ΞΕΝΟΓΛΩΣΣΕΣ ΑΝΑΚΟΙΝΩΣΕΙΣ (ABSTRACT) ΕΛΛΗΝΩΝ ΕΡΕΥΝΗΤΩΝ

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● G0712

EXPRESSION OF FASL AND PS2 MOLECULES ON GASTRIC EPITHELIAL CELLS IS INDEPENDENT OF HELICOBACTER PYLORI (HP) INFECTION.

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Interaction between Fas and FasL seems to be involved in apoptosis mainly in the immune system; pS2 is a trefoil peptide involved in gastrointestinal-(GI)mucosa repair. In this prospective work the expression of FasL and pS2 in HP gastritis before and after eradication was studied. Patients-Methods. Twenty four symptomatic patients were studied. Upper GI endosopy was performed and at least 4 biopsies were obtained (antral:2, body:2) for CLO test and histology(Sydney classification);18 patients were HP(+)and 6 HP(-) by both Eradication therapy was given to HP(+)patients and all were endoscoped 102 ± 5.3d after. Formalin-fixed paraffin embedded tissue sections were stained by the ABC immunoalkaline phosphatase method and Fast-Red.Rabbit polyclonal anti-FasL(Santa-Cruz)and anti-pS2 (YLEM) antibodies were used.FasL antigen unmasking was performed by heat treatment in microwave oven.Staining intensity was graded from 0–2+. Results.At baseline, FasL and pS2 were steadily expressed by gastric epithelial cells in both HP(+) and HP(-) patients; however, pS2 was not expressed in areas of intestinal metaplasia. The pattern of expression for both molecules remained almost the same after eradication. Conclusions. 1.FasL and pS2 were expressed by gastric epithelial cells irrespective of HP infection. 2. HP infection does not seem to influence epithelial restitution through a pS2 - dependent mechanism. 3. FasL possibly contributes: a. to epithelial homeostasis by inducing apoptosis in Fas(+) epithelial cells (unpublished data); b.to downregulation of inflammation by inducing apoptosis in Fas(+) mucosa infiltrating activated lymphocytes.

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● G1104

RANDOMIZED STUDY TO INVESTIGATE IF PROLONGATION OF OMEPRAZOLE (O) TREATMENT ADDS TO THE HEALING RATE OF DUODENAL ULCERS (DU).

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S. Michopoulos, P. Tsibouris, A. Balta, M. Sotiropoulou*, M. Economou, G. Galanopoulos, I. Vougadiotis, N. Kralios. Gastroenterology and Pathology* Units, Alexandra University Hospital, Athens, Greece. Although H. pylori eradication (HPE) treatment is universally accepted for patients with DU, it is not clear if prolongation of antisecretory treatment beyond the end of antibiotic administration is necessary. Aim of the study: To evaluate if prolongation of O treatment could influence healing results. Patients and methods: 202 DU patients (mean age 52.3± 14.3 years, 114male, 72smokers) HP(−) after finishing a 10 days HPE regimen with O 20mg bid, clarithromycin 500mg bid & amoxycilline 1g bid, were randomly assigned to continue on O 20mg qd for 7 (GroupI-62 patients), 21(GroupII-64 patients) or 35 days (Group III-76 patients) respectively. Groups were comparable for sex, age and smoking. Endoscopy was performed one month after treatment completion and HPE was considered successful when both histology & CLO-test*(Delta West LTD) were negative. For conflicting results ^{13}C-urea breath test was performed. For unhealed ulcers serum gastrin was evaluated. Stat: X^2 -test, t-test. Results: All patients had a follow up endoscopy.HPE rates and healing rates among patients with successful HPE are shown on the table: No patient had high gastrin in serum. Conclusions: 1) Ulcer healing after successful HPE is high. 2) Prolongation of antisecretory treatment for more than a week following HPE regimen, does not increase ulcer healing rate.

	Group I	Group II	Group III	Stat
HPE%(CI)	87.1(78.5-95.7)	90.6(83.3-97.7)	93.4(87.7-99.1)	NS
Healed%(CI)	92.5(85.4-99.6)	93.1(86.4-99.8)	90.1(83.0-97.2)	NS

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● G1103

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

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S. Michopoulos, P. Tsibouris, A. Balta, M. Sotiropoulou*, M. Economou, G. Galanopoulos, I. Papaspyrou*, N. Kralios. Gastroenterology and Pathology* Units, Alexandra University Hospital, Athens, Greece. Prolongation of acid suppression after HPE treatment is disputable. Aim of the study: To evaluate the influence of comorbid diseases on UHR. Patients and 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 20mg 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 (32patients, 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 mellitus, extragastric neoplasias, neuropathies and pneumonopathies did not affect ulcer healing. Excluding patients with chronic renal failure, NSAID use and cardiovascular diseases, UHR after successful HPE increased 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.

