ΠΡΟΣΚΕΚΛΗΜΕΝΕΣ ΞΕΝΟΓΛΩΣΣΕΣ ΑΝΑΚΟΙΝΩΣΕΙΣ ΕΛΛΗΝΩΝ ΕΡΕΥΝΗΤΩΝ

● EPITHELIAL CELL TURNOVER IN NON-DYSPLASTIC GASTRIC MUCOSA ADJACENT TO EARLY AND ADVANCED GASTRIC CANCER

K. Triantafyllou[1], P. Kitsanta[2], D.G. Karamanolis[3], C. Kittas[4], S.D. Ladas[1]
[1]Hepatogastroenterology Unit, Attikon University General Hospital, Athens, Greece, [2]Histopathology Department, Sheffield Teaching Hospitals, Sheffield, United Kingdom, [3]Gastroenterology Department, Tzaneion Hospital of Piraeus, Piraeus, [4]Laboratory of Histology - Embryology, Medical School, Athens University, Athens, Greece

Introduction: Epithelial cell turnover alterations, as well as, p53 and Bcl-2 protein expression play important role during gastric oncogenesis.

Aims & Methods: The aim of the study was to investigate cell apoptosis and proliferation rates, p53 and Bcl-2 protein expression in non-dysplastic tissue (NDT) adjacent to early (EGC) and advanced gastric carcinomas (AGC).

We examined 17 EGC and 15 AGC, and NDT specimens five cm from the margin of each tumor. Cell proliferation, p53 and Bcl-2 expression were detected immunohistochemically using MIB-1, anti-p53 and anti-Bcl-2 monoclonal antibodies. Apoptosis was measured by TUNEL method. The rate of the positive stained cells was count using image analysis technique (SABA).

Results: No differences were observed of either median apoptotic (2 vs 2) or median proliferation (8 vs 12) index between NDT adjacent to early and advanced tumors. While both indices were significantly higher in tumors than in the NDT, no difference was observed of either apoptotic (10 vs 10) or proliferation (35 vs 25) index between EGC and AGC, as well. However, both p53 (4 vs 2, $p = 0.004$) and Bcl-2 (15 vs 5, $p = 0.05$) protein expression was higher in the NDT adjacent to advanced tumors. *H. pylori* positive as compared to *H. pylori* negative gastric mucosa showed higher both p53 (3 vs 1, $p = 0.01$) and Bcl-2 (15 vs 10, $p = 0.05$) immunoreactivity.

Conclusion: 1. Cell turnover is not different between NDT adjacent to EGC and AGC. 2. Both p53 and Bcl-2 protein accumulation is more intense in NDT adjacent to AGC and 3. p53 and Bcl-2 immunoreactivity is related to *H. pylori* infection.

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● SOLUBLE TRIGGERING RECEPTOR EXPRESSED ON MYELOID CELLS (STREM-1): A NEW MEDIATOR INVOLVED

V. Koussoulas[1], S. Vassilliou[1], E.J. Giamarellos-Bourboulis[2], G. Tassias[1], M. Demonakou[3], M. Mouktaroudi[3], H. Giamarellou[3], C. Barbatzas[1]
[1]Gastroenterology, Sismanoglion General Hospital, [2]4th Dept. of Internal Medicine, Athens Medical School, [3]Pathology, Sismanoglion General Hospital, Athens, Greece

Introduction: Triggering receptor expressed on myeloid cells (TREM-1) is a promoter of cytokine production triggered by microbial components. To investigate the significance of sTREM-1 for the pathogenesis of peptic ulcer, sTREM-1 was associated with parameters of gastritis and lipid peroxidation.

Aims & Methods: Forty patients with dyspepsia were enrolled; twenty with peptic ulcer and 20 controls without any macroscopic abnormalities. All patients were endoscoped; gastric juice was aspirated and biopsy specimens were collected from antrum and body. sTREM-1 was estimated by a hand-made enzymeimmunoassay. Lipid peroxidation, indexed by malondialdehyde (MDA), was estimated by the thiobarbitouric assay, after passage through an HPLC system.

