К 5 –й группе относят сочетание изменения ДПК, внепеченочных желчных протоков, протока поджелудочной железы, толстой кишки и др. Рекомендуют способы зашивания культи ДПК, применяемые для ликвидации внутреннего свища.

Выводы и предложения. Таким образом, нами предложен новый способ мобилизации двенадцатиперстной кишки и намечен один из путей подхода к созданию классификации способов закрытия культи ДПК при различных изменениях в пилородуоденальной зоне с учетом пластических возможностей передней, задней, медиальной и латеральной стенок ДПК.

Список литературы:

1. Литтман И. // Брюшная хирургия. — Будапешт, 1970;

2. Матяшин И.М. Глузман А.М. Справочник хирургических операций. — Киев, «Здоров'я », 1979;

3. Навроцкий И.Н. Ушивания культи двенадцатиперстной кишки. — М,, 1972;

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STUDY OF LIPID SPECTRUM INDICATORS IN BLOOD OF PREGNANT WOMEN WITH CHRONIC B, C VIRUS HEPATITIS

Abstract. One of the main functions of the liver is the synthesis and regulation of lipid metabolism. Metabolic activity in the liver during pregnancy, especially after the II trimester, causes changes in lipid levels in the body. HBV and HCV damage to the liver negatively affect lipid metabolism. There are no scientific reports on the effects of chronic HBV and HCV infections on lipid parameters during pregnancy.

Objective of the research: to study the level of total cholesterol in blood, triglycerides, A1- apolipoprotein, the main protein in high-density lipoprotein, low-density lipoproteins in pregnant women with chronic HBV and HCV.

Object of the research: The research work was carried out at the II Department of Obstetrics and Gynaecology at the Azerbaijan Medical University, Baku, Azerbaijan. The research was based on prospective material for 2016-2018. The object of the study was 150 pregnant women aged 18-45 years. I group - trial group (practically healthy pregnant women; n = 50); II group - pregnant women with chronic HBV infection (n = 55); III group - pregnant women with chronic HCV infection (n = 45). The principles of randomization were not violated in the course of the study. The groups were identical in terms of gestation and parity.

Research methods: Diagnostics of viral hepatitis (B, C) were performed by express card, I immune-enzyme analysis (Automated Biochemical ECLIA Analyzer - Cobas 4000 e411), HBV, HCV Quantitative and Qualitative analysis with PZR Reaction (Real Time PZR Detection Systems). Determination of total cholesterol and triglyceride levels in blood plasma was defined by enzyme-colorimetric and low-density lipoproteins by homogenous enzymatic colorimetric method. The blood content of Apolipoprotein A1, the main protein component of high-density lipoproteins, was analyzed by immunoturbidimetric analysis. The liver enzymes (ALT, AST), CRP determination in the blood were performed in the biochemical automated analyzer - the Roche-Hitachi Cobas 4000 c311 device Cobas (c 501/502).

Mathematical and statistical methods: Variation (U-Mann-Whitney), dispersion (F-Fisher), discriminant (χ2-Pearson Chi Square) and correlation analysis (ρ-Spearman) were used in the study. Statistical analyzes were performed in the MS EXCEL-2013 spreadsheet and in the SPSS-20 statistical package software. “O” hypothesis was rejected when statistical analyzes were $p \leq 0.050$.

Results: The results of the study showed that the level of total cholesterol in the blood in infected pregnant women increased by 1.5 times compared with practically healthy pregnancies ($F = 52{,}039$; $p_F < 0.001$; $p_U < 0.001$). A positive correlation between total cholesterol and CRZ was found in the blood of pregnant women with B, C virus hepatitis ($\rho = 0.341$, $p = 0.001$). Statistical quality analysis of triglyceride indices revealed an increase in lipid levels in the main group compared to the control group ($\chi2=6{,}518$; $p=0{,}0384$). The level of Apolipoprotein A1 in the blood of infected pregnant women decreased 2.1 times compared with control group ($p\ F = 149{,}916$; $p_F < 0.001$; $p_U < 0.001$). Statistical analysis of the study showed a negative correlation between ApoA1 and major hepatic transferases (ALT, AST) in pregnant women with HBV, HCV infections (with ALT $\rho = -0{,}238$, $p=0{,}018$; with AST $\rho = -0{,}230$, $p=0{,}023$). In pregnant women with viral hepatitis, levels of low-density lipoproteins in the blood increased 1.4 times compared to the control group ($p < 0.001$; $F = 22{,}759$; $p_F < 0.001$; $p_U < 0.001$).

Conclusion: The presented scientific work may not only confirm the effects of HCV infections on the lipid spectrum during pregnancy but may also help to understand the pathogenesis of the parental hepatitis during gestation.

Keywords: HBV, HCV infections, pregnancy, lipid spectrum

Relevance: Triglycerides play a key role in maintaining fat tissues within the human body. Physiologists have likened cholesterol for its function, composition and consistency to bile. The word

"cholesterol" from Greek is also translated as "solid bile". For the first time, a French scientist has proven that cholesterol is a fat-containing alcohol, so it is called "cholesterol" in foreign literature.

(1) Cholesterol fulfils following functions in the body: Regulation of metabolism, maintenance of cell structure, protection of cell membranes from toxins, maintenance of synthesis of corticosteroids and other hormones, maintenance of bile synthesis; conversion of 7-dehydrocholester to vitamin D (under ultraviolet rays); other (2) Cholesterol reduction may be related to hunger, cachexia, sepsis, extensive burns, tuberculosis, liver cirrhosis, some types of anaemia, taking some drugs (estrogens, interferon, etc.) and other causes. In general, since lipoproteins cannot perform lipid metabolism independently, they implement transport function. The following fractions of cholesterol in blood are: High-Density Lipoproteins (HDL); Low-Density Lipoproteins (LDL); Very Low-Density Lipoproteins (VLDL); Triglycerides (TG). In popular terms, HDL is called "good cholesterol" and LDL "bad cholesterol". “Good” lipoproteins are mostly synthesized in the liver. 200-300 mg of cholesterol enters the body with daily food. Blood cholesterol levels vary depending on the patient's age, sex, weight, nutritional ration, congenital or acquired diseases (liver, gall bladder and other digestive diseases, metabolic syndrome, etc.), as well during pregnancy. According to scientific sources, the limits of these changes are based on certain figures. Data on cholesterol and triglyceride levels during pregnancy are not exact. For example, Geraghty A. and fellows (2017) believe that serum cholesterol levels in non-pregnant mature women should be less than 5 mmol/ l and triglyceride levels less than 2 mmol /l. (3) During pregnancy, there is an increase in blood cholesterol levels due to metabolic changes and hormonal regeneration processes. For example, the thyroid stimulating hormone maintains cholesterol levels in the blood. This is one of the hormonal causes of cholesterol increase during pregnancy. On the other hand, an increase in progesterone during pregnancy is manifested by an increase in the weight and fat tissues of a pregnant woman. Increase in estrogens levels results in increased lipoproteids rich in triglycerides and accelerating the lipogenesis.