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G0738

LANSOPRAZOLE VS RANITIDINE BISMUTH CITRATE (RBC) BASED SHORT-TERM TRIPLE THERAPIES FOR HELICOBACTER PYLORI (HP) ERADICATION: A RANDOMISED STUDY WITH 6-MONTH FOLLOW-UP

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The aim of our study was to compare the eradication rates and the safety profiles of two short-term therapies containing Lansoprazole (LAN) or RBC. **Patients-Methods:** Eighty-five patients (median age 46, range 17–82) with documented Hp infection (by CLO-test and histology) and either with endoscopically proven peptic ulcer (n=52) or dyspepsia (n=33) were randomized to receive LAN 30 mg bid plus Clarithromycin (CL) 500 mg bid plus Amoxycillin (AMO) 1 gr bid for one week (Group A, n=43), or RBC 400 mg bid plus CL 500 mg bid plus AMP 1 gr bid for one week (Group B, n=42). Hp eradication was assessed 4 weeks and 6 months after the completion of treatment (by CLO-test and histology). A CL sensitivity test was carried out in the cultured pre treatment (57/85, 67.1%) and post treatment (9/12, 75%) Hp strains. **Results:** Two (3.5%) Hp strains exhibited primary and only one secondary CL resistance. The eradication rates according to Intention To Treat (ITT) and Per Protocol (PP) analyses are summarized in the table. All side effects in both groups were mild and no patient discontinued treatment due to adverse events. **Conclusion:** Short-term therapies based on LAN or RBC proved equally effective during a 6 month follow-up. A trend towards a decreasing efficacy at 6 months was only observed in RBC based treatment.

Hp eradication	ITT (1 mo)	PP (1 mo)	ITT (6 mo)	PP (6 mo)
Group A	35/43 (81.4%)	35/39 (89.7%)	31/43 (72.1%)	31/35 (88.6%)
Group B	34/42 (81.0%)	34/40 (85.0%)	27/42 (64.3%)	27/35 (77.1%)

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● G1290

EFFECTIVENESS OF ANTI-SECRETORY THERAPY IN PREVENTING GASTROESOPHAGEAL REFLUX DISEASE (GERD) AFTER SUCCESSFUL TREATMENT OF *HELICOBACTER PYLORI* INFECTION.

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There is increasing evidence that *Helicobacter pylori (H. pylori)* eradication might predispose to gastroesophageal reflux disease(GERD). However, no studies exist examining the usefulness of acid suppression in preventing GERD after *H. pylori* eradication. The aim of this prospective study was to examine the effectiveness of anti-secretory treatment, after successful *H. pylori* eradication, in preventing GERD. **Methods:** Eighty initially *H.pylori*(+) patients, without GERD at the time of *H.pylori* eradication [50 peptic ulcer (PU) and 30 non-ulcer (NU), 55M, 25F, median age 38 yrs, range 19–57] after successful *H.pylori* eradication were randomized to recieve either Omeprazole 20mg daily (group A)for one year or no treatment (group B). All patients underwent upper GI endoscopy at 6 and 12 months or when GERD symptoms occured. **Results:** There were 40 patients in group A and 40 patients in group B. There were no statistically significant differences between the two groups as far as sex, age, body weight and other demographic data were concerned. During follow up 7 patients from group A and 5 patients from group B were lost and therefore there were 33 and 35 patients respectively in groups A and B who completed the protocol. One out of 33 patients in group A(3%) and 10/35 (28.5%) in group B developed GERD symptoms during follow-up(p=0.0065). The respective values for esophagitis were 0/33(0%) and 6/35(17.1%) (p=0.0249, Fisher's exact test). **Conclusion:** *H. pylori*(−) patients after successful eradication and without anti-secretory treatment develop GERD in a significantly higher proportion than patients who receive anti-secretory treatment. This and other relative evidence stress the need for critical evaluation of the indications for elimination of this organism.

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● G1384

HLA-DR AND EXPRESSION OF COSTIMULATORY MOLECULES (CM) (B7–1,B7–2,ICAM-1) ON GASTRIC EPITHELIAL CELLS IN H PYLORI(HP) GASTRITIS.

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There is evidence,that HP infection upregulates HLA-II molecules on gastric epithelial cells(GEC). In this study we prospectively evaluated whether GEC are capable of "complete" antigen presentation through additional expression of CM in HP gastritis.Patients-Methods. Thirty symptomatic patients were studied. Upper GI endosopy was performed and at least 4 biopsies were obtained (antral:2, body:2) for CLO-test and histology (Sydney classification); 23 patients were HP(+) and 7 HP(-)controls- by both methods. HP eradication therapy was given to HP(+) patients and all patients were endoscoped 116 ± 9d after. Formalin-fixed parafin embedded tissue sections were stained by the ABC immunoalkaline phosphatase method. Mouse monoclonal anti HLA-DR (a chain,DAKO), goat polyclonal anti B7–1, anti B7–2(Santa-Cruz), mouse monoclonal anti-ICAM-1(Zymed) were used. Antigen unmasking was performed by heat treatment in microwave oven. Results. 20/23patients eradicated HP.In HP gastritis HLA-DR was expessed and correlated with disease activity(Spearman,p=0.002). No HLA-DR expression was observed in controls.In HP eradicated patients significant inhibition of HLA-DR was found(Wilcoxon,p=0.0001). ICAM-1, expressed on GEC in 85% of HP(+),did not correlate with gastritis parameters and subsided after eradication.B7–1, B7–2 expressed in controls and HP(+)patients with a predominance of B7–1;no significant inhibition observed after eradication. Conclusions-.GEC acquires APCs properties in HP infection through the de novo expression of HLA-DR and CM.Thus phenomenon seems to attenuate after eradication and resolution of mucosal inflammation.

