1C: Non-gastric diseases related to *H. pylori* infections

1C:01 HELICOBACTER PYLORI AND REFUX OESOPHAGITIS

R.J.L. Loffeld, B.F.M. Wedermuller. Department of Internal Medicine, Ziekenhuis De Heel, Zoandam, The Netherlands

The main pathogenetic factor in reflux oesophagitis apparently is the reflux of acidic stomach contents. From a theoretical point of view it could be postulated that HP indirectly has an effect on reflux oesophagitis by making the reflux more aggressive.

A prospective study was done in consecutive patients presenting with reflux disease.

During one year all consecutive patients undergoing upper GI endoscopy were studied for the presence of HP. Inclusion criterion was the presence of hiatal hernia and/or reflux oesophagitis, and Barrett’s oesophagus. Exclusion criterion was the presence of oesophageal or gastric cancer and the presence of concomitant peptic ulcer disease or macroscopic gastritis.

Biopsy specimens were taken for HE stain, urease test and culture. A serum sample was taken for determination of IgG antibodies against HP using a home-made ELISA. As a reference group patients in whom endoscopy did not show abnormalities were included. Reflux oesophagitis was present in 118 patients (68 C, 50 Q, mean age 55.3 years). Hiatal hernia without oesophageal inflammation was seen in 109 patients (43 C, 66 Q, mean age 57.3 years), and Barrett’s oesophagus in 13 patients (8 C, 5 Q, mean age 61.5 years). The reference group consisted of 399 patients (166 C, 233 Q, mean age 48.4 years).

HP was present in 27% of oesophagitis patients, in 23% of Barrett’s oesophagus, in 32% of hiatal hernia, in 28% of oesophagitis with hiatal hernia and 25% of oesophagitis without hiatal hernia, and in 51% of controls. Prevalence of HP was significantly lower in all groups compared with the reference group (p < 0.001). There was no difference between the different groups. Patients with oesophagitis and HP were significantly older than oesophagitis patients without HP, 61.5 years (SD 17) versus 53 years (SD 17) (p < 0.001). The same was true if helicobacter positive and negative references were compared, 52 years (SD 17) versus 44.8 years (SD 17) (p < 0.0001).

It is concluded that the prevalence of HP in patients with reflux disease is significantly lower than in a reference group with normal oesophagus. The patients with reflux oesophagitis and HP are older than those without the infection indicating that the age cohort effect is present. In case of reflux oesophagitis it is essential to know whether the patients is helicobacter positive, because long-term acid suppressive therapy will lead to atrophic gastritis. HP has no direct or indirect role in the pathogenesis of reflux oesophagitis.

1C:02 WHAT IS THE INFLUENCE OF HELICOBACTER PYLORI INFECTION ON MUCOSAL LESIONS DEVELOPMENT IN PATIENTS TAKING ACETYLSALICYLIC ACID?

H. Kordecki, M. Kurowski, D. Plecika, R. Kosik. M. Curie Hospital, Szczecin, Poland

Acetylsalicylic acid (ASA) is one of the drugs widely taken for ischaemic heart disease. But up to now the influence of ASA on gastric and duodenal mucosa is still not known. Recently published reports suggest that repeated doses of ASA increase mucosal tolerance and ASA could cause the mucosal injury only during the first days of treatment. However these studies were performed in rats.

The aim of our study was to evaluate the frequency of mucosal lesions (m.l.) in the upper digestive tract as well as the frequency of Helicobacter pylori (H.p.) infection in patients chronically receiving acetylsalicylic acid in the doses ranged from 70-300 mg daily. ASA was administered for prevention of ischaemic heart disease attacks in 60 patients treated in the Department of Cardiology in our hospital.

These patients were 31-72 years old (mean 50.5 y). They were sent for endoscopic examination of upper digestive tract because of the planned heart surgery. None of them reported any complaints from the upper abdomen.

The control group comprised the same number of patients of similar age treated for ischaemic heart disease, who did not take ASA due to various contraindications.

In 52 (87%) patients suffering from ischaemic heart disease and taking ASA the presence of mucosal erosions in the stomach (some of them were haemorrhagic) and/or in the duodenal bulb were disclosed. 12 (20%) patients had gastric ulcer and the microscopical examination revealed active gastritis in 39 (65%) patients. 76% of patients receiving ASA were infected with H.p. (positive rapid urease test confirmed by microscopical examination). In the control group comprised patients not taking ASA only 4 patients (7%) the prevalence of single mucosal erosions was revealed. 54 patients had no changes in the upper digestive tract. The prevalence of H.p. was disclosed in 16 (27%) patients.

The study we performed failed to confirm the views that long-term ASA therapy is free of gastric and duodenal m.l. Although the study group was relatively small we cannot share the opinion that gastric and duodenal ulcers occurring during ASA therapy are not hazardous for patients. However it should be stressed that significantly higher percentage of H.p. infection appears in patients treated with ASA in comparison with control group, what in our opinion is favouring factor of m.l. development.

1C:03 CHANGES IN ANTIBODIES TO HELICOBACTER PYLORI (HP) AND IN JOINT MANIFESTATIONS IN PATIENTS WITH RHEUMATOID ARTHRITIS (RA) AFTER ERRADICATION FROM HP PRELIMINARY RESULTS

L.B. Graff 1, L.P. Andersen 2, A. Bremmelgaard 1, O. Bonnevie 3, S. Bondesten 1, A. Gernow 1, A. Savnik 1, J. Bloem 3, N. Heiby 3, B. Danneskiold-Samsøe 1, Frederiksberg Hospital, 2 Rigshospitalet, 1 Statens Serum Institut, Copenhagen, Denmark

**Aims:** To evaluate the effect of eradication from HP in patients with RA with regard to IgG antibodies and to joint manifestations.