During physiological pregnancy, human placental lactogen (HPL) promotes insulin resistance, reduces low-density lipoproteins, and promotes delivery of free fatty acids to the liver.

(4) The higher prevalence of cholestasis in pregnant women and those who are not pregnant makes the problem clear. Scientific sources report increased cholestasis in liver diseases, especially in hepatitis B and C. (5), (6).

Although there are few scientific studies on the incidence of B, C virus hepatitis in women, specificities of course of pregnancy, birth in women with chronic HBV and HCV infections, there is no dissertation on lipid spectrum in pregnant women. (7, 8,9,10) There are no studies on lipid metabolism in pregnant women with chronic B, C virus hepatitis in the domestic literature as well as in foreign literature.

Objective of the research: to study the main indicators of lipid metabolism in blood in pregnant women with chronic B, C virus hepatitis, and to determine the correlation dependency of these indices with the liver enzymes, CRZ. For this purpose, levels of apolipoprotein A1 (Apo A1), the main fraction of total cholesterol (TC), triglycerides (TG), high-density lipoproteins (HDL) in plasma of pregnant women, low-density lipoproteins (LDL), ALT, AST, viral load in blood, CRZ have been studied.

Object and methods of research: The research work was carried out at the Department of Obstetrics and Gynecology of the Azerbaijan Medical University, Baku, Azerbaijan. The research work was carried out on the basis of collected material for 2016-2017. The main focus of the study was 100 pregnant women with chronic B, C virus hepatitis in the 18-45 year age group, and 50 practically healthy pregnant women in the trial group. Pregnant women with severe genital and extragenital infections, diabetes, high glucose tolerance test, and severe preeclampsia were not included in the group.

Pregnant women in the study group were identical to their pregnancy duration and parity. Diagnostics of viral hepatitis (B, C) were performed by express card, immune-enzyme analysis, (Automated Biochemical ECLIA Analyzer - Cobas 4000 e411), HBV, HCV Quantitative and Qualitative PZR Reaction (Real Time PZR Detection Systems).

PZR- 1IU = 4.5 copies for hepatitis B virus; PZR-1IU = 2.5 copies for Hepatitis C virus (National Institute of Biological Standards and Hepatitis B Control for NIBSC WHO International Standard: 97/746) Determination of TC, TG in blood plasma was carried out by enzymatic-colorimetric, LDL homogenous colorimetric method. Amount of ApoA1, a key protein component of HDL in the blood was performed by immunoturbidimetric analysis.

Norms of blood lipids content were calculated according to NCEP standards (2001). (11) Liver enzymes, CRZ determination in the blood were performed on a biochemical automated analyzer - Roche-Hitachi Cobas 4000 c311 device cobas (c 501/502). For biochemical analysis, patients' blood samples were taken from elbow veins as 3-5 ml after hunger in morning hours (800-1,100), centrifuged 3,000 times in 15 minutes, and serum separated and placed in special test bottles and set for a biochemical analyzer.

Statistical analysis: To analyze the obtained quantitative and qualitative digital data, methods of variational, disperse, discriminant and correlation analyzes were applied.

In the variational analysis, the mean values of the obtained samples (M), their standard deviations (± □), standard errors (± m), 95% confidence intervals (95% CI), minimum (min) and maximum (max) values of the series were calculated. For comparison and statistical evaluation of the difference between the quantitative indicators, analysis of disperse was used - the ANOVA

test, using the F-Fisher criterion. For the final decision on accepting or rejecting the "null hypothesis", the non-parametric ranked Wilcoxon U-test (U-Mann-Whitney) was additionally applied.

For the analysis of qualitative characteristics in the studied groups, cross-tables of 2x2 and 2xn were preliminarily compiled. In each cell, the sample fractions (p) and their errors (mp) were calculated for an alternative grouping option. To determine the degree of conjugation between qualitative characteristics, the $\chi 2$-Pearson (Pearson Chi-Square) criterion was used. At the boundary values ($p \approx 0.050$), the statistical significance was additionally assessed by the criteria of Continuity Correction, Likelihood Ratio, Fisher's Exact Test and Linear-by-Linear Association. For the final decision on accepting or rejecting the "null hypothesis", the results of most criteria were used.

To assess the tightness of the relationship of both qualitative and quantitative signs observed in the groups, a correlation analysis was performed. As an analysis, the nonparametric rank correlation coefficient of ρ-Spearman (ρ-Spearman) was used. The statistical significance of the correlation coefficient was evaluated by a 2-sided criterion.

The critical level of significance for the adoption of the "null hypothesis" in all statistical analyzes was adopted $p \leq 0,050$. The calculations were performed on the MS EXCEL-2013 spreadsheet (IBM USA) and the SPSS-20 statistical package.

Results of the study: The object of the study was 150 pregnant women. The patients studied were divided into 3 groups: I group - trial group (n = 50). The test group was made by practically healthy pregnant women. II group - HBV-positive pregnancies (n = 55), III group - HCV-positive pregnancies (n = 45). Only pregnant women with monocyesis were included in the study. There were no women smoking and drinking among pregnant women in the research group.