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● G3337

SHORT-TERM REMISSION OF CROHN'S DISEASE AFTER TREATMENT OF *H. PYLORI* INFECTION.

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Aim: To compare anti-*Hp* treatment and prednisolone in the induction of remission of Crohn's disease (CD). **Methods:** 70 consecutive patients with a first mild-to-moderate attack of ileal or ileocolonic CD were endoscoped. Biopsies were taken from the gastric antrum, body and fundus to assess blindly for *Hp* infection, *Hp* gastritis and focally enhanced gastritis (focal periglandular CD3+, CD45RO−, CD68+ inflammatory infiltrates). Patients found to be *Hp*+ by CLO-test and immunohistochemistry received OAC14 (omeprazole 20mg bd, amoxycillin 1g bd, clarithromycin 0.5g bd for 14 days) and were re-endoscoped 8–10 weeks later. *Hp*- patients received prednisolone (0.75mg/kg tapering 5mg/week). The CDAI was calculated before and 8–10 weeks after treatment onset. **Results:** 30 patients were *Hp*+ (43%, group A) and 40 *Hp*- (group B). No significant differences were found in any demographic, clinical, laboratory, endoscopic (erythema, erosions, aphthae) or histologic (granulomas, microgranulomas, aphthoid ulcers, focal gastritis) data between groups A and B. No peptic ulcer disease was found. Focal gastritis was detected in 27 *Hp*- and 19 *Hp*+ patients before treatment and was evenly distributed in the antrum and body/fundus. *Hp*+ patients had in addition typical features of *Hp* gastritis. After treatment clinical remission was achieved in all patients. The CDAI fell from 310(260–345)(mean,range) to 133(120–145) in group A and from 312(255–345) to 125(120–138) in B (p<0.0001). OAC14 eradicated *Hp* in 28(93%) patients; this resulted in healing of aphthae in 4/5 and erosions in 11/12 patients, remission of *Hp* gastritis and identification of focal gastritis/microgranulomas in 4 more patients. Thus, OAC14 achieved at least short-term clinical remission of CD comparable to prednisolone and by healing Hp gastritis revealed previously undetected features of CD gastritis.

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G3338

THE VALUE OF GASTRIC BIOPSIES IN THE DIAGNOSIS OF CROHN'S DISEASE

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The aim of this prospective study was to assess the value of gastric biopsies in the diagnosis of Crohn's disease (CD). Gastroscopy was performed before any treatment in consecutive patients with irritable bowel syndrome (IBS, 80), newly diagnosed ulcerative colitis (UC, 35), a first attack of ileocolonic CD (70), microscopic colitis (MC, 25) and celiac disease (25). Biopsies taken from the duodenum and the gastric antrum, body and fundus were examined blindly for the presence of *H. pylori*, *Hp* gastritis (Houston Update System) and features thought to be characteristic of CD gastritis [aphthoid ulcers, granulomas, microgranulomas and focally enhanced gastritis (CD3+/CD45RO+/CD68+ inflammatory infiltrates)]. *Hp* was found in 30(44%) patients with CD, 17(48%) with UC, 43(53%) with IBS, 12(48%) coeliacs and 13(52%) with MC (p>0.05). Peptic ulcer disease was not found in any patient. Erythema was the only endoscopic finding in the stomach of non-CD patients. Erosions or aphthae were found in 13(22%) and erythema in 25(36%) CD patients. Granulomas were found in 18(10*Hp*+), microgranulomas in 29(16*Hp*+) and focal gastritis in 46 (66%), 19*Hp*+/27Hp-) of 70 CD patients. Granulomas were predominantly distributed in the antrum whereas focal gastritis was evenly distributed in the antrum, body and fundus. None of the non-CD patients had evidence of granulomas or focal gastritis; in contrast, CD3+/CD45+/CD68+ cells, if present, were diffusely distributed in the stomach. *Hp*+ patients showed features of *Hp* gastritis but its severity and grade were similar between patient groups. The positive and negative predictive value of focal gastritis for the diagnosis of CD was 100% and 87.3% respectively. Thus, focal gastritis is a reliable indicator of CD, which may be of particular value in the differential diagnosis of intermittent diarrhea or indeterminate colitis.

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G3626

DOES H.PYLORI INFLUENCE THE COURSE OF ULCERATIVE COLITIS?

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H Pylori (HP) induces the immune response of the host and has been implicated to various extragastric diseases. The aim of this study was to investigate the influence of HP on the clinical course of Ulcerative Colitis (UC). Therefore, all patients (pts) with UC followed at the IBD clinic from September 1994 to November 1998, with adequate follow-up and inactive disease for at least one month before entry, were included. IgG antibodies to HP were detected at the historical sera, whereas patients who had taken eradication therapy, were excluded. The observed (O) relapses were classified according to their yearly occurrence during the follow-up period and were compared to the expected (E) ones, that were estimated by multipling the whole number of O relapses by the weighted value (WV. WV=No of pts at risk of relapse yearly ÷ No of pts/years of the follow-up period). Fifty nine consecutive pts (37men - 22women, mean age 50--19y), followed-up for a mean of 34 (range 12–51) months, accounting for 161 persons/year, were included during the study period. Twenty pts (33.9%) were found to be HP(+). HP(+) and HP(-) pts were almost identical as far as the duration of the disease is concerned and the duration of follow-up. Ninety three relapses of UC were observed with a mean number of 2 (range 0–5) per patient (O/E: 53/34 the 1st year, 22/31 the 2nd year, 11/18 the 3rd year and 7/10 the 4th year). Fourteen patients (23.7%) remained in remission during the whole study period and did not differ according to the HP status (p=ns). Seventeen pts had been followed up for 48 months consecutively and no significant difference of relapses was observed in concern to HP status (p<0.5). The observed relapses of the HP(+) pts did not differ from the expected ones (p<0.36). Therefore we concluded that HP did not influence the clinical course of ulcerative colitis.