**Methods:** 54 RA patients participate in the study, 17 have been found HP-culture positive and 8 patients, 6 women and 2 men, median age 62.5 years (39–79 y) and median disease duration 12.5 years (2–35 y) have been followed for 18 weeks. The patients were examined for IgG antibodies (LMW IgG) to HP by ELISA before and after eradication from HP. Biopsies were taken and examined according to the Sydney classification of gastritis before and 6 weeks after ended antibiotic treatment. The patients’ joints were examined for pain at palpation and for swelling and the joints were giving scores from 0 to 3 in each category. The serological and the clinical examination were performed before treatment, 6 and 18 weeks after.

**Results:** Median LMW-IgG was before treatment 284.5 Eu (145–840 Eu), after 6 weeks 163 Eu (119–832 Eu) and after 18 weeks 97 Eu (0–374), (p2 weeks = 0.02), (p18 weeks < 0.01). The median score of joint pain was 30 (4–95) before treatment, 28 (2–112) after 6 weeks and 34.5 (3–107) after 18 weeks (p2 weeks = 0.04), (p18 weeks = 0.09). The median score of joint swelling was 7 (2–21) before treatment, 2 (0–40) after 6 weeks and 4 (0–23) after 18 weeks (p2 weeks = 0.20), (p18 weeks = 0.32).

**Conclusion:** A significant change was found in IgG to HP after eradication (p < 0.01), but the changes in joint pain and joint swelling showed no significance (p18 weeks = 0.09).

1C:04 HP PREVALENCE IN IBD PATIENTS: ROLE OF IBD TREATMENT

M. De Reuck, M. Brad, J. Otero, A. Cuzzoli, P. Denis, E. De Koster, M. Deltenre. Bruggman University Hospital, Brussels, Belgium

A lower prevalence of HP in IBD patients (pts), associated to sulfapyridine (SP) treatment, has been suggested (El Omar Gut 1994; 35: 1385–8). We determined HP prevalence in 130 IBD pts: 85 Crohn’s disease (CD), 45 ulcerative colitis (UC), using serology (78), Breath Test (21), histology (31). IBD treatment: none (NT), enteric coated SP, 5-ASA. We compared with 155 Non Ulcer Dyspepsia (NUD) pts. Results: HP prevalence is shown in Table 1.

Prevalence of HP in IBD (23%) lower than in NUD (47%) p < 0.0001. No difference CD (19%)-UC (31%) (ns). UC NT (46%) not different from NUD (47%), higher than treated UC (14%), p = 0.05 or treated CD (18%, p < 0.01). No difference SP (16%) to 5-ASA (18%). Untreated CD (23%) not significantly lower than untreated UC (46%) (ns). Conclusions: We confirm the lower HP prevalence in treated IBD patients, both CD and UC. HP prevalence in untreated UC patients is not different from NUD patients. HP prevalence in untreated CD patients may be lower than in untreated UC patients, but our series is too small to reach a statistically significant difference. We find no difference in HP prevalence between pts treated with
CAG is a condition characterized by atrophy of oxyntic mucosa hypo/chlorhydria and fasting hypergastrinemia. It has recently been observed that a significant proportion of CAG patients are Hp infected and that Hp infection is significantly associated with the development of corporal atrophy in patients treated long-term with proton pump inhibitors. No studies until now have been performed specifically to determine if Hp infection is present in CAG patients, if it is possible to treat the infection since the lack of acid secretion could determine less favourable condition to his eradication. 19 consecutive patients (15 F, 4 M aged 34–72) with Hp positive CAG (histology, culture, IgG; at least two of these tests positive) were treated for 4 wks with bismuth subsalicylate 240 b.i.d.; amoxicillin 1 g tid-post-prandially and metronidazole 250 mg tid post-prandially were given during the first 2 wks of therapy. Endoscopy was again performed after 6 mos, to evaluate Hp status by histology and culture in antrum (n = 5) and body biopsies (n = 5). The gastritis status was graded according to the Sydney system. Gastric atrophy levels (pg/ml; specific RIA) and title of IgG Hp (U/I; Elisa GAP test, Biord) were also determined. Results are expressed as median, range, non-parametric test for paired data was used for statistical evaluation. Results: Overall eradication rate was 78.9% (15/19 pts). Minor side effects were recorded in about half of patients, but none determined therapy withdrawal. Gastrin levels decreased dramatically in eradicated patients [220 (49–1400) vs 42 (10–285); p < 0.001] as IgG Hp [80 (10–100) vs 31 (0–65); p < 0.005] Corpal chronic atrophy and gastritis were significantly reduced after eradication [respectively: 2 (1–3) vs 1 (0–2); p = 0.001 and 2 (1–3) vs 1 (0–3); p = 0.031]. Improvement of gastritis activity completely regressed [1 (0–3) vs 0 (0–0)]. Conclusions: These data that is possible to efficaciously treat Hp infection in CAG patients, determining a pronounced reduction of gastrin levels and a significant reduction in the score of corporal chronic gastritis.

1C:06 DOES HELICOBACTER PYLORI INFECTION HAVE A ROLE IN PERNICIOUS ANEMIA?

