ΞΕΝΟΓΛΩΣΣΕΣ ΑΝΑΚΟΙΝΩΣΕΙΣ (ABSTRACTS) ΕΛΛΗΝΩΝ ΕΡΕΥΝΗΤΩΝ

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Abstract no.: 06.18

Helicobacter pylori in Tonsil Tissue of Greeks

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Background and Aim. *Helicobacter pylori* is a gram-negative, rod- or spiral-shaped bacterium that infects the gastric mucosa, causing chronic active gastritis, gastroduodenal ulcers, and gastric malignancies. Tonsil tissue has been proposed as an extragastric reservoir for *H. pylori*, though data in this regard are conflicting. The socioeconomic status of the subjects is one of the most important factors that should be taken into account in the studies. The aim of this study was search by immunohistochemical method, *H. pylori* presence in tonsil tissue obtained from Greek patients.

Material and Method. A total of 36 consecutive patients aged 4–62 years who had undergone a tonsillectomy procedure, were included in the study. Consecutive sections were stained using hematoxylin/eosin and a polyclonal antibody directed against *H. pylori* (Biocare) using an immunoperoxidase technique following heat induced antigen retrieval.

Results. Histologically, the diagnosis of chronic nonspecific tonsillitis was made in all patients. Immunohistochemically, *H. pylori* was detected in 24 (66.7%) sections stained with the antibody. No correlation was found among the degree of inflammation, the age and the sex of the patients, and the presence of *H. pylori*.

Conclusions. Our research suggests that *H. pylori* can be found in tonsil tissue, supporting the oral-oral transmission route for *H. pylori*. To our knowledge, this is the first documented study by immunohistochemical method on detection of *H. pylori* in tonsil tissue in Greece.

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Cell Cycle Regulatory Proteins Expression (bcl-2, bcl-x, cyclin D1) and Apoptosis In *Helicobacter pylori* Induced Gastritis

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Aim. To assess apoptosis and expression of Bcl-2, Bcl-x and Cyclin D1 in patients with *H. pylori* gastritis before and after *H. pylori* eradication.

Endoscopic biopsies from the body and antrum of the stomach from 30 patients with histologically confirmed *H. pylori* gastritis were studied. Apoptotic activity was assessed with the TUNEL method, Bcl-2, Bcl-x and Cyclin D1 expression with immunoperoxidase staining. Bcl-x expression was studied before *H. pylori* eradication only, while 10 patients with reactive NSAID-induced gastritis served as controls for Bcl-x expression.

Mean value of apoptotic index before and after *H pylori* eradication was 3.5% (0.6–11.7%) and 12% (4.8–32.9%) for the body and 5.02% (2.03–19.29%) and 7.4% (1.9–16%) for the antrum, respectively ($p < 0.05$). Bcl-2 expression was observed in the antrum only in 8/30 (27%) and 26/30 (87%) patients before and after *H. pylori* eradication, respectively ($p < 0.05$). Cyclin D1 expression was found in ~1% of cells, both before and after *H. pylori* radication, being increased (in ~5–10% of cells) in foci of regenerative activity. Bcl-x expression was observed in 10/10 (100%) patients with Grade 3 and in 6/8 (75%) with Grade 2 gastritis and G3 and G2 *H. pylori* load, respectively. All patients (12/12, 100%) with Grade 1 gastritis were negative for Bcl-x epression as the 10/10 (100%) control patients.

Conclusions. (a) Apoptotic activity remains increased, particularly in the body of the stomach (b) expression of the antiapoptotic protein Bcl-2 is increased in the antral mucosa only (c) cyclin D1 expression remains unchanged (d) Bcl-x expression is associated with high *H. pylori* load and severe gastritis.

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Should Biopsies be Taken from Functional Dyspepsia Patients Under 45 Years of Age?

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Aim. To assess whether histology influences the management of functional dyspepsia (FD).

Study Design. Four year prospective single center.

Inclusion Criteria. FD patients < 45 years (negative gastroscopy).