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G4465

HELICOBACTER PYLORI (HP) INFECTION DOES NOT AFFECT GASTRIC EMPTYING IN PATIENTS WITH FUNCTIONAL DYSPEPSIA (FD).

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The purpose of this study was to determine whether gastric emptying in patients with FD is affected by Hp infection and its eradication. Gastric emptying of a standard semisolid meal was evaluated by the acetaminophen absorption technique (Heading et al. Br J Pharmacol 1973; 47: 415–421). Biopsy urease test and serology (Hp IgG ELISA) assessed Hp status. The study consisted of two parts. First was a comparison of gastric emptying in Hp-positive and Hp-negative patients with FD and asymptomatic controls. The second was a therapeutic trial investigating the effect of Hp eradication on gastric emptying in Hp-positive patients with FD four weeks after treatment. 33 patients with FD (24 Hp-positive and 9 Hp-negative) and 17 controls where evaluated. Patients with FD had significantly prolonged gastric emptying compared with controls: mean maximum plasma acetaminophen concentration divided by body weight (C_{max}/BW) was 0.173 and 0.224 μg/ml-kg respectively (p=0.02). The area under the plasma acetaminophen concentration-time curve divided by body weight (AUC/BW) was 18.4 and 24.4 μg·min/ml-kg respectively (p=0.01). Gastric emptying did not differ significantly between Hp-positive and Hp-negative patients with FD. Mean C_{max}/BW was 0.176 and 0.167 μg/ml-kg respectively (p=0.9). Mean AUC/BW was 18.8 and 17.4 μg·min/ml-kg respectively (p=0.5). Hp eradication in 14 Hp-positive patients with FD did not affect gastric emptying significantly. Mean C_{max}/BW was 0.171 and 0.160 μg/ml-kg (p=0.6); mean AUC/BW was 17.4 and 18 μg·min/ml-kg (p=0.9) before and after Hp eradication respectively. In conclusion, although gastric emptying is prolonged in patients with FD, neither Hp infection nor its eradication affect gastric empeying.

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10/03 **RANDOMIZED STUDY COMPAIRING IF PROLONGING OMEPRAZOLE (O) TREATMENT AFTER HP ERADICATION INCREASES DUODENAL ULCER (DU) HEALING RATE**

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The need for prolongation of antisecretory treatment after HP eradication treatment is disputable. The aims of our study were: 1) to evaluate if prolongation of antisecretory treatment could influence healing and 2) to estimate the influence of comorbid diseases on ulcer healing.

Patients and Methods: 280 patients with DU and HP after finishing a 10 days eradication regimen with O 20 mg bid, clarithromycin 500 mg bid, amoxycilline 1 g bid, were randomly assigned to continue O 20 mg qd for 7 (Group I--87 patients), 21 (Group II--92 patients) or 35 days (Group III--101 patients) respectively. Groups were comparable for sex, age and smoking. Endoscopy was performed to access DU healing 4–6 weeks after treatment completion. HP eradication was verified by histology, CLO-test and ^{13}C-urea breath test. Complete clinical and laboratory evaluation to access comorbid diseases, was performed. For unhealed ulcers serum gastrin was evaluated, before starting O 20 mg qd. Analysis was performed only to those who accepted endoscopy.

Stat: X^2-test, t-test, univariate ($p < 0.10$) and multivariate analysis ($p < 0.05$).

Results: 262 patients had a follow up endoscopy (mean age 52.7±14.7 years, 150 males, 90 smokers). Eradication rates were: Group I: 85.4% (CI 77.6–93.2), Group II: 86% (CI 78.6–93.5), Group III: 86.2% (CI 79.1–93.3). $p > 0.1$. Healing rates after successful eradication were: Group I: 92.9% (CI 73.8–91.9), Group II: 84.3% (CI 78.5–94.5), Group III: 92.6% (CI 86.8–98.3). $p > 0.1$. Healing rates for those not eradicated: Group I: 16.7% (CI 8.1–41.4), Group II: 80% (CI 49.8–100), Group III: 61.5% (CI 30.9–92.1). $p < 0.01$. One patient presented gastrinoma. In all other patients ulcers healed within 3 months. Age, female sex, chronic renal failure and NSAID's use were related in univariate analysis with failure in ulcer healing after successful eradication. Only chronic renal failure (n = 14, healing rate 28.6%, $p < 0.001$) and NSAID's use (n = 46, healing rate 37%, $p < 0.001$) were significant in multivariate analysis. When patients with chronic renal failure, NSAID's use and high serum gastrin were excluded, healing rates after successful eradication increased to 100% for all three groups.

