ΠΡΟΣΚΕΚΛΗΜΕΝΕΣ ΞΕΝΟΓΛΩΣΣΕΣ ΑΝΑΚΟΙΝΩΣΕΙΣ ΕΛΛΗΝΩΝ ΕΡΕΥΝΗΤΩΝ

● 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.

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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* 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* infections was found in 13 out of 25 (52%) patients, whereas only one out of six (16.7%) 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.

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

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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 1gx5d.

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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• 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±13.8 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 diagnosis 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 analysis history of gastrointestinal bleeding due to peptic ulceration was risk factor for more severe reflux disease (OR=11.61, 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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● 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, amoxyciline 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 (TNFa), 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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● *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 non-specific 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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