ΠΡΟΣΚΕΚΛΗΜΕΝΕΣ ΞΕΝΟΓΛΩΣΣΕΣ ΑΝΑΚΟΙΝΩΣΕΙΣ ΕΛΛΗΝΩΝ ΕΡΕΥΝΗΤΩΝ

● HIGH RESOLUTION MAGNIFICATION ENDOSCOPY CAN RELIABLY IDENTIFY THE NORMAL GASTRIC MUCOSA, *HELICOBACTER PYLORI* INFECTED STOMACH AND GASTRIC ATROPHY

G.K. Anagnostopoulos[1], K. Yao[2], P. Kaye[3], P. Fortun[2], E. Fogden[2], A. Shonde[2], S. Folley[2], S. Sunil[2], J. Atherton[2], C. Hawkey[2], K. Ragunath[2]
[1]Endoscopy, Mitera General Hospital, Athens, Greece, [2]Endoscopy, [3]Histopathology, Wolfson Digestive Diseases Centre, Nottingham, UK

Introduction: Endoscopic visualisation of *H. pylori* infection and gastric atrophy is not always feasible with conventional endoscopy. The history of magnifying endoscopy for observing the surface microstructure of the GI mucosa has its roots in Japanese studies. However, the Japanese findings have not been validated in the Western world. The aim of our study was to describe the magnified endoscopic findings in human stomach, to correlate them with *H. pylori* gastritis and gastric atrophy, and to evaluate the inter-/intra-observer agreement in the assessment of the magnified endoscopic patterns seen.

Aims & Methods: Consecutive patients who underwent upper GI endoscopy for dyspeptic symptoms were included in the study. Endoscopies were performed using a high resolution magnifying endoscope (GIF Q240-Z, Olympus, X115 magnification). The endoscopists classified the magnified endoscopic findings in gastric body into the following 4 patterns: (1) Honeycomb-type subepithelial capillary network (SECN) with regular arrangement of collecting venules (CV) and regular round pits; (2)a honeycomb type SECN with regular round pits, with or without sulci but loss of CVs, (3) loss of normal SECN/CVs with white enlarged pits surrounded by erythema, and (4) loss of normal SECN and round pits with irregular arrangement of CVs. 4 biopsies were obtained for histological analysis and 2 for CLOtest. Targeted biopsies were taken from areas with type 4 pattern. All endoscopies were digitally recorded and still images were captured. 5 endoscopists were invited to assess 200 pictures for the magnified endoscopic types. The assessment was repeated after 1 week, when the same images but in a different order were shown to the endoscopists.

Results: 95 consecutive patients (52 male, mean age 58.6 years) were enrolled in the study. *H. pylori* infection was demonstrated in 26 patients and gastric atrophy was found histologically in 18 patients. The sensitivity, specificity, positive and negative predictive value of: (a) type 1 for predicting: the normal gastric mucosa was 92.7%, 100%, 100%, 83.8%, (b) types 2-3 for predicting the *H. pylori*-infected stomach was 100%, 92.7%, 83.8%, 100% and (c) type 4 for predicting gastric atrophy was 90%, 96%, 85.7%, and 97.3% respectively. The kappa values for inter- and intra-observer agreement in predicting normal gastric mucosa, *Helicobacter pylori* gastritis and gastric atrophy were 0.864, and 0.913 respectively.

Conclusion: High resolution magnification endoscopy can reliably identify the normal gastric mucosa, *H. pylori*-associated gastritis and gastric atrophy in a Western population.

● **INCIDENCE OF ACUTE NONMALIGNANT GASTROINTESTINAL BLEEDING IN GREEK PATIENTS AND ITS ASSOCIATED FACTORS**

N.E. Tsesmeli, P. Kotsaftis, C.G. Savopoulos, A.I. Hatzitolios, G.D. Kaiafa, M. Apostolopoulou, D.T.H. Karamitsos
1st Medical Propedeutic Dept, AHEPA Hospital, Aristotle University of Thessaloniki, Thessaloniki, Greece

Introduction: Natural history of acute nonmalignant gastrointestinal bleeding (GIB) has been influenced by the increasing population age, wide use of non-steroidal anti-inflammatory drugs (NSAID) or antiplatelet agents (low-dose aspirin) and *Helicobacter pylori* (*Hp*) seroprevalence.

