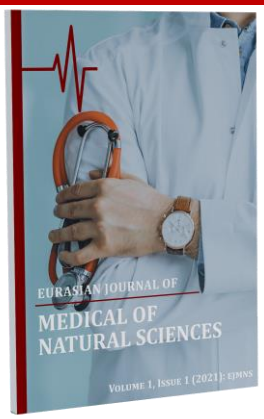

ASSESSMENT OF RISK FACTOR MODIFICATION IN PATIENTS WITH IHD

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ABSTRACT

Evaluation of the quantitative change of trimethylamine-N-oxide in patients with IHD in relation to risk factors such as age, body weight, genetic predisposition, diet is considered one of the urgent tasks today. Reducing heart disease mainly involves eliminating behavioral risk factors such as smoking, unhealthy diet, obesity, physical inactivity, and alcohol. In order to provide timely treatment, it is necessary to identify the risk factors that cause cardiovascular diseases early. Disruption of gut microbiota exacerbates these risk factors and increases complications.

Cardiovascular disease is one of the leading causes of death worldwide. By the end of 2019, 17.9 million people died from diseases of the cardiovascular system, which is 32% of all deaths worldwide. 85% of them are from heart attacks and strokes. More than three-quarters of cardiovascular diseases occur in low- and middle-income countries. Of the 17 million non-infectious diseases in 2019, 38% of premature deaths (under 70 years) are cardiovascular diseases. Reducing heart disease mainly involves eliminating behavioral risk factors such as smoking, unhealthy diet and obesity, physical inactivity, and alcohol. Early detection of cardiovascular diseases is necessary for timely treatment.

Cardiovascular diseases are the leading cause of death in people of all ages. [5, p. 19-287; 3, p. e38]. One of the main reasons for the development of cardiovascular diseases is atherosclerosis, that is, chronic inflammatory disease and lipid oxidation are observed in elastic and muscular arteries [4, p. 791–804; 1, p. 713–735]. In fact, the narrowing of the intravascular opening is associated with the accumulation of cholesterol and lipoproteins in the intima of the vessels, and the damage to the endothelium of the vessels determines the initial period of atherosclerosis [2, p. 620–636.].

The purpose of the study; development of preventive measures for assessment, treatment and prevention, taking into account that changes in intestinal microbiota affect the production of proatherogenic metabolite trimethylamine-N-oxide in patients with ischemic heart disease

In order to evaluate the quantitative changes of trimethylamine-N-oxide in patients with IHD in relation to risk factors such as age, body weight, genetic predisposition, diet, 90

patients and 30 control groups who were treated in bed with the diagnosis of angina pectoris due to ischemic heart disease in ASMI clinic 1 therapy department were studied. .

Body weight, index Ketle, smoking, genetic predisposition, eating pattern and structure are studied in all patients. They were taken from venous blood to study the quantitative changes of cholecystokinin-8, trimethylamine-N oxide in their blood. table 1.

Table 1. Indicators of risk factors in patients with IUD and control group

Variables		1,2,3 groups (N=90)	4 gropup (N=30)	P	Жами (N=120)
consuming tobacco	Does not smoke	37 (41.1%)	17 (56,7%)	0.067	44 (36.7%)
	smokes	53 (58.9%)	13 (43.3%)		76 (63.3%)
Genetic predisposition	genetic predisposition exist	53 (58.2%)	6 (20.7%)	<0.001	59 (49.2%)
	no genetic predisposition	38 (41.8%)	23 (79.3%)		61 (50.8%)
Body mass index	normal	2 (3.3%)	19 (65.5%)	<0.001	22 (18.3%)
	obesity I degree	60 (66.7%)	4 (13.8%)		64 (53.3%)
	obesity II degree	21 (23.3%)	1 (3.4%)		22 (18.3%)
	obesity III degree	4 (4.4%)	0 (0%)		4 (3.3%)
	overweight	3 (3.3%)	6 (20.0%)		8 (6.7%)

When we examined the risk factors, the most observed factors associated with increased TMAO among patients with IHD were smoking 53 58.9% and 13 recipients in the control group did not have increased TMAO 43.3%; When determining the predisposition to heart disease, increased TMAO was observed in 58.2% of 53 patients with a diagnosis of CKD, only 6 patients in the control group had increased TMAO (20.7%). ($p<0.01$).

