ΠΡΟΣΚΕΚΛΗΜΕΝΕΣ ΑΝΑΚΟΙΝΩΣΕΙΣ ΕΛΛΗΝΩΝ ΕΡΕΥΝΗΤΩΝ

● *HELICOBACTER PYLORI* (*HP*) STATUS, COMPLEMENT C3 PHENOTYPES AND OTHER FACTORS (SOCIOECONOMIC-SOMATOMETRIC) IN PATIENTS WITH DUODENAL ULCER (DU).

A. Archimandritis, D. Kalogeras, M. Chronaki, N. Toupadaki, S. Souyioultzis, P. Apostolopoulos, M. Tzivras.
University of Athens, Medical School, Athens, Greece.

Objective: To investigate the relationship of *HP* status with the complement C3 phenotypes and other, socioeconomic and somatometric, factors in patients with DU.

Methods: Prospective study. One hundred (100) consecutive patients (men=73, age: 19-84 y, mean ± sd = 48 ± 15) with endoscopically, for the first time, diagnosed DU were included. One hundred (100) clinically healthy people, absolutely matched with DU patients with regard to sex, age, and various socioeconomic parameters (income, education, area of residence and number of rooms during the last 10 years), served as controls. C3 phenotypes were determined by electrophoretic immunofixation on suitable cellulose acerate strips using specific antiserum. *Hp* status was determined by searching anti-*Hp* IgG antibodies in the sera (ELISA). Socioeconomic factors (profession according the calories expenditure per day, income, area of residence, education, number of rooms) were studied using a suitable questionnaire. Body weight in Kg and height in cm were measured. Conditional regression analysis. Fisher's exact test and chi-square test were used for statistical analysis.

Results: 1. The prevalence of (C3) F + FS phenotypes was significantly higher in DU patients (p=0.005, RR=2.61, 95% CI: 1.33-5.12). 2. Significantly more patients than controls were *HP*(+) (p=0.0006, RR=4.31, 95% CI: 1.86-9.95). 3. Although *HP* status did not differ in patients and controls concerning C3 phenotypes, significantly more patients (C3) F+FS were *HP*(+) than the controls (Fisher's exact test, p=0.006, RR=1.308, 95% CI: 1.073-1.594). 4. Significantly more *HP*(+) patients than controls had body weight ≤75 Kg and body height ≤175 cm (p=0.002, RR=4.83, and p=0.00001, RR=4.96 respectively). No differences were found in patients and controls, as far as the other examined parameters was concerned.

Discussion: 1. There was an obvious predominance of phenotypically (C3) F+FS, *HP*(+) people among patients with DU. It has been found, (C3) F to have an enhanced capacity to bind with the respective receptor on mononuclear cells in vitro. 2. Significantly more *HP*(+) patients than controls had body weight and height ≤ than 75 Kg and 175 cm respectively, possibly related to some degree of malnutrition.

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● EXPRESSION OF THE Ki-67 PROLIFERATION ANTIGEN IS NOT AFFECTED BY *HELICOBACTER PYLORI* INFECTION IN PATIENTS WITH CHRONIC OR CHRONIC ACTIVE GASTRITIS.

I. Gikonti, P. Karakitsos, E. Spathi, I. Georgoulakis, A. Daritsis, E. Karavanakis, J.A. Karagiannis.
"Agia Olga" Hospital, Athens, Greece.

Aim of the study was to assess the expression of cell proliferation antigen Ki-67 in gastritis patients with or without *HP* infection. Forty dyspeptic patients with endoscopic findings of gastritis (28 male, 22 female, age range 18-68 years) were studied. Multiple endoscopic biopsies and brushing were taken from the body and antrum of the stomach for histology and cytology studies. 11/40 patients were defined by histology as having chronic gastritis (Group A) while 29/40 chronic active gastritis (Group B). 6/11 from group A and 16/29 from group B were *HP*(+) (by histology, cytology and CLO test). For Ki-67 expression the monoclonal antibody MIB1 (Ylem, Italy) was used in paraffin sections for immuno-histochemistry with the ABC technique using DAB as chromogen and methyl-green for counter-staining. In every case at least 200 serial nuclei from intact pits were counted. Quantitation of Ki-67 expression was performed as percentage of positive cell nuclear area with the image analysis system CAS 100. Expression of Ki-67 was observed mainly in the neck and fundus of the pits.