Aims & Methods: The aim was to quantify the incidence of acute GIB in Greek patients.It is a retrospective data collection from 151 patients (mean age 59.73 years) presenting with hematemesis, melena or hematochezia in a Greek hospital during a six-month period. Patients with malignant causes were excluded. The results were analysed by x2 method.

Results: The two medium age groups (41-65 and 66-80 years) included most patients with acute GIB, 50 (33.11%) and 58 (38.41%) respectively, whereas only few ones (7.95%) were over 81 (p <0.001). 110 (72.85%) patients presented upper GIB, 31 (20.53%) lower GIB and the remaining 10 (6.62%) no identifiable source. 48.34% patients with GIB were males and 51.66% females, 52.73% with upper GIB were males and 42.27% females and 35.48% with lower GIB were males and 64.5% females. 47/110 (42.73%) with upper GIB were on NSAIDS or low-dose aspirin treatment with a drug ratio 1:1 (p >0.1). Peptic ulcer (55.45%) was the major cause. 39 (63.93%) ulcers were located in the duodenum and 22 (36.07%) in the stomach. Based on histology, 32 (52.46%) peptic, 20 (51.28%) duodenal and 12 (54.54%) gastric ulcers, respectively, were *Hp*(+). 21 (34.43%) peptic, 12 (30.77%) duodenal and 9 (40.09%) gastric ulcers, respectively, were *Hp*(+) with concomitant use of NSAIDS or low dose aspirin. 10 (16.39%) peptic ulcers were non-*Hp* non-NSAID/low-dose aspirin ones. Gastroduodenal erosions (17.27%), followed by varices (14.55%), were the second cause of upper GIB. Colonic diverticula (38.71%), with a predominance of women (66.67%), were the principal diagnosis of lower GIB and ischemic colitis (29.03%) the second one.

Conclusion: Very advanced ages were found to be the smallest patient group with acute GIB. The increased incidence in females, with a significant statistical difference in case of lower GIB, was attributed to the high prevalence of diverticulosis and increasing use of antiplatelet therapy and NSAIDS. NSAIDS were not any more the commonest cause of drug-induced upper GIB, when being compared with low dose aspirin. The relatively low frequency of *Hp* infection might be explained by the number and site of gastric biopsies, false negative results because of bleeding, antisecretory treatment or even geographical variation.

● **INVESTIGATING THE ROLE OF SOLUBLE TRIGGERING RECEPTOR EXPRESSED ON MYELOID CELLS AS AN INDICATOR IN EFFICACY OF TREATMENT OF PEPTIC ULCER DISEASE**

S. Vassiliou[1], V. Koussoulas[1], E.J. Giamarellos-Bourboulis[2], H. Tzepi[2], C. Kranidioti[2], G. Tassias[1], H. Giamarellou[2], C. Barbatzas[1]
[1]Gastroenterology, Sismanoglion General Hospital, [2]4th Dept. of Internal Medicine, Athens Medical School, Athens, Greece

Introduction: Acid secretion is associated with peptic ulcer disease. Several environmental and host factors modify acid secretion. Soluble triggering receptor expressed on myeloid cells (sTREM-1) is a novel cytokine implicating to peptic ulcer disease. Present study was focused on the investigation of the probable role of sTREM-1 as a marker of the efficacy of peptic ulcer disease treatment.

Aims & Methods: Thirty patients with peptic ulcer disease were enrolled; Patients were endoscoped and gastric juice was aspirated; biopsy specimens were collected from antrum and corpus. All patients were received antisecretory treatment in the proper dosage and time sequence. Concomitant eradication treatment for *Helicobacter pylori* was applied. After the end of the treatment a second look endoscopy was proceed; in an attempt to elucidate alterations to sTREM-1 in gastric juice. Pathologic score of chronic gastritis was recorded both before and after treatment according to updated Sydney score. sTREM-1 was estimated by a hand-made enzymeimmuno assay.

