is known about postnatal CMV (pCMV) infection. Although pCMV infection in term healthy infant is mostly asymptomatic, serious gastrointestinal symptoms (vomiting, diarrhea, abdominal distension, hepatosplenomegaly, blood stools) are described in literature.

We describe two cases of infant hematemesis, focusing on the challenging differential diagnosis between pCMV gastritis and non IgE-mediated Cow's Milk Protein Allergy (CMPA) enteropathy.

Case 1: a 3-month-old female infant presented with growth impairment, hematemesis and melena. Blood and stool analysis (bacterial, viral and parasites panels) resulted normal. Cow's milk specific-IgE were negative.

Viral serologies revealed recent CMV infection with positive CMV-DNA Polymerase Chain Reaction (PCR) on urine and blood samples. Congenital CMV infection was ruled out through negative CMV-DNA PCR on the first day of life saliva sample. Esophagogastroduodenoscopy (EGD) revealed petechial elements in antral and duodenal-bulb mucosa; at biopsies normal eosinophils count and negative morphological research of *Helicobacter pylori* (HP) were found. Intranuclear CMV inclusion bodies were not detected and CMV immunostaining was negative.

Case 2: a 2-month-old male infant presented with dehydration, bloody diarrhoea, vomiting and feeding refusal. Blood analysis revealed severe hypoalbuminemia, anaemia and hypertransaminasemia. Stool examinations (bacterial, viral and parasites panels) and Mycobacterium Tuberculosis screening were negative. Allergological and immunological investigations resulted normal. CMV-DNA PCR on urine, blood and maternal milk samples were positive. CMV-DNA PCR on Guthrie card was negative. EGD and rettosigmoidoscopy revealed exudative active inflammation in duodenal mucosa. HP research was negative while CMV immunostaining visualized duodenal cells viral inclusions.

Discussion Paediatric hematemesis is mainly caused by foreign bodies ingestion, CMPA, infectious gastritis (*Helicobacter pylori*, CMV, parasites), drug-induced gastritis (steroids and FANS) and eosinophilic gastropathy. In our cases the differential diagnosis focused on pCMV infection and non IgE-mediated CMPA. Both infants had a partial clinical improvement after starting a cow's milk protein free diet. However, due to the concomitant pCMV infection and the absence of cow's milk specific-IgE, a definitive diagnosis could not be established.

In conclusion, paediatric hematemesis differential diagnosis turns out particularly challenging when considering non IgE-mediated CMPA and pCMV gastropathy. In fact, neither the absence of cow's milk specific-IgE

and comparison of gluten-degrading microorganisms (GDM) from feces and saliva of adolescent patients with coeliac disease (CD) and healthy controls (HC). Additionally, we compared genomes of the same bacterial species isolated from samples of feces and saliva obtained from the same individual.

Feces and saliva were obtained from 5 CD patients (2 female, 3 male) on gluten-free diet (GFD) and 5 HC (3 female, 2 male) aged 13-18 years. Samples were inoculated on culturing medium (MCG3) with gluten as a major source of carbon and nitrogen. All colonies with lysis zone were further isolated in pure culture and identified using MALDI Biotyper (Bruker Daltonics). In 4 samples (3 CD, 1 HC), Whole genome sequencing (WGS) was performed on MiSeq platform (Illumina) on all strains that belonged to the same species and were isolated from fecal sample and from saliva in the same individual.

In the CD group 10 GDM strains were isolated (5 were not identified): 2 from feces and 8 from saliva. In contrast to the HC group, where 16 GDM were isolated (1 was not identified): 7 from feces and 10 from saliva, 1 GDM was isolated from both samples (saliva and feces). GDM isolated from CD samples belong to 3 genera of bacteria and 1 yeast (*Candida albicans*). The latter was also isolated in the HC group along with bacteria from 12 different genera. That indicates higher GDM diversity in HC compared with the CD group.

Three bacterial species were isolated from feces and saliva of the same individual: *Veillonella parvula*, *Lactobacillus paracasei*, *Lactobacillus rhamnosus*. WGS showed identical genomes only in *L. rhamnosus*. That could indicate transmission between oral cavity and gut.

We found that cultivable GDM are diverse and more often present in feces and saliva of HC than CD, which could be the effect of GFD the CD patients were on. Genomically identical lactobacilli were detected in saliva and in feces of the same individual.

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HELICOBACTER PYLORI INFECTION IN CHILDREN WITH CELIAC DISEASE

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Aim: to reveal the effect of *H. pylori* on course of celiac disease (CD) in children.

Methods 58 children with histologically confirmed CG and newly diagnosed CD were examined. Children were divided into two groups according to presence of *H. pylori* infection: the first group-12 *H.pylori*-positive and the second group – 46 *H.pylori*-negative subjects. All patients underwent histological examination of gastric and duodenal biopsies, histological verification of *H. pylori* infection and biopsy urease test. Tissue transglutaminase antibodies (tTG IgA, IgG) anti- H+/K+ ATPase and anti-intrinsic antibodies, were measured by ELISA. **Results** Mean age of patients was 11.33 ±3.06 years in group1 and 10.38±1.43 years in group 2 (p=0.582). Manifestation of CD didn't differ statistically significantly in groups.