G.J. Pérez-Pérez, R. Carmel, M.J. Blaser. Vanderbilt University Department of Veterans Affairs Medical Center, Nashville TN and University of Southern California Los Angeles CA

Previous studies indicate that patients with pernicious anemia (PA) are infected with H. pylori (Hp) less than often are age-matched controls. However, Hp infection may be present prior to the development of PA and gradually become absent due to the development of the associated type A atrophic gastritis. To determine whether evidence of Hp infection may disappear during the course of the illness, we studied 47 PA patients (mean age 58.2 years and 36.2% male) prospectively. The diagnosis of PA was established by the presence of macrocytic anemia, progressive development of the characteristic tongue (3–4), pernicious acidosis (PA), neuropathy (PN) and chronic and ANA. 19/32 (61%) patients showed evidence of Hp infection. Comparison of the 12 Hp-seropositive (Hp+) and 35 Hp-seronegative (Hp–) PA patients showed that the former group had significantly lower gastrin (mean 686 vs 1293 pg/ml, p = 0.045) and higher pepsinogen A levels (mean 27.1 vs 11.6 pg/ml, p = 0.05), factors consistent with less severe atrophic gastritis. The 35 Hp– patients were followed for a total of 204 person-years; none showed seroreversion. The 12 Hp+ were followed for a total of 63 person-years, (mean 5.25 years); during this time, 4 (33%) of the patients seroreverted. Thus, among patients with PA, the seroreversion rate was greater than 6% per year. Those who seroreverted were younger (52 vs 69 y, p = 0.06) and had lower pepsinogen A levels (9 vs 39, p = 0.057) than those remaining positive. These data are consistent with the hypothesis that Hp-infection precedes at least some cases of PA and that the infection reverses with time, and presumably with progressive gastritis. This pattern may be especially associated with a subset of PA characterized by less severe gastritis and pepsinogen abnormalities in infected persons. The development of atrophic gastritis as part of PA leads to gradual elimination of the organism and failure to detect a past Hp infection. Prospective studies, in the pre-PA stage of gastritis are needed to clarify the putative role of Hp in causing PA.

1C:07 ROLE OF H. PYLORI INFECTION IN PEPTIC ULCERATIONS IN 50 PATIENTS WITH CIRRHOSIS

L. Prigent-Delecourt, D. Lamanque, F. Roodot-Thoraval, R. Akremi, M.T. Chaumette, J.P. Richardart, J.C. Delchier. Hospital Henri Mondel, 94000 Creteil, France

The pathogenesis of gastric and duodenal ulcers is one of the main problems facing patients in liver cirrhosis. The aim of this study was to determine the pathogenic factors associated with peptic ulcerations in patients with cirrhosis. Patients with histologically proven cirrhosis and not recently treated by antibiotics, antisecretory or anti-inflammatory drugs, were enrolled and referred for upper gastrointestinal endoscopy. Upper digestive tract bleeding within the last week was an exclusion criteria. Age, gender, smoking habit, recent alcohol intake, etiology of cirrhosis, Child–Pugh grade were recorded and basal gastrinemia was determined. Esophageal varices were graded from 0 to 3. Severity of the bacteriological load in the antrum was graded from 0 to 3 for erythema, edema and snake-skin mosaic pattern (maximum score: 18). H. pylori status was determined from rapid urease test and histology on biopsy samples or 13C-urea breath test.

Fifty patients were included. There were 42 males and 8 females, mean age ± SD: 56 ± 10 years. Cirrhosis was alcoholic in 35, grade A, B and C in the Child-Pugh classification in 13, 18 and 19 respectively. Twenty seven (54%) were Hp positive. Peptic ulcerations were present in 18 (36%): gastric ulcer in 7, duodenal ulcer in 7, gastric erosions in 5, duodenal erosions in 3. Univariate analysis showed that peptic ulcerations were not significantly related with age, gender, smoking habit, etiology of cirrhosis, Child–Pugh grade, esophageal varices grade. Hp positivity (8/18 vs 19/32, p = 0.47), and basal gastrinemia. Univariate and multivariate analysis showed that they were significantly related to a high hypertensive gastropathy score (10.7 ± 4.4 vs 7.1 ± 4.4, p < 0.02) and to a recent alcohol ingestion (67% vs 29%, p < 0.01).

Conclusion: Peptic ulcerations in cirrhosis are unrelated to Hp despite high prevalence of infection. They are significantly and independently related to hypertensive gastropathy severity and to recent ingestion of alcohol.

1C:08 CORRELATION BETWEEN GASTRIC INFECTION WITH HELICOBACTER PYLORI AND PLASMA LEVELS OF FIBRINOGEN, PLASMINOGEN ACTIVATING INHIBITOR (PAI) AND VON WILLEBRAND FACTOR (vWF) ANTIGEN

L. Berti, C. Cernuschi, C. Abbiati, G. Beccari, R. Di Battista, R. Marchi, A. Federici, B. Bottasso, N. Di Rocco, R. de Franceschi, P.M. Mannucci, 1st Gastroenterology service, University of Milan, IRCCS Ospedale Policlinico, Milan, Italy; A. Bianchi Bonomi Hemophilia and Thrombosis Centre, University of Milan, IRCCS Ospedale Policlinico, Milan, Italy

There is evidence suggesting that patients at risk for coronary heart disease (CHD) are more likely to suffer from Helicobacter Pylori (Hp) gastric infection than controls. In addition, preliminary data suggest that patients with Hp infection may have high levels of plasma fibrinogen (Fg) to determine whether Hp infection is associated with increased plasma levels of fibrinogen and other risk factors for CHD such as PAI and vWF antigen.

Methods: consecutively patients undergoing upper GI endoscopy were included. Statistical analysis was carried out by means of Student’s t test. Results: 130 patients (66 Hp positive and 64 Hp negative) were enrolled. There was no difference between the two groups in sex, age, smoking history, hormonal therapy in females, arteriosclerosis, dizziness, value of acute phase reactants, family history of CHD. Plasma fibrinogen levels (mg/dL) were 318 ± 76 in the Hp+ group vs 291 ± 65 in the Hp– group (p = 0.037); plasma PAI levels (mg/dL) were 41.2 ± 29 in Hp+ and 35.8 ±
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24 in HP—patients (p = N.S.); vWF antigen levels (U/dL) were 148 ± 53 in the HP group vs 114 ± 52 in the HP—group (p = 0.01). Conclusion: Plasma vWF and fibrinogen levels were significantly higher in HP+ than in HP—patients. PAI levels were also increased in HP+ patients, although the difference did not reach statistical significance. Further studies on larger patient populations are required to clarify whether HP+ patients are indeed at higher risk for CHD.