Demographic data and clinical characteristics of the patients indicated that all of the pregnant women were Azerbaijani. The average age of the test group was 26.7 ± 0.6; in the main group was 28.8 ± 0.5 years. BKI in practical healthy pregnancies was 24.5 ± 0.5 kg / m²; in the main group, 25.7 ± 0.3 kg / m² (it should be noted that BKI in pregnant women was calculated based on weight gain in the second trimester of pregnancy, taking into account pre-pregnancy indicators). Clinical-laboratory characteristics of the studied groups on other indicators are given in Table 1,2,3,4.

Table 1

Crosstab					
			Groups		Total
			Control	HBV and HCV	
Rh	Rh -	Count	8	9	17
		% within group	16,0%	9,0%	11,3%
	Rh +	Count	42	91	133
		% within group	84,0%	91,0%	88,7%
Total		Count	50	100	150
		% within group	100,0%	100,0%	100,0%

Chi-Square Tests					
	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1,625[a]	1	,202		
Continuity Correction[b]	1,003	1	,316		
Likelihood Ratio	1,554	1	,213		
Fisher's Exact Test				,274	,158
Linear-by-Linear Association	1,615	1	,204		
N of Valid Cases	150				
a. 0 cells (0,0%) have expected count less than 5. The minimum expected count is 5,67.					
b. Computed only for a 2x2 table					

Table 2

Crosstab					
			Groups		Total
			Control	HBV and HCV	
Blood groups	O(I)	Count	13	37	50
		% within group	26,0%	37,0%	33,3%
	A(II)	Count	18	33	51
		% within group	36,0%	33,0%	34,0%
	B(III)	Count	10	20	30
		% within group	20,0%	20,0%	20,0%
	AB(IV)	Count	9	10	19
		% within group	18,0%	10,0%	12,7%
Total		Count	50	100	150
		% within group	100,0%	100,0%	100,0%

Chi-Square Tests			
	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	2,982[a]	3	,394
Likelihood Ratio	2,947	3	,400
Linear-by-Linear Association	2,354	1	,125
N of Valid Cases	150		
a. 0 cells (0,0%) have expected count less than 5. The minimum expected count is 6,33.			

Table 3

Crosstab					
			Group		Total
			Control	HBV and HCV	
Count of pregnancies	First pregnancy	Count	32	42	74
		% within group	64,0%	42,0%	49,3%
	Multiple pregnancy	Count	18	58	76
		% within group	36,0%	58,0%	50,7%
Total		Count	50	100	150
		% within group	100,0%	100,0%	100,0%

Chi-Square Tests					
	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	6,454[a]	1	,011		
Continuity Correction[b]	5,604	1	,018		
Likelihood Ratio	6,517	1	,011		
Fisher's Exact Test				,015	,009
Linear-by-Linear Association	6,411	1	,011		
N of Valid Cases	150				
a. 0 cells (0,0%) have expected count less than 5. The minimum expected count is 24,67.					
b. Computed only for a 2x2 table					

Table 4

Crosstab					
			Group		Total
			Control	HBV and HCV	
Paritet	Nulliparous women	Count	39	58	97
		% within group	78,0%	58,0%	64,7%
	Multiparous women	Count	11	42	53
		% within group	22,0%	42,0%	35,3%
Total		Count	50	100	150
		% within group	100,0%	100,0%	100,0%

Chi-Square Tests					
	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	5,835[a]	1	,016		
Continuity Correction[b]	4,993	1	,025		
Likelihood Ratio	6,097	1	,014		
Fisher's Exact Test				,019	,012
Linear-by-Linear Association	5,797	1	,016		
N of Valid Cases	150				
a. 0 cells (0,0%) have expected count less than 5. The minimum expected count is 17,67.					
b. Computed only for a 2x2 table					

The mean values of ALT, AST enzymes in infected pregnant women were 27% ± 2.5 U /l (range 6-187) (pF = 0.026) respectively; 32.0 ± 2.6 U / l (range 3-194) (pF = 0.016) (in test group - 19.5 ± 0.7 U / l (range 11-28); 22.9 ± 0.5U / l (range 16-28). However, it should be noted that only 11.0 ± 3.1% of pregnant women with parenteral hepatitis showed an increase of ALT, 17.0 ± 3.8%- increase of AST; for 89% of patients had no ALT level changed , and for 83%- the level of AST.

Although different levels of viremia were found in the blood. According to the results of examination PZR was negative on 14 patient at virologic examination of the blood of pregnant women with HBV (25.5 ± 5.9%); viral load was <2000 IU / mL in 20 patients (36.4 ± 6.5%), viral load was 21,000 (38.2 ± 6.6%)> 2000 IU / ml in 21 pregnant women. PZR was negative in 9 HCV-infected pregnant women(20.0 ± 6.0%), 14 patients (31.1 ± 6.9%) had a viral load <4x10^5 IU / ml, 6 pregnant (13.3 ± 5.1%) - 4 x10^5 IU / ml - 8x10^5 IU / ml; high viremia (> 8x10^5 IU / ml) was found in 16 pregnant women (35.6 ± 7.1%).

In accordance with the objective of the study, lipid spectrum indices were studied in comparison in practical healthy pregnant women and in women with chronic HBV and HCV (Table 5, 6).

Table 5

Comparison of blood lipid parameters in test and basic research groups

Parameters	Groups	Number of patients (N)	Mean values	Standard Deviation	Stand Error	95% Lower bound	95% Upper bound	Min	Max	P (Fischer)	P (Mann-Whitney)
XS, mmol/l	control	50	3,78	0,81	0,11	3,55	4,01	1,80	6,00	<0,001	<0,001
	patient	100	5,71	1,80	0,18	5,35	6,07	1,88	9,90		
TG, mmol/l	Control	50	2,98	0,62	0,09	2,80	3,15	1,90	4,50	0,280	0,017
	Patient	100	2,78	1,22	0,12	2,54	3,02	0,80	6,90		
ApoA1, g/l	Control	50	3,37	1,03	0,15	3,08	3,67	1,09	5,10	<0,001	<0,001
	Patient	100	1,62	0,70	0,07	1,48	1,76	0,01	3,65		
LDL, mmol/l	Control	50	2,43	0,41	0,06	2,31	2,55	1,78	3,35	<0,001	<0,001
	Patient	100	3,52	1,58	0,16	3,21	3,83	0,30	8,80		

