difference (p <0.05). It was found that in patients with NASH F0 stage of fibrosis was registered in 35.7% against 16.7% in NASH with DIOS (p <0.05). F1 stage was registered in 38.1% of patients with NASH against 27.7% of cases of NASH with DIOS (p> 0.05). In patients with NASH F2, the stage of fibrosis was registered in 23.8% against 38.9% in NASH with DIOS (p <0.05). At the same time, F3 stage of fibrosis in patients with NASH was registered in 2.4% against 16.7% in NASH with DIOS (p <0.05).

Thus, in patients with NASH, the following patterns of liver fibrosis were established: activation of collagen synthesis processes (in the presence of DIOS 1.6 times (p <0.05), in the absence - 1.3 times (p <0.05)), a slight increase the intensity of collagen breakdown in NASH with DIOS- 1.2 times (p> 0.05); increase in CLA by 13.8% (p <0.05) for DIOS, however, in its absence, CLA in NASH was reduced by 21.3% (p <0.05). For patients with NASH is characterized by an increase in the content of hexosamines in the blood: for DIOS in 1.3 times (p <0.05) against 1.2 times (p <0.05), the content of sialic acids, respectively - in 1.4 against 1, 2 times (p <0.05), and accelerated degradation of fucoglycoproteins (1.8 to 1.6 times, respectively) (p <0.05). The consequence of the registered processes was an increase in the integrated Fibro-test for NASH with DIOS- 2.1 times compared to the indicator in PHIs (p <0.05), for NASH without DIOS- 1.6 times (p <0.05).

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THE COINFLUENCE OF THE STATE OF THE BLOOD LIPID SPECTRUM AND CONTENT OF ADIPOKINES ON THE CLINICAL COURSE OF NON-ALCOHOLIC FATTY LIVER DISEASE IN THE PRESENCE OF COMORBID CHRONIC KIDNEY DISEASE

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The purpose of the study was to find out the probable mutual influence of the state of the blood lipid spectrum of and content of adipokines in blood: leptin, adiponectin on the clinical course of non-alcoholic fatty liver disease combined with obesity depending on its form and the presence of comorbid chronic kidney disease. 444 patients were examined: of which 84 patients with non-alcoholic fatty liver disease with grade I obesity (group 1), which contained 2 subgroups: 32 patients with non-alcoholic hepatic steatosis and 52 patients with non-alcoholic steatohepatitis; 270 patients with non-alcoholic fatty liver disease with comorbid obesity of the I degree and chronic kidney disease of the I-III stage (group 2), including 110 patients with non-alcoholic steatosis of the liver and 160 patients with non-alcoholic steatohepatitis. The control group consisted of 90 patients with chronic kidney disease stage I-III with normal body weight (group 3). The mean age of patients was (45.8±3.81) years.

The study shows that patients with non-alcoholic steatohepatitis and obesity without concomitant chronic kidney disease are characterized by the following changes in the blood lipid spectrum: maximum increase in blood triacylglycerols (by 2.1 times, p <0.05), a probable increase in total cholesterol (by 1.4 times, p <0.05) and proatherogenic low-density lipoproteins (by 1.6 times, p <0.05), a probable decrease in anti-atherogenic high-density lipoproteins (by 1.6 times, p <0.05), which with the addition of comorbid chronic kidney disease are likely to deepen (within 1.5-1.8 times, p <0.05), in addition to hyper triacylglycerol. According to the results of the study, the content of leptin in the blood was significantly increased by 1.4 times (p <0.05) compared with almost healthy individuals, which differed significantly from patients with non-alcoholic steatosis of the liver with chronic kidney disease and non-alcoholic steatohepatitis with chronic kidney disease (p <0.05). The content of adiponectin in the blood was significantly reduced by 1.4 times compared with almost healthy individuals (p <0.05) and also differed significantly from patients with non-alcoholic hepatic steatosis with chronic kidney disease and non-alcoholic steatohepatitis with chronic kidney disease (p <0.05).

Based on the results, it was found that significant metabolic prerequisites for the development of non-alcoholic steatohepatitis against the background of obesity and chronic kidney

disease are probable postprandial hyperglycemia, hyperinsulinemia, increased glycosylation of hemoglobin. Hyperleptinemia and hypoadiponectinemia are also factors in the burden of non-alcoholic steatohepatitis and obesity due to the progression of mesenchymal inflammation and cytolysis of hepatocytes.

Biriuk I.G.

PECULIARITIES OF DEVELOPMENT OF THE COLON TOPOGRAPHY AT THE END OF THE FETAL PERIOD OF HUMAN ONTOGENESIS

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At the current stage of development of abdominal surgery accurate data concerning individual anatomical peculiarities of sizes, shape and location of the colon are essential. A number of issues dealing with causes stipulating development of pathological processes of both the colon and abdominal organs on the whole remain unclear. The size of the colon and its spatial interrelations with adjacent complexes of organs and the abdominal wall influences on the development of its topography. Meanwhile, any disorders of such interrelations can become a morphological precondition promoting formation of developmental defects of this intestinal portion.

A doctor of any specialty facing certain pathological signs of developmental defects in the abdominal organs in children should understand the fact that genesis of an ailment is of congenital character and it requires conceptual ways to correct defects in children.

The research was carried out on 37 specimens of human neonates by means of the methods of macroscopy, thin section under the microscope МБС-10 control, and radiography. Examination of the colon found that its ascending portion was located in the right lateral portion of the abdominal cavity passing from the ceacum to the hepatic flexure. In 21 cases it was pressed against the lateral abdominal wall, that is, was located in the lateral position. In 10 cases the ascending portion of the colon was located proximally, that is, it was displaced to the side of the middle line, and in 6 cases it was located in the middle towards the right lateral abdominal wall and the middle line. Practically in all the cases the ascending portion of the colon was located in the mesoperitoneal position. Its length from the ileac fossa to the liver was from 54,5 mm to 78,0 mm.

Examination of the hepatic flexure of the colon detected three main positions towards the inferior border of the liver: 1. On 19 specimens the right flexure of the colon arose from the inferior border of the liver. 2. On 12 specimens it was located under the inferior liver border. 3. In 6 cases the flexure was half covered with the inferior liver border.

The transverse portion of the colon on the material examined was directed from the right to the left and a little distally. Close to the left lateral region of the abdominal cavity it formed left or splenic flexure. The length of the transverse portion of the colon changes within the limits from 118,0 to 200,5 mm. The transverse portion of the colon on the fetal specimens has two main positions: 1. Superior (on 21 specimens) – in its middle part the transverse portion of the colon touched the inferior border of the liver. 2. Inferior (in 16 cases) – the middle portion of the colon deflects to the umbilicus. In the majority of cases the colon is rather mobile and possesses its own mesentery from 24,5 to 43,5 mm long. The inferior border of the spleen is adjacent to the posterior border of the splenic flexure. On the majority of specimens this flexure (24 specimens) arose from under the left liver lobe, and in others (13 cases) – it was covered by this lobe.

Examination of the descending portion of the colon found that it was located in the left lateral side of the abdominal cavity. The descending portion is from 44,5 to 87,5 mm long. In the majority of cases (25 specimens) this portion of the colon similar to the ascending portion is located in the mesoperitoneal position. Meanwhile, in 12 cases the inferior part of the descending portion of the colon had its mesentery. Close to the mesenteric crest the descending portion of the colon passes into the sigmoid one.

Thus, the shape and size of the sigmoid at the end of the fetal period of human ontogenesis are individually variable. Filling of the colon with meconium produces a substantial effect on its position, mobility, diameter and color of this intestinal portion.