Exclusion Criteria. Peptic ulcer, GORD, gallstones, past *H. pylori* infection, recent antibiotic, H2RA, PPI or NSAIDs treatment, malignancy.

Methods. Patients underwent abdominal US and gastroscopy. Biopsies taken from the gastric antrum, angularis, and body were processed for CLO-tests and histology (H&E, Giemsa) to assess *H. pylori* status and gastritis (Houston System). Additional diagnostic tests were performed when indicated (e.g. parietal cell autoantibodies).

Results. Eighty-five patients (25 males), 34.9 (14–44) years (mean, range) were assessed. 45/85 (53%) were smokers. The main endoscopic findings were focal/diffuse erythema and/or mucosal nodularity. *H. pylori* was diagnosed in 64/85 (75%) patients. Histology on *H. pylori*-patients revealed reactive gastritis (6/21), autoimmune gastritis (1/21), or no active inflammation (14/21). 32/64 (50%) *H. pylori* + patients had mild-to-moderate chronic active antral gastritis (12/32 pangastritis). The other 32/64 (50%) *H. pylori* + patients had more severe chronic gastritis with lymph follicles [22/64 (34%)], atrophy [27/64 (42%)], and IM [22 (34%, type III in 14). One patient had dysplasia. The age of patients with atrophic gastritis and IM was 33 years (28–44). No endoscopic signs predicted the presence of atrophy or IM. Overall 27/85 patients (32%) had developed worrying histological abnormalities.

Conclusion. As part of an evidence-based approach patients with FD should probably undergo gastric biopsies not only to detect *H. pylori* but evaluate and grade gastritis, as well. Histology may then reveal advanced pathology that will probably need long-term close surveillance.

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Abstract no.: 08.16

Anti-Inflammatory Activity of the Probiotic Lactobacillus Casei Strain Shirota in C57BL6 Mice Infected with *Helicobacter pylori* SS1 Strain

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Aim. To study the potential inhibitory effect and mode of action of Lactobacillus casei strain Shirota (LcS, Yakult®, Japan) on *Helicobacter pylori* (*H. pylori*) utilizing the in vivo HpSS1 mouse infection model, over a period of 9 months.

Materials and Methods. LcS was administered orally through the water supply over a period of 9 months to C57BL/6 mice previously infected by the *H. pylori* SS1 strain (study group n = 25). Appropriate control groups of *H. pylori* infected yet untreated animals (n = 25), or uninfected animals given the LcS (n = 25) were also included in the study. *H. pylori* colonization and development of gastritis was assessed at 1, 2, 3 6 and 9 months postinfection. *H. pylori* colonization was evaluated by quantitative culture, serology, histology and PCR. The effect of LcS on cytokine production (IL-10, IL-12, TNF-α and IFN-γ) by stimulated PBMCs was assessed in vitro by ELISA.

Results. A significant decrease in the *H. pylori* colonization levels in the antrum and fundus was observed, compared with the control *H. pylori* infected group. This was accompanied by significant decline in the associated chronic and active gastric mucosal inflammation observed, at each time point, throughout the observation period. In concordance with the in vivo results, high levels of anti-inflammatory IL-10 were detected following incubation of stimulated PBMCs with live LcS cells. Further immunohistochemistry evaluation for detection of released cytokines in vivo is under way.

Conclusion. The anti-*Helicobacter pylori* activity of LcS in vivo may be linked to a strong anti-inflammatory reaction elicited by the probiotic strain.

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Abstract no.: 10.17*

Molecular and Immunohistochemical Evaluation of the Expression of Telomerase in Gastric Carcinomas: Correlation with *H. pylori* Infection

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Introduction. Telomerase is an enzyme associated with cellular immortalization and plays an important role in carcinogenesis. Telomerase adds hexameric repeats of 5'-TTAGGG-3' to the ends of chromosomal DNA called telomere, the length of which increase proportionally to the cellular activation rate. Telomerase consists of 2 subunits, Telomerase RNA template (hTR) and Telomerase reverse transcriptase protein (hTERT).

