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Liver Steatosis

The features of carbohydrate and lipid metabolism in patients co-infected with HIV/HCV

K. Iurko[1]. V. Kozko[1], A. Adeyemi[1]

[1]Kharkov national medical university, infectious diseases, Kharkov, Ukraine

Background: Among the factors indicative of the progression of chronic hepatitis C (CHC), the leading position belongs to hepatic steatosis and insulin resistance (IR), which may be virus-induced, and metabolic. The main risk factors for the metabolic syndrome in HIV-infected individuals are high viral load, use of drugs lopinavir/ritonavir and didanosine, increased body weight and levels of Low density lipoproteins (LDL) and/or triglyceride (TG) levels, patient age, and co-infection with human immunodeficiency virus (HIV) and hepatitis C virus (HCV). Thus, HCV-infection in HIV-infected individuals is one of the major risk factors for metabolic disorders.

Materials & Methods: The study of insulin in the blood serum was conducted with immunofluorescence assay, the determination of glycosylated hemoglobin (HbA1C) was carried out by ion-exchange chromatography. Determination of glucose in the blood serum was carried out by a colorimetric method using a reagent kit from the company 'SpaynLab' (Spain). The HOMA IR index was determined, which was calculated by the formula: [(fasting glucose) x (fasting insulin)] mmol/l / 22.5.
The study of lipid metabolism of blood (total cholesterol (TC), TG, High-density lipoproteins (HDL), LDL) was carried out by the enzymatically-colorimetric method with diagnostic kits from the company 'SpaynLab' (Spain). The content of Very low density lipoproteins (VLDL) in blood serum was determined by the formula: VLDL=TG/5. Atherogenic coefficient (AC) was calculated by the formula: AC=(TC-HDL)/HDL. Statistical analysis was performed using the software package «Statistica for Windows», 8.0.

Results: In the investigated patients identified carbohydrate metabolism disorders as an increase in serum glucose, insulin, HbA1C, level of HOMA IR. The greatest manifestation of disorders of carbohydrate metabolism was established in patients co-infected with HIV/HCV, and the lowest - in HIV-infected individuals.
TC in patients of all groups had no significant difference with that of the control group in patients. The patients studied, compared to the control, there was a significant increase of TG, AC, LDL, VLDL and reduction HDL. Significantly higher levels of TG ($p<0.001$) was observed in patients co-infected with HIV/HCV compared to patients with chronic hepatitis C and HIV separately.

Conclusions: In HIV-infected patients with chronic hepatitis C and co-infection with HIV/HCV significant increases in serum glucose, insulin, HbA1C and HOMA IR index values were observed. This indicates a violation of carbohydrate metabolism in patients examined. The most significant manifestations of disorders of carbohydrate metabolism were observed in patients co-infected with HIV/HCV ($t=27,4$; $p<0.001$), which exceeds the specified changes in patients with CHC in 1.53 times ($t=17,9$; $p<0.001$) and HIV-infected patients ($t=12,8$; $p<0.001$) 2.14 times. The studied patients had lipid metabolism disorders, namely increases in the serum TG, AC, LDL, VLDL and HDL reduction. Significantly higher TG levels ($p<0.001$) was observed in patients co-infected with HIV/HCV compared to patients with CHC and HIV separately.

No conflict of interest