In group A Ki-67 expression was found 6-9% (7.17+1.12, mean+SD) while in group B 11-45% (27.76+10.6). Taking 10% as cut-off value for enhanced Ki-67 expression, there was no significant difference between *HP*(+) (22/40) and *HP*(-) (18/40) samples from both groups A and B (chi-square, p=0.85). Looking at each group separately, there was also no significant, difference between *HP*(+) and *HP*(-) samples with the proportion test (6/11 *HP*+ and 5/11 *HP*-, z=0.298 and p=0.38 in group A and 16/29 *HP*+ and 13/29 *HP*-, z=0.37 and p=0.35 in group B).

In summary, Ki-67 expression was found higher in chronic active gastritis, but was not affected by the presence or not of *Helicobacter pylori*.

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● DETECTION OF *H. PYLORI* IN PATIENTS WITH ACUTE UGI HAEMORRHAGE FROM ULCERS WITH STIGMATA OF RECENT BLEEDING.

A. Konstantinidis, A. Germanopoulos, E. Kaneliopoulou, A. Pantes, V. Vamvakousis, V. Ekonomou, J. Karubalis, P. Georgopoulos, J. Drikos, N. Skandalis.
Gastroenterologist Unit, General Hospital "G. Gennimatas", Athens, Greece.

Aim: To asses the prevalence of *HP* in patients with acute UGI haemorrhage from ulcers with endoscopic stigmata of recent bleeding, as well the proper time of biopsying for CLO test and histologic detection of *HP*.

Method: Eight three patients (58 male, 25 female, aged 22-82 years, mean age 54.5 y) presenting with acute UGI bleeding from ulcers with stigmata of recent haemorrhage.

Method: Emergency UGI endoscopy was performed and once an ulcer with stigmata of haemorrhage (slow rate bleeding, visible vessel, adherent clot) was encountered, haemostasis was done injecting epinephrine 1:10000 +/-1-2 cc ethanolamine oleate 5%. Biopsies were taken from the antrum and corpus for both CLO test and histology. In group A patients biopsies were taken right after the haemostasis, in group B during a 2nd endoscopy 4-8 days later.

Results: In group B patients the prevalence of *HP* was very low in CLO test and in histologic *HP* positive patients the number of *HP* was very low almost in all cases (see Table 1).

Conclusions: In patients with acute UGI haemorrhage from ulcers with stigmata of recent bleeding, the rate of *HP* detection by CLO test and/or histology is high, if biopsies were taken during the emergency endoscopy.

Table 1.

Patients	**ulcers**		***H. pylori***			
no	Bulb	Stomach	CLO test	Histology	Total	NSAIDs
Group A 44	33	11	32 (72.7%)	38 (86.4%)	41 (93.2%)	26 (59%)
Group B 39	29	10	8 (20.5%)	25 (64.1%)	25 (64.1%)	17 (43.6%)

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● IS THE ACTIVITY AND SEVERITY OF GASTRITIS AND *HELICOBACTER PYLORI* LOAD RELATED TO THE OUTCOME OF ANTI-*H. PYLORI* TREATMENT?

G.J. Mantzaris, Em. Archavlis, P. Amberiadis, D. Kourtessas, Kal. Petraki, G. Triantafyllou.

Dept of Gastroenterology, Evangelismos Hosp., Athens, Greece.

Aim: To test the hypothesis that anti-*H. pylori* (*Hp*) treatment is more effective in patients with more severe and active *Hp* gastritis we evaluated the outcome of OAC_{10} (omeprazole 20 mg bd, clarithromycin 0.5 g bd and amoxycillin 1 g bd for 10 days) in relation to the pretreatment grading of severity and activity of gastritis and the *Hp* load in patients with active duodenal or gastric ulcer (DU, GU) or non ulcer dyspepsia (NUD).

Study design: Prospective, single center, investigator-blind.