1C:09 REFLUX ESOPHAGITIS: A COMPLICATION OF HELICOBACTER PYLORI ERADICATION THERAPY? N. Sacca, A. De Medici, S. Rodinò, M. De Siena, A. Giglio. Servizio di Endoscopia Digestiva, Ospedale Cisano, Catanzaro, Italy

Many therapeutic approaches in Helicobacter Pylori eradication have been proposed in the last years. Recently some authors reported in a preliminar study an high incidence of reflux esophagitis in patients after HP eradication therapy [1]. Aim of our study was to evaluate this phenomenon in our endoscopical population during the last year. Methods: 276 patients affected by peptic ulcer and HP infection were treated with different therapeutic regimens (various antisecretory drugs: omeprazole or ranitidine and various antibiotics: claryctomycline (A) + tinidazole, A + metronidazole (B), amoxycillin (C) + bismuth, C + B. The patients were investigated clinically and endoscopically at 1–6 months after therapy and when dyspeptic symptoms occurred, HP status was assessed by urease test and histology. Results: 169 (61.2%) were eradicated at 6 months after therapy: 24 of them developed an endoscopically proven reflux esophagitis which was mild (grade 1) in all the patients. Conclusions: our study confirms the evidence of reflux esophagitis development in patients treated for HP infection. Such evidence was explained by different theory: changes eating and drinking habits that can reduce lower esophageal sphincter pressure or the interruption of a chronic therapy with antisecretory drugs for peptic disease. We believe that this phenomenon isn’t usual finding in patients without previous evidence of reflux esophagitis and that further studies are needed to clarify this phenomenon.

Reference

1C:10 THE ASSOCIATION BETWEEN HELICOBACTER PYLORI AND ISCHAEMIC HEART DISEASE (IHD) ASSESSED BY CORONARY ANGIOGRAPHY

N. Ossei Gernig, P. Moayyed 1, A.T.R. Axon 1, P.J. Grant, Academic Unit of Medicine, Leeds General Infirmary; 1 Centre for Digestive Diseases, Leeds General Infirmary, Leeds, UK

Introduction: H. pylori infection has been reported to increase the risk of IHD. The evidence for this association is conflicting as the methods for evaluating IHD have been imprecise (history and/or ECG changes). We have determined the association of H. pylori status with IHD assessed by coronary angiography in a large group of patients. Methods: Consecutive caucasian patients undergoing coronary angiography were recruited. H. pylori status was assessed by serology (Helico G). Coronary angiography was assessed by an experienced cardiologist who was blinded to H. pylori status and IHD was defined as > 50% stenosis of at least one vessel. Results: 288 patients were recruited (median age 59, age range 33–83 years, 95 female). 204 (70%) patients had IHD and 185 (64%) patients were H. pylori positive. 68% patients with IHD were H. pylori positive compared with 50% without IHD (2 χ 2 = 0.003). When adjusted for age, sex, smoking, cholesterol, hypertension and social class H. pylori infection was still associated with IHD (relative risk = 2.4; 95% CI = 1.2–5.1; p = 0.02).

Conclusion: The study suggests that H. pylori is a risk factor for IHD.

1C:11 H. PYLORI INFECTION IN PATIENTS WITH AND WITHOUT CORONARY ARTERY DISEASE

N. Vakil, A. Khurshid, T. Fenske, T. Bajwa. Univ of Wisconsin, Milwaukee W7

The aim of this study was to determine the sero-prevalence of H. pylori infection in patients with suspected ischemic heart disease with normal and abnormal coronary angiograms. Methods: 150 patients undergoing coronary angiography for suspected coronary artery disease were prospectively studied. Angiograms were read by experienced invasive cardiologists blinded to the results of H Pylori serology, which was determined by a multiwell ELISAs assay. Patients with occlusive coronary artery disease were assessed with normal chest x-rays (controls) and sub-group analysis was performed in patients with single vessel, two-vessel and three vessel disease. The study was designed to have the statistical power to detect a 20% difference with a power of 90% at p = 0.05. Results: Mean ± SEM, chi square test or Fisher’s exact test for differences between groups. The age of the study population ranged from 37–91 years (Mean = 62 ± 1). 69 patients were sero-positive (46%). Of 31 patients with normal coronary arteries, 17 (55%) were sero-negative and 14 (45%) were sero-positive. Of 119 patients with abnormal coronary arteries, 54 (46%) were sero-positive (46%; P = 0.09). There were no significant differences between patients with mild moderate and severe disease. 11 of 29 patients (38%) with single vessel disease were sero-positive. The odds ratios for H. pylori infection as a risk factor were not statistically significant: for the presence of coronary artery disease (1.04), for a previous myocardial infarction (1.04), for triple vessel disease (0.99), for severe disease (0.94). Correction for known risk factors did not alter the results. Conclusions: H. pylori infection rates are similar in patients with normal and abnormal coronary arteries, one, two or three vessel disease, mild and severe disease. Other factors similar in men were smoked to and with and without previous myocardial infarction. Our data argue against a causal role for H. pylori infection in ischemic heart disease.