Results: Median ($\pm$ SE) of sTREM-1 before and after treatment in patients with ulcer was 43.78$\pm$11.61 pg/ml and 3.01$\pm$0.81 pg/ml respectively (P <0.001). Correlation between sTREM-1 and scores of neutrophils, monocytes infiltration of gastric mucosa and total gastritis score was found significant (P: 0.022, 0.040 and 0.045, respectively).

Conclusion: sTREM-1 could be used as a marker for the activity of peptic ulcer disease and chronic gastric inflammation. The role of sTREM-1 as an indicator of the activity of chronic gastric mucosal inflammation needs to be further clarified.

● LEVOFLOXACIN-BASED TRIPLE THERAPY VERSUS BISMUTH-BASED QUADRUPLE THERAPY AS A SECOND LINE TREATMENT FOR THE ERADICATION OF *H. PYLORI* INFECTION

S. Karatapanis[1], S. Georgopoulos[2], N. Papantoniou[3], P. Lisgos[1], S. Kalathenos[3], S. Afantenou[1], L. Skorda[1], C. Savidis[1], T. Anagnostis[1], C. Psellas[1], A. Zervos[1], K. Komnianides[1]
[1]1st Dept of Internal Medicine, General Hospital of Rhodes, Rhodes, [2]Gastroenterology Clinic, Medical Center, Phaliron, Athens, [3]Gastroenterology, General Hospital of Rhodes, Rhodes, Greece

Introduction: Levofloxacin-based triple therapy has been suggested as an alternative second line treatment to bismuth-based quadruple therapy for persistent *Helicobacter pylori* (*H. pylori*) infection.

Aims & Methods: *AIM:* Was to compare levofloxacin-based triple therapy (levofloxacin + amoxicillin + PPI) to bismuth-based quadruple therapy (bismuth + tetracycline + metronidazole + PPI) as a second line treatment for the eradication of *H. pylori*.

Methods: In our study there were included 77 patients who failed to eradicate *H. pylori* following 7-10 days previous treatment with PPI plus amoxicillin plus clarithromycin. All patients were randomly assigned either to levofloxaxin triple (levofloxacin 500 mg bid+amoxicillin 1 gr bid + lansoprazole 30 mg bid) therapy (Group A, N = 39) or to classical bismuth based quadruple regimen (bismuth 120 mg X 4 + tetracycline 500 mg tid + metronidazole 500 mg tid + lansoprazole 30 mg bid) (N = 38, Group B). Both groups were treated for 10 days. Eradication of *H. pylori* was assessed by ^{13}C-urea breath test 4-6 weeks after therapy.

Results: The *H. pylori* eradication rates on the intention to treat analysis (ITT) were 37/39 (94.87%) in Group A and 30/38 (78.9%) in group B (P <0.05). The per protocol eradication rates were 97.3% and 85.7% respectively (NS). Side effects were significantly higher in the quadruple regimen (3 patients discontinue treatment due to side effects versus none in the levofloxacin regimen).

Conclusion: A 10-day course levofloxacin triple therapy appeared more effective and better tolerated than a 10-day bismuth-based quadruple therapy in the treatment of persistent *H. pylori* infection.

● **MAY SOLUBLE TRIGGERING RECEPTOR EXPRESSED ON MYELOID CELLS (STREM-1) BE CONSIDERED AN ANTI-INFLAMMATORY MEDIATOR IN THE PATHOGENESIS OF CHRONIC GASTRITIS?**

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

Introduction: Soluble triggering receptor expressed on myeloid cells (sTREM-1) is a novel mediator involved in the pathogenesis of peptic ulcer disease. To investigate the potential role of sTREM-1 to the anti-inflammatory response in chronic gastritis, sTREM-1 was compared with other anti-inflammatory mediators of gastritis.

Aims & Methods: Forty patients with dyspepsia were enrolled; twenty with peptic ulcer and 20 controls without any macroscopic abnormalities. All patients were examined by endoscopy; gastric juice was aspirated and biopsy specimens were collected from antrum and corpus of the stomach. sTREM-1 was estimated by a hand-made enzymeimmuno assay. Interleukin-8 and interleukin-10 were estimated by ELISA.