Aim. To define hTR activity in the serum of *H. pylori* related gastric carcinoma patients and to compare these findings with the immunohistochemical expression of Telomerase in bioptic material taken from the same patients.

Materials and Methods. Telomerase activity was measured by using the QIAGEN OneStep RT-PCR kit in human serum of 45 *H. pylori*-related gastric carcinoma cases (22 diffuse and 23 intestinal type). Histologically, Intestinal Metaplasia (IM) type I was observed in five cases, type II in 8 and type III in 12 cases. The expression of Telomerase in the tissues was detected immunohistochemically using a anti-Telomerase Moab (Novocastra, UK). Positive and negative controls were also included.

Results. Increased hTR activity as well as immunohistochemical expression of tissue Telomerase detected in 43/45 cases examined. In normal gastric mucosa, weak hTR expression was noted, limited to basal cells of gastric glands. hTR activity and Telomerase expression found to be higher in IM of type III than in type I and II (4 : 2 : 1 accordingly). *H. pylori*-negative controls constantly expressed very low levels of hTR activity.

Conclusion. *H. pylori* infection may be a strong trigger for hTR overexpression possibly through activation of epithelial 'stem cells' during the procedure of intestinal metaplasia.

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Abstract no.: 10.21

Helicobacter pylori and Noncardia Gastric Cancer

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Aim. To test the hypothesis that *H. pylori* is implicated in the pathogenesis of noncardia gastric cancer (NCGCs).

Patients. NCGCs patients; non-NSAID induced duodenal ulcers (DU) (positive controls) or irritable bowel syndrome (IBS) patients (negative controls).

Methods. Detailed demographic, social, medical, and family history was obtained. Patients and controls underwent gastroscopy. Biopsies were taken from lesions and endoscopically healthy gastric mucosa; the latter in order to grade gastritis (Houston System) and seek for *H. pylori* [CLO-tests, histology (C & Å, Giemsa)]. If negative, serological evidence of *H. pylori* was sought (IgA/G anti-*H. pylori*, RIA). Conditions predisposing to NCGCs were excluded.

Results. No significant baseline clinical or demographic differences were seen between patients and controls except for age. 115 patients with diffuse or enteric type NCGCs were studied. Histology from 'healthy' gastric mucosa revealed diffuse atrophy and intestinal metaplasia (predominantly type III). Active or past *H. pylori* infection was detected in 94% (108/115) NCGCs patients. CLO-tests were positive only in biopsies from non cancerous tissues [75/115 (65%) patients]. Histology revealed *H. pylori* in 107/115 (93%) patients whereas serology was positive in 1/15 histologically *H. pylori*– patients. All DU patients and 58/85 (60%) IBS patients were *H. pylori*+ ($p < 0.001$ vs. NCGCs and DUD). Only 20/98 (20%) *H. pylori*+ DU and 13/51 (25%) *H. pylori*+ IBS patients had focal atrophic gastritis with or without IM ($p < 0.0001$ vs. NCGCs). No other patient- or disease-related factor was associated with the development of NCGCs.

Conclusion. In this cohort of patients *H. pylori* appears to be the leading aetiological factor for NCGCs through the development of atrophy and IM.

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Abstract no.: 11.07

H. pylori Infection Increases the Risk of Bleeding in Regular Aspirin and NSAIDs Users

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Aim. Assess the risk for gastric bleeding in *H. pylori*+ aspirin or NSAID users.

Methods. *H. pylori* was prospectively sought in a cohort of 215 regular aspirin and/or NSAID users who at endoscopy were found to have peptic ulcer(s) and/or erosions. 100 patients were bleeders and 115 had epigastric pain. Patients clinical and endoscopic data were recorded. *H. pylori* infection was diagnosed by CLO-test, histology and serology (RIA method). Exclusion criteria were peptic ulcer before the use of aspirin/NSAIDs, recent H2RA, PPIs or antibiotic treatment, and Crohn's disease.