Conclusions: 1) Ulcer healing after successful HP eradication is high. 2) Prolongation of antisecretory treatment for more than a week after finishing successful HP eradication, does not increase ulcer healing rate. 3) Chronic renal failure and NSAID's use are associated with ulcer healing impairment. For those patients protongation of antisecretory treatment needs farther evaluation.

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10/05 **HERPES SIMPLEX VIRUS TYPE 1 (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-Methods: 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 endoscopically 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 the 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.

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11/06 CLINICAL SPECTRUM OF GASTROESOPHAGEAL REFLUX DISEASE (GERD) AFTER HP ERADICATION IN PATIENTS WITH GASTRODUODENAL ULCER (GDU)

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Esophagitis is reported to be more frequent after successful HP eradication in patients with gastroduodenal ulcer (GDU). The aim of the study was to investigate prospectively the incidence and severity of gastroesophageal reflux (GERD) and esophagitis (E) in patients with prior GDU 3 months after treatment completion.

Patients and Methods: 280 patients with prior GDU were contacted 3 months after HP eradication treatment was accomplished. 262 (mean age 52.7±14.7 years, 150 male, 90 smokers) accepted to give information and undergo an upper GI endoscopy. HP eradication was verified by histology, CLO-test and ^{13}C-urea breath test. 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, multivariate analysis

Results: 37 patients were positive for HP (10 with GERD - 2E). Analysis was performed only for successful HP eradication. Results are resumed in the following table.

GERD	PreRx	PostRx*	E	PreRx	PostRx**
0	132	116	0	202	184
1	32	43	1	19	32
2	25	32	2	3	8
3	36	34	3, 4	1	3

*p = 0.33, **p = 0.006, PreRx & PostRx: Pre and post eradication period

From those who had GERD before (n = 93), 10 had no symptoms, 22 had less severe GERD, 42 had the same symptoms and 19 presented an aggravation, while from those who were free of symptoms (n = 132), 28 reported GERD after treatment. From those with prior E (n = 23), 15 had no E, 3 was better, 5 without any difference and 7 had aggravated. From those with no E (n = 202), 28 presented E after treatment. Age, smoking, alcohol consumption and hiatal hernia were not predictive factors either for GERD or for E. There was a strong correlation between GERD and E after HP eradication (p < 0.01).

Conclusions: 1) DU patients develop more frequently E, but not GERD three months after eradication treatment. 2) Pre-treatment GERD is aggravated in 20% of patients and first appearing GERD is reported in another 21% but symptoms of 34% of patients are improved. 3) Pre-treatment E is aggravated in 30% of patients and first appeared E is presented in 14%. Nevertheless E is of a rather mild form.

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12/14 TREATMENT OF *H. PYLORI* INFECTION MAY INDUCE REMISSION OF CROHN'S DISEASE COMPARABLE TO PREDNISOLONE

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Objectives: To compare anti-*Hp* treatment and prednisolone in the induction of remission of Crohn's disease (CD).

Methods: 75 consecutive patients with a first mild-to-moderate attack of ileal or ileocolonic CD were endoscoped. Biopsies were taken from the gastric antrum, body and fundus to assess blindly for *Hp* infection, *Hp* gastritis and focally enhanced gastritis (focal periglandular CD3+, CD45RO+, CD68+ inflammatory infiltrates). *Hp*+ patients by CLO-test, histology and immunohistochemistry received OAC$_{14}$ (omeprazole 20 mg *bd*, amoxycillin 1 g *bd*, clarithromycin 0.5 g *bd* for 14 days) and were re-endoscoped 8–10 weeks later. *Hp*– patients received prednisolone (0.75 mg/kg tapering 5 mg/week). The CDAI was calculated before and 8–10 weeks after treatment onset.

Results: 33 patients were *Hp*+ (44%, group A) and 42 were *Hp*– (56%, group B). No significant differences were found in any demographic, clinical, laboratory, endoscopic (erythema, erosions, aphthae) or histologic (granulomas, microgranulomas, aphthoid ulcers, focal gastritis) data between groups A and B. No peptic ulcer disease was found. Focal gastritis was detected in 29 *Hp*– and 22 *Hp*+ patients before treatment and was evenly distributed in the antrum and body/fundus. *Hp*– patients had in addition typical features of *Hp* gastritis. After treatment clinical remission was achieved in all patients. The CDAI fell from 312 (261–347) (mean, range) to 132 (112–144) in group A and from 315 (250–346) to 125 (119–136) in group B ($p < 0.0001$); differences in CDAIs between groups A and B were not significant. OAC$_{14}$ eradicated *Hp* in 30/91% patients; this resulted in healing of aphthae in 5/8 and erosions in 12/13 patients, remission of *Hp* gastritis and identification of focal gastritis/microgranulomas in 6 more patients.

Discussion: OAC$_{14}$ resulted in clinical remission of CD comparable to prednisolone and by healing *Hp* gastritis revealed previously undetected features of CD gastritis.

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11/07 EFFECTIVENESS OF ANTI-SECRETORY THERAPY IN PREVENTING GASTROESOPHAGEAL REFLUX DISEASE (GERD) AFTER SUCCESSFUL TREATMENT OF HELICOBACTER PYLORI INFECTION

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There is increasing evidence that Helicobacter pylori (H pylori) eradication might predispose to gastroesophageal reflux disease (GERD). However, no studies exist examining the usefulness of acid suppression in preventing GERD after H. pylori eradication.