Results: Correlation between ratios of IL-8/sTREM-1and IL-8/IL-10 was found positively significant (r: +0.911, P: 0.021). Median (± SE) of sTREM-1 of controls and patients with ulcer was 3.91±0.57 pg/ml and 44.27±241.55 pg/ml respectively (P: 0.006). Median (±SE) concentrations of IL-10 in subjects with _Helicobacter pylori_ positive and negative gastritis were 18.78±9.81 and 16.34±7.22 pg/ml, respectively (P: 0.011). Correlation between IL-8/sTREM-1 ratio and score of neutrophils infiltration of gastric mucosa was found significant (r: +0.798, P: 0.006). sTREM-1 was negatively correlated with IL-8 and positively correlated with IL-10 (P: 0.037 and 0.042, respectively); IL-10 was negatively correlated with IL-8 (P: 0.043).

Conclusion: sTREM-1 might be a novel factor contributing to the anti-inflammatory response in chronic gastric inflammation. The latter involvement might indicate sTREM-1 as an independent factor to the gastric inflammatory process that is positively correlated to histopathologic abnormalities of gastritis.

● REVEALING THE SIGNIFICANCE OF SOLUBLE TRIGGERING RECEPTOR EXPRESSED ON MYELOID CELLS IN THE INFLAMMATORY PROCESS OF PEPTIC ULCER DISEASE

V. Koussoulas[1], V. Karagianni[2], E.J. Giamarellos-Bourboulis[2], S. Vassiliou[1], A. Pelekanou[2], C. Barbatzas[1], H. Giamarellou[2]
[1]Gastroenterology, Sismanoglion General Hospital, [2]4th Dept. of Internal Medicine, Athens Medical School, Athens, Greece

Introduction: Soluble triggering receptor expressed on myeloid cells (sTREM-1) is a novel mediator implicating to the pathogenesis of peptic ulcer disease. Aim of the present study was to investigate the pattern of sTREM-1 release from the inflamed gastric mucosa.

Aims & Methods: Forty five patients with peptic ulcer were enrolled; Patients were endoscoped and gastric juice was aspirated; biopsy specimens were collected from antrum and corpus. Three biopsy specimens from antrum were cultured; the first incubated with the addition of lipopolysaccharides (LPS), the second with the addition of gastric juice of each patient and the third served as control. Supernatants were collected and sTREM-1 and TNFα were measured. All patients were received antisecretory treatment in the proper dosage and time sequence. Eradication treatment for *Helicobacter pylori* was applied. After the end of the treatment a second look endoscopy was proceed. Pathologic score of chronic gastritis was recorded both before and after treatment according to updated Sydney score. sTREM-1 was estimated by a hand-made enzymeimmuno assay and TNFα with ELISA.

Results: Median ($\pm$SE) of sTREM-1 before and after treatment in patients' control, LPS and gastric juice incubated supernatants were $63.91\pm13.58/3.90\pm0.80$ pg/ml (P <0.001), $108.78\pm18.81/14.81\pm3.61$ pg/ml (P <0.001) and $33.91\pm9.32/3.81\pm0.96$ pg/ml (P: NS), respectively. Respective values of TNFα were $9.79\pm1.48/6.70\pm1.21$ pg/ml (P = 0.007), $14.57\pm3.31/7.51\pm2.61$ pg/ml (P <0.001) and $9.59\pm2.81/7.31\pm1.96$ pg/ml (P = 0.006). Comparison of sTREM-1 between control and LPS incubated supernatants in patients with peptic ulcer was found significant (P = 0.014). Correlation between sTREM-1 and score of monocytes infiltration of gastric mucosa and total gastritis score was found significant (P = 0.012 and 0.025, respectively). Correlation between sTREM-1 and TNFα was found significant (P <0.001).

Conclusion: sTREM-1 release from gastric mucosa in patients with peptic ulcer disease seems to be depended from the activity of mucosal inflammation. The increase of sTREM-1 secretion is triggered from the inflammatory cells infiltration and is related with the increase of the main pro-inflammatory cytokine. Similar alterations of sTREM-1 concentrations in post treated patients were not observed. Implication of sTREM-1 in peptic ulcer disease needs to be further investigated.