Results. Of 100 bleeders [42 males, mean age (range) 53 (18–83) y, 38 smokers] 39 were using aspirin, 50 NSAIDs and 11 both; Endoscopy revealed gastric ulcers/erosions in 50, duodenal ulcers/erosions in 42 and both in eight patients. Of 115 nonbleeders [48 males, age 51 (17–86) y, 48 smokers], 39 were using aspirin, 58 NSAIDs and 11 both; Endoscopy revealed GU/erosions in 54, DU/erosions in 40 and both in 11 patients. Multifactorial analysis showed that bleeding was associated with *H. pylori* infection but not with demographic data, smoking, social drinking, coffee, comorbidities, ulcer location, duration and dose of aspirin/NSAID use. *H. pylori* was diagnosed in 86/100 (86%) patients bleeders and 78/115 (68%) nonbleeders. The RR for bleeding was × 2.08 higher in *H. pylori*+ aspirin/NSAID users [OR 2.91 (95% CI 2.27–3.55)]. The RR for bleeding in *H. pylori*+ aspirin users was × 3.1 higher than in *H. pylori*-users [OR 6.24 (95% CI 4.92–6.56)].

Conclusion. *H. pylori* infection increases the risk of bleeding in aspirin/NSAID users. This risk is higher for aspirin than NSAID users.

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Histologic Presence of Esophagitis in Patients with Negative-Endoscopic Gastro-esophageal Reflux Disease (NERD)

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Objective. No data seem to exist on the histologic evaluation of the esophageal mucosa in patients with NERD. In the present study we conducted thorough histologic examination in NERD patient's esophageal mucosa.

Patients and Methods. Thirty-one patients (nine males and 22 females, age range 19–81 years) with endoscopy proven NERD underwent histologic evaluation of the esophageal mucosa for detection of inflammatory infiltrations. During upper GI endoscopy were obtained: (1) biopsy specimens above ($n = 4-6$) and below ($n = 4-6$) the gastroesophageal junction for examination of: (a) presence of esophagitis and specialized intestinal metaplasia (goblet cells), and (b) presence of *H. pylori* (*H. pylori*) histologically, and (2) antrum and corpus biopsy specimens ($n = 2 + 2$) for assessment of (a) and (b).

Results. In 25 out of 31 (80.64%) patients histological lesions of esophagitis were detected (lymphocytic and/or neutrophilic inflammatory infiltrations). Histologic presence of *H. pylori* infection was found in 13 out of 25 (52%) patients, whereas only one out of six (16.67%) patients without inflammatory infiltrations was *H. pylori* infected. Moreover, three patients (without *H. pylori* infection) with NERD exhibited Barrett's esophagus, low-grade mucosal dysplasia, and eosinophilic esophagitis, respectively.

Conclusions. In patients with NERD, histologic presence of esophagitis with concomitant *H. pylori* infection seems to be common.

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Abstract no.: 13.06

Helicobacter pylori Identified Immunohistochemically in Resected Gallbladder Mucosa and in Remnants of Cholesterol Gallstones: a Case Report

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Introduction. Recently, several studies have revealed the presence of *Helicobacter pylori* in bile and cholesterol gallstones. Furthermore, it has proposed that *H. pylori* present in bile may predispose to cholesterol gallstone formation. However, whether this microorganism might colonize the gallbladder mucosa is not clearly documented. We present a case of chronic follicular lithiasic cholecystitis with localized *H. pylori* infection.

