

Assessment of Vitreous Spectrum Parameters in Patients with Vitreous Destruction in the Course of Treatment

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Abstract Opacities of the vitreous body that occur with age are the result of a violation of the structure of the vitreous body of the processes of its destruction, liquefaction and wrinkling and are currently an urgent problem of ophthalmology that determines the quality of life of patients. According to the literature, about 76% of people have floating "flies" in front of their eyes, and 33% associate them with a decrease in vision. Vitreous opacities are one of the manifestations of vitreous destruction with the formation of seals that shield light, cast a shadow on the retina, and, as a result, reduce not only the quality of vision of patients, but also their quality of life in general.

Keywords Ophthalmology, Vitreous, Ultrasound examination

1. Introduction

Under normal conditions, the concentration of cholesterol in the blood significantly exceeds the concentration of cholesterol in the vitreous body. One of the suggested mechanisms of scintillation synchysis pathogenesis is the release of cholesterol from lysed red blood cells [2,5,6,9]. This could explain the cases associated with vitreous hemorrhage, since extravascular blood degeneration releases excess cholesterol, which precipitates as crystals from the vitreous solution.

However, this mechanism of cholesterol development due to blood degeneration is not sufficient to explain cases of scinchysis that occur without hemorrhage, such as cases of retinal detachment or overripe cataracts [1,3,4,5]. It is assumed that in retinal detachment, cholesterol is formed from the subretinal fluid that diffuses into the posterior chamber through retinal tears [3,8]. A recent study shows that oxidative stress and lipoperoxidation are the causes of the formation of intraocular cholesterol crystals [4,7,11-0]. This suggests that redox imbalance and increased oxidative modifications in patients with vitreous degradation play a role in pathogenesis, possibly due to changes in the hemato-water or hemato-vitreous barrier [6,7,8].

Treatment of vitreous opacities is currently attracting the attention of doctors and researchers [5,7,8,9,10]. There are many advanced solutions to this problem. In our work, we included the drug atoris in the treatment regimen, and depending on the chosen therapy, we divided all patients into 2 groups.

Objective: to determine the level of the lipid spectrum in patients with vitreous destruction.

2. Material and Methods of Research

The study included 102 patients with vitreous destruction. The diagnosis of vitreous destruction was made according to the International Classification of Diseases (ICD-10) - H43. Patients were divided into 2 groups depending on the chosen therapy. Main group 1 consisted of 50 patients with vitreous destruction who received atoris as a background of basic therapy. Comparison group 2 included 52 patients with vitreous destruction who received only basic therapy. Also, to compare laboratory parameters, a control group was created, which included 20 practically healthy individuals. All patients were taken blood to study the parameters of the lipid spectrum. The blood lipid spectrum included: total cholesterol, very low-density lipoproteins (VLDL), low-density lipoproteins (LDL), and high-density lipoproteins (HDL), triglycerides, and the atherogenicity index over the course of treatment.

3. Research Results

The results of the study showed (Table 1) thatami revealed a disturbed lipid spectrum in patients with vitreous destruction.

When evaluating the analysis of the lipid spectrum in the control group, we found results that did not differ from the reference values: namely, total cholesterol was 3.5 ± 0.1 mmol/L; VLDL- 0.6 ± 0.02 mmol/L; LDL- 3.1 ± 0.1 mmol/L; triglycerides- 1.1 ± 0.1 mmol / L and atherogenicity index – 3.2 ± 0.2 ; reduced from normal: HDL – $1.6 \pm 0.02 \times$ mmol/l.

In patients of the main group (n=102), total cholesterol was 8.1 ± 0.2 mmol/L; VLDL- 1.5 ± 0.03 mmol/L; LDL- 5.1 ± 0.1 mmol/L; triglycerides- 2.7 ± 0.1 mmol/L and atherogenicity index- 7.3 ± 0.1 ; reduced from the norm: HDL – 1.0 ± 0.02 mmol/L.

Thus, analyzing the parameters of the lipid spectrum, we found a deviation from the norm of the indicators of the main group (Table 1).

Table 1. Analysis of the lipid spectrum of the studied individuals of the examined groups, $M \pm m$

Indicator (norm)	Control group (n=20)	Main group (n=102)
TCh (0.5-5.2 mmol / l)	3.5 ± 0.1	$8.1, 1 \pm 0.2^{***}$
VLDL (0.16-0.85 mmol / l)	0.6 ± 0.02	$1.5 \pm 0.03^{***}$
LDL (2.5-4.1 mmol / l)	3.1 ± 0.1	$5.1 \pm 0.1^{***}$
HDL (1.42-male; 1.68-female mmol / L)	1.6 ± 0.02	$1.0 \pm 0.02^*$
TG (0.3-1.5-male; 0.4-1.8-female mmol/L)	1.1 ± 0.1	$2.7 \pm 0.1^{***}$
AI (2-3-negative, 3-4-moderate, 4-8-high)	3.2 ± 0.2	$7.3 \pm 0.1^{***}$

Note: * - significance of indicators in relation to the control group (differences are significant: * - $p < 0.05$, * * * - $p < 0.001$).