● CHARACTERIZATION OF THE NUMBER AND TYPE OF REPEATING EPIYA PHOSPHO- RYLATION MOTIFS IN THE CARBOXYL TERMINUS OF CAGA PROTEIN IN *HELICO- BACTER PYLORI* CLINICAL ISOLATES

E.G. Panayotopoulou[1], K. Petraki[1], A. Kalliaropoulos[1], K. Papadakos[1], S. Michopoulos[2], D.N. Sgouras[1], A.F. Mentis[1], A. Archimandritis[3]
[1]Institut Pasteur Hellenique, [2]Gastroenterology Clinic, Alexandra General Hospital, [3]Second Department of Internal Medicine, Athens University School of Medicine, Athens, Greece

We aimed to map the EPIYA tyrosine phosphorylation motifs (TPMs) *A*:EPIYAKVNK, *B*:EPIYAQVAKK, and *C*:EPIYATIDDLG in CagA protein, which have been proposed to enhance cagA-dependent pathogenecity. Sixty-five *Helicobacter pylori* clinical strains isolated from adults with nonulcer dyspepsia (n=13), esophagitis (n=12), gastric ulcer (n=11), and duodenal ulcer (n=29) were analyzed. In the 48 cagA-positive strains, the 3' variable region of cagA gene was amplified and sequenced and the EPIYA motifs were mapped in the deduced protein sequences. *H. pylori* colonization and the associated gastritis were evaluated by the modified Sydney system and statistical analysis performed by χ^2 test and Fisher's exact test.

The majority of strains harbored the *ABC* (54.5%) and the *ABCC* combination of TPMs (13.6%). Only four strains were found to harbor additional TPM in the *ABCCC* (n=3) or *ABABC* (n=1) combinations. Eighty-five percent of strains isolated from gastroduodenal ulcers harbored *ABC* or *ABCC* combinations of TPMs. EPYIA presence in the CagA protein was correlated significantly with the development of gastroduodenal ulcer (χ^2=11.617, p=.0007). and in particular with the presence of duodenal ulcer (p=.0016). There was significant positive association with the severity of chronic inflammatory infiltration (p=.039) and the activity of chronic gastritis (p=.013) in the antrum, but not with higher levels of *H. pylori* colonization (p=.136). In conclusion, the severity of chronic inflammatory infiltration and the activity of chronic gastritis developed in the antrum of *H. pylori*-positive patients may be associated with the presence of EPYIA TPMs in the CagA protein, irrespective of the levels of *H. pylori* colonization in the gastric mucosa.

● **META-ANALYSIS ON THE RELATIONSHIPS BETWEEN *HELICOBACTER PYLORI* INFECTION AND EXTRA-GASTRIC GI CANCERS**

T. Rokkas, D. Pistiolas, P. Sechopoulos, J. Robotis, G. Margadinis
Henry Dunant Hospital, Athens, Greece

Background: *Helicobacter pylori* is an important causative factor in gastric carcinogenesis. However, its role in extra-gastric gastrointestinal (GI) malignancies, such as esophageal and colonic cancer, is controversial. The main aim of this study was to explore the relationship between *H. pylori* infection and these malignancies by meta-analyzing all relevant cohort and case-control studies. Secondary aims were to investigate the possible sources of heterogeneity between studies and to look for the existence of publication bias.

Methods: Extensive Medline English language medical literature searches for human studies were performed through May 2006, using suitable keywords. Pooled estimates were obtained using fixed or random-effects models as appropriate. Heterogeneity between studies was evaluated with the Cochran Q test, whereas the likelihood of publication bias was assessed by constructing funnel plots. Their symmetry was estimated by the Begg and Mazumdar adjusted rank correlation test and by the Egger's regression test.

Results: For colon cancer the pooled odds ratio (OR) with 95% confidence intervals (CI) were 1.44 (1.2-2.02), test for overall effect $Z=2$, $p=0.038$. The heterogeneity Q value was 10.5, $I^2=52.3$, $p=.062$. There was no publication bias (Begg and Mazumdar adjusted rank correlation test p two-tailed value 0.7, Egger's regression test p value .09). For esophageal cancer the pooled OR with 95% CI were 0.54 (0.41-0.72), test for overall effect $Z=-4.17$, $p<.0001$. The heterogeneity Q value was 10.93, $I^2=36$, $p=.14$. There was no publication bias (Begg and Mazumdar adjusted rank correlation test p two tailed value 0.27, Egger's regression test $p=.19$).

