ΠΡΟΣΚΕΚΛΗΜΕΝΕΣ ΑΝΑΚΟΙΝΩΣΕΙΣ ΕΛΛΗΝΩΝ ΕΡΕΥΝΗΤΩΝ

● THE ROLE OF GASTRIC CYTOLOGY IN THE INVESTIGATION OF *HELICOBACTER PYLORI*

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Introduction: *Helicobacter pylori* has been implicated in the pathogenesis of chronic gastritis, gastric and duodenal ulcer, and possibly gastric carcinoma. The organism may be detected by invasive and non-invasive methods with variable sensitivity. The aim of this prospective study was to evaluate the role of direct brush taken smears, imprints and liquid phase cytology in the investigation of *HP.*

Material and Methods: The study was carried out on cytology smears taken during endoscopy from 108 patients with gastritis and gastric ulcer. Paired gastric biopsy and gastric brush specimens were collected. One biopsy was tested for urease using the CLO test, the other was processed to paraffin and consecutive sections were stained with haematoxylin and eosin, modified Giemsa. The brush and imprints specimens were stained with Papanicolaou and Giemsa stains. The brush was also immersed in CytoLyt (Cytyc) and two ThinPrep (Cytyc, Marlborough, MA) slides were made. The ThinPrep smears stained using Papanicolaou and Giemsa techniques.

Results: In 92 out of the 108 cases a correlation between the histologic examination was found (φ=0.71). In 45 out of 50 cases a correlation between the conventional cytologic examination (smears and imprints) and CLO was found (φ=0.76). In 94 out of 108 cases a correlation between the cytologic and histologic examination was found (φ=0.84). The application of the proportion test failed to reveal any statistically significant difference between the conventional cytologic examination and the liquid phase cytology (z=-0.350, p=0.37).

Conclusions: Cytology appears to be an accurate method for the identification of *HP.* Moreover, liquid phase cytology offers the opportunity to preserve material for further investigations using molecular biology and analytical cytology techniques. However, in some cases low cellularity samples were obtained, due to the difficulty to extract the obtained mucus material from the brush.

Δημοσιεύθηκε στο Gut 1999;45(Suppl III):A130.

● IS *H. PYLORI* PRESENT IN DENTAL PLAQUE OF CHILDREN AND THEIR FAMILIES?

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Background: *H. pylori* infection and intrafamilial spread is common among Greek pediatric population.

Aim: To investigate the presence of *H. pylori* in dental plaque of children with upper GI symptoms who underwent endoscopy and their family members.

Methods: The study sample consisted of 35 children aged 4 to 14 years old and 49 family members (mother and/or father). Gastric biopsies, gastric juice and serum were collected from all children. Dental sampling was done in parents and in children before endoscopy. Each subgingival plaque sample was collected with sterile paper pointers from 4 healthy and 4 diseased gingival crevices and a PCR method was used for the detection of *H. pylori.*

Results: 15 out of 35 (43%) children were considered *H. pylori* infected by at least one method (CLO, histology, culture). Specifically, *H. pylori* was detected in antral biopsies of 100%, 93% 77% of *H. pylori* positive children by CLO, histology and culture, respectively. Gastric juice was positive for *H. pylori* in 66% and 57% of patients by PCR and culture. Serum IgG antibodies were found in 12/15 (80%) of *H. pylori* biopsy positive children. In 11/15 families of *H. pylori* positive children, at least one member was found to be *H. pylori* infected by serum antibodies and/or by urea test and/or endoscopy. *H. pylori* was detected in dental plaques of 6/15 biopsy positive children. It was also detected in one biopsy negative child. *H. pylori* was detected -at least in one member- in dental plaques of 7/15 families of *H. pylori* positive children. Children who had *H. pylori* identified in their dental plaque belonged to families that also had *H. pylori* in dental plaque.

Conclusions: *H. pylori* is detected in dental plaque of children and their families and that may play an important role -acting as a "reservoir"- for the intrafamilial spread and transmission.

Δημοσιεύθηκε στο Gut 1999;45 (Suppl V):A182.

● PEPTIC ULCER DISEASE IN CIRROSIS. THE ROLE OF *HELICOBACTER PYLORI*

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Background: Although *Helicobacter pylori (Hp)* is the major pathogenetic factor for peptic ulcer disease (PUD) in the general population and its successful eradication significantly eliminates the risk of ulcer recurrence the role of *Hp* for PUD in cirrhotic patients (CP) remains controversial.

Aims: To evaluate the prevalence, of *Hp* infection in PUD and the effect of *Hp* eradication in ulcer recurrence in CP.

Methods: Fourteen patients [12M, 2F, mean age 61.6y (range 48-71)] with cirrhosis (9 alcoholic, 2 HBV, 2 HCV, 1 cryptogenic - Child Pough A:7, B:5, C:2) and peptic ulcer (5G, 9D) diagnosed at endoscopy for investigation of varices (6p) epigastric pain (4p) or bleeding (4p) and fourteen non CP with PUD matched in sex, age and ulcer characteristics with the CP were included in the study. All patients were not using NSAIDs/aspirin and had never previously received *Hp* eradication treatment. Patients underwent endoscopy at entry, four weeks after eradication therapy and in case of clinical relapse. *Hp* infection was diagnosed by CLO test and histology. All patients were followed for 12 months with start point of the follow-up period 2 months after the inclusion endoscopy. *Hp*(+) patients received *Hp* eradication therapy for one or two weeks followed by two to three week omeprazole administration, *Hp*(-) received a four week omeprazole regimen. X^2 (Yate's correlation) was used for statistical analysis.

