SECTION: HELICOBACTER PYLORI

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HELICOBACTER PYLORI 1999–2000 ERADICATION AND RESISTANCE

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Background

In years 1999–2000, we examined 83 patients from the region of Eastern Slovakia and tried to set the cause of the failure in treatment of Helicobacter pylori [HP]. Apart from the serology, microscopy, cultivation, and rapid urea test [RUT], we examined also the sensitivity in vitro on antibiotics [ATB], which are used in eradication schemes of treatment Hp. It includes: C aritromycin [CLA], Azitromycin [AZI], Amoxycilin [AMO], and Metronidazol [MET].
Into the group, we also involved patients who were at least once [43.3 %], twice [37.2 %], or more times [6.3 %] re-cured by the triple combination – two ATB with blockation of proton pump [PPI], at our or some other surgery within the district, and at whom the positivity of at least two tests on HP after the cure overlasted. We failed in stating the number of HP eradication in 13.2 % of patients.

Aim

It was to state out, which drug of the triple combination was "responsible" for the failure of the treatment, it means, causes the lasting positivity on HP after the cure.

Methods and Results

Out of 83 cultivation examinations, 47 were positive, and we succeeded to state the sensitivity at 12 stems of HP, which makes 26.6 %. We found no resistant stem to AMO. The resistance to AZI and CLA was equal, 16.6 %. High resistance to MET was found – up to 74.9 %, which leads us to a conclusion that this one is the main cause of the failure of triple combination treatment. At present, we do not use it at our surgery in the above-mentioned indications.

Conclusion

Transporting grounds also influence the success of examination. While at the transport in Stuart transporting medium, most of the HP stems had died, in the grounds of Portagem, pylori stems had survived quite well.
Because four tested stems of HP were sensitive in vitro on each ATB, the failure of eradication treatment raises some doubts about keeping to the treatment regime, or/and it brings up a question of entering some other elements, important for the success of the treatment.
Even though there is some evidence of slow fall of prevalent infection of HP in Slovakia, the above mentioned results are the reason for being aware and careful of introducing any kind of HP eradication, except for the first indication group, as set in Maastricht 2000.

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CHANGES IN THE PREVALENCE OF H. PYLORI INFECTION IN FREQUENTLY OCCURRING GASTROENTEROLOGICAL DISEASES OVER THE PAST 14 YEARS AND THEIR CLINICAL CONSEQUENCES

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Introduction

In the last decade the prevalence of Helicobacter pylori (HP) infection among the Czech population shows signs of falling. This leads to consideration not only about changes in the prevalence of this infection in gastroenterological diseases, but also about changes in the prevalence of HP associated gastroenterological diseases.

Goal

To study changes in the prevalence of HP infection among the patients with duodenal (DU) and gastric (GU) ulcer disease, with functional dyspepsia (FD) and gastroesophageal reflux disease (GERD) over the past 14 years.

Patients and Methods

The above mentioned diseases were monitored as a part of the 2.250 consecutive cases and were examined in 3 time periods: 1st period 1988–1991, 2nd period 1996–1998, 3rd period 2001–2002 (750 cases in each period). HP was estimated using rapid urease test and/or histologically from antral biopsy particles.

Results

HP prevalence: in DU patients: 1st period 95.7 % (from 209 patients), 2nd period 83.1 % (from 167 patients), 3rd period 80.0 % (from 85 patients), in GU patients: 1st period 39.6 % (from 53 patients), 2nd period 72.6 % (from 16 patients), 3rd period 83.3 % (from 6 patients), in FD patients: 1st period 41.4 % (from 379 patients), 2nd period 25.2 % (from 436 patients), 3rd period 22.6 % (from 527 patients), in GERD patients: 1st period 52.7 % (from 74 patients), 2nd period 38.0 % (from 237 patients), 3rd period 30.1 % (from 362 patients). In GERD patients the prevalence of HP was the same in nonerosive esophagitis as in erosive esophagitis of various grade.

