

Influence of Childhood Obesity on the Hepatobiliary System

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Abstract: Of the children we examined, 58 were obese and 20 had normal weight. We conducted a series of complex tests to determine their obesity and the state of the hepatobiliary system. The tests we conducted consisted of clinical, laboratory and instrumental studies. In this case, we conducted a series of biochemical analyses, determination of carbohydrates and lipids in the blood serum, and ultrasound examination of the hepatobiliary system. Studies have shown that most children with obesity have non-alcoholic fatty liver disease and an increase in liver enzymes by one third, as well as changes in the colloidal composition of bile.

Keywords: children with obesity, hepatobiliary system, metabolic syndrome.

Of the children we examined, 58 were obese and 20 had normal weight. We conducted a series of comprehensive examinations to determine the state of the hepatobiliary system in obesity and normal conditions. The examinations we conducted included clinical, laboratory and instrumental studies. We conducted a series of biochemical analyses, determination of the carbohydrate and lipid spectrum of blood serum, and ultrasound examination of the hepatobiliary system. As a result of the examinations, we found that most children with obesity had non-alcoholic fatty liver disease, and a third had an increase in the level of liver enzymes and a change in the colloidal composition of bile.

Introduction. Obesity in children is a symptom complex of various metabolic disorders and conditions associated with obesity, the only pathogenetic mechanism for the development of this pathological condition, which has been in the center of attention of representatives of various fields of medicine in recent years. Despite the successes achieved in the mechanism of its development, metabolic syndrome is one of the most controversial issues in modern medicine [1,2]. Today, in this regard, the symptom complex begins to form in childhood and remains asymptomatic for a long time, obesity and metabolic syndrome in children are a pressing problem [3, 4]. Today, the conclusions have been proven that the digestive system organs play a key role in the pathogenesis of metabolic and hormonal disorders, in which they themselves become target organs, and with increasing obesity, the condition of the digestive system organs worsens [5, 6, 7, 8]. It has been established that any component of metabolic syndrome leads to secondary disorders in the form of non-alcoholic fatty liver disease in adults, while views on this pathological process differ: some authors consider liver steatosis to be a "harmless condition" that does not affect the state of hepatocytes to one degree or another and disappears after the elimination of the etiological factor [9]. Other authors believe that liver damage is an important factor in the development of metabolic syndrome, and with the development of nonalcoholic fatty liver disease, bile formation and bile secretory activity are impaired, which is manifested in the state of the biliary system [10]. Among children, 68% of children with obesity are diagnosed with non-alcoholic fatty liver disease, and 84% of children with metabolic syndrome are diagnosed with non-alcoholic fatty liver disease [11]. The purpose of this study is to examine the impact of obesity on the hepatobiliary system of children.

Materials and methods of the study

78 children aged 9 to 16 years were examined: the main group consisted of 58 children (31 boys, 27 girls) with signs of metabolic syndrome (IDF, 2007), [14], the comparison group - 20 children with normal body weight (12 boys, 8 girls), who were treated in hospital for diseases of the hepatobiliary system (dysfunction of the biliary tract, cholelithiasis). The examination was carried out on the basis of

clinical, laboratory and instrumental research methods and included the determination of renal enzymes, carbohydrate and lipid spectrum of blood serum, determination of contractile activity of the organs of the hepatobiliary system, UTT, computed tomography. Statistical analysis of the results was carried out using the STATISTICS 6.0 package. The reliability of the difference in quantitative characteristics was calculated using the Student t-test and the Mann-Whitney criterion. The chi-square test was used to analyze the statistical significance of the difference in qualitative characteristics. When comparing the difference indicators, the indicator r < 0.05 was considered reliable.

Examination results and their analysis:

According to the criteria of instrumental studies (increased exogeneity of the organ parenchyma and diffuse unevenness, opacity of the vascular pattern, refraction of ultrasound rays), non-alcoholic fatty liver disease was observed mainly in children of the main group (38-66.6%). Computed tomography of 23 patients with non-alcoholic fatty liver disease showed that the liver parenchyma is uneven, the densitometric density of the unchanged area ranged from 55 to 60 H. units, the local density of individual foci ranged from 15 to 43 H. units, whose contours were not determined in all zones during local examination. When studying the pulse sequence using SPAIR, a low signal was observed in hypodense zones. At all stages of contrast enhancement, including during examination with a scanner, minor changes in their density were noted in the parenchyma, which did not change. At all stages of contrast enhancement during a delayed examination, their density did not differ significantly in the parenchyma areas, which did not change. It was found that children in the main group had a higher average level of alanine aminotransferase, bilirubin, gamma-glutamyl transpeptidase than in the comparison group, which indicates a violation of the process of bile formation and excretion (Table 1). It should be noted that 22 (37.9%) children in the main group had an increase in ALT levels, in 3 (11.1%) of them their indicators increased by 2 or more times, which indicates the development of an inflammatory process such as non-alcoholic steatohepatitis in the liver parenchyma. Half of the children in all groups had various anomalies in the development of the bile ducts. Structural changes in the gallbladder wall were detected in 27 (47.2%) children of the main group and in 3 (15.0%) of the comparison group (r<0.05); in all groups, these changes were diffuse (up to 99%). Cholestasis of the gallbladder with preserved lithogenic effect was observed only in 3 (5.5%) of 14 patients of the main group; all the others had a concomitant disease of cholelithiasis at different stages. It should be noted that 17 (33.3%) children of the main group and 7 (30.0%) children of the comparison group (children with cholelithiasis were examined) had a violation of the lithogenic effect of the bile composition, accompanied by the formation of bile deposits of varying severity and gallstones. In the comparison group, cholelithiasis was most common - 6 out of 58 (11.0%) in the main group and 8 out of 20 (40%) in the comparison group (p < 0.05); bile deposits were observed in 48 (83.3%) in the main group and 5 (25%) in the comparison group (p < 0.05). No significant differences were found in the presence of one or more gallstones in the study group: in the main group, these figures were 2 (10%) and 4 (20%), in the comparison group - 10 (30.5%) and 8 (40%). The effect of impaired lithogenic effect of the drug on waist circumference was analyzed. It was found that the waist circumference in children with bile deposits averaged 109.7 ± 6.3 cm, which is significantly higher than in children with gallstones (93.5 \pm 4.1 cm) (r < 0.05) and in children with unchanged colloidal composition of bile (100.6 \pm 9.1 cm) (p < 0.05), indicating that children with abdominal obesity are at risk for developing gallstones. It turned out that children with non-alcoholic fatty liver disease more often had gallstone disease of different stages than children without hepatosis (15 of 36 children (41.6%) and 2 of 20 children (10%) p < 0.05).