Conclusion: The results of our study showed a small statistically significant relationship between *H. pylori* infection and colon cancer. On the contrary, there was an inverse statistically significant relationship between *H. pylori* infection an esophageal cancer, suggesting that *H. pylori* infection has no etiological role in this malignancy.

● INTRAGASTRIC BALLOON TOLERANCE IS INDEPENDENT OF *HELICOBACTER PYLORI* STATUS IN PATIENTS WITH MORBID OBESITY

N.G. Mathou, K.D. Paraskeva, K.J. Zografos, J.A. Karagiannis
Gastroenterology Unit, "Agia Olga" Hospital, Athens, Greece

Endoscopic intragastric balloon consists a method for weight loss. However, most patients experience poor balloon tolerance. It has not studied if the presence of *Helicobacter pylori* infection is a factor affecting tolerability.

Thirty-three patients (median body mass index, 38.1), 23 women, 10 men, age range 20-65 years were studied. In all patients, an intragastric balloon (INAMED, USA) was inserted endoscopically and was filled up to a median volume of 550 mL. *H. pylori* status was confirmed during screening Endoscopy (rapid urease test plus histology). Exclusion criteria were peptic ulcer, severe gastritis, or chronic nonsteroidal anti-inflammatory drug use. All patients were followed up daily in the first 7 days and monthly thereafter up to removal (6 months later) by a standard questionnaire. Patients were allowed to on-demand H_2-RA/PPI's and/or prokinetics. Nausea and/or vomiting and/or crampy epigastric pain were characterized as mild to moderate (nausea, vomiting <10/day, duration <10 days and/or pain without necessitating further management) or severe (intractable nausea and/or vomiting >10 day, duration >10 days and/or severe pain necessitating further management and/or premature removal of the balloon.

Fourteen patients were *H. pylori* positive (42.4%) whereas 19 were negative (57.6%). All patients, independently to their *H. pylori* status, experienced mild to moderate symptoms with a mean duration of 2 days. Seven (21%) experienced severe symptoms requiring further management (three *H. pylori* positive, four *H. pylori* negative) and in four of them (12%) the balloon had to be removed within 1 month (three *H. pylori* negative, one *H. pylori* positive). These findings were not statistically significant, against or for, *H. pylori* status.

Therefore, *H. pylori* eradication is not justified prior to balloon insertion.

● **EXPRESSION OF iNOS IN PRE-NEOPLASTIC AND NEOPLASTIC CONDITIONS OF GASTRIC MUCOSA AFTER *HELICOBACTER PYLORI (H. PYLORI)* INFECTION: A MOLECULAR AND IMMUNOHISTOCHEMICAL STUDY IN TISSUE MICROARRAYS (TMAS)**

A. Karameris[1], E. Tsiambas[1], S. Gazi[2], K. Gerontopoulos[2], G. Vilaras[1], D. Stefanou[2], T. Rokkas[3]

[1]Department of Pathology, 417 VA Hospital (NIMTS), Athens, [2]Department of Pathology, University of Ioannina, Ioannina, [3]Gastroenterology Clinic, Henry Dunant Hospital, Athens, Greece

Background: Reactive oxygen and nitrogen species may play a crucial role in human carcinogenesis. The reactive nitrogen species are derived from the synthesis of NO, stimulated by iNOS, in a variety of cell types including activated macrophages and neutrophils. Recent studies have revealed that *Helicobacter pylori* (*H. pylori*) infection leads to the formation of nitrotyrosine, which may contribute to DNA damage and apoptosis in gastric mucosa.

Aim: To analyze the expression of iNOS by immunohistochemical and molecular methods in *H. pylori* infected preneoplastic and neoplastic conditions of the gastric mucosa using Tissue Microarrays (TMAs), in a effort to correlate the possible role of active nitric species and *H. pylori* infection in the progression of gastric carcinogenesis.

