GP142 LEVEL OF 25(OH)-VITAMIN D AND VITAMIN D RECEPTOR (VDR) GENE POLYMORPHISMS IN ADOLESCENTS WITH CHRONIC GASTRODUODENITIS

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Introduction Chronic gastroduodenitis in adolescents is often accompanied by a decrease in bone density. The mechanisms of this relationship are not fully understood.

Objective To study the level of 25 (OH)-vitamin D, vitamin D receptor gene polymorphisms and bone metabolism in adolescents with chronic gastroduodenitis in dependency of helicobacter pylori (HP) infection.

Materials and methods 51 adolescents aged from 12 to 15 years old were examined. All patients had a morphologically checked chronic gastroduodenitis condition.

Group 1 included 19 patients with HP infection and other 32 children without HP infection were put in a group 2. All children had a gastroenterological examination including urease test and taking a biopsy sample from the stomach. 25 (OH)-vitamin D in serum was determined by ELISA. Polymorphisms (ApaI, TagI, BsmI) of vitamin D receptor gene (VDR) were determined by PCR with restriction digest. Dual-energy x-ray absorptiometry of the lumbar region of the spine L1-L4 was carried out in all patients on a Hologic QDR 4500C densitometer equipped with pediatric reference base in order to study osteocalcin (OK) and β -Cross-Laps.

Results A 25 (OH)-vitamin D deficiency was observed with an equal frequency in both groups (5.26% and 6.25%, $p > 0.05$). The correlation between vitamin D levels and the presence of HP- infection was not found ($r_s = 0.417$, $p < 0.05$). At the same time, the frequency of occurrence of genotypes containing the allele B of the VDR BsmI gene was higher among patients without HP infection compared with children with HP infection ($p < 0.05$, $\chi^2 = 6.03$). In both groups, the carriers of the VDR BsmI gene B had a significantly higher level of osteocalcin in comparison with children without it (90.43 ± 36.03 ng/ml and 63.02 ± 34.12 ng/ml, $p < 0.005$). The carriers of the ApaI gene A allele in both groups had high bone mineral density and high levels of 25 (OH)-vitamin D, osteocalcin, and β -cross-laps in comparison to children without this allele. Children with hp-infection, carriers of the t + genotype of the TagI gene of the VDR gene had a higher bone mineral density ($p = 0.001$).

Conclusion In adolescents with chronic gastroduodenitis, the bone metabolism and the level of 25 (OH)-vitamin D are related to genetic polymorphism gene VDR, and the presence of HP- infection as well.

GP143 COELIAC DISEASE AND IGA DEFICIENCY IN PAEDIATRIC DIABETES – HOW OFTEN SHOULD WE BE SCREENING OUR PATIENTS?

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Aims Coeliac disease [CD] and IgA deficiency are more common in patients with Type 1 Diabetes Mellitus [T1D] than in

the general population. Optimal screening for these conditions varies worldwide. Current guidance from the International Society for Paediatric and Adolescent Diabetes [ISPAD] is to screen at T1D diagnosis and every 1–2 years thereafter. As IgA deficiency can cause a false negative, CD screening includes measuring IgA Anti-Tissue Transglutaminase antibodies [TTG], serum IgA, and, with IgA deficiency, IgG TTG antibodies. The first aim of our audit was to compare the current screening practice at our centre against ISPAD guidance. Our second aim was to evaluate the need to screen for these conditions every 1–2 years by evaluating changes in TTG and IgA levels following T1D diagnosis.

Methods The study design is a single center, retrospective study of CD screening carried out on 150 consecutive patients attending diabetes services at TUH and diagnosed with T1D over a fifteen year period (2003 – 2018).

Results CD was diagnosed in 15 patients (10%). TTG was tested at T1D diagnosis in 98 patients (65.3%) and 1–2 yearly in 88 patients (58.7%). Four patients (2.7%) with initially negative TTG results subsequently developed elevated TTG levels and were diagnosed with CD. IgA was measured at diagnosis in 83 patients (55.3%) and 1–2 yearly in 44 patients (29.3%). Ten patients (6.7%) have not received IgA testing to date. Two patients (1.3%) were diagnosed with IgA deficiency on first IgA testing. Of the 138 patients with normal first IgA levels (92%), none have developed IgA deficiency during a mean 5.5 years of follow-up testing.

Conclusion Our study shows a lack of consistency in CD screening. On the basis of this study, we have initiated a clear CD screening protocol to be used at diabetes out-patient clinics. The results support continuing to monitor TTG levels every 1–2 years to detect changes in TTG levels as four patients with initially negative CD screening subsequently developed positive TTG and IgA EMA results. They also support measuring IgA levels at diagnosis of T1D to detect IgA deficiency. The results do not provide evidence for continuing to monitor IgA levels every 1–2 years as the IgA status of those with normal IgA levels at T1D diagnosis did not change. We believe a multi-centre prospective study is warranted to further evaluate the need to continue to screen for IgA deficiency after T1D diagnosis.

GP144 QUALITY OF LIFE AND THE IMPACT OF CARING FOR A CHILD WITH PRADER WILLI SYNDROME

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Aims To evaluate the family impact of caring for a child with Prader Willi Syndrome (PWS), the caregiver burden and the parents perceived impact on the affected child's Quality of Life (QOL).

Methods All children diagnosed with PWS, attending the service were invited to participate ($n=44$). QOL was evaluated using the PedsQL questionnaire. The Family Impact module was used to measure the impact of PWS on the family unit. Parent Proxy Reports were used to evaluate the parent's perception of the child's QOL. The PWS Burden questionnaire was used to assess additional challenges associated with managing a child with PWS.