Patients: 80 DU patients [45M, 35F, mean age 46y (17-68), 40 smokers, 16 NSAID users], 16 GU patients [7M, 9F, 49y (31-69), 6 smokers, 5 NSAID users] and 27 NUD patients [12M, 17F, 53y (18-69), 5 smokers, 5 NSAID users].

Methods: Patients were endoscoped before and 2 months after the completion of treatment. The presence of *Hp* and its eradication were assessed by CLO tests and histology (H&E, modified Giemsa) on 4 antral and 4 body/fundic biopsies. The severity and activity of gastritis and the *Hp* load were graded (0-3, Houston classification).

Results: Following treatment, all ulcers healed. *Hp* infection was eradicated in 52/80 (65%) DU, 11/16 (69%) GU and 20/27 (74%) NUD patients. Smoking, NSAIDs or a past bleeding episode did not adversely influence the outcome of OAC_{10}. The mean (SE) values for pretreatment grading of severity and activity of gastritis and *Hp* load were respectively 2.54(.06)/2.46(.07)/2.77(.05) for DU, 2.50(.13)/2.44(.16)/2.75(.11) for GU, and 2.41(.11)/2.48(.11)/2.59(.11) for NUD patients; scores were higher for DU and GU than NUD patients but differences were not significant. The mean (SE) scores for pretreatment grading of severity/activity/*Hp* load were respectively: for DU/*Hp*- and DU/*Hp*+ patients 2.52(.07)/2.44(.04)/2.75(.06) and 2.57(.1)/2,54(.1)/2.86(.05); for GU/*Hp*- and GU/*Hp*+ patients 2.36(.15)/2.2(.19)/2.73(.14) and 2.80(.2)/2.80(.2)/2.8(.2); for NUD/*Hp*- and NUD/*Hp*+ patients 2.54(.14)/2.30(.15)/2.55(.14) and 2.29(.18)/2.29(.18)/2.71(.18); for all *Hp*- and *Hp*+ patients irrespective of treatment group 2.48/2.39/2.71 and 2.52/2.52/2.83 (mean value). Comparison of values for pretreatment grading of activity/severity of gastritis or *Hp* load did not reveal any significant differences between *Hp*- and *Hp*+ patients within or between groups. Ulcer patients had higher gastritis and *Hp* load scores than NUD patients as did *Hp*+ than *Hp*- patients within each group but differences were not significant.

Conclusion: Pretreatment scores for severity and activity of gastritis and *Hp* load did not affect the outcome of OAC_{10}. Virulent *Hp* factors may be important in determining not only the course of *Hp* infection but also the outcome of treatment.

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● *HELICOBACTER PYLORI* INFECTION AND REFLUX ESOPHAGITIS: IS THERE ANY ASSOCIATION?

S. Manolakopoulos, A. Christidou, A. Katrsanika, D. Tzourmakliotis.
Department of Gastroenterology, Polyclinic General Hospital of Athens, Athens, Greece.

Helicobacter pylori (*Hp*) infection is closely associated with peptic ulcer disease, but its role in the pathogenesis of reflux esophagitis remains obscure. The aim of our study was to determine whether there was a different prevalence of *Hp* infection in cases with esophagitis. We retrospectively studied 218 (male/female=141/77, mean age ±sd=49.7 ± 14) consecutive patients (pts) undergoing upper gastrointestinal (UGI) endoscopy. Pts were selected from the cohort of pts attending our Endoscopy Unit on the basis of *a.* a diagnosis of duodenal ulcer, esophagitis, hiatus hernia without esophageal inflammation and an endoscopy without abnormalities *b.* the availability of quick urease test and/or antral biopsy. Duodenal ulcer (group 1) was diagnosed in 117 pts, hiatus hernia without inflammation (group 2) was diagnosed in 36 pts and erosive esophagitis (group 3) in 46 pts. Nineteen pts had normal UGI endoscopy (group 4). Prevalence of *Hp* infection was as follows: Group 1=90.6%, Group 2=60%, Group 3=54.3% and Group 4=55%. We did not find any difference in the prevalence of *Hp* infection between group 2, 3 and 4 ($p=0.8$). Pts with duodenal ulcer had a significantly higher incidence of colonization by *Hp* infection than other groups ($p<0.0001$). The comparison of *Hp* prevalence between pts with grade 1 esophagitis and those with grade 2-4, did not reveal any difference ($p=0.7$).