1C:12 HELICOBACTER PYLORI AND HEART

M. Caselli 1, M. Kubbagej 2, M. Ruina 3, P. Gaudenzi 1, L. Trevisani 1, M.G. Marangon 1, H. Heras Saltve 1, G.F. Baggioni 2, V. Alvisi 1. 1 School of Gastroenterology, University of Ferrara, Italy; 2 Institute of Cardiology, Hospital of Comacchio, Italy

H. pylori infection has recently been associated with an increased risk of coronary heart disease (CHD). We studied 83 Italian patients with CHD and 26 control patients matched for sex and age. We enrolled 13 patients with myocardial infarction as evaluated by ECG, typical enzimatic increase and echocardiography, and 70 ischemic patients as evaluated by coronary angiography in presence of a stenosis of more than 70% in at least one coronary artery. Exclusion criteria comprised presence of gastritis, peptic ulcer or positive history for dyspeptic symptoms as well. A seropositivity anti-H. pylori by using the Helori-test IgG kit (Eurospol S.p.A.) was detected in 73 (87.9%) of the ischemic patients in comparison with 12 patients (46.1%) in the control group (p < 0.002). We do not find any significant difference within the group of ischemic patients neither in relation to the sex or the type of pathology. Successively we have carried out a multivariated analysis in order to evaluate the relative role of different risk factors in our population: H. pylori increases the risk of heart disease 1.07 to 1.56, in the presence smoking from 0.50 to 2.20, familiarity from 0.47 to 1.57 and the social state from 0.11 to 3.73. A successfull multivariated analysis demonstrated that H. pylori plays a role indepedently from the other risk factors. We conclude that H. pylori should really represent a risk factor for CHD independent from the known ones.

1C:13 HELICOBACTER INFECTION AND THE RISK OF CORONARY OCCLUSION

A. Aromas 1, P. Knekt 1, A. Reunanen 1, H.J. Rautelin 1, T.U. Kosunen 1, 1National Public Health Institute and Social Insurance Institution, Helsinki, Finland; 2 University of Helsinki, Helsinki, Finland

Certain infections including those caused by helicobacters, may increase the risk of cardiovascular diseases. Our purpose was to analyse the association of helicobacter infection with myocardial infarction in the general population.

Methods: A health examination survey was carried out in 12 Finnish cohorts during 1973–76 with a follow-up until the end of 1985. 3471 men, aged 45–64 years, participated in the baseline survey. During the follow-up a new fatal or non-fatal myocardial infarction was observed in 276 subjects who were free of cardiovascular disease at baseline and in 165 subjects who had cardiovascular disease at baseline. A nested case-control study based on these cases and two matched controls formed. A total of 842 controls were matched for age, sex and municipality and cardiovascular disease at baseline. Serum samples taken at baseline were analyzed for Helicobacter IgG and IgA antibodies.

Results: 79% of the subjects with cardiovascular disease at baseline and myocardial infarction during the follow-up had elevated titers of helicobacter IgG antibodies, versus 77% in their control subjects, which resulted in a relative risk (RR) of 1.16; 95% confidence interval (CI) 0.73–1.86. Of those who initially did not have cardiovascular disease but had first myocardial infarction during the follow-up, 83% had IgG antibodies at the baseline, versus 78% in the control subjects; RR 1.37, (95% CI 0.94–2.00). Overall IgG positivity of the 441 subjects with myocardial infarction was 82%, versus 78% in the control subjects, RR 1.29 (95% CI 0.96–1.73). The respective figures for IgA antibodies were 68% and 67%, RR 1.05 (95% CI 0.71–1.54).

Conclusion: The present data indicate a possible although statistically nonsignificant association between the risk of myocardial infarction and...
helicobacter infection as measured by IgG positivity. However, this possible risk seems to be small.

**1C:14 HELICOBACTER INFECTION IN DEVELOPING ATROPHIC GASTRITIS AMONG SMOKING MEN**
T.U. Kosunen1, P. Sipponen2, K. Varis1, I.H. Rautein1, J. Virtamo3, M. Härkönen1, O.P. Heinonen1, 1 University of Helsinki, 2 Jori Hospital, Expp, 3 National Public Health Institute, Helsinki, Finland

Active chronic gastritis, known to be caused by helicobacter infection, is followed by atrophic gastritis after a mean of 20 years. Low serum pepsinogen I (PG I) level indicates atrophy in the body of stomach. Our purpose was to study the rate of the association of helicobacter infection with the development of atrophic gastritis.

Methods. Serum samples from 475 smoking men with different levels of PG I at three year intervals were tested for H. pylori IgG and IgA antibodies by enzyme immunoassay. Endoscopies with histological examination of the biopsies were carried out after the second sample on subjects with gastric complaints and those with low PG I.

Results. Helicobacter antibodies were most common in men with low PG I in second or both samples (95% and 94% respectively).

Antibodies were less common in men with persistent normal PG I (79% in men with gastric symptoms and 66% in men selected randomly).

IgA antibodies were most common (77%) in men whose pepsinogen I fell below 25 ug/l during the observation period. IgG and IgA antibody titers fell most frequently in men with low PG I level at the 3-year follow-up and only 10% of them were bacteria positive histologically.

Conclusions. Development of atrophic gastritis is regularly associated with helicobacter infection. The progress of gastritis leads first to the disappearance of detectable bacteria, then to decreasing antibody titers and finally to seronegativity.

**1C:15 PREVALENCE AND CLINICAL SIGNIFICANCE OF H. PYLORI INFECTION IN ELDERLY POPULATION**
T.E. Strandberg, R.S. Tilvis, M. Vuoristo, T.U. Kosunen. University of Helsinki, Helsinki, Finland

Our purpose was to study the association of H. pylori infection with common disease states in randomized age cohorts of elderly subjects of the Helsinki Ageing Study.

Methods. Helsinki Ageing Study is a comprehensive health examination of age cohorts of 75, 80 and 85 years. In 1989-90 456 females and 168 males were examined in general health care using questionnaires for symptoms, general diseases (like diabetes, cancer and cardiovascular diseases), use of drugs, interviews, physician’s examination and selected laboratory investigations. 624 subjects were studied for helicobacter IgG, IgA and IgM antibodies. 69 subjects were endoscoped for epigastric symptoms.

