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LIPID METABOLISM PARAMETERS AND LEVELS OF ANTIOXIDANTS IN MONGOLOID GIRLS WITH OBESITY

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to analyze the state of lipid metabolism and antioxidant status in Mongoloid girls with obesity Studies were conducted in 22 girls (mean age – 15.06 ± 1.53 years) with the first degree exogenously constitutional obesity of and in 48 girls of control group (mean age – 14.25 ± 2.42 years). All girls by ethnicity were Mongoloids. Lipid components (total cholesterol, triglycerides, high-density lipoproteins, low-density lipoproteins) and components of antioxidant defense (total antioxidant activity, α -tocopherol, retinol, reduced and oxidized glutathione, superoxide dismutase activity, glutathione peroxidase activity, glutathione reductase activity and glutathione S-transferase activity) in the blood were determined. Spectrophotometric and fluorometric methods were used.

Mongoloid girls with obesity had higher values of total cholesterol (1.22 times higher, $p=0.017$), triglycerides (2.16 times higher, $p<0.0001$) and lower values of HDL (1.26 times, $p=0.0018$), compare to the control. In the antioxidant defense system a decrease in α -tocopherol (1.41 times, $p=0.0262$), retinol (1.12 times, $p=0.0306$), superoxide dismutase activity

(1.28 times, $p=0.0004$) and glutathione S-transferase activity (1.71 times, $p<0.0001$) were noted in comparison with the control group with the absence of statistically significant changes in other components.

The study revealed changes in lipid metabolism and antioxidant defense parameters in Mongoloid girls with exogenously constitutional obesity, consisting of the presence of dyslipidemia, a decrease in fat-soluble vitamins and antioxidant-enzymes activity. Based on results obtained corrective measures recommended for Mongoloid girls with exogenously constitutional obesity to stabilize the lipid metabolism and antioxidant status by increasing the content of products containing polyunsaturated fatty acids in the diet and administration of antioxidants complex.

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CLINICAL AND GENETICAL CHARACTERISTICS OF CELIAC DISEASE IN KOSOVAR CHILDREN

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Coeliac disease develops in genetically susceptible individuals who, in response to unknown environmental factors, develop an immune response that is subsequently triggered by the ingestion of gluten. The disease has many clinical manifestations, ranging from severe malabsorption to minimally symptomatic or non-symptomatic presentations. The aim of this study is to evaluate the genetical and clinical presenting patterns of celiac disease in pediatric patients from Kosovo.

Study subject were children aged 0-18 treated for celiac disease at Pediatric Clinic, University Clinical Center of Kosovo (UCCCK), as a referral center for celiac disease. The diagnosis of celiac disease was established according to the

90's or 2012 ESPGHAN criteria. Personal data and clinical presentation at time of diagnosis were collected through individual interviews.

A total of 60 unrelated patients with celiac disease were evaluated.

Children's age at diagnosis ranged from 17 month to 18 years, with a mean age 5.5 years ($SD \pm 3.31$). The classical celiac disease presented with (diarrhea, abdominal distention and weight loss) occurred in 78% of the cases while non-classical form in 22% ($p<0.001$). Presenting features were diarrhea, abdominal distention and weight loss, each with 47 cases (78%), abdominal pain ($n = 42$, 70%), anorexia ($n = 48$, 80%); vomiting ($n = 28$, 46%), anemia ($n = 46$, 76%), constipation ($n = 14$, 23%), failure to thrive ($n = 17$, 28%), short stature ($n = 18$, 30%). All children were positive for IgA-tTG, while 52 of them (87%) had tTG titers levels >10 times of upper limit of normal. The HLA-DQ heterodimer pattern of these 60 patients was: heterozygous DQ2.5 heterodimer ($n = 35$), heterozygous DQ2.5/DQ2.2 heterodimer ($n = 12$), homozygous DQ2.5 heterodimer ($n = 3$), heterozygous DQ2.5/DQ8 heterodimer ($n = 3$), heterozygous DQ8 heterodimer ($n = 2$), heterozygous DQ2.2 heterodimer ($n = 2$). Three patients, biopsy proven, were negative for DQ2.5, DQ2.2 and DQ8 heterodimers. In the present study, 18 (30.0%) patients had a dual dosage of HLA-DQ-susceptible genotypes; we found no correlation between HLA double dose and/or tTG IgA level with clinical presentation.