Conclusions: There is no significant difference in *Hp* prevalence between pts with reflux esophagitis, hiatus hernia and controls. The severity of esophagitis does not correlate with *Hp* infection. Our data suggest that *Hp* is unlikely to be etiologically important in cases with reflux esophagitis.

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● CLINICAL SPECTRUM OF GASTROESOPHAGEAL REFLUX DISEASE AFTER *HP* ERADICATION IN PATIENTS WITH DUODENAL ULCER.

S. Michopoulos, P. Tsibouris, A. Balta, H. Bouzakis, G. Galanopoulos, N. Kralios.
Gastroenterology Unit, "Alexandra" University Hospital, Athens, Greece.

Esophagitis is reported to be more frequent after successful *Helicobacter pylori* (*HP*) eradication in patients with duodenal ulcer (DU).

The **aim of the study** was to investigate whether *HP* eradication affects the incidence and severity of gastroesophageal reflux disease (GERD) and esophagitis (E) in patients with prior DU.

Patients and methods: 163 patients with prior DU were contacted 24±0.75 (14-45) months after *HP* eradication was accomplished. 151 (85 men, 71 smokers) consented to giving information, out of whom 101 (57 men, 47 smokers) agreed to undergo an upper GI endoscopy. A biopsy from the antrum was set to CLO®test (Delta West LTD), while 2 biopsies from both antrum and fundus were stained with H&E and modified Giemsa. A 4 grade rating scale was used to evaluate symptoms of GERD (0=none, 1=symptoms could be ignored, 2=symptoms cannot be ignored and 3=symptoms influence concentration or interrupts daily activities). E was graded according to Savary-Muller classification.

Stat: Wilcoxon pair test, chi-square.

Results:

GERD:	**PreRx**	**PostRx***	**E:**	**PreRx**	**PostRx****
0	123	91	0	147	91
1	18	32	1	3	8
2	6	15	2	1	2
3	4	13	3,4	0	0

*p<0.001, **p<0.01 PreRx & PostRx: Pre and post eradication period

From those who had GERD before (28), 11 have no symptoms, 6 (2E) have the same symptoms and 11 (1E) present an aggravation while those who were free of symptoms (123) initially, 32 (10E) report GERD after treatment. Analysing the pairs' differences among the various degrees of GERD and E, we found (Wilcoxon): p<0.001 for GERD and p<0.05 for E. Age, smoking, alcohol consumption and hiatal hernia are not predictive factors, neither for GERD appearance nor for E. Eight patients were positive for *HP*, 2 of them with GERD (1E).

Conclusion: 1) DU patients develop more frequently GERD and E after *HP* eradication. 2) Pre-treatment GERD is aggravated in 40% of patients and first appearing GERD is reported in another 26%. 3) E is also more frequent but usually of mild degree at least 2 years after eradication.

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● A PROSPECTIVE, RANDOMIZED, CONTROLLED, INVESTIGATOR-BLIND, SINGLE-CENTRE TRIAL COMPARING TWO ONE-WEEK OMEPRAZOLE TRIPLE THERAPIES FOR HEALING PEPTIC ULCER AND ERADICATION OF *H. PYLORI* INFECTION.

C. Tzathas, Kal. Petraki, P. Amberiadis, D. Kourtessas, P. Christoforidis, N. Florakis, Em. Archavlis, G. Triantafyllou.
Evangelismos Hospital, Athens, Greece.

Aim: To evaluate the efficacy of two one-week triple therapies in healing peptic ulcer and eradicating *Hp* infection.