Results: Nine CP were infected with *Hp* (64.3%) in comparison to all fourteen non-CP who were *Hp*(+) ($p<0.001$). Successful eradication was achieved in seven CP (77.7%) and in eleven controls (78.5%). Two CP and two out of three controls who remained *Hp*(+) after eradication therapy relapsed in the follow-up period (NS). Six out of eleven *Hp*(-) CP (50%) relapsed two of them rebled and one died. In contrast, only one *Hp*(-) control (9%) relapsed in the follow-up period ($p<0.05$).

Conclusions: 1. *Hp* prevalence in PUD is significantly lower in CP than in controls. 2. *Hp* eradication does not abolish the risk of ulcer recurrence in the CP as it does in the controls. 3. This may implicate the need for maintenance antisecretory treatment in all CP with PUD especially in high risk subgroups.

Δημοσιεύθηκε στο Gut 1999;45(Suppl V):A184.

● HERPES SIMPLEX VIRUS TYPE I (HSV-1) AS A POSSIBLE FACTOR CAUSING PEPTIC ULCER AND ITS RELATIONSHIP WITH *HELICOBACTER PYLORI*

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Aim: of the study was the identification of HSV-1 from biopsy specimens of patients with active peptic ulcer as well as the investigation of a possible relationship between HSV-1 and *Helicobacter pylori (HP)* for the development of a subset of peptic ulcer disease.

Materials-Method: 90 patients, 34 with prepyloric and 56 with duodenal ulcer as well as 50 persons with no evidence of peptic ulcer considered as a control group were examined. Biopsies were taken from the crater and rim of the ulcer, 3 cm away from the ulcer, as well as from exdoxopically healthy mucosa area of the patients and control group. Malignancy in patients with prepyloric ulcer was excluded histologically. The method used for the identification of HSV-1 was polymerase chain reaction (PCR). Moreover the detection of the *HP* was achieved by CLO test at samples taken from the antrum and the body of the stomach.

Results: Using PCR method for the presence of HSV-1, positive results were found in 28 out of 90 patients (31%) and specifically in 17 out of 56 patients with duodenal ulcer (30.4%) and in 11 out of 34 patients with prepyloric ulcer (32.4%). Positive samples for HSV-1 were obtained only from the crater or rim of patients with peptic ulcer disease. Statistically significant difference was found between peptic ulcers cases positive for both HSV-1 and *HP* (68%) and those negative for HSV-1 and positive for *HP* (91.9%) (P value 0.009). This difference becomes larger in cases of prepyloric ulcers positive for HSV-1, where the percentage of positive results for *HP* is limited to 36.4% of cases.

Conclusions: The above results suggest that HSV-1 is possibly associated with a subset of peptic ulcers. Moreover HSV-1 may act independently from *HP,* causing peptic ulcer disease.

Δημοσιεύθηκε στο Gut 1999;45(Suppl III):A76.

● INFLUENCE OF COMORBID DISEASES ON ULCER HEALING RATE (UHR) AFTER SUCCESSFUL *H. PYLORI* ERADICATION (HPE)

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Prolongation of acid suppression after HPE treatment is dusputable.

Aim: To evaluate the influence of comorbid diseases on UHR.

Patients-Methods: In 181 patients (mean age 52.2±14.1 years, 105 male, 66 smokers) successfully eradicated with various 10 day regimens, endoscopy was performed 1 and 3 months after successful HPE, to access UHR. HPE was verified by histology, CLO-test and ^{13}C-urea breath test. Complete clinical and laboratory evaluation for comorbid diseases was performed. For unhealed ulcers serum gastrin was evaluated and omeprazole 20 mg qd was given. Stat: X^2 -test, t-test.

Results: UHR after successful HPE was: 89% (CI 84.3-93.6). No patient had high serum gastrin. All ulcers healed within 3 months of follow up. Chronic renal failure (10 patients-none on NSAID, UHR: 40%, p<0.001), cardiovascular diseases (28 patients, UHR: 79%, p=0.05) and NSAID use (32 patients, UHR: 69%, p=0.01) were related with reduced UHR, after successful HPE. Healing failure for cardiovascular diseases, remained significant despite NSAID use exclusion (p=0.01). Gastrointestinal bleeding (50 patients, p=0.78), rheumatic diseases, endocrinopathies, diabetes melitus, extragastric neoplasias, neuropathies and pneumonopathies did not affect ulcer healing. Excluding patients with chronic renal failure, NSAID use and cardiovascular disease. UHR after successful HPE increase to 100%.