Case presentation. A 73-year-old-woman with a previous medical history of repeated episodes of lithiasic pancreatitis was admitted to hospital because of right hypochondrial pain. Laparoscopic cholecystectomy was carried out under the diagnosis of gallstones and cholecystitis. Grossly, there was a large number of mixed stones in the gallbladder. The cystic wall was moderately thickened. Microscopically, the diagnosis of chronic follicular lithiasic cholecystitis with extensive areas of pseudopyloric gland metaplasia was made. A microorganism closely resembling *H. pylori* (stained with Giemsa) was detected incidentally on the surface of the gallbladder mucosa and in the pseudo-pyloric glands. Immunohistochemical study using a polyclonal antibody to *H. pylori* antigens (Biocare) confirmed the nature of this microorganism. Also, remnants of cholesterol gallstones were strongly *H. pylori* positive.

Conclusions. These data support the presence of *H. pylori* in gallbladder mucosa, reflecting either that *H. pylori* is an indigenous part of a flora in the stone-containing gallbladder or, alternatively, that *H. pylori* colonization in the biliary tree predisposes to gallstone formation.

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GASTRO-OESOPHAGEAL REFLUX DISEASE IS MORE SEVERE IN PATIENTS WITH A PREVIOUS HISTORY OF GASTROINTESTINAL BLEEDING DUE TO ULCERATION

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INTRODUCTION: Patients with a bleeding ulceration are considered to be more prone to oesophagitis after successful eradication.

AIMS & METHODS: Aim. To evaluate the significance of history of previous gastrointestinal bleeding due to peptic ulceration in the severity of gastro-oesophageal reflux disease, when H. pylori has not been eradicated. Patients and Methods: 522 consecutive patients (mean age 62.2±13.6 years, 248 men, 130 smokers (22±37 PY), 140 daily drinkers) with regurgitation and acid reflux were offered and finally undergone an endoscopy. All patients completed a standardised questionnaire before endoscopic evaluation. A history of gastrointestinal bleeding due to peptic ulceration was recorded when the episode of bleeding has been investigated by either endoscopy or radiology. Oesophagitis diagnose was done according to the LA classification. Stat: t-test, X2, logistic regression analysis.

RESULTS: At least one episode of gastrointestinal bleeding due to peptic ulceration had 20/200 (10%) without oesophagitis, 26/168 (15%) grade A, 14/70 (20%) grade B, 4/21 (19%) grade C, 9/21 (43%) grade D oesophagitis. Gastrointestinal bleeding due to peptic ulceration was less frequent in patients without oesophagitis or grade A oesophagitis (46/368 (13%) than in those with oesophagitis grade higher than A (40/154 (26%) ($p=0.0002$). There was no difference between bleeders and non-bleeders in current smoking (bleeders 22/86 vs. non-bleeders 108/436, $p=0.87$), daily alcohol consumption (bleeders 28/86 vs. non-bleeders 112/436, $p=0.19$) or body mass index >25 (bleeders 74/86 vs. non-bleeders 342/436, $p=0.11$). In logistic regression analyse history of gastrointestinal bleeding due to peptic ulceration was risk factor for more severe reflux disease (OR=11.51, $p<0.0001$), as it happened for age (OR=7.37, $p=0.01$), daily alcohol consumption (OR=6.80, $p=0.02$), presence of hiatus hernia (OR=9.15, $p=0.002$) and gastrectomy (OR=10.18, $p<0.001$). Three months after successful eradication of H. pylori oesophagitis was improved in 5/86 patients with a history of gastrointestinal bleeding, aggravated in 12/86 and remained unchanged in 69/86

CONCLUSION: 1) Gastro-oesophageal reflux disease is more severe among patients with a previous history of gastrointestinal bleeding due to peptic ulceration. 2) In this patient group the severity of gastro-oesophageal reflux disease tends to be unchanged three months after successful H. pylori eradication. Thus we should speculate a higher acidity of gastric fluid in this patient group

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Increased levels of *Helicobacter pylori* IgG Antibodies in the Aqueous Humor and Serum of Patients with Primary Open-Angle and Exfoliation Glaucoma

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Objective. We investigated the levels of anti-*H. pylori* specific IgG antibodies in the aqueous serum of patients with POAG and patients with exfoliation glaucoma (XFG) and compared these with age-matched patients with cataract.