Patients and Methods: Eight six unselected, consecutive patients (60 males, 26 females, mean age 48.5 years, range 16-73) with endoscopically diagnosed active duodenal (DU, n=72) or gastric ulcer (GU, n=14) and *Hp* infection were randomly allocated to receive for one week either omperazole (20 mg bd), amoxycillin (1 g bd) and clarithromycin (0.5 g bd) (OAC_{500}, n=43) or omeprazole (20 mg bd), metronidazole (0.5 mg bd) and clarithromycin (250 mg bd)(OMC_{250}, n=43). All patients received omeprazole (20 mg a day) for further two weeks. Patients were re-endoscoped 2 months after the completion of treatment. At each endoscopy, the presence of *Hp* and its successful eradication were assessed respectively by positive and negative CLO tests, histology (H&E, modified Giemsa) and immunohistochemistry (rabbit anti-*Hp* mcAb, Dakko) on 3 antral and 3 body biopsies.

Results: There were no significant differences in any patient or disease related parameters between study groups. Six patients in the OAC_{500} and 12 patients in the OMC_{250} were lost to follow up. Two patients could not tolerate OMC_{250} and were withdrawn from the study; minor adverse events were recorded in five and 3 patients in the OAC_{500} and the OMC_{250} groups, respectively. Ulcer healing and *Hp* eradication rates are given in the table (n, %).

Treatment	**Intention-to-treat (ITT)**		**Per-protocol (PP)**	
group	Ulcer healing	*Hp* –ve	Ulcer healing	*Hp* –ve
OAC_{500}	35/43 (81%)	30/43 (70%)	35/37 (95%)	30/37 (81%)
OMC_{250}	26/43 (61%)	21/43 (49%)	26/29 (90%)	21/29 (72%)

There were no significant differences between study groups (Fisher's exact test).

Conclusion: This study by mirroring a "real" world (single centre, unselected patients, ITT analysis, etc) emphasizes the fact that eradication rates of *Hp* infection are far from those reported in the literature.

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● *HELICOBACTER PYLORI* DENSITY AS A PREDICTOR OF GASTRIC CELL PROLIFERATION.

*K. Patikos, [1]A. Zizi-Serbetzoglou, K. Triantafyllou, G. Katsoras, I. Milonakou, [1]I. Elemenoglou, D.G. Karamanolis.

Gastroenterology and [1]Pathology Departments "Tzaneion" Hospital, Piraeus, Greece.

Helicobacter pylori (*Hp*) seems to increase gastric cell proliferation and in this way may play a role in gastric carcinogenesis. The aim of this study was to reveale predictive factors of gastric cellular proliferation status in *Hp*(+) patients and to investigate the effect of eradication therapy in this status. Forty-one patients with *Hp*(+) pangastritis were included in the study. Two biopsies from gastric antrum and 2 from gastric body at diagnosis and 1 month after the end of eradication treatment were obtained endoscopically to evaluate the degree of gastritis, gastric atrophy, intestinal metaplasia and *Hp* density. Ki-67 Antigen was studied immunohistochemically with MIB 1 (YLEM) monoclonal antibody and expressed as the labelling index per cent (LI%). Total antrum LI% (mean±SE) decreased from 13.41±2.73 at diagnosis to 1.39±0.36 post-treatment ($p=10^{-5}$). Total body LI% decreased from 8.23±1.64 to 1.24±0.29 post-treatment ($p=10^{-4}$). The difference between pre- and post- treatment antrum and body LI% was statistically significant in both eradicated ($p<10^{-5}$) and not eradicated (p=0.0002) patients. Multivariate analysis where age, degree of gastritis, gastric atrophy, intestinal metaplasia and *Hp* density were included, revealed that *Hp* density is the only independent predictive factor of gastric cell proliferation status in post-treatment period (Odds Ratio=2.8, 95% CI: 1.8-4.4, p=0.024). On the contrary none of the above factors influences gastric cell proliferation in pre-treatment period.

Conclusions: 1) Eradication therapy decreases gastric cell proliferation in both eradicated and not eradicated patients in early post-treatment period. 2) Post-treatment *Hp* density in gastric mucosa is a predictor of gastric cellular proliferation.

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Το Διοικητικό Συμβούλιο και η Οργανωτική Επιτροπή του Συνεδρίου
ευχαριστούν τις ακόλουθες φαρμακευτικές εταιρείες
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