The following changes were found when body weight was measured in all patients with IHD with increased TMAO; 66.7% of 60 patients had 1 degree obesity, 23.3% 21 degree obesity and 4.4% 4.4% 3 degree obesity. In the control group, there was no increase in uric acid and TMAO, excess body weight was observed in 6 patients (20.0%), 1st degree obesity was observed in 4 patients (13.8%), 2nd degree obesity was observed in 1 patient (3.4%). ($p<0.001$).

When we studied these factors in four groups, the following indicators were observed. Table 2.

Table 2. Risk factors in 4 groups

Variables		1-гурух (N=30)	2-гурух (N=30)	3-гурух (N=30)	4-гурух (N=30)	p	Жами (N=120)
consuming tobacco	Does not smoke	12 (40.0%)	16 (53.3%)	10 (33.3%)	6 (20.0%)	0.067	44 (36.4%)
	smokes	18 (60.0%)	14 (46.7%)	20 (66.7%)	24 (80.0%)		77 (63.6%)
Genetic predisposition	genetic predisposition exist	20 (66.7%)	16 (53.3%)	17 (56.7%)	6 (20.0%)	<0.001	59 (48.8%)

	no genetic predisposition	10 (33.3%)	14 (46.7%)	13 (43.3%)	24 (80.0%)		62 (51.2%)
Body mass index	normal	0 (0%)	2 (6.7%)	1 (3.3%)	19 (63.3%)	<0.001	23 (19.0%)
	obesity I degree	13 (43.3%)	23 (76.7%)	23 (76.7%)	5 (16.7%)		64 (52.9%)
	obesity II degree	14 (46.7%)	3 (10.0%)	4 (13.3%)	1 (3.3%)		22 (18.2%)
	obesity III degree	3 (10.0%)	0 (0%)	1 (3.3%)	0 (0%)		4 (3.3%)
	overweight	0 (0%)	2 (6.7%)	1 (3.3%)	5 (16.7%)		8 (6.6%)

As we can see in the table, the main number of smokers is observed in the 1st, 2nd, 3rd groups with ischemic heart disease - 40%, 53.3%, 33.3% r<0.067, 66.7%, 53, 53. 3%, 56.7% according to groups, obesity 1 degree 43.3%, 76.7%, 76.7% according to groups, 2 degree 46.7%; 10.0%; 13.3% corresponding to groups, 3 levels 10.0%; 0% ; 3.3% were observed according to the groups. (<0.001).

The influence of various factors on the increase in TMAO concentration was studied and the result was that the probability of increase in TMAO concentration in "Tobacco" smokers increased by 64% compared to non-smokers [OR = 0.36], the confidence interval calculated on the basis of Euler's constant and Fisher's r value also showed that the obtained results were statistically significant [CI 95% (0.36–0.93) p-value = 0.045].

The effect of genetic predisposition on the increase of TMAO concentration was studied, and it was clinically proven that the probability of pathological increase of TMAO in "predisposed" individuals was 5.35 times higher than in "non-predisposed" individuals [OR = 5.35], the confidence interval calculated based on Euler's constant and Fisher's r value were also obtained. showed statistical significance [CI 95% (2.09-15.63) p-value = 0.001].

The influence of body mass index on the increase in TMAO concentration was studied, the "normal" category was taken as a reference group, and it was clinically proven that the probability of TMAO increase at a pathological level is 95 times higher among individuals who are in the "1st degree of obesity" [OR = 95.00]. The confidence interval calculated based on Euler's constant and Fisher's r value also showed that the obtained results were statistically significant [CI 95% (22 -564.45) p-value < 0.001]. In addition, it was clinically proven that the probability of TMAO increase in the pathological level is 133 times higher in individuals entering the "2nd degree of obesity" [OR = 133.00]. The confidence interval calculated based on Euler's constant and Fisher's r value also showed that the obtained results were statistically significant [CI 95% (18.39 -2908.92) p-value < 0.001]. No correlation was found between other categories of body mass index and pathological elevation of TMAO "obesity III degree" [OR = 2695; (CI 95% (0 -NA) p-value < 0.992]; "overweight" [OR = 3.8; (CI 95% (0.56 - 27) p-value < 0.164).

It was found that there is a correct correlation between the increase in body weight and the increase in TMAO (r = 0.438; r=0.001) in patients with IUD. 67.7% smoking, 48.8% genetic predisposition, 1st degree obesity 52.2%, 2nd degree obesity 18.2%, 3rd degree obesity 3.3% were observed together with increased TMAO.

An increase in TMAO leads to the development of atherogenesis in patients with an effect on risk factors.

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