Conclusion

Over the past 14 years decreased rate of prevalence of DU and GU patients and increased rate of FD and GERD patients were noted. The prevalence of HP in DU, FD and GERD patients is decreasing, whilst in GU patients in the 2nd and 3rd period it was paradoxically

higher. (This is explainable by our more accurate diagnosis of HP infection in these patients within the last two periods). The decreasing rate of HP infection prevalence in FD patients and the increasing rate of FD patients in the resent years can be taken as another evidence, that HP infection is not involved in the etiology of FD.

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PEPSINOGEN I AND CHRONIC GASTRITIS – HELICOBACTER PYLORI POSITIVE

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Background

Helicobacter pylori are one of the most important etiological agents causing chronic gastritis. The prevalence of Helicobacter pylori is about 45 %, increasing with the age of population and with the social and economy conditions.

Aim

Diagnosis of Helicobacter pylori positive – chronic gastritis has been established on the endoscopic examination including histological findings of bioptic specimens of gastric mucosa, the determination of Helicobacter pylori infection and serological level of pepsinogen I. These examinations were performed in our group of 67 patients suffering from Helicobacter pylori gastritis.

Results

Histological findings revealed chronic gastritis with a partial and/or multi-focal glandular atrophy in 41.8 % of the patients. The mean value of pepsinogen I level in serum of these patients was significantly lower ($p < 0.001$) compare to what we found in patients with chronic gastritis without any histological signs of atrophy.

Conclusion

Low level of serum pepsinogen I in patients with the chronic Helicobacter pylori positive gastritis provide more precise information about etiology, morphology and function changes of gastric mucosa. The level of serum pepsinogen I can help us in differential diagnose of other chronic gastric pathology.

References

Graham, D. Y. Helicobacter pylori infection is the primary couse of gastric cancer. J. Gastroenterol., 2000, 35, Suppl. 12, 90–97.

Axon, T. Are all helicobacters equal? Mechanismus of gastroduodenal pathology and their clinical implications. Gut, 1999, 45, Suppl. 1, 1–4.

Fixa, B., Malfertheiner, P., Tuschy P *et al.* Infekce Helicobaster pylori a žaludeční imunita – společné etiopatogenetické mechanismy u chronické gastritidy? Čes. a Slov. Gastroent., 1995, 5, 147–152.

Gritti, I., Roi, G. S. Pepsinogen: physiology, pharmacology, pathophysiology and exercise. Pharmacol. Res., 2000, 41, 265–281.

Fixa, B., Komárková, O., Nožička, Z., Nožička, J., Petrová, J., Faller, G. Vztah žaludeční autoimunity ke stavu žaludeční sliznice a k infeci Helicobacter pylori. Čes. a Slov. Gastroent., 2000, 3, 83–88.

Ozasa, K., Kurata, J. H., Higashi, A., Watanabe Y. *et al.* Helicobacter pylori infection and atrophic gastritis: a nested case-control study in rural town in Japan. Dig. Dis. Sci., 1999, 44, 253–256.

033

HELICOBACTER PYLORI INFECTION IN SYMPTOMATIC AND ASYMPTOMATIC CHILDREN AND ADOLESCENTS IN THE CZECH REPUBLIC

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Introduction

There are inconsistent results of studies on the relationship between Helicobacter pylori (H. pylori) infection and dyspepsia or recurrent abdominal pain in children and adolescents.
Compare the epidemiological features of H. pylori infection between children and adolescents with dyspepsia or recurrent abdominal pain (symptomatic) and without these symptoms (asymptomatic), living in the Czech Republic.

Methods

Cases include children and adolescents (2–18 years) with upper gastrointestinal symptoms attending the paediatrics gastroenterology department from 1994 to 1999. An age, sex, socio-economic matched control group consisted of children visiting the general paediatrics service for symptoms unrelated to the upper gastrointestinal tract. Demographic and socioeconomic factors evaluated including patent educational level, place of residence, living conditions, type of drinking water and pets in their household. In addition, the symptomatic children and adolescents had endoscopic evaluation and biopsies from stomach antrum and corpus for quick urease test, histology/histoscopy and H. pylori culture. H. pylori infection was stated when at least two tests were positive.