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GLUTEN-DEGRADING MICROORGANISMS IN ADOLESCENTS WITH COELIAC DISEASE

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Oral and gut microbiota play an important role in the pathogenesis of coeliac disease. The aim of our study was isolation

The prevalence of the classical form was 58.3% in group 1 and 66.7% group 2 ($p=0.736$); atypical CD 41.7% vs 31.1% ($p=0.509$). Moderate and severe epigastric pain was common in both groups: 50.0% vs 31.1% ($p=0.309$). Significantly elevated level of anti- tTG antibodies ($>100\text{RU/ml}$) was detected equally in groups: 33.3% vs 47.7% ($p=0.516$). The prevalence of elevated level of antiparietal antibodies in groups was the same: in group 1- anti- H+/K+ ATPase antibodies in 2 children and in 3 children in group 2; and anti-intrinsic antibodies in 1 person in group 1 as in group 2. Thus, 25% vs 8.7% ($p=0.078$).

Conclusion There are conflicting studies results regarding the association between *H. pylori* and CD. Whether *Helicobacter pylori* triggers, doesn't affect or protects against CD is currently the subject of research. This study didn't reveal any effects of *H. pylori* on course of celiac disease in children despite the fact, that *H. pylori* is suspected as possible trigger of autoimmunity.

264 SCREENING FOR ANTIBODIES, ASSOCIATED WITH AUTOIMMUNE LIVER DISEASES IN CHILDREN WITH CELIAC DISEASE

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Objective to determine the prevalence of autoimmune liver diseases in children with CD.

Methods We observed 45 children aged 3 to 16 years. CD in all patients was diagnosed according to the ESPGHAN criteria. Serological examination with the determination of antibodies to tissue transglutaminase (anti-tTG IgG, IgA); histological examination of the duodenal mucosa; genetic typing for HLA-DQ2/DQ8 were carried out. Duodenal histology having Marsh grade III features were eligible for the study. Antibodies to hepatic antigen cell nuclei, skeletal muscle, cell nuclei, mitochondria, smooth muscles of IgG class were determined by indirect immunofluorescence (nRIF) using reagent kits. The antigen were biochips of primate's liver, primate's musculus iliopsoas, human Hep2 epithelial cells, liver, kidney, and stomach of rats.

Normal titer $<1:80$. Anti-Parietal Cell Antibody (PCA), IgG was determined by nRIF using biochips primate's stomach as antigen. This kit of reagents detects antibodies for the diagnosis of such diseases as autoimmune hepatitis(AIH) 1, 2 and 3 types, primary biliary cirrhosis, primary sclerosing cholangitis (PSC), overlap syndrome (combination of AIH and PSC), autoimmune gastritis.

Results We didn't obtain elevated levels of antibodies associated with autoimmune liver disease in all children with CD. In 1 person we observed elevated levels of anti-parietal cell antibodies. It was a 15-year-old girl with a typical form of celiac disease, additionally suffering from primary oligomenorrhea, autoimmune diabetes mellitus (type 1). Further examination revealed non- *Helicobacter pylori* atrophic gastritis. Thus, autoimmune gastritis was diagnosed.

Conclusion Antibodies, associated with autoimmune liver diseases were uncommon in children with CD. Probably, due to insufficient amount of participants of the study. On the other hand, anti-parietal cell antibodies have been found.

265 SCFA (SHORT CHAIN FATTY ACIDS) PROFILE IN CHILDREN WITH COELIAC DISEASE

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The intestinal microbiota ferments complex carbohydrates that have not been broken down or absorbed during digestion into short-chain fatty acids (SCFA). SCFA are involved in different metabolic processes and the functioning of the immune system. Recent studies have shown changes in the SCFA profile in people with celiac disease (CD). Here we compared the composition of SCFA in the faecal water of the paediatric population with CD and the healthy children (HC).

Faecal samples were obtained from 5 individuals with CD (2 female, 3 male) on a gluten-free diet (GFD) and 5 HC (3 female, 2 male) aged 13-18 years. To determine the SCFA concentrations sterile filtered fecal water was prepared.

The SCFA profile was determined by gas chromatography.

We compared the average SCFA concentrations between groups with CD and HC, for which we used the Student's t-test. We also calculated the total SCFA and the fermentation index $FI = (\text{acetic acid} - (\text{propionic acid} + \text{butyric acid})) / (\text{the sum of SCFA})$.

The comparison of concentrations of individual SCFA fractions showed a statistically significantly lower value of acetic acid ($p = 0.04$) and a statistically marginal increase in caproic acid concentration ($p = 0.089$) in the CD group. The averages of other SCFA were higher in healthy individuals but without statistical significance. Total SCFA was statistically significantly higher ($p = 0.047$) in HC. Fermentation index was 0.092 in HC and 0.079 in CD patients.

The literature regarding the profile of SCFA in the paediatric population CD is scarce. Our results differ from the current literature, which reports a significant increase of acetic acid, total SCFA, and fermentation index in the paediatric population with CD compared to the control group. An increase in caproic acid in CD has not been reported yet. Recent studies have suggested that the study groups' age difference or the amount of gluten in the diet could explain the discrepancies in our results.

266 EFFECT OF DIETARY PROTEIN INTAKE ON BODY MASS INDEX AMONG PRIMARY SCHOOL CHILDREN

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Recent studies have suggested that excessive dietary protein intake, especially animal-source protein, in infancy could affect health outcomes (e.g., obesity) in childhood. However, the effect of dietary protein intake in school-age children on growth and development is still unclear. The aim of this study was to assess dietary protein intake and its association with body mass index (BMI) in primary school children in Croatia.

Anthropometric measurements of children ($n=156$; 50% boys) aged 8.3 ± 0.5 years from primary schools in Zagreb City were performed according to standard protocols. Sex- and age-standardized BMI z-scores were obtained using AnthroPlus software. A dietary record for 3 non-consecutive