Design. Prospective, nonrandomized, consecutive comparative study.

Participants. Twenty-six patients with POAG, 27 patients with XFG, and 31 age-matched patients with senile cataract.

Methods. Aqueous humor was aspirated at the beginning of glaucoma surgery from 26 POAG and 27 XFG patients, and at the beginning of phacoemulsification cataract surgery from 31 age-matched nonconsecutive cataract patients. Serum samples were obtained the day of surgery.

Main Outcome Measure. Anti-*H. pylori* IgG concentration in the aqueous and serum as measured by an enzyme-linked immunosorbent assay.

Results. The mean concentration of anti-*H. pylori* specific IgG antibodies was significantly greater in the aqueous samples from patients with POAG (14.27 ± 3.06 U/ml) and XFG (14.25 ± 3.39 U/ml) compared with that from control patients (4.67 ± 1.07 U/ml); ($p = 0.006$ and $p = 0.003$, respectively). No difference was observed in the levels of *H. pylori* antibodies between POAG and XFG aqueous samples ($p = 0.3$). Similarly, the serum concentration of anti *H. pylori* antibodies was significantly greater in patients with POAG (69.96 ± 9.69 U/ml); ($p = 0.013$), and XFG (81.37 ± 10.62 U/ml); ($p = 0.002$), compared with the cataract controls (44.16 ± 6.48 U/ml).

Conclusions. *H. pylori* antibody levels are significantly increased in the aqueous humor and serum of patients with POAG/XFG. These findings support a role for *H. pylori* infection in the pathobiology of these glaucomas.

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MOLECULAR AND IMMUNOHISTOCHEMICAL EVALUATION OF THE EXPRESSION OF TELOMERASE IN GASTRIC CARCINOMAS: CORRELATION WITH HP INFECTION

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INTRODUCTION: Telomerase is an enzyme associated with cellular immortaliazation and plays an important role in carcinogenesis. Telomerase adds hexameric repeats of 5'-TTAGGG-3' to the ends of chromosomal DNA called telomere, the length of which increase proportionally to the cellular activation rate. Telomerase consists of 2 subunits. Telomerase RNA template (hTR) and Telomerase reverse transcriptase protein (hTERT).

AIMS & METHODS: The aim of the study was to define hTR activity in the serum of Hp related gastric carcinoma patients and to compare these findings with the immunohistochemical expression of Telomerase in bioptic material taken from the same patients. Telomerase activitity was measured by using the QIAGEN OneStep RT-PCR kit in human serum of 45 Hp-related gastric carcinoma cases (22 diffusa and 23 intestinal type). Histologically, Intestinal Metaplasia (IM) type I was observed in 5 cases, type II in 8 and type III in 12 cases. The expression of Telomerase in the tissues was assessed immunohistochemically using a anti-Telomerase Moab (Novocastra, UK). Positive and negative controls were also included.

RESULTS: Increased hTR activity as well as immunohistochemical expression of tissue Telomerase detected in 43/45 cases examined. In normal gastric mucosa, weak hTR expression was noted, limited to basal cells of gastric glands. hTR activity and Telomerase expression found to be higher in IM of type III than in type I and II (4:2:1 accordingly). Hp-negative controls constantly expressed very low levels of hTR activity.

CONCLUSION: Hp infection may be a strong trigger for hTR overexpression possibly through activation of epithelial ``stem cells" during the procedure of intestinal metaplasia.

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EFFECT OF THE HELICOBACTER PYLORI ERADICATION ON SERUM LIPIDS LEVELS, INFLAMMATORY PARAMETERS AND FACTORS INFLUENCING HAEMOSTASIS

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INTRODUCTION: Epidemiologic data suggest a causative role of chronic Helicobacter pylori (Hp) infection in the pathogenesis of the ischemic heart disease, through changes on serum lipids levels and on inflammatory and thrombotic parameters, but this is not widely acceptable.