Conclusion:

66.6% of obese children show signs of non-alcoholic fatty liver disease, in which structural changes in the liver parenchyma are characterized by diffuse focal changes, accompanied by the development of steatohepatitis against the background of foci of fatty degeneration of various sizes and unchanged parenchyma. It was noted that in children with metabolic syndrome, the indicators of cytolytic enzymes and cholestasis markers were higher than in children with normal body weight, which indicates a sharp disruption in the process of bile formation and excretion of bile.

Every third (33.3%) child with obesity showed an increase in ALT, which indicates the development of non-alcoholic steatohepatitis. In children with metabolic syndrome, concomitant severe disorders of the functional state of the biliary tract are formed, in which, along with the physicochemical composition of bile, its motor-evacuation activity is also disrupted. Every third (36.7%) obese child has abnormalities in the colloidal composition of bile, which manifests itself in the form of cholelithiasis with a predominance of the stone formation stage. Every third (33.3%) obese child has signs of diffuse cholestasis of the gallbladder, which is accompanied by cholelithiasis at various stages. It has been established that there is a correlation between abnormalities in the lithogenic composition of bile and waist circumference, which indicates that children with abdominal obesity may be predisposed to the development of cholelithiasis and are prone to timely preventive measures.

Иктибослар | Сноски | References:

- 1. Диденко В.И. Современные достижение в оценке стеатоза печени. ГУ «Институт гастроэнтерологии НАМН Украины», г.Днепропетровск Журнал «Гастроэнтерология» № 3 (57) 2015г.стр.94-100
- 2. Бокова Т., Урсова Н. Состояние гепатобилиарной системы у детей с ожирением и метаболическим синдромом. Журнал «Гастроэторология» №1, 2011. стр.59-62
- 3. Дворяковская Г.М., Кутырева Е.Н. Неалкогольная жировая болезнь печени у детей по данным ультразвукавой диагностики. Журнал «Гастроэторология» №4, 2012г. стр. 53-62.
- 4. Гусева А.А., Гурова М.М., Состояние гепатобилиарной системы и клиникометаболические особенности у детей с избытком массы тела и ожирением / С.П. Гусева, И.В. Зоря // Актуальные проблемы педиатрической науки и практики: материалы Всерос. конф. с междунар. участием, посвящ.
- 5. Бокова Т. А. Метаболический синдром у детей: особенности формирования и клинического течения, подходы к диагностике, профилактике и лечению. автореф. дис. докт. мед. наук. Москва, 2014. 48 с.
- 6. Леонтьева И. В. Диагностика и лечение метаболического синдрома в практике педиатра // Доктор. Ру. 2011. № 2. С. 13–23.
- 7. Синицын П. А., Щербакова М. Ю., Ларионова В. И. Метаболический синдром у детей и подростков. Клинико-генетические параллели. LAP LAMBERT Academic Publishing, 2011. 153 с.
- 8. Nelson R. A., Bremer A. A. Insulin resistance and metabolic syndrome in the pediatric population // Metab. Syndr. Relat. Disord. 2010. Vol. 8. № 1. P. 1–14.
- 9. Лазебник Л. Б., Звенигородская Л. А. Метаболический синдром и органы пищеварения. М.: Анархис, 2009. 184 с.
- 10. Павловская Е. В., Строкова Т. В., Каганов В. С. Метаболический синдром у детей и подростков. Взгляд гастроэнтеролога // Вопросы детской диетологии. 2012. Т. 10. № 5. С. 50–56.
- 11. Stephen A. Harisson. Повышение активности печеночных ферментов и жировая дистрофия печени // Клиническая гастроэнтерология и гепатология. 2009. Т. 2. № 3. С. 26—31.
- 12. Fu J. F., Shi H. B., Liu L. R. et al. Non-alcoholic fatty liver disease: An early mediator predicting metabolic syndrome in obese children?//World J. Gastroenterol. 2011. Vol. 14. № 17 (6). P.735–742.
- 13. Звенигородская Л. А., Овсянникова О. Н., Хомерики С. Г. Холестероз желчного пузыря и атерогенная дислипидемия: этиология, патогенез, клинические проявления, диагностика, лечение // Эффективная фармакотерапия. 2012. № 2. С. 8–13.
- 14. Григорьева Н. И. Желчнокаменная болезнь: ещё один компонент метаболического синдрома? // Медицинский совет. 2012. № 9. С. 92–93