Table 6

Comparison of lipid parameters in HBV and HCV groups

Parameters	Groups	Number of patients (N)	Mean values	Standard Deviation	Stand Error	95% Lower bound	95% Upper bound	Min	Max	P (Fischer)	P (Mann-Whitney)
XS, mmol/l	HBV	55	5,70	1,77	0,24	5,22	6,18	1,88	9,90	0,952	1,000
	HCV	45	5,72	1,85	0,28	5,17	6,28	2,78	9,00		
TG, mmol/l	HBV	55	2,58	1,08	0,15	2,29	2,88	0,80	6,90	0,079	0,157
	HCV	45	3,01	1,33	0,20	2,61	3,41	0,91	5,70		
ApoA1, g/l	HBV	55	1,61	0,75	0,10	1,40	1,81	0,01	3,65	0,839	0,964
	HCV	45	1,64	0,65	0,10	1,44	1,83	0,01	3,50		
LDL, mmol/l	HBV	55	3,32	1,21	0,16	2,99	3,64	0,30	6,50	0,157	0,601
	HCV	45	3,77	1,94	0,29	3,19	4,35	1,20	8,80		

In practical healthy pregnant, while the mean value of TX in blood was M = 3.78 ± 0.11 mmol / l, in patients infected with hepatitis B, C this value was M = 5.71 ± 0.18 mmol / l. In infected pregnant women, the TX in blood increased by 1.5 times compared with the healthy pregnant women (F = 52,039; pF <0.001;). There was no statistical difference in this indicator for HBV and HCV groups (pF = 0.952; pU = 1,000) (Figure 1).

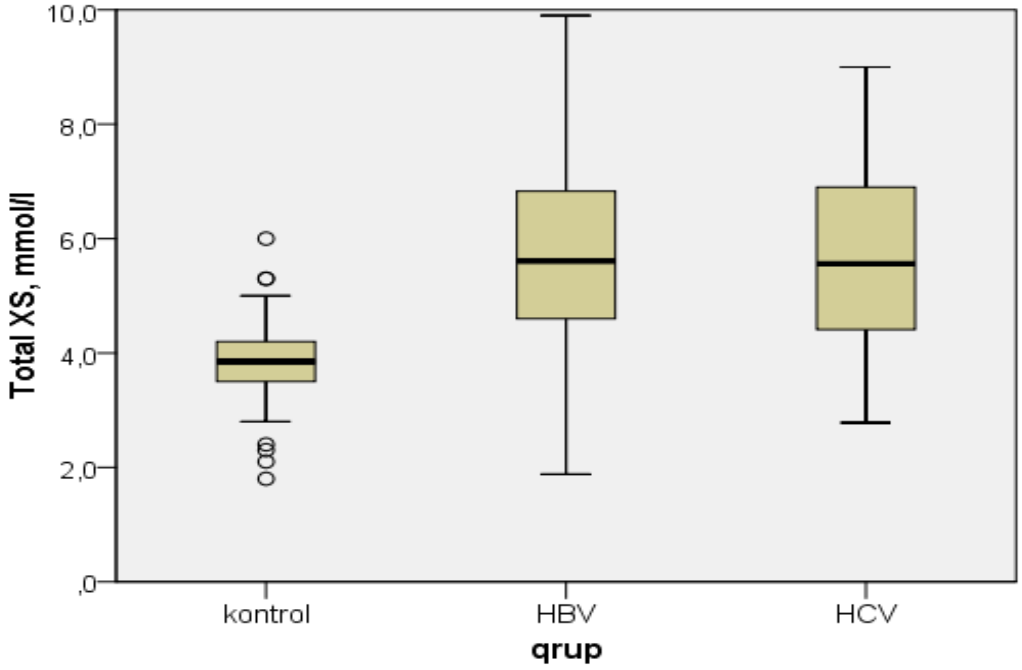

Figure 1. Total cholesterol levels in the control groups

In our study, we found a positive correlation between TX and CRZ in blood (ρ = 0.341, p = 0.001) (Figure 2)

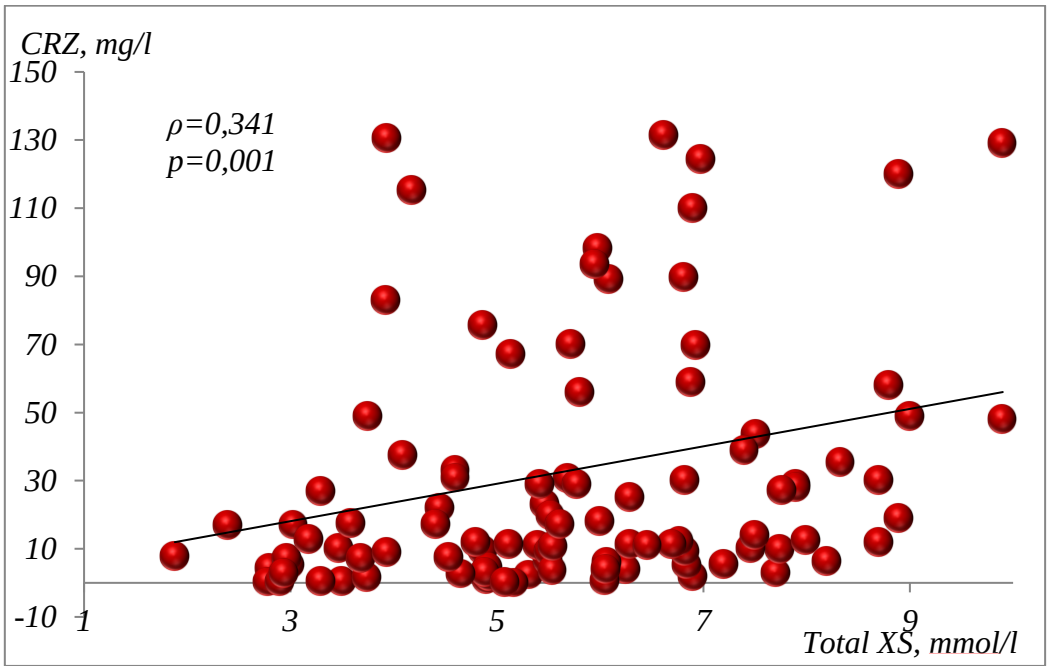

Figure 2. Correlation between total XS and CRZ in infected pregnants

In pregnant women with chronic B, C hepatitis virus, the mean value of blood TG was lower than that of practically healthy pregnant women. (2.78 ± 0.12g / l, respectively (2.98 ± 0.09g / l); pF = 0.280). As can be seen, although there was no statistically exact difference in the mean values in the examined groups, but it was possible to detect this difference in individual variables in qualitative analyzes. Thus, if only 1 person (2.0 ± 2.0%) TG norm exceeded the norm in the practically healthy group, this increase was found in the

15 pregnant women (15.0 ± 3.6%) in the infected groups. Comparison of HBV and HCV groups on TG value showed that in the HCV group the amount of blood TG was 1.17 times higher than that of the HBV group (Figure 3).