AIMS & METHODS: The aim of our study was to investigate the influence of Hp eradication on the serum lipids concentration, on several inflammatory parameters and on certain thrombotic factors.

We studied 42 patients [33M, 9F, of mean age 53,2±14,5 (range: 20-77) years] with histologically confirmed chronic active Hp gastritis, before and 3 months after an Hp eradication treatment (omeprazole 20mg tid, amoxycilline 1gr tid, clarithromycin 500mg tid). The hematocrit value, white blood cells (WBC) count and the platelets count, the serum levels of C-reactive protein (CRP), tumor necrosis factor (TNFα), interleukine-6 (IL-6), lipoprotein-A, cholesterol, triglycerides, HDL and LDL as well as prothrombin time, APTT, the serum levels of fibrinogen and antithrombin III and the protein C and S, were determined before and after Hp treatment. Patients with known ischemic heart disease and those under anticoagulant and antilipid therapy were excluded. Paired t-test was used for statistical analysis.

RESULTS: Three months after the treatment, Hp eradication was established in 31 out of 42 (73,8%) patients. Among the patients with no Hp eradication a significant increase of the serum triglycerides (139,6±53,29 vs 110,2±37,48 mg/dl, p<0,01) and a decrease of the protein-S (88,6±24,19 vs 103,2±23,63%, p<0,05) were found. Among the patients with Hp eradication a significant increase of the hematocrit value (44,3±5,21 vs 42,2±6,57%, p<0,01) and levels of HDL (58,6±11,88 vs 51,9±10,89 mg/dl, p<0.001) and a decrease of the WBC count (6.567±1.852 vs 7.158±2.245/UL, p<0,01), serum levels of fibrinogen (554±64,7 vs 402±100,9 mg/dl, p<0,05) and triglycerides (91,3±42,53 vs 103,5±49,37 mg/dl, p<0,05) were observed. None of the other investigated parameters was significantly changed.

CONCLUSION: Hp eradication is associated with significant early changes in certain indices of systemic inflammation (hematocrit value, WBC count, serum fibrinogen), thrombotic factors (fibrinogen, protein-S) and serum lipids (triglycerides, HDL), which are considered protective against the ischemic heart disease.

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ROLE OF GROWTH FACTORS, APOPTOSIS AND COX-1 AND 2 EXPRESSION IN GASTRIC CARCINOGENESIS - CORRELATION WITH H. PYLORI INFECTION

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Introduction: Several epidemiologic studies demonstrated a correlation between gastric carcinogenesis and infection with *H. pylori*. The exact mechanism that is responsible for the transformation of the gastric mucosa still remains obscure. It is postulated that excessive mucosal cell proliferation may eventually result in gastric atrophy, mutation and transformation of epithelial cells in a more malignant phenotype. These processes seems to be complicated by the expression of COX-2 as an inflammatory enzyme to release excessive amounts of PGE2, leading to further proliferation, reduction of apoptosis, angiogenesis and tumor growth. So the aim of this study was to investigate the possible role of Growth Factors such as TGFs and HGF in gastric carcinogenesis in correlation with *H. pylori* infection, as well as to study the expression of genes such as COX-1 and 2 and apoptosis related molecules Bax and BCL-2. Material and Methods: Bioptic specimens and serum from 50 patients with gastric carcinoma were used. Molecular biology techniques were implicated for the study of the expression of TGFs, HGF, COX-1 and 2, and immunohistochemistry for the detection of Bax and BCL-2 proteins. Negative controls were used in all stages of the process. Results: 40/50 (80%) patients were positive for H. pylori. CagA+ strains were detected in 35/40 (87.5%) H. pylori (+) cases. HGF and TGFs are expressed more frequently in gastric carcinomas than in normal tissues (10 and 15-folds respectively). No statistically significant difference was observed in the COX-1 expression between diseased and normal tissues. On the other hand, COX-2 expression was exclusively seen among gastric carcinoma cases. Only the Bax protein correlated with 50% of the H. pylori (+) cancer cases. Conclusion: Infection with *H. pylori*, especially with strains expressing CagA+, seems to be a well documented pathway to developing gastric carcinoma. Upregulation of growth factors and COX-2, and dysregulation of the Bax/BCL-2 system in *H. pylori* infected patients, may contribute to neoplastic transformation.