Results

There were 829 children and adolescents examined, 624 cases and 205 controls. The prevalence OF H. pylori infection was 33 % aminy symptomatic children vs. 7.5 % aminy controls (OR = 6.2, $p < 0,001$) and was similar among boys and girls (32 % vs. 34.5 %, respectively). H. pylori prevalence increased with the age among symptomatic children (10 % for children < 6 years to 37 % ages of 11 and 16 years) ($p < 0,001$) but tended to fall with age among asymptomatic children 11 % for children < 6 years vs. 6 % for children 10 years (OR = 2.0, 95 % CI = 0.7–6.2). There was an inverse correlation between mother's educational level and H. pylori status. The H. pylori prevalence was 10 % among asymptomatic children who drank city or well water vs. 3 % among those who drank bottled water (OR = 4.95 % CI = 1.1 to 18, $p < 0.05$).

Conclusion

H. pylori infection was more prevalent among symptomatic children and adolescents vs. asymptomatic children and adolescents within the same population. H. pylori prevalence increased with age among symptomatic children and adolescents and tended to fall among controls, showing that there were Fundamentals differences between the two groups. Any other of studied factors was a significant

risk factor for acquirement the H. pylori infection with exemption of the group of the asymptomatic children and adolescents who drank bottled water.

References

Bode G., Rothenbacher D., Brenner H., Adler G.: H. pylori and abdominal symptoms: a population based study among preschool children in Southern Germany. Pediatrics, 101, 1998, 634–637
Gold B. D.: New Approaches to Helicobacter pylori infection in children. Curr. Gastroenterol. Rep., 3, 2001, 235–247
Graham D. A.: Can therapy by denied for H. pylori infection. Gastroenterology, 133, 1997, 51173–51177
Malaty H. M., Graham D. Y.: Importance of childhood socioeconomic status of the current prevalence of H. pylori infection, Gut, 35, 1994, 742–745

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CLINICAL COURSE OF HELICOBACTER POSITIVE GASTRITIS IN PATIENTS WITH PREVIOUSLY DIAGNOSED CELIAC DISEASE

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Introduction

In 10–20 % of patients with a diagnosis of coeliac sprue with involvement of jejunal mucosa grade 3c according to Marsh criteria and positive specific antibodies against EMA, we have observed T lymphocytic gastritis with dominant antral involvement. An unresolved question still remains whether Helicobacter infection precedes coeliac disease and/or coeliac disease is a consequence of Helicobacter infection.

Methods

In two girls aged 14 and 16 with proven coeliac disease we postulated the presence of HP+ gastritis on the basis of clinical presentation. This was subsequently confirmed by positive serology (CagA antibodies), positive stool test and INFAI breath test. Coeliac disease was diagnosed beforehand because of the presence of growth retardation at their ages of 12 and 15, respectively. In both, we observed strongly increased score of antibodies against gliadin IgG,A and ARA IgA, tTG IgA and IgG and positive HLA Dq alpha 1*0501/beta1*02 and by enterobiopsy, Marsh score was 3c and more.

Results

Using gastrofibroscopy, we demonstrated the presence of typically active gastritis grade II, type B. In both we observed serological relapse mostly in the class IgA gliadin and tTG and also relapse of enterobioptic finding. After eradication of Helicobacter gastritis using the standard three-drug combination, we observed persistence of serological relapse lasting 3 months while HP-noninvasive tests were negative. At that time we also observed constant persistence of celiac findings despite rigorous gliadin-free diet. We decided to administer Prednisone in one of these patients to normalize both serological and enterobioptic findings.

Conclusion

Our case report may stimulate further study of the relation between Helicobacter infection and the presence of celiac disease. The cases we show here – relapsing celiac disease and change of type from originally classic age-related to refractory during HP gastritis – are not explainable from contemporary knowledge point of view.˝