Children diagnosed as having celiac disease in Pediatric Clinic at the University Clinical Centre of Kosovo, presented namely with gastrointestinal manifestations and contains very few patients with atypical symptoms. While there was found no correlation between HLA double dose and/or tTG IgA level with clinical presentation.

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THE ROLE OF RAPID QUALITATIVE IMMUNOCHROMATOGRAPHIC ANTIBODY TEST FOR SCREENING OF COELIAC DISEASE IN CHILDREN

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Coeliac disease (CD) is an immune-mediated inflammatory disease triggered by dietary gluten and related proteins in genetically predisposed individuals.

Standard serology testing and small bowel biopsy are used to establish the diagnosis of CD. New and improved point-of-care (POC) methods are non-invasive and could help reduce the diagnostic delay of CD. We aimed to determine the prevalence (using rapid POC test) and clinical characteristics of CD found by screening in first-grade schoolchildren.

A rapid qualitative immunoassay POC test (Simtomax Blood Drop DGP) designed for detection of immunoglobulin (Ig) A and IgG deamidated gliadin antibodies (DGP) as well as total IgA (to identify IgA deficient patients) in whole blood was used to test healthy, first-grade schoolchildren who consumed gluten regularly without restrictions. All children with positive POC test were examined by paediatric gastroenterologist and referred to do total IgA and coeliac serology testing – IgA antibodies against type-2 tissue transglutaminase (tTG) in IgA sufficient children and IgG DGP in those with IgA deficiency. Finally, the diagnosis of CD was established according to

diagnostic guidelines of European Society for Paediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN).

Out of 1404 children (51% female, mean age 7.23 y) tested, 85 (6.05%) had positive rapid POC test. All those were referred to do serology testing but

2 were excluded from the analysis as were older than first-graders and 4 refused. Out of 79 tested, 8 had positive IgA tTG (6 with values of more than 10x upper limit of normal (ULN)). Finally, 7 children were diagnosed with CD (0.5%): one with no-biopsy approach according to ESPGHAN guidelines and 6 with positive biopsies (one had Marsh 2 and 5 Marsh 3). One child (tTG was 4x ULN) had normal biopsy and CD was not confirmed. There was no significant difference in children with CD and the children with negative serology in age (median 7.08 vs 7.25 years, $P > 0.05$), BMI (median 16.39 vs 15.36, $p > 0.05$), presence of symptoms (57% vs 44%, $p > 0.05$) nor in positive family history for CD (none vs one).

Children diagnosed with CD did not have significantly more symptoms nor lower BMI, and could not have otherwise been diagnosed clinically. In our study, the prevalence of CD in first-grade children was overall 0.5%. This is higher prevalence of CD than reported earlier for Croatia, showing the benefit of IgA and IgG DGP based POC test in screening.

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COLECTOMY RATE IN PAEDIATRIC-ONSET ULCERATIVE COLITIS – ONE CENTRE EXPERIENCE

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Paediatric-onset ulcerative colitis (UC) is often more extensive than in adults, and as disease severity is associated with disease extent, children are more prone to refractory severe episodes, sometimes requiring colectomy.

Previous population-based studies in patients with UC revealed variable colectomy rates. However, a decrease in colectomy rates was observed during the last two decades. The aim of our study was to assess the colectomy rate in paediatric patients with UC and to compare the clinical features of children who had to those who did not have colectomy.

In our hospital, data on children diagnosed with inflammatory bowel disease have been prospectively collected since January 2004. Retrospectively we analysed data (disease history, age at diagnosis, sex, baseline characteristics, and course of disease) on all children diagnosed with UC ($n = 170$) from 2004 to January 2018. Four children were lost to follow-up and were not included into analysis.