Results. H. pylori antibodies were found in 68-70% of the cohorts without differences between sexes. Seropositivity was not associated with common disease states, self-reported history of ulcer disease or the use of non-steroidal anti-inflammatory drugs. Histology was normal in nine subjects; all free of H. pylori infection. Chronic gastritis was found in 87% of those endoscoped; these included all 20 with verified (serology, histology or culture) infection but also 39 without evidence of the infection. A subject was found to have gastric cancer, but the biopsy was not evaluated for gastritis and bacteria; the subject was seropositive.

Conclusions. Helicobacter infection was very common in elderly general population of Helsinki but in this population it was not associated with an increased risk for any common disease. Chronic gastritis was found in many elderly subjects without detectable H. pylori infection.

**1C:16 H. PYLORI INFECTION AND CARDIOVASCULAR DISEASES IN ELDERLY POPULATION**
T.E. Strandberg, R.S. Tilvis, M. Vuoristo, T.U. Kosunen. University of Helsinki, Helsinki, Finland

According to some reports, certain infections including those caused by helicobacters, may increase the risk of cardiovascular diseases. Our purpose was to examine the association between H. pylori infection and cardiovascular diseases in a cross-sectional, prospective, population-based study in elderly subjects.

Methods. Serla from 624 female and male citizens of Helsinki, aged 75, 80 and 85 years were studied for H. pylori IgG, IgA and IgM antibodies at the baseline. All subjects were followed up for five years for patient histories, clinical manifestations, and electrocardiographic or echocardiographic changes related to cardiovascular diseases. Data on total and cardiovascular mortality were based on death certificates.

Results. H. pylori antibodies were found in 68% of the subjects. Seropositivity was not associated with the occurrence of electrocardiographic or echocardiographic changes and it was not related to well established cardiovascular risk factors except high density lipoprotein cholesterol, which was lower in seropositive subjects. Seropositivity was not related to total or cardiovascular mortality.

Conclusions. Helicobacter antibodies were very common in elderly general population of Helsinki but in this population they were not associated with an increased risk for cardiovascular diseases and mortality.

**1C:17 LACK OF ASSOCIATION BETWEEN HELICOBACTER PYLORI INFECTION AND ANGIOGRAPHICALLY DOCUMENTED CORONARY HEART DISEASE**

Background: Recent data have linked chronic infection with Helicobacter pylori (HP) to coronary heart disease (CHD). Since symptoms like angina, palpitation or chest pain as well as non-invasive testing for CHD and in 68% of the subjects. Seropositivity was not associated with any of the subjects. Seropositivity was not associated with any of the etiologic groups of CU: food allergy (FA) (n = 18), false food allergy (FFA) (n = 37), physical urticaria (PU) (n = 9), abnormal microbial hypersensitivity (MH) (n = 5), general diseases (GD) (n = 6), autoimmune diseases (AD) (n = 13) and idiopathic urticaria (IU) (n = 16). Hp infection was established by gastric biopsies (urease test, Giemsa staining, culture) in 70 patients and/or by serology (Pyloristat, BioWhitaker) in 53 subjects. Atopy was diagnosed by positive skin prick-tests and total serum IgE level was measured. After age adjustment there was no significant association of Hp infection prevalence with any of the etiologic groups of CU: total: 36%; FA: 11%; FFA: 46%; PU: 33%; MH: 20%; GD: 67%; AD: 31%; IU: 38% (p = 0.23). Hp infection was not correlated with serum IgE levels, and atopy was correlated with the absence of Hp infection (p = 0.006).

Conclusions. The results of our study clearly show that Hp infection is hypotized by others.

**1C:18 CHRONIC URTICARIA AND HELICOBACTER PYLORI INFECTION**
M. Morisset1, J.D. de Korwin1, A. Lozinski2, G. Kanny1, P. Pienat3, D.A. Moneret-Vautrin4, 1 Service de Médecine D, 2 Labaratoire de Bactériologie, 3 Laboratoire d’Anatomie Pathologique, Hôpital Central, CHU, 54035 Nancy Cedex, France

The role of gastric pathology in chronic urticaria (CU) has been suspected since the beginning of the century. Recently, the role of Helicobacter pylori (Hp) was pointed out in a few cases. The aim of this retrospective study was to determine the prevalence of Hp infection in 104 patients (mean age: 37 years) with CU. Seven groups of patients were defined according to the etiology of CU: food allergy (FA) (n = 18), false food allergy (FFA) (n = 37), physical urticaria (PU) (n = 9), abnormal microbial hypersensitivity (MH) (n = 5), general diseases (GD) (n = 6), autoimmune diseases (AD) (n = 13) and idiopathic urticaria (IU) (n = 16). Hp infection was established by gastric biopsies (urease test, Giemsa staining, culture) in 70 patients and/or by serology (Pyloristat, BioWhitaker) in 53 subjects. Atopy was diagnosed by positive skin prick-tests and total serum IgE level was measured. After age adjustment there was no significant association of Hp infection prevalence with any of the etiologic groups of CU: total: 36%; FA: 11%; FFA: 46%; PU: 33%; MH: 20%; GD: 67%; AD: 31%; IU: 38% (p = 0.23). Hp infection was not correlated with serum IgE levels, and atopy was correlated with the absence of Hp infection (p = 0.006).