A comparative analysis of the results of the lipid spectrum showed that the study subjects of the main group had statistical differences in the indicators of OH ($P < 0.001$), VLDL ($P < 0.001$), LDL ($P < 0.001$), TG ($P < 0.001$) and HDL ($P < 0.001$), IA ($P < 0.001$) in relation to the persons of the control group.

Patients with vitreous destruction had a disturbed lipid spectrum.

When evaluating the analysis of the lipid spectrum in the studied individuals, results were different from those before treatment: namely, in patients of the main group, total cholesterol before treatment was 7.9 ± 0.1 mmol/L, after treatment- 5.0 ± 0.2 mmol/L. VLDL before treatment was

1.4 ± 0.03 mmol/L, after treatment- 0.6 mmol/L. Pretreatment LDL was 4.9 ± 0.1 mmol / L, posttreatment- 3.6 ± 0.1 mmol/L. Pretreatment HDL was 1.1 ± 0.02 mmol/L, posttreatment HDL increased to 1.5 ± 0.02 mmol/L. Triglycerides before treatment were 2.9 ± 0.1 mmol/L, after treatment - 1.2 ± 0.1 mmol/L. The atherogenicity index before treatment was equal to 6.8 ± 0.3 , after treatment- 3.3 ± 0.2 (Table 2).

In patients of the comparison group, total cholesterol before treatment was 8.1 ± 0.2 mmol/L, after treatment- 7.2 ± 0.1 mmol/L. VLDL before treatment was 1.5 ± 0.03 mmol/L, after treatment- 1.2 ± 0.03 mmol/L. LDL before treatment was 4.5 ± 0.1 mmol/ L, after Pretreatment HDL was 4.2 ± 0.1 mmol/L. Pretreatment HDL was 1.2 ± 0.02 mmol/L, posttreatment HDL increased to 1.3 ± 0.02 mmol/L. Triglycerides before treatment were 2.8 ± 0.1 mmol/L, after treatment - 2.2 ± 0.09 mmol/L. The atherogenicity index before treatment was 6.4 ± 0.1 , after treatment- 5.8 ± 0.1 .

Thus, analyzing the parameters of the lipid spectrum in the dynamics of treatment, we found a significant positive dynamics in patients of the main group, compared with the comparison group.

We found lipid metabolism disorders in patients with vitreous destruction in both groups before treatment, i.e. hypercholesterolemia, hypertriglyceridemia, mixed hyperlipoproteinemia, and low HDL cholesterol. Reducing LDL cholesterol using statin therapy with atoris is a powerful approach to primary and secondary prevention of not only cardiovascular diseases, but also eye diseases [4,7,8].

Statins are currently the standard drug for the treatment of any type of atherosclerosis and, in the presence of appropriate risk factors, are also prescribed for both primary and secondary prevention [2,8].

4. Conclusions

The use of the drug atoris in patients of the main group with vitreous destruction compared to the comparison group who did not receive statins, there was a decrease in flies in front of the eyes and floating opacities of the vitreous body.

Table 2. Comparative analysis of the lipid spectrum of the examined groups in the dynamics of treatment, $M \pm m$

Indicator (norm)	Main group (n=50)		Comparison group (n=52)	
	Pretreatment	Posttreatment	Pretreatment	Posttreatment
TCh (0.5-5.2 mmol / l)	$7.9 \pm 0.1^{***}$	5.0 ± 0.2	$8.1, 1 \pm 0.2^{***}$	7.2 ± 0.1
VLDL (0.16-0.85 mmol / l)	$1.4 \pm 0.03^{***}$	$0.6 \pm 0.03^{***}$	1.5 ± 0.03	1.2 ± 0.03
LDL (2.5-4.1 mmol / l)	$4.9 \pm 0.1^{***}$	$3.6 \pm 0.1^{***}$	4.5 ± 0.1	4.2 ± 0.1
HDL (1.42-male; 1.68-female mmol / L)	$1.1 \pm 0.02^*$	$1.5 \pm 0.02^*$	1.2 ± 0.02	$1.3 \pm 0.02^*$
TG (0.3- 1.5-male; 0.4-1.8-female mmol / l)	$2.9 \pm 0.1^{***}$	$1.2 \pm 0.1^{***}$	2.8 ± 0.1	2.2 ± 0.09
AI (2-3-negative, 3-4-moderate, 4-8-high)	$6.8 \pm 0.3^{***}$	3.3 ± 0.2	6.4 ± 0.1	5.8 ± 0.1

Note: * - significance of indicators between groups (differences are significant: * - $p < 0.05$, * * * - $p < 0.001$).

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