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RELATIONSHIP BETWEEN ACUTE INFLAMMATORY DEMYELINATING POLYNEUROPATHY (AIDP) AND H. PYLORI INFECTION (HP-I)

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INTRODUCTION: AIDP is an autoimmune process characterized by progressive weakness and mild sensory changes, possibly caused by an immunologic attack directed against myelin components, resulting in demyelinating polyneuropathy. The aim of this pilot study was to assess the prevalence of HP-I and evaluate the endoscopic and histologic findings of upper gastrointestinal tract in AIDP patients.

AIMS & METHODS: 10 patients (5 men: 5 women, mean age 54.2±22.7 years) with AIDP [diagnosed by increased CSF protein without increased WBC count (albuminocytologic dissociation), electrodiagnostic testing, and nerve conduction examination] underwent upper gastrointestinal endoscopy to obtain: 1) biopsy specimens above and below the gastroesophageal junction for examination of: (a) presence of specialized intestinal metaplasia (goblet cells), and (b) presence of HP histologically and by CLOtest, and 2) antrum and corpus biopsy specimens for assessment of (a) and (b). All the patients received sandoglobulin 0.04mg/kgx5d, and 2 patients received methylprednisolone 1grx5d.

RESULTS: HP-I was established in 4 out of 10 patients by CLOtest and in 9 out of 10 patients by histology. Anti-HP antibodies were not found in the CSF in any patient. Endoscopic findings of gastritis were present in all patients, and duodenitis was found in 5 patients. Histological presence of gastritis was observed in 9, and intestinal metaplasia in 4 patients.

CONCLUSION: HP-I is increased in AIDP patients as confirmed by histology. Molecular mimicry between ganglioside-like epitopes of the HP lipopolysaccharide and peripheral nerve gangliosides in nerve is a possible proposed mechanism. Larger studies are needed to elucidate the implication of HP-I in the development of AIDP

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GASTRIC MUCOSA CELL TURNOVER FROM PATIENTS WITH EARLY AND ADVANCED GASTRIC CANCER

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Introduction: Early gastric cancer (EGC) is a different biologic entity than advanced cancer (AGC). Epithelial cell turnover alterations play important role during oncogenesis.

Aim: To investigate cell apoptosis and proliferation rates in early and advanced gastric cancer and in the gastric mucosa adjacent to cancer.

Methods: We examined tissue biopsies from 17 EGC, 15 AGC and 18 *H. pylori* positive dyspeptic patients (DPT). We also examined non-dysplastic tissue specimens 5 cm apart from the margin of each tumor. *H. pylori* status and cell proliferation were studied immunohistochemically with an anti-*H. pylori* and MIB-1 by the Avidin-Biotin Complex method. Apoptosis was measured by TUNEL method. The rate of the positive stained cells was count using image analysis technique (SABA).

Results: *H. pylori* was detected in 16/17 and 11/15 early advanced gastric cancers, respectively. Median apoptosis index was significantly higher in EGC (10) and AGC (10) than in DPT (3) (p<0.001). Median proliferation index was not significantly different among EGC (35), AGC (25) and DPT (29) (p=0.3). No significant differences were observed of either apoptosis or proliferation indexes between EGC and AGC. Median apoptosis index was significantly lower in non-dysplastic tissue adjacent to EGC (25) and AGC (18) than in DPT (31) (p=0.05). Median proliferation index was significantly lower in EGC (8) and AGC (12) than in DPT (29) (p<0.001).

Conclusion: Cell turnover is not different between early and advanced gastric cancer, but it is lower in non-dysplastic tissue adjacent to the tumour in comparison with that of chronic *H. pylori* gastritis.