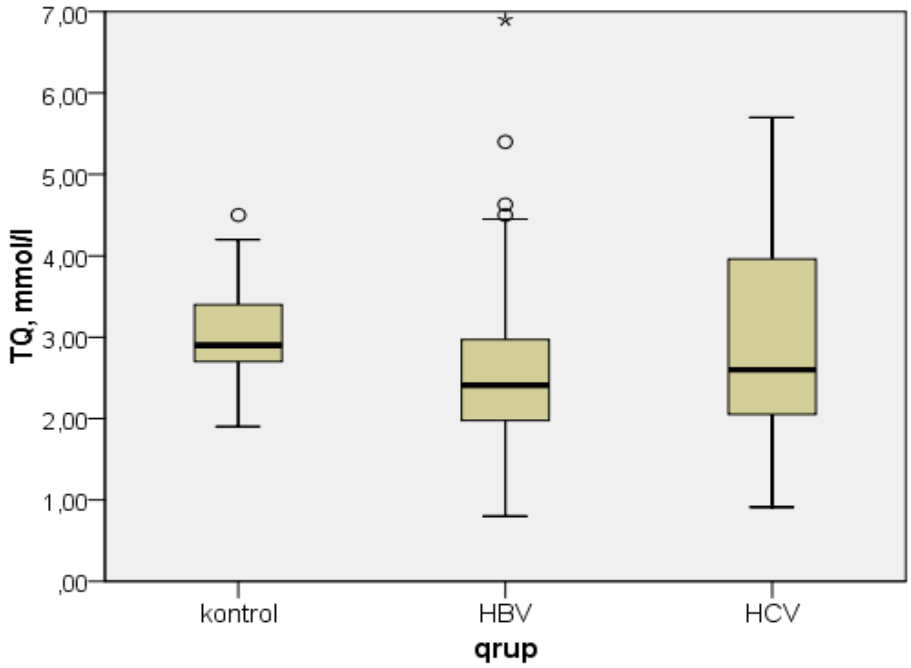

Figure 3. Triglyceride levels in the control groups

It is mentioned in the scientific sources, that (Bartels A. and co-workers, 2012) during pregnancy the level of HDL was 0,9- 3,7 mmol/l, the amount of LDL can change in intervals 1,3- 6,1 mmol/l. During our research it was determinated, that the major blood plasma respresentative LDL is ApoA1. According to the analysis the main value of ApoA1 in the second trimester of pregnancy in practically health pregnancies was M= 3,37±0,15 g/l.

The results of the study showed that level of apolipoprotein A1 in blood were reduced by 2.1 times in pregnant women infected with hepatitis B and C compared with control group (pF = 149,916; p_F <0.001; p_U <0.001) (Figure 4).

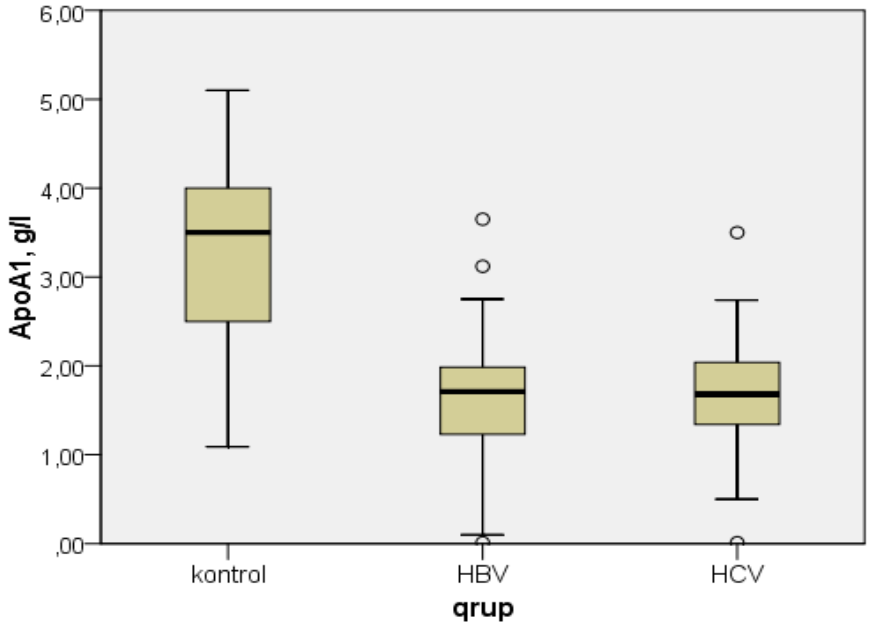

Figure 4. Level of ApolipoproteinA1 in the control groups

The comparative analysis of HDL and LDL blood serum of pregnant women with B, C infection were studied by us. It is known that the main function of ApoA1 is to deliver cholesterol, triglycerides to the tissues by releasing them from the vascular wall, and on the other hand, levels of ApoA1 can change in inflammatory processes such as acute phase protein. Scientific sources have reported that there is information on neutralization of HDL's DNA and RNA-containing viruses. It is assumed that HDL inhibits virus-induced cellular compounds. (13)

Statistical analysis of our study revealed a negative correlation between ApoA1 and the major liver transferases (ALT, AST) in the blood of pregnant women with HBV, HCV infection (with ALT ρ= -0,238, p=0,018; with AST = 00.230, *p* = 0.023).) (Figure 5,6)