Conclusions: 1) UHR after successful HPE is high. 2) Chronic renal failure, cardiovascular diseases and NSAID use are related with reduced UHR despite successful HPE. Those patients need prolongation of antisecretory treatment.

Δημοσιεύθηκε στο Gastroenterology 1999;116(4):A253.

● PCNA INDEX IN *H. PYLORI* POSITIVE GASTRITIS BEFORE AND AFTER ERADICATION OF *H. PYLORI*

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Background: PCNA is a suitable tool to study cell proliferation rate. *H. pylori (HP)* infection has been suggested to increase gastric epithelial cell proliferation.

Aims: To investigate PCNA expression by gastric epithelial cells in *HP* gastritis before and after *HP* eradication and to look for any correlation with gastritis, parameters (Sydney classification).

Patients-Methods: We studied 30 dyspeptic patients (age: 27-81). Multiple antral and body biopsies were taken for CLO test, histopathology (H&E, Giemsa, Alcian blue) and immunohistochemistry (PCNA, clone PC10, mouse monoclonal, NOVOCASTRA), at presentation and 116±9.5 days after the initial endoscopy; eradication therapy was given in *HP*(+) patients. PCNA was studied in 3 zones: 1=surface + upper 1/3 of the gastric pit (gp), 2=rest 2/3 of gp, 3=glands. Positive nuclei were counted by image analysis (software: image tools); results were expressed as number of positive nuclei per studied zone (PCNA index).

Results: 23/30 were *HP* (+); in 20/23, *HP* was eradicated. 7/30 *HP* (-) served as controls (age matched). PCNA index in *HP* (+): antrum: 1=8.82±2.2=16.35±3.6, 3= 5.11±1.22 (ANOVA, p=0.008), body: 1=5.8±1.4, 2=10.15±2.54, 3=2.85±0.9 (ANOVA, p=0.018). After eradication: antrum: 1=9.8±2.35, 2=11.88±2.9, 3=1.35±0.8 (ANOVA, p=0.0035), body: 1=4.5±1.36, 2=6.35±1.92, 3=1.43±0.56 (ANOVA, p=0.034). In 7 *HP* (-) patients PCNA index was: in antrum: 1=2.57±1.67, 2=3.57±2.84, 3=0.57±0.57 (ANOVA, p=0.520) and differed significantly per zone from *HP*(+) (Mann-Whitney, 1: p=0.041, 2: p=0.038, 3: p=0.053). No correlation was found between PCNA index and gastritis parameters.

Conclusions: Gastric epithelial cell proliferation rate is increased in *HP* infection as compared with *HP* (-). This seems to decrease after eradication, though not significantly at least for the studied period.

Δημοσιεύθηκε στο Gut 1999;45(Suppl V):A61.

● RANDOMISED STUDY OF TWO SECOND-LINE QUADRUPLE THERAPIES AFTER FAILED 10-DAYS *H. PYLORI* TREATMENT WITH OMEPRAZOLE, CLARITHROMYCIN AND AMOXYCILLINE (OCA-10)

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Aim: There is no defined second-line treatment after failure to cure *H. pylori* by OCA-10 regimen. The aim of this study was to evaluate the efficacy of two quadruple regimens as salvage therapy of *H. pylori* and to assess the impact of microbial resistance on the efficacy of the second line treatment.

Patients-Methods: Fifty consecutive patients (aged 18-79 years, mean 46.5, 29 men, 23 smokers) with duodenal ulcer (n=19) or non-ulcer dyspepsia (n=31) and persistent *H. pylori* infection (confirmed by histology and culture) were randomly assigned to one of the two quadruple schemes for 7 days: Omeprazole (O) 20 mg bid+Bismuth subcitrate (B) 125 mg qid+ Metronidazole (M) 500 mg bid+Tetracyclin (T) 500 mg qid (group OBMT, n=23) or Clarithromycin (C) 500 mg bid (group OBMC, n=24). Eradication was assessed 4-6 weeks post-treatment. Antibiotic sensitivity test was carried out prior to the second-line treatment using the agar dilution method.

Results: 47 patients completed the project. The *H. pylori* cure rates (overall and according to pre-treatment microbial resistance) were:

Resistance pattern (ITT)	***H. pylori* cure**	
	OBMT	**OBMC**
MET_S CLA_S 29/50 (58%)	15/15	11/13
MET_S CLA_R 4/50 (8%)	2/2	1/2
MET_R CLA_S 11/50 (22%)	3/4	3/5
MET_R CLA_R 6/50 (12%)	2/2	1/4
Overall Per Protocol (PP)	22/23 (95.6%)[a]	16/24 (66.7%)[a]
Overall Intention To Treat (ITT)	22/24 (91.7%)[b]	16/26 (61.5%)[b]

a: $p<0.05$, b: $p<0.05$

No patient discontinued treatment due to usually mild side effects.

Conclusions: Quadruple therapy with O+B+M+T is more effective than O+B+M+C as a second line *H. pylori* treatment after eradication failure with OCA-10. Excluding previously failed antibiotics from the quadruple scheme may be necessary independently of *H. pylori* resistance pattern.

Δημοσιεύθηκε στο Gut 1999;45(Suppl V):A272.