References

Diamanti A, Maino C, Niveloni S, Pedreira S, Vazquez H, Smecuol E, Fiorini A, Cabanne A, Bartellini MA, Kogan Z, Valero J, Maurino E, Bai JC. (1999) Characterization of gastric mucosal lesions in patients with celiac disease: A prospective controlled study. Am J Gastroenterol. 94(5):1313–9.
Chen MH, Lien CH, Yang W, Wu CL. Helicobacter pylori infection in recurrent abdominal pain in children – a prospective study. (2001) Acta Paediatr Taiwan. 42(5):278–81.
De Giacomo C, Gianatti A, Negrini R, Perotti P, Bawa P, Maggiore G, Fiocca R. (1994) Lymphocytic gastritis: a positive relationship with celiac disease. J Pediatr. 124(1):57–62.
Luzza F, Mancuso M, Imeneo M, Mesuraca L, Contaldo A, Giancotti L, La Vecchia AM, Docimo C, Pensabene L, Strisciuglio P, Pallone F, Guandalini S.(1999) Helicobacter pylori infection in children with celiac disease: prevalence and clinicopathologic features. J. Pediatr Gastroenterol Nutr. 28(2):143–6.
Ernst PB, Takaishi H, Crowe SE.(2001) Helicobacter pylori infection as a model for gastrointestinal immunity and chronic inflammatory diseases. Dig Dis. 19(2): 104–11.

035

RELATION BETWEEN HELICOBACTER PYLORI AND COLONIC POLYPS

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Introduction

H. pylori plays a significant role in the pathogenesis of gastroduodenal ulcer and MALT lymphoma. It also participates in the multifactorial pathogenesis of gastric carcinoma. Its presence is also indicated in the origin of certain extragastric diseases. Adenomatous polyps and colorectal carcinoma related to H. pylori are included. Information based on the knowledge that H. pylori increases the level of gastrin, which, due to its trophic effect, causes mucous proliferation. This clinically important finding also deserves attention, in particular because the results published hitherto have been very often controversial.

Methods

Our coloscopic examination of 50 symptomatic patients (71 ± 2 years of age) forming our study group clearly showed the presence of colonic polyps, while the control group of 50 hospitalised patients (69 ± 2 years of age) did not manifest any organic gastric disease; neither inflammatory nor malignant disease of the colon. These patients were not treated with antibiotics.
Antibodies against H. pylori and the level of gastrin were ascertained in the blood samples taken on an empty stomach after a polypectomy and during the taking of the first blood sample by means of the ELISA (Eia H. pylori) and RIA (GASK-PR) methods.
Statistical processing of the results was carried out by means of ANOVA (post hoc test) using Sheffe‘s test for independent values.

Results

The largest number of polyps (78 %) was found in the left colon, the remaining 22 % being in the transverse and the right colon. Solitary polyps prevailed (62 %) over multiple ones (38 %).

Tubulovilous polyps (52 %) were the histologically most frequent types. Tubular polyps occurred in 27.2 % and hyperplastic ones in 10.4 % of cases. Polypous carcinoma was found in 8.4 % of patients.

In the case of polypous patients the level of IgG antibodies against H.pylori was 2.92 ± 0.34, while in the control group it was 2,51 ± 0.34 (NS). IgA antibodies reached the value of 0.73 ± 0.10 in the former group and 0.79 ± 0.09 (NS) in the control patients. Non-significant differences were also found when examining for IgM antibodies (0.57 ± 0.09 vs 0.57 ± 0.06).

Our observation of the gastrin level likewise showed non-significant differences between the patients with polyps and control group (99.51 ± 40.5; 140.65 ± 28.67 µU/l; NS).

Conclusion

Our study did not show any significant changes of the seroprevalence of H. pylori in patients with adenomatous polyps.

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References

Breuer-Katschinski, B., Nemes, K., Marr, A., Rump, B., Leiendecker, B., Breuer, N, Goebell, H.: Helicobacter pylori and the Risk of Colonic Adenomas.Digestion 1999, 60, 210–215

Siddheshwar, R. K, Gray, J. C, Kelly, S. B.: Plasma level of progastrin but not amidated gastrin or glycine extended gastrin are elevated in patients with colorectal carcinoma. Gut 2001, 48, 47–52

Siddeshwar, R. K., Muhhammad, K. B., Gray, J. C., Kelly, S. B.: Seroprevalence of Helicobacter pylori in Patients with Colorectal Polyps and Colorectal Carcinoma. Am.J.Gastroenterol. 2001, 96, 84–88