Objective: The aim of this prospective study was to examine the effectiveness of anti-secretory treatment, after successful H. pylori eradication, in preventing GERD.

Methods: Eighty initially H. pylori(+) patients, without GERD at the time of H. pylori eradication (50 peptic ulcer (PU) and 30 non-ulcer (NU), 55 M, 25 F, median age 38 yrs, range: 19–57) after successful H. pylori eradication were randomized to receive either Omeprazole 20 mg daily (group A) for one year or no treatment (group B). All patients underwent upper GI endoscopy at 6 and 12 months or when GERD symptoms occured.

Results: There were 40 pts in group A and 40 pts in group B. There were no statistically significant differences between the two groups as far as sex, age, body weight and other demographic data were concerned. During follow up 7 pts from group A and 5 pts from group B were lost and therefore there were 33 and 35 pts respectively in groups A and B who completed the protocol. One out of 33 pts in group A (3%) and 10/35 (28.5%) in group B developed GERD symptoms during follow up (p = 0.0065). The respective values for esophagitis were 0/33 (0%) and 6/35 (17.1%) (p = 0.0249, Fisher's exact test).

Discussion: H. pylori(+) pts after successful eradication and without anti-secretory treatment develop GERD in a significantly higher proportion than patients who receive anti-secretory treatment. This and other relative evidence, stress the need for critical evaluation of the indications for elimination of this organism.

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14/49 RANDOMIZED COMPARISON OF OMEPRAZOLE (O) AND RANITIDINE (R) WHEN USED FOR H. PYLORI (HP) ERADICATION IN "QUAD" TREATMENT (SECOND LINE) OF PATIENTS WITH EROSIVE DUODENITIS (ED) OR DUODENAL ULCER (DU)

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Few data are available on the efficacy of eradication regimens in patients with ED. The aim of our study was to compare the efficacy of either O or R combined in a "quad" regimen including tripotassium dicitratobismuthate (B), metronidazole (M) and tetracycline hydrochloride (T) as a second choice treatment for HP eradication in patients with ED or DU, whose initial treatment had failed.

Patients and Methods: 119 patients with DU (mean age: 48±13.7 years, 64 men, 56 smokers) and 37 with ED (mean age: 47.8±13.2 years, 24 men, 12 smokers), who have failed to eradicate HP with double (ED = 22, DU = 58) or triple regimens (ED = 15, DU = 61), none of which contained M, were randomly assigned to receive B 600 mg bd + M 500 mg tid + T 500 mg tid combined with either O 20 mg bid (Group Ia: 19 ED, Group Ib: 59 DU patients) or R 300 mg bid (Group IIa: 18 ED, Group IIb: 60 DU patients) for 14 days. Groups were comparable for sex, age and smoking. Endoscopy was performed 4–6 weeks after treatment completion. HP eradication was verified by histology, CLO-test and ^{13}C-urea breath test (UBT).

Stat: t-test, X2-test

Results: Endoscopy was performed in 27 ED and 80 DU patients. 6 ED and 30 DU patients performed only UBT. Eradication rates are: A) Intention to treat (n = 156): Group Ia: 68.4% (45.4–91.4)-Group IIa: 94.4% (82.7–100) [p = 0.06], Group Ib: 79.7% (69.1–90.2)-Group IIb: 70% (58.1–81.9) [p = 0.22]. Globally ED: 81.1% (67.8–94.3) - DU: 74.8% (66.9–82.7) [p = 0.43]. B) Per protocol analysis (n = 107): Group Ia: 83.3 (58.6–100)-Group IIa: 100 [p = 0.10], Group Ib: 85.4 (75.8–95.1)-Group IIb: 73.7 (59–88.4) [p = 0.14], Globally ED: 96.3 (88.7–100)-DU: 83.1 (74.9–91.4) [p = 0.08]. Age, sex, smoking and alcohol consumption did not influence eradication rate. Mild side-effects (nausea, metallic taste) were frequent. 11 patients did not complete therapy (1 pseudomembranous colitis-2 bismuth toxicity- 3 allergy- 2 oral candidiasis-2 nausea-1 diarrhea). 13 patients were lost in follow up.

Conclusions: R 300 mg bid and O 20 mg bid are equally effective when used as antisecretory agents combined with B, M, T in ED and DU patients who had failed to eradicate HP. Nevertheless there is a trend (p = 0.06) in intention to treat analysis in favor of R, only for ED and not for DU patients.

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14/57 LANSOPRAZOLE (LAN) VS RANITIDINE BISMUTH CITRATE (RBC) BASED SHORT-TERM TRIPLE THERAPIES FOR *HELICOBACTER PYLORI* (*H. PYLORI*) ERADICATION: A RANDOMISED STUDY WITH 6-MONTH FOLLOW-UP

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Objective: We have compared the eradication rates and the safety profiles of two short-term therapies containing LAN or RBC.