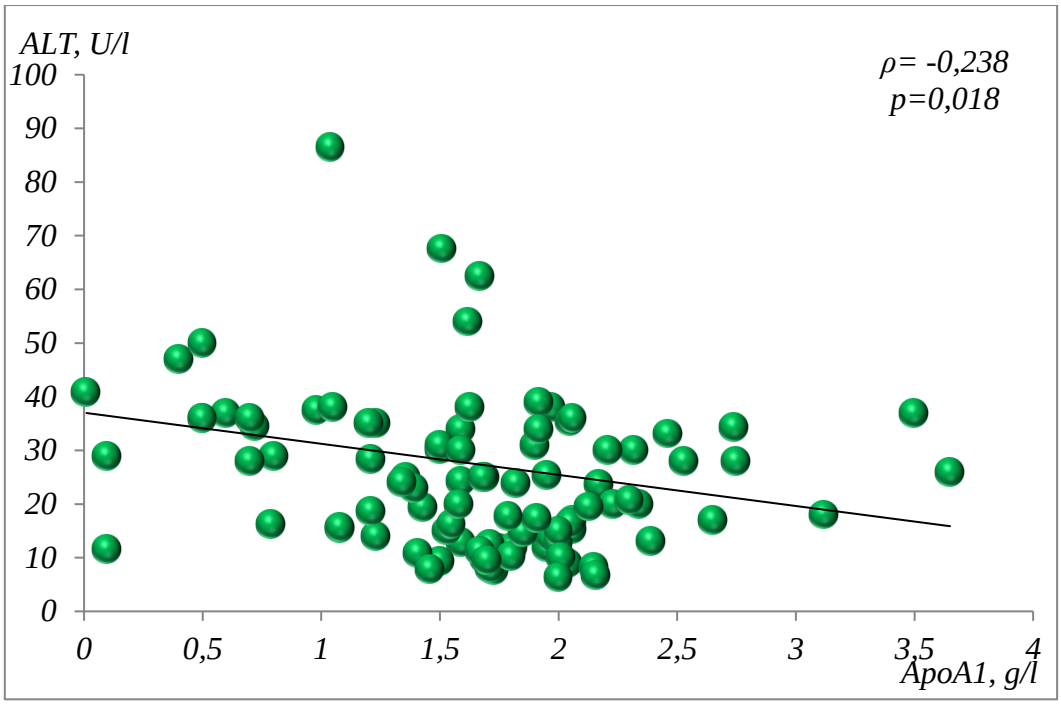

Figure.5 Correlation dependency between ALT and ApoA1 in infected pregnant women

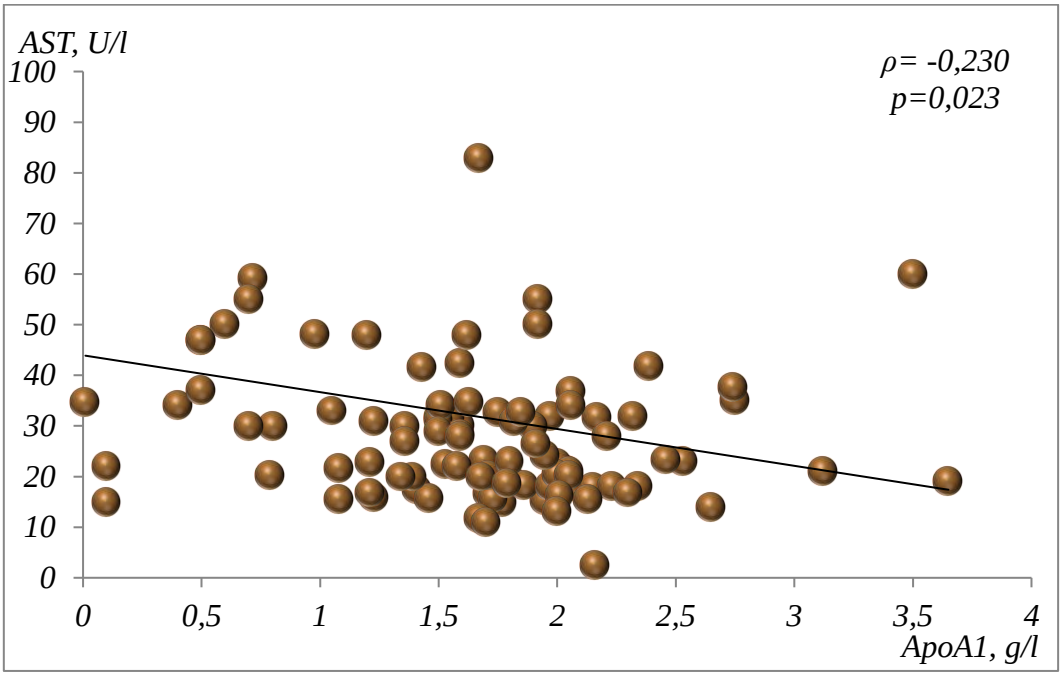

Figure 6. Correlation between AST and ApoA1 in infected pregnant women

The results of the study showed that dyslipidemia in infected pregnant manifested in the form of HDL reduction and increase of LDL. Statistical analyzes showed that LDL blood levels increased 1.4 times in pregnant women with viral hepatitis compared with the control group (p <0.001; F = 22,759; p_F <0.001; p_U <0.001) (Fig. 7).