Conclusions. The results of our study clearly show that Hp infection is hypotized by others.
1C: Non-gastric diseases related to H. pylori infections

1C:19 H. PYLORI (HP) INFECTION AND TRANSMISINASES
N. Figura, C. Gennari. Inst. of Internal Medicine, University of Siena, Siena, Italy

The production of granulating toxin (GT) by Helicobacter hepaticus has been associated with the development of hepatic and hepatic tumours in mice. HP also produces GT, in addition to the vacuolating toxin called VacA which is mostly expressed together with the cytotoxin-associated protein CagA. However, no association has yet been made between HP infection and liver damage. Patients and Methods. Levels of glutamic oxaloacetic and glutamic pyruvic transaminases (GOT, GPT) in serum samples of 38 HP positive adult patients were retrospectively compared with those of 23 HP negative age-matched controls. HP positive patients were tested for serological CagA antibodies and HP by Western blotting. All patients had a negative history of liver and muscular diseases, were negative for markers of virus hepatitis A, B, and C, and did not take hepatotoxic drugs. Results. Mean levels ± SD (IU/L) of serum GOT and GPT were 22.61 ± 9.54 and 21.82 ± 11.09 in infected patients, and 23.57 ± 14.90 and 19.78 ± 10.57 in uninfected patients (non significant). GOT and GPT levels in the 19 CagA+ infected patients were 25.63 ± 11.75 and 24.37 ± 13.35 versus 19.58 ± 5.44 (P = 0.049, significant) and 19.26 ± 7.81 (P = 0.158, non-significant) in the 19 CagA− infected patients. Conclusions. GT, if produced in vivo, does not seem to injure the liver. Persons with CagA+ HP infection might have a mild degree of liver damage. The HP vacuolating toxin, should it reach the liver, could alter cell permeability and/or influence cell growth factors and injure the hepatocytes. Increased GOT levels, however, could also derive from gastric epithelial cells which in CagA+ patients seem more damaged.

1C:20 FOOD ALLERGY AND H. PYLORI INFECTION
N. Figura 1, A. Perrone 2, C. Gennari 1, M. Vagliasindi 1, P. Rottoli 2, 1 Int. Medicine Insts., Siena Univ., Italy; 2 Resp. Diseases Insts., Siena Univ., Italy

Most antigens get to the immune system through mucosa. Gastrointestinal mucosa is a barrier for alimentary antigens. Inflammatory processes could alter the integrity of the barrier, increase the permeability of mucosa, and enhance crossing of food antigens. Many H. pylori (HP) infected persons test positive for basophilic-bound and serum specific IgE. As HP can also induce IgE-mediated histamine release from human basophils in vitro and mucosal IgE positive mast cells are involved in food allergy (FA), we investigated whether HP infection could be associated with FA. 38 adult patients with symptomatic FA and 53 age-matched controls (31 patients with respiratory allergy and 22 healthy subjects) were studied. Allergies were determined by titrating IgE to alimentary or respiratory antigens using Pharmacia CAP System RAST FELIA. HP infection was diagnosed serologically by an ELISA. Anti-CagA antibodies were investigated in HP positive persons by Western blotting. Results:

<table>
<thead>
<tr>
<th>Patient group</th>
<th>HP positive</th>
<th>CagA positive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Food allergy</td>
<td>42.1%</td>
<td>62.5%</td>
</tr>
<tr>
<td>Respiratory allergy</td>
<td>45.1%</td>
<td>28.5%</td>
</tr>
<tr>
<td>No allergy</td>
<td>47.8%</td>
<td>27.2%</td>
</tr>
</tbody>
</table>

CagA seropositivity in FA infected patients was significantly more prevalent than in infected controls (P = 0.030, Mantel-Haenszel test). We postulate that serious inflammatory gastric mucosal lesions could enhance the epithelial permeability and render non-selective the passage of allergens which, in atopic persons, could directly stimulate an IgE response. CagA+ HP infection could trigger or worsen FA.

1C:21 GALLSTONES AND H. PYLORI (HP) INFECTION
N. Figura 1, D. Vaira 2, R. Vermill 3, J.E. Crabtree 3, F. Bazzoli 2, 1 Univ. of Siena; 2 Univ. of Bologna, Italy; 3 Univ. of Leeds, UK

Gallstones consist of cholesterol crystals and calcium salts. Several proteins, including immunoglobulins (IgG), have been reported to have cholesterol crystallisation-promoting activity. In addition, accumulation of bacterial products in bile can be associated with hydrolysis of bilirubin conjugates and precipitation of insoluble cholesterol. HP infection and gallstones are conditions very common and the prevalence of both increases with age. Their possible association has never been considered. IgG and IgA to HP were investigated by Western blotting (WB) in serum samples and in bile samples obtained at cholecystectomy from 15 patients with gallstones disease (GD) at 1:200 and 1:50 dilutions. The presence of HP antigens in bile was investigated by WB: proteins were separated by electrophoresis in polyacrylamide gel and transferred onto nitrocellulose which was blotted with antibodies to CagA, urease, and whole HP antigens (provided by Bionico, Siena). Ten patients were seropositive for HP infection; 7 patients also had serum antibodies to CagA. Nine bile samples from infected patients tested positive for anti-HP IgA. No bile sample from uninfected patients contained anti-HP IgA. Immune reactivity to HP antigens of IgA in bile and IgG in serum was similar. Two bile samples from CagA seropositive patients contained anti-antigen of 60 kDa ca. which immunoreacted solely with the anti-CagA antibody.

Conclusions. Bile samples from HP seropositive GD patients contain HP antigens and related antibodies. Biochemical characterisation and evaluation of potential nucleating (or antinucleating) activity of these proteins may help in understanding if HP infection has a role in the pathogenesis of GD.