Methods: Eighty-five *H. pylori* positive patients (CLO-test and histology) (median age 46, range 17–82) with peptic ulcer (n = 52) or non ulcer dyspepsia (n = 33) were randomized to receive LAN 30 mg bid, Clarithromycin (CL) 500 mg bid and Amoxycillin (AMO) 1 gr bid for one week (Group A, n = 43), or RBC 400 mg bid, CL 500 mg bid and AMP 1 gr bid for one week (Group B, n = 42). *H. pylori* eradication was assessed (by CLO-test and histology) at 4 weeks and 6 months post treatment. Clarithromycin sensitivity tests are carried out in the cultured pre treatment (57/85, 67.1%) and post treatment (9/12, 75%) *H. pylori* strains.

Results: The regimen failed to eradicate two (3.5%) *H. pylori* strains (one in each treatment arm), which exhibited primary CL resistance. Secondary CL resistance was recorded only in one out of the six (16.7%) treatment failures with the RBC based combination. Patients in both groups were assessed at 1 and 6 months post treatment. The eradication rates according to intention-to-treat (ITT) and per protocol (PP) analyses are summarized in the following table:

Hp erad.	ITT (1 mo)	PP (1 mo)	ITT (6 mo)	PP (6 mo)
Group A	35/43 (81.4%)	35/39 (89.7%)	31/43 (72.1%)	31/35 (88.6%)
Group B	34/42 (81.0%)	34/40 (85.0%)	27/42 (64.3%)	27/35 (77.1%)

Side effects in both groups were mild and no patient discontinued treatment due to adverse events. There was no difference in *H. pylori* eradication rate between the two groups at 1 and 6 months post treatment.

Discussion: We conclude that short-term therapies based on LAN or RBC are equally effective to eradicate *Helicobacter pylori*.

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73.07 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 dyspepsic 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). PCNAindex 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.

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15/28 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.

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83.08 SPECIALISED INTESTINAL METAPLASIA AT THE OESOPHAGO-GASTRIC JUNCTION: PREVALENCE AND CORRELATION WITH CLINICAL, ENDOSCOPIC AND HISTOLOGICAL FINDINGS

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Background: Specialised intestinal metaplasia (SIM) around the oesophagogastric junction (OGJ) is considered to be premalignant.

Aim: To examine prospectively the prevalence of SIM at the OGJ and to correlate its presence with clinical, endoscopic and histological findings.

Methods: 101 patients (40 women, 61 men; mean age 55 yr, range 20–79 yr) with symptoms of the upper gastro-intestinal tract underwent endoscopy and note was made for endoscopic evidence of short segment Barrett's oesophagus (SSBO). Biopsies were taken from gastric type mucosa just distal to the OGJ. They were stained with hematoxylin/eosin and alcian blue/periodic acid – Schiff for the detection of SIM and inflammation of the cardiac mucosa (carditis) and with Warthin-Starry for H. pylori presence.

Results: SIM around the OGJ was detected in 27 patients (26.7%). Multiple logistic regression analysis revealed that metaplasia was associated significantly with age (odds ratio [OR], 2.8; 95% confidence interval [CI], 1.2–6.6), endoscopic suspicion of SSBO (OR, 3.6; 95% CI, 2.2–6.9), detection of H. pylori (OR, 2.8; 95% CI, 1.1–7) and presence of carditis (OR, 6.4; 95% CI, 2.8–16.8). Patients with SIM and endoscopic picture of SSBO were more likely to have symptoms of gastro-oesophageal reflux disease (p = 0.03) and endoscopic oesophagitis (p = 0.01).

Conclusions: This study confirms that the prevalence of SIM around the OGJ is high in symptomatic patients. Age, endoscopic evidence of SSBO and histological presence of H. pylori and carditis are independent risk factors associated with SIM.

P0135 PREVALENCE OF *HELICOBACTER PYLORI* INFECTION AND ENDOSCOPIC FINDINGS IN PATIENTS WITH DIABETES MELITUS

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Background: A high prevalence of dyspeptic symptoms is described in diabetic patients. It has been suggested that Helicobacter pylori (Hp) infection may significantly contribute to gastric and duodenal disorders in such patients.

Aim: To evaluate the prevalence of Hp infection in a group of diabetic patients with dyspepsia vs a control group and to associate it with endoscopic findings.

Methods: 37 diabetic patients (M 23, F 14) and 53 non-diabetic patients (M 34, F 19) all with important dyspeptic symptoms were enrolled. Gender (M 62% vs 64%) and mean age (65.2 vs 62.6 years) did not differ significantly between the two groups of patients (diabetic vs non-diabetic). In all patients upper GI endoscopy was performed and endoscopic biopsies were examined by the urease rapid test.

Results: Hp infection was detected in 17 diabetic patients (45.9%) and 23 patients in the control group (43.3%), the prevalence in the two groups being not significant (0.8). GI endoscopy documented that the prevalence of gastritis was similar in both groups (65% vs 62.3% p n.s.); peptic ulcer prevalence was 16.2% in diabetic patients and 17% in control group (p n.s.). When examining only the patients with Hp infection gastritis prevalence did not differ significant between the two groups (53% in diabetic patients vs 61% in control; p n.s.). Peptic ulcer prevalence was higher in the control group (34.8%) than in diabetics (23.6%) with Hp infection, but the difference was not significant (χ^2 0.6 p 0.4) statistically.