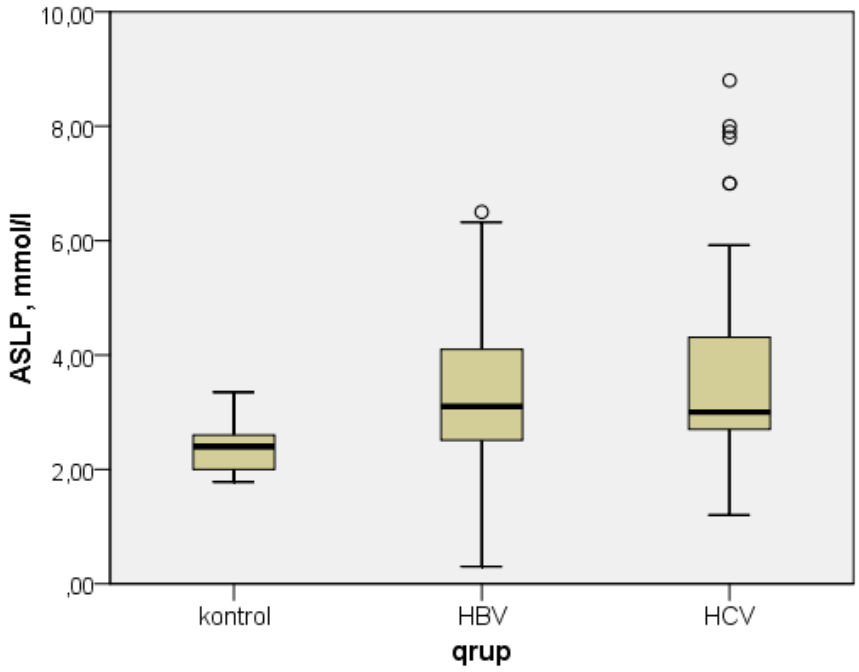

Figure 7. Level of ASHL in the control groups

Discussion: Increased levels of estrogens due to placental steroidogenesis during normal pregnancy cause a decrease in LDL. (14)

However, the increase in LDL levels in HBV and HCV-infected pregnant women can be attributed to the variability of lipid spectrum as a result of inflammatory cytokines activity caused by parenteral viruses. Recent experimental studies (Tao Wu et al. 2017) have suggested that the metabolic changes of lipids involved in the structure of hepatocyte membranes during HBV infection can lead to development of hepatitis and cirrhosis (15)

It is known that hepatitis and liver damage are not caused by the direct effects of HBV, HCV on hepatocytes, but by the immune response of the liver cells to viruses. Osboume Q et al (2019) agrees that inflammatory cytokines accelerate lipogenesis during HBV infection and increase LDL by reducing the synthesis of HDL. (16). It is worth noting that experimental studies also come up with another idea: LDL can block the incorporation of HCV (in vitro) into human fibroblasts. (17). The authors hypothesize that increased LDLs may serve to cure from HCV infection.

From our findings, we conclude that HBV and HCV infections cause dyslipidemia due to metabolic changes in the liver, resulting in increased levels of TX, TG, and LDL in infected pregnancies, and decreased HDL levels. According to some scientists, LDL receptors play a major role in the transition of HCV to the liver cell. It is believed that antibodies against LDL-receptors can block HCV cell migration. Studies of these scientists show that the reduction of LDL-receptors leads to a weakening of the infectious properties of virions. (18,19,20). Studies conducted by perinatologists indicate that pathological pregnancy results were higher in pregnant women with XS, TG, LDL over 75 percentile and lower than 25 percentile of HDL in blood during less pregnancy period . For example, studies by Wang C., and colleagues (2017) show that premature birth was less common in pregnant women with LDL <1.89 mmol / l. (21). Given the fact that the LDL mean value in infected pregnant women in our study groups is M = 3.52 ± 0.16 mmol / l, this could have a negative effect on pregnancy outcome. Although some literature indicates the less importance of lipid spectrum determination during pregnancy, Maria E. et al. (2013) note that HDL levels in blood are directly related to reproductive outcomes. (22). According to a study by Alyse S. and colleagues (2012), pregnancy-related hypertiglyceridemia is rare case, and this can be dangerous for life of genetically predisposed patients. Clinical studies show that women who have a high blood TG with empty stomach over than 4mmol / l (up to 10mmol / l) before pregnancy should start treatment and those patients should be monitored during pregnancy because of the risk of hypercoagulation and preeclampsia. (23). In our study, we have not yet investigated the effects of dyslipidemia on pregnancy outcomes in patients with hepatitis B, C, but we think that our research work can contribute to studies in this subject.

Conclusion: Increased blood cholesterol, triglycerides, LDL levels in pregnant women with HBV and HCV, and decrease of Apolipoprotein A1, a major component of HDL compared with practical pregnant women, proves the importance of the issue of keeping those pregnant women under supervision as risk group in terms of liver related complications. The presence of dyslipidemia and main hepatic transaminases in infected pregnant women, the presence of a positive correlation between CRZ confirms the effects of HBV and HCV infections on the lipid spectrum during

pregnancy. As the scientific literature lacks information on lipid spectrum changes in pregnant women with hepatitis B and C, we believe that our research work can help to understand the pathogenesis of HBV and HCV during pregnancy.

LITERATURE:

1. Akira ENDO A historical perspective on the discovery of statins. Proc Jpn Acad Ser B Phys Biol .Sci. 2010 May 11; 86(5): 484–493. doi: 10.2183/pjab.86.484

2. Н.Б.Багорова «Холестерин и его влияние на организм» Международный научный журнал « Инновационная наука» №2 2017 ISSN 2410-6070 стр.209-210. (N.B.Bagorova "Cholesterol and its influence at the body" International magazine "Innovative science" №2 2017 ISSN 2410-6070, pp. 209-210.)

3. Geraghty AA, Alberdi G, O'Sullivan EJ, O'Brien EC, Crosbie B, Twomey PJ, McAuliffe FM Maternal and fetal blood lipid concentrations during pregnancy differ by maternal body mass index: findings from the ROLO study. BMC Pregnancy Childbirth. 2017 Oct 16;17(1):360. doi: 10.1186/s12884-017-1543-x.)

4. Grimes SB, Wild R. Effect of Pregnancy on Lipid Metabolism and Lipoprotein Levels Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000-.2018 Feb 20.)

5. Huseyin Begenik, Ahmet Cumhur Dulger, Ramazan Esen, Habib Emre Coexistence of Intrahepatic Cholestasis of Pregnancy Accompanied by Hepatitis B Flare in a Pregnant Woman and the Subsequent Development of Postpartum Thyroiditis and HBS Antigen Loss: A Case Report Journal of Medical Cases, Volume 3, Number 4, August 2012, pages 240-242.)