Results: Mean (±SE) of sTREM-1 of controls and patients with ulcer was 5.53±0.78 pg/ml and 174.02±98.28 pg/ml respectively (P = 0.006). Mean (±SE) of sTREM-1 concentrations in subjects without evidence of gastritis, *H. pylori* positive gastritis and *H. pylori* negative gastritis were 47.31±16.67 pg/ml, 77.36±43.38 pg/ml and 125.95±103.35 pg/ml, respectively (PNS between patients). sTREM-1 was positively correlated with the degree of mucosal atrophy (P = 0.009) but it was not correlated either with the activity of gastritis or the degree of intestinal metaplasia or MDA levels or with the density of *Helicobacter pylori*.

Conclusion: Results revealed that sTREM-1 might be an independent factor leading to the ulcerative inflammatory process.

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HIGHER REINFECTION RATE IN CELIAC DISEASE PATIENTS AFTER SUCCESSFUL *HELICOBACTER* ERADICATION FOR PEPTIC ULCER DISEASE

K.D. Paraskeva[1], N. Mathou[1], N. Giannakou[2], A. Michalopoulou[2], I. Ghiconti[2], C. Spiliadi[2] and J.A. Karagiannis[1]
[1]Gastroenterology Unit, [2]Department of Pathology, Agia Olga Hospital, Athens Greece

Eradication regimes for *Helicobacter pylori* infection have been proven particularly effective and with low reinfection (or recrudescence) rate that is estimated ~1-2% annually.

Aim of the study was to assess maintenance of the eradication in patients with celiac disease. In 29 patients (16 male, 13 female, age range 19-67 years) with celiac disease, all in clinical and histologic remission on a gluten-free diet, benign peptic ulceration (20 duodenal, 9 gastric) was diagnosed endoscopically. None was on long-term NSAIDs.

Of the 29 patients, 23 were *Hp*+ by histology and *Campylobacter*-like organism (CLO) test and received for both healing and *H. pylori* eradication first-line triple schemes (PPI, clarithromycin, amoxicillin) and those who failed (7/23) to become *Hp*- second-line quadruple schemes (PPI, bismuth compounds, amoxicillin, metronidazole), both at the recommended dose and duration. All had their ulcers healed, 20 of 23 became *Hp*- by histology/CLO test and/or ^{13}C-UBT, whereas three of 23 remained *Hp*+, *Hp*- patients were reevaluated for their *H. pylori* status with 13C-UBT after a mean observation period of 55 months (range 22-68 months). Twelve of twenty (60%) remained *Hp*-, whereas 8/20 (40%) became *Hp*+. Reinfection rate for matched nonceliac patients and for similar observation period was ~10%. Nine of twenty patients, four *Hp*- and five *Hp*+, were re-endoscoped because of dyspeptic symptoms and in three of nine, all *Hp*+, recurrence of duodenal ulcer was diagnosed.

Even if the number of patients studied is small, it seems that patients with celiac disease have higher than expected reinfection rate. Genetic factors influencing susceptibility to *H. pylori* infection by modulating the host immune response might be implicated to explain this observation.

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RABEPRAZOLE 7-DAYS VS RABEPRAZOLE 10-DAYS VS RABEPRATOLE 14 DAYS TRIPLE THERAPY IN THE ERADICATION OF *H. PYLORI* INFECTION – A RANDOMIZED STUDY

S. Karatapanis[1], S. Georgopoulos[3], L. Skorda[4], N. Papantoniou[2], K. Komnianides[1], P. Lisgos[1], C. Psellas[1], A. Mentis[5], H. Tsibidakis[1], H. Kouvidou[4], K. Lois[1], D. Kipraios[1], V. Artikis[4]
[1]1st Department of Internal Medicine and [2]GI Department, General Hospital of Rodos, Rodos, [3]GI Department, Athens Medical, P. Faliro Hospital, Athens, [4]"Elpis" General Hospital, Athens, [5]Department of Medical Microbiology, Institut Pasteur Hellenique, Athens, Greece.

Objective: To evaluate the efficacy and safety of three triple therapies based on rabeprazole (RAB).