Of 166 children diagnosed with UC, 12 had colectomy (7.2%). Patients with colectomy, compared with UC patients who did not have colectomy, did not significantly differ in gender (girls 58% vs. 48%), age at diagnosis (12.27 vs. 12.62 years), body mass index at the time of diagnosis (median -1.2 vs. -0.22), Paediatric Ulcerative Colitis Activity Index (PUCAI) at the diagnosis (median 32.5 vs. 40), proportion of patients with extensive disease (E4) (75% vs. 57%) nor in extraintestinal manifestations (for all p was > 0.05). There was a significant difference in family history positive to IBD (25% vs. 7%, $p < 0.05$), in the highest PUCAI each patient had during the observation period (65 vs. 40, $p < 0.05$), number

of patients treated with azathioprine (92% vs. 28%, $p < 0.01$) and anti-TNF therapy (58% vs. 3.9%, $p < 0.01$). We found a significant decrease of colectomy rate (from 2004 to 2010 rate was 13.5% (7/52) vs. 4.4% (5/114) in the period from 2011 to 2018, $p < 0.05$). During the same time, the proportion of children with UC treated with anti-TNF therapy increased, although not significantly ($p > 0.05$): from 2004 to 2010, two children (3.8%) were treated with anti-TNF therapy, and both required colectomy, while in later period, 11 children (9.7%) received biologics, and 5 of them underwent colectomy.

Our paediatric patients presented with extensive form of the ulcerative colitis in nearly 60% of patients, requiring colectomy in overall 7.2% of patients. However, the colectomy rate decreased significantly during the observed period.

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ACUTE LIVER FAILURE IN NEWLY DIAGNOSED WILSON'S DISEASE – RECOVERY WITHOUT TRANSPLANTATION

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Wilson's disease (WD) is an autosomal recessive genetic disorder that leads to the impairment of cellular copper metabolism.

Clinical presentation is heterogeneous, with predominantly hepatic, neurological and psychiatric manifestations. Acute decompensated WD presenting as fulminant liver failure is a life-threatening condition for which liver transplantation is the ultimate treatment.

14-year-old girl presented with acute abdominal pain and peripheral oedema lasting two weeks before onset of abdominal pain. On initial examination, patient was febrile, complaining of periumbilical pain, dyspnea, cough, presenting with anasarca, extensive limb oedema and ascites (gained 17 kg), without encephalopathy. Laboratory evaluation revealed Coombs-negative haemolytic anaemia (Hb 101 g/L, Rtc $125 \times 109/L$), thrombocytopenia ($79 \times 109/L$), leukocytosis ($19.27 \times 109/L$), mildly elevated inflammatory markers, hypergammaglobulinemia (IgG 21.7g/L) with reduced complement components (C3 0.24g/L, C4 $< 0.08g/L$), coagulopathy (INR > 2.5), marked hypoalbuminemia (> 15 g/L), mildly elevated bilirubin (49 $\mu\text{mol/L}$), slightly elevated liver enzymes (AST 63 U/L, GGT 98 U/L), borderline serum ammonia (52.3 $\mu\text{mol/L}$), normal ALT and alkaline phosphatase. Abdominal ultrasound showed large amounts of ascites, with mildly enlarged, structurally altered liver (irregular surface, nodular parenchyma) and splenomegaly, with no signs of hepatic vein thrombosis on Color Doppler and MRI. Heart ultrasound was normal. Kayser-Fleischer ring was negative on slit lamp examination. Viral serology (HAV, HBV, HCV, HEV, EBV, CMV, and Parvo B19) was negative as well as autoantibodies (ANA, SMA, LKM, ANCA, anti dsDNA). Serum alpha-1 antitrypsin level was normal. Diagnosis of Wilson's disease was practically confirmed after demonstrating pronounced cupriuria (17.8 $\mu\text{mol}/24\text{h}$); serum ceruloplasmin was low (0.13 g/L) and serum copper levels slightly reduced (10.1 $\mu\text{mol/L}$). Initial treatment was supportive (albumin infusions, vitamin K, fresh frozen plasma, diuretics, lactulose, antibiotics). Liver transplant