1C:22 H. PYLORI (HP) INFECTION AND THYROID DISEASES
N. Figura, E. Guarino, A. Gragnoli, G. Di Cairano, F. Lorè, D. Cataldo, C. Gennari. Institute of Internal Medicine, University of Siena, Siena, Italy

Thyroid diseases (TD) are frequently associated with the production of autoantibodies to components or products of the thyroid gland like colloid and peroxidase. As HP infection can stimulate an autoimmune response, we investigated the possible role of HP infection and of infection by HP strains which express the cytotoxin-associated protein CagA in the development of TD. A group of 46 women with TD and autoantibodies to thyroglobulin (mean age 48.81 ± 19.92 years) and 35 control women (mean age 48.17 ± 16.06) were studied. Serum samples were diluted 1:200 and blotted with a HP cell suspension (previously denatured in Laermilni) run electrophoretically in a polyacrylamide gel with sodium dodecyl sulphate, and transferred onto nitrocellulose. Tests were considered positive for the presence of 5 or more bands of reaction. Results. Thirty-four TD patients (68.7%) tested seropositive for HP infection vs. 16 controls (48.4%; P = 0.043, Mantel-Haenszel test). The prevalence of IgG to HP CagA, ureB, and HP in infected patients with TD was 70.5%, 58.8%, and 55.8% vs. 50.3% (P = 0.16), 50.0% and 31.2% (P = 0.107) in infected controls. Levels of autoantibodies to thyroglobulin in HP positive and negative TD patients were similar (P = 0.747), as well levels of TSH (P = 0.950) and FT3 (P = 0.751). Conclusions. The association observed between TD and HP infection deserves further investigations. Comparisons between the levels of autoantibodies to thyroglobulin before and after bacteriological eradication could help to assess a possible role of HP infection in thyroid diseases.

1C:23 IS HELICOBACTER PYLORI (HP) INFECTION ASSOCIATED WITH A TENDENCY TOWARDS A PROCOAGULANT STATE IN HEALTHY INDIVIDUALS?
F. Parente, V. Imbesi, G. Maconi, E. Rossi 1, G. Bianchi Porro. Department of Gastroenterology, 1 Department of Haematology, L. Sacco University Hospital, Milan, Italy

The influence of HP on circulating levels of coagulation factors related to ischaemic heart disease (IHD) remains uncertain. The aim of this study was, therefore, to investigate the possibility for HP to induce a tendency towards a procoagulant state, thereby influencing the risk of IHD. 368 asymptomatic blood donor were initially admitted to the study. Exclusion criteria included: age > 51 years, recent intake of any compound capable of interfering with blood coagulation, use of oral contraceptives, previous HP eradication therapy, diagnosis of IHD, peptic ulcer or any systemic chronic illness. 300 subjects (259 males), aged 20-51 years (mean 34.7) fulfilled the above criteria and were enrolled into the study. They were questioned about past medical history, diet, alcohol and cigarette consumption, education and socio-economic status. Their blood was analyzed for total and HDL cholesterol, ESR, CR reactive protein, fibrinogen, PT, FTT, factor VII:C, haemoglobin, platelet and leucocyte count. In addition, prothrombin cleavage fragment (FI-2), which is an index of prothrombin activation, was assayed. HP status was then determined in all subjects by means of a specific IgG antibodies assay. The overall prevalence of HP infection was 53.5% (158/300); as expected, HP-positive patients were significantly older (37.0 ± 7.9) than HP-negative group (32.2 ± 7.4). The two groups did not significantly differ as for alcohol and cigarette consumption, total and HDL cholesterol, plasma fibrinogen, total leucocyte and platelet count, PT and PTT, ESR and C-reactive protein. In contrast, FVII:C and FII + FV levels were significantly higher in HDL- and platelet-positive subjects (1.04 ± 0.4 vs 0.89 ± 0.3); however, after adjustment by multiple regression for age, sex, socio-economic status and smoking habits there was no longer any evidence of a significant association between HP and these factors. In conclusion, we found no evidence in healthy individuals to support the hypothesis that HP infection is associated with a tendency towards a procoagulant state.
1C:24 PREVALENCE OF H. PYLORI (HP) INFECTION AND HP-RELATED UPPER GI LESIONS IN PATIENTS WITH INFLAMMATORY BOWEL DISEASES (IBD). A CASE-CONTROL STUDY
F. Parente, P. Molteleni, S. Bollani, G. Maconi, B. Rembacken 1, A. Axon 1, G. Bianchi Porro, GI Units, L. Sacco Hospital, Milan, Italy; 2 LGI, Leeds, UK

Although a reduced prevalence of HP infection has been reported in IBD patients (pts), the clinical significance of HP infection in this setting remains unclear. The aim of this study was, therefore, to evaluate the prevalence of HP infection in a large series of IBD pts as well as the frequency of gastroduodenal lesions in those accepting to undergo upper GI endoscopy.

216 consecutive IBD pts (122 with Crohn’s disease (CD) and 94 with ulcerative colitis (UC)) had their anti-HP IgG titres measured using a commercial ELISA kit. 216 blood donors matched for age, sex, place of birth in Italy and socio-economic status served as controls. All pts were offered the possibility of undergoing upper GI endoscopy. The overall seroprevalence of HP infection was 48% in IBD pts vs 59% in the control group (p < 0.01), with a significantly lower frequency in CD vs UC pts (p < 0.01). After adjustment for age, education and socio-economic status CD remained associated with a significantly lower risk of HP infection. No association was observed between reduced HP seropositivity and previous use of NSAIDs, GA, steroids or azathioprine, whereas a weak relationship between previous antimicrobial use and reduced risk of HP infection was found. 179 pts (106 with CD and 73 with UC) underwent endoscopy; the prevalence of peptic ulcer was similar in both groups (4.7% in CD and 5.5% in UC pts), but 11 more CD pts had ulcers which were interpreted as CD-related. 7 of them had a history of forget complaints but the remaining had never suffered from dyspepsia. 64% of these CD-associated gastroduodenal ulcers were HP-ive. Our findings show that CD but not UC pts have a reduced prevalence of HP infection as compared to matched healthy controls. This is partly due to a greater use of antimicrobials. 10% of CD pts had also a gastroduodenal localisation of their disease, which is often asymptomatic. Most of these CD-associated ulcers are unrelated to HP infection.

1C:25 FIBRINOGEN AND H. PYLORI IN ASYMPTOMATIC POST MI PATIENTS AND HEALTHY CONTROLS
J. Rajput-Williams 1