Methods: 142 *H. pylori* positive patients (CLO-test, histology) median age 48, range 18-79) with peptic ulcer (n=79) or non-ulcer dyspepsia (n=63) were randomized to receive RAB 20 mg bid, Clarithromycin (CL) 500 mg bid, and Amoxycillin (AMO) 1 gr bid for 1 week (Group A, n=47), or RAB 20 mg bid, CL 500 mg bid and AMO 1 gr bid for 10 days (Group B, n=48) or RAB 20 mg bid, CL 500 mg bid and AMO 1 gr bid for 14 days. *H. pylori* eradication was assessed 4 weeks after completion of treatment (by CLO-test and histology). Clarithromycin sensitivity tests were carried out in the cultured pre-treatment (96/142, 67.6%) *H. pylori* strains.

Results: The eradication rates according to intention to treat analysis (ITT) were 35/47 (74.4%) in Group A, 38/48 (79.1%) in Group B and 43/47 (91.5%) in Group C ($P<0.05$ between Group A and C), and according to per-protocol analysis (PP) 37/47 (78.7%) in Group A, 40/48 (83.3%) in Group B and 44/47 (93.6%) in Group C ($P<0.05$ between group A and C). Primary CL resistance was 9/96 (9.4%). The eradication rate in the CL resistant *H. pylori* strains was 3/9 (33.3%), whereas in the CL sensitive strains 81/97 (93.1%) $P<0.0005$. Side effects in all groups were generally mild and only two patients discontinued treatment due to adverse effects (1 in Group B and 1 in Group C).

Conclusion: We conclude that 14-days triple therapy based on rabeprazole proved more effective than the 7-days regimen in the eradication of *Helicobacter pylori* infection.

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● THE IMPACT OF ERADICATION OF *HELICOBACTER PYLORI* ON THE EVOLUTION OF INTESTINAL METAPLASIA AND GASTRIC DYSPLASIA

H. Tsibidakis[1], N. Papantoniou[2], S. Karatapanis[1], G. Terezakis[1], A. Farmakidis[1], P. Lisgos[1], A. Ponzetto[3]

[1]1st Department of Internal Medicine and [2]Gastroenterology Department, General Hospital of Rodos, Rodos, Greece, [3]Gastroenterology Department, University of Torino, Torino, Italy

Aim: The study aims to investigate the evolution of intestinal metaplasia and gastric dysplasia in patients after successful eradication of *Helicobacter pylori* (*HP*).

Patients and Methods: 103 patients (61 males, 42 females, mean age 65 years) with *HP* positive chronic atrophic gastritis were included in our study. All patients underwent upper GI Endoscopy and biopsies were taken form the antrum and body of the stomach to estimate the degree of atrophy, intestinal metaplasia (IM) and dysplasia. The presence of *HP* was established by histology (Giemsa), serology (ELISA) and with the urea breath test (UBT-test). All patients received *HP* eradication with triple regimens. Eradication was assessed 4 weeks after completion of treatment by UBT and HP histology. All patients were re-examined endoscopically at 6, 12, and 24 months after successful eradication of *HP* to assess the evolution of intestinal metaplasia and gastric dysplasia.

Results: At initial endoscopy we noted type I intestinal metaplasia in 74/103 patients (72%), type IIa in 7/103 (7%), and type IIb in 22/103 (21%). A slight degree of dysplasia was also observed in 15/103 patients (14%). 24 months after the eradication of *HP*, in the patients with IM of type I, we found regression of IM in 11/74, a change to type IIb in 2/74, and to IIa in 3/74 while in 58/74 IM remained unchanged. In the patients with IM of type IIa we observed regression in 3/7, change to IIb in 2/7 while in 2/7 IM remained unchanged. In the patients with IM of type IIb we noted regression in 3/22, change to IIa in 3/22, change to type I in 7/22, and to change in 9/22. At 24 months after successful eradication of *HP* we noted disappearance of dysplasia in 14/15 patients.

Conclusions: The data of our study show that after successful eradication of *HP* there is regression of intestinal metaplasia and dysplasia of the stomach in a significant number of patients.

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