6. Wijarnpreecha K, Thongprayoon C, Sanguankeo A, Upala S, Ungprasert P, Cheungpasitporn W Hepatitis C infection and intrahepatic cholestasis of pregnancy: A systematic review and meta-analysis. Clin. Res. Hepatol .Gastroenterol. 2017 Feb;41(1):39-45. doi: 10.1016/j.clinre.2016.07.004. Epub 2016 Aug 16

7. Дадашева А.Э. «Инфекции, вызванные вирусами гепатитов В и С, у иммунокомпрометированных лиц из групп С высоким риском инфицирования» Автореф.дисс….док. мед.наук: Баку,2015. -48 стр.(7. Dadasheva A.E. "Infections caused by the hepatitis B and C viruses in immunocompromised individuals from groups with high risk of infection" Abstract of a thesis.... doc. of medical science: Baku, 2015. p. 48)

8. Əlifəttahzadə S.Ə. “ Azərbaycanda yaşayan talassemiyalı xəstələrdə hepatit B və C virusları ilə törədilən infeksiyalar” Diss. ...avtoref.tibb üzrə fəlsəfə dokt: Bakı,2015.-22 səh. (Alifatahzade S.A. “Infections caused by hepatitis B and C virus in thalassemia patients living in Azerbaijan”. Abstract of PhD of medicine: Baku, 2015.p.22)

9. Агаева Г.Ш. «Метаболические изменения у больных хроническим вирусным гепатитом С и их влияние на противовирусную терапию Автореф.дисс. …док.филос.по мед.наук: Баку. 2010.- 20 стр. (Agaeva G.Sh. "Metabolic changes in patients with chronic viral hepatitis C and their effect on antiviral therapy. Author. Diss. ... doc. Phylos. In medical science: Baku. 2010 pp.- 20.)

10. Сеидалиева С.М. «Особенности течения беременности и родов у женщин с хроническими вирусными гепатитами В и С» Дисс….автореф. на соискание ученой степени доктора философии по медицинским наукам, Баку. 2011, 22с.(Seyidalieva S.M. “Features of the course of pregnancy and childbirth in women with chronic viral hepatitis B and C” Diss .... Abstract. for the degree of Doctor of Philosophy in Medical Sciences, Baku. 2011, p.22)

11.Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) NIH Publication No 01-3670; May 2001

12. Bartels Ä, Egan N, Broadhurst DI, Khashan AS, Joyce C, Stapleton M, O'Mullane J, O'Donoghue K. Maternal serum cholesterol levels are elevated from the 1st trimester of pregnancy: a cross-sectional study. J Obstet Gynaecol. 2012 Nov;32(8):747-52. doi: 10.3109/01443615.2012.714017) .

13. Гребенников И.Н., Куликов В.А. «Липопротеины высокой плотности: не только обратный транспорт холестерина » Ж. «Биохимия» Вестник ВГМУ, 2011, том10,№2 стр.12-19 (13. Grebennikov I.N., Kulikov V.A. “High-density lipoproteins: not only the reverse transport of cholesterol” J. “Biochemistry” Bulletin of the Voronezh State Medical University, 2011, volume 10, No 2, pp. 12-19)

14. Deepak Parchwani, Digisha Patel “ Status of lipid profile in pregnancy ” National Journal of Medical Research. Vol 1 Issue 1 July – Sept 2011 : ISSN 2249 -4995 Page 10-12

15. Tao Wu, Xiaojiao Zheng, Ming Yang, Aihua Zhao, Meng Li, Tianlu Chen, Jun Panee, Wei Jia, andGuang Ji Serum lipid alterations identified in chronic hepatitis B, hepatitis B virus-associated cirrhosis and carcinoma patients Sci Rep. 2017; 7: 42710. doi: 10.1038/srep 42710

16. Osbourne Quaye, Benjamin Godfried Amuzu, Samuel Mawuli Adadey, and Emmanuel Ayitey Tagoe “Effect of Hepatitis (HBV) infection on lipid profile in Ghanaian patients” J. Virology: Research and Treatment “Volume 10: 1–5, 2019 ; DOI: 10.1177/1178122X19827606). 15. Alyse S. Goldberg Robert A. Hegele Severe Hypertriglyceridemia in Pregnancy . The Journal of Clinical Endocrinology & Metabolism, Volume 97, Issue 8, 1 August 2012, Pages 2589–2596, https://doi.org/10.1210/jc.2012-1250

17. Monazahian M. Bohme I, Bonk S., Koch A, Scholz C, Grethe S, Thomesen R. “Low density lipoprotein reseptor as a candidate reseptor for hepatitis C virus” J. Med. Virol. 1999. Mar;57 (3) 223-9.

18. Agnello V, Abel G, Elfahal M, Knight GB, Zhang QX. Hepatitis C virus and other flaviviridae

viruses enter cells via low density lipoprotein receptor. Proc Natl Acad Sci USA. 1999;96:12766–12771131.

19. Molina S, Castet V, Fournier-Wirth C, Pichard-Garcia L, Avner R, Harats D, Roitelman J, Barbaras R, Graber P, Ghersa P, Smolarsky M, Funaro A, Malavasi F, Larrey D, Coste J, Fabre JM, Sa-Cunha A, Maurel P. The low-density lipoprotein receptor plays a role in the infection of primary human hepatocytes by hepatitis C virus. J Hepatol. 2007;46:411–419

20. Owen DM, Huang H, Ye J, Gale M., Jr Apolipoprotein E on hepatitis C virion facilitates infection through interaction with low-density lipoprotein receptor. Virology. 2009; 394:99–108

21. Wang C., Zhu W., Wei Y., Su R., Feng H., Hadar E., Hod M. The associations between early pregnancy lipid profiles and pregnancy outcomes. J. Perinatol. 2017 Feb;37(2):127-133. doi: 10.1038/jp.2016.191. Epub 2016 Oct 27 .

22. Maria E. Baardman Wilhelmina S. Kerstjens-Frederikse Rolf M.F. BergerMarian K. Bakker Robert M.W. Hofstra Torsten Plösch The Role of Maternal-Fetal Cholesterol Transport in Early Fetal Life: Current Insights.

Biology of Reproduction, Volume 88, Issue 1, 1 January 2013, 24, 1-9,https://doi.org/10.1095/biolreprod.112.102442

23. Alyse S. Goldberg Robert A. Hegele Severe Hypertriglyceridemia in Pregnancy . The Journal of Clinical Endocrinology & Metabolism, Volume 97, Issue 8, 1 August 2012, Pages 2589–2596, https://doi.org/10.1210/jc.2012-1250

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