Conclusions: Our preliminary data, that showed no difference in the prevalence of Hp infection between diabetics and control group, do not support an association between H. pylori infection and diabetes melitus. The endoscopic findings in patients with Hp infection did not differ between diabetic and control group.

P0531 DIABETES MELLITUS AND ERADICATION OF *H. PYLORI* INFECTION

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Introduction: Diabetes Mellitus may increase general susceptibility to acute and chronic infections which is likely to be multifactorial. The general effects of hyperglycaemia are probably important, and there may be additional immune disturbances especially in IDDM. H. Pylori (H.P.), the main aetiologic factor for gastritis and peptic ulcer, is the most common gastric infection worldwide.

Aim: The aim of this study was to compare the effectiveness of a combination therapy containing Lansoprazole, Amoxicillin and Clarithromycin for one week in the eradication of H.P. in pts with IDDM and in dyspeptic pts.

Material-Methods: A group of 20 pts with IDDM, (12 M, 8 F) aged 28–45 yrs, affected by H.P. infection and a group of 20 dyspeptic pts (10 M, 10 F), aged 30–50 yrs, were inluded in our study. Documentation of H.P. infection in studied pts was made by rapid urease test and histology. All the pts of two groups were treated with Lansoprazole 30 mgr bid, Amoxicillin 1 gr bid and Clarithomycin 500 mgr bid for one week. Patients were assessed for eradication, defined as absence of bacteria in rapid urease test and histology, at least 4 weeks after completion of treatment.

Results: Eradication rate in pts with IDDM compared to dyspeptic pts was significantly lower (19/20 pts, 95% in dyspeptic pts group vs 14/20 pts, 70% in IDDM pts group, $p < 0.002$). Significant clinical improvement of symptoms was observed in those pts of both groups where eradication therapy was successful. Two patients of each group (10%) expierenced side-effects. All adverse events were mild and no patient discontinued treatment.

Conclusion: Eradication rate of H. Pylori seems to be significantly lower in IDDM patients in comparison to that observed in dyspeptic patients.

P0532 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 breath 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.

P0544 PEPTIC ULCER DISEASE IN CIRRHOSIS. 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 [12 M, 2 F, mean age 61.6 y (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 (6 p) epigastric pain (4 p) or bleeding (4 p) 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.

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P0578 **A PROSPECTIVE STUDY COMPARING VARIOUS ANTI-*H. PYLORI* TREATMENT REGIMENS FOR PATIENTS WITH PEPTIC ULCER DISEASE (PUD)**

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Objectives: Prospective, randomized, single center study to compare various anti-*Hp* regimens.

Methods: Consecutive patients with *Hp* related peptic ulcer disease are randomized to one of the following anti-*Hp* regimen. Patients are re-endoscoped 8 weeks after completion of therapy. The diagnosis and successful eradicaton of *Hp* were assessed by CLO-test, histology (mod. Giemsa, H&E) and immunohistochemistry (rabbit anti-*Hp* monoclonal antibody, DAKO) in biopsy specimens taken from the gastric antrum and body/fundus. Treatment related side effects and compliance with treatment were recorded.

Results are given in the table:

Treatment Regimen	n	Age (yr) (range)	Treatment-related side effects	Treatment-related compliance	Eradication rate ITT n (%)	Eradication rate PP n (%)
OCM_7	43	46 (16–73)	7 (22%)	41 (95%)	21 (49)	21/29 (72)
OAC	86	42 (15–78)	8 (10%)	86 (100%)	64 (74)	64/77 (83)
OAC_{10}	78	40 (16–69)	7 (10%)	77 (99%)	61 (78)	61/69 (88)
OAC_{14}	55	41 (17–68)	6 (11%)	54 (93%)	45 (82)	45/49 (92)
OAM_{14}	70	44 (20–76)	8 (15%)	68 (97%)	45 (64)	45/50 (90)
$OBMT_{10}$	71	40 (17–70)	9 (13%)	64 (90%)	50 (78)	50/61 (82)

O, omeprazole; C, clarithromycin; A, amoxycillin; M, metronidazole; B, bismuth; T, tetracyclin. OAM: O, 20 mg *bd;* A 1 g *bd;* M, 0.5 g *tid.* OCM: O, 20 mg *bd;* C, 0.25 g *bd;* M, 0.5 g *bd.* OAC: O, 20 mg *bd;* A, 1 g *bd;* C, 0.5 g *bd.* OBMT: Omeprazole (20 mg *bd*)-classical triple therapy. PP analysis: OAC_{14} *vs* OCM_7, p = 0.03; OAM_{14} *vs* OCM_7, p = 0.06; OAK_{10} *vs* OCM_7, p = 0.07.

All other comparisons were not significant although a type II error cannot be excluded. *C. difficile* colitis develop in a patient receiving OAC_{10}; no other serious side-effects of treatment were seen. OBMT was the less well tolerated regimen and did not meet our expectations for the highest eradication rate. The prevalence of M- and C-resistant strains in our country is reported 56% and 5%, respectively.

Conclusion: In communities with a high prevalence of M-resistant strains OCM_7 should probably not be given. The cost effectiveness of $OAC_{-7, -10 \text{ or } -14}$ should be assessed in a large-scale study. Differences in ITT and PP reflect the "real-life" of a single center.

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P1026 **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.