Material and Methods: 45 formalin fixed, paraffin embedded gastric carcinoma cases were studied for determination of the expression of iNOS (25 *H. pylori*+ and 20 *H. pylori*-). Intestinal metaplasia (IM) type I were observed in 5 *H. pylori*+ cases, IM II in 8 and IM III in 12 of them. Controls included 5 *H. pylori*- IM I, 6 IM II and 10 IM III cases. The TMArrayer apparatus (Chemicon, USA) was used for the construction of TMAs. iNOS expression in molecular level was determined by differential-PCR in microcore samples taken by TMArrayer and properly analyzed with an Image Analysis System (DIS-200, Digital Image Systems, Hellas). The expression of iNOS molecule was studied by immonochistochemistry using a polyclonal anti-IgG human iNOS antibody (Santa Cruz, USA) and the evaluation of the result was done by DIS-200.

Results: 12/13 of intestinal and 10/12 of diffuse *H. pylori*+ gastric carcinomas overexpressed iNOS as indicated by differential PCR and immunohistochemistry. 2/5 IM, 8/12 IM and 7/8 IM *H. pylori*+ cases were also expressed increased iNOS levels as well as 5 diffuse and 5 intestinal type *H. pylori*- carcinomas. In addition 0/5 *H. pylori*- IM I, 1/6 *H. pylori* IM II and 2/10 *H. pylori*- IM III cases were also expressed high iNOS levels. A statistical significant difference concerning the expression of iNOS between *H. pylori*+ Ca and *H. pylori*- Ca (<0.01), between IM and (<0.05), as well as between *H. pylori*+ and *H. pylori*- IM and (<0.01) was also observed.

Conclusion: Our data suggest that iNOS expression in gastric mucosa is of significant importance for the initiation and promotion of gastric carcinogenesis. The observation that high iNOS levels in IM type II and III correlated well with *H. pylori* infection, may be a strong evidence for long term follow up.

● **HELICOBACTER PYLORI INFECTION AS A CAUSE OF IRON DEFICIENCY ANAEMIA**

N. Papantoniou[1], S. Karatapanis[2], P. Lisgos[2], E. Spanos[3], Y. Papantoniou[1], K. Kladakis[1], H. Tsibidakis[1], K. Komnianidis[2], C. Psellas[2], S. Manolitsis[2], G. Manolakas[2], I. Stouraitis[2]
[1]Department of Gastroenterology, [2]1st Department of Internal Medicine, [3]Department of Hematology, Rhodes General Hospital, Rhodes, Greece

Background: It is estimated that 30% of patients presented with iron deficiency anemia remain undiagnosed despite complete upper and lower GI Endoscopy and other tests. *Helicobacter pylori* (*H. pylori*) infection is the main cause of chronic gastritis, peptic ulcer and gastric cancer. *H. pylori* infection has a relation with iron metabolism but the exact mechanism is unknown.

Aim: To investigate the role of *H. pylori* infection in patients with iron deficiency anemia of unknown aetiology.

Patients and Methods: In the present study we investigated 115 consecutive patients presented with iron deficiency anemia during the period 2003-2004. We excluded patients with nutritional problems, with IBD, with a history of upper GI surgery, patients receiving NSAIDs or aspirine, pregnant women and patients with malignancy. Presence of *H. pylori* was assessed by histology (Giemsa), CLO-test and/or UBT. Iron deficiency anemia was diagnosed in the presence of low plasma ferritin and iron level and low transferring saturation.

Results: 54 out of the initial 115 patients excluded from further evaluation (due to medication 33, due to malignancy 6, due to pregnancy 2 and 3 due to recent episode of GI bleeding). From the remaining patients 38/61 (62.29%) had established *H. pylori* infection, while 23/61 (37.7%) were negative to *H. pylori*. Mean haemoglobin levels were similar in both group of patients (8.9 gr% vs 8.7 gr%). On the contrary the mean values of serum iron and ferritin levels and transferin saturation were significantly lower in those patients harboured *H. pylori* infection as compared with those negative to *H. pylori* testing.

Conclusion: Chronic atrophic gastritis as a consequence of chronic *H. pylori* infection correlates with iron deficiency anemia of unknown aetiology. The interaction of *H. pylori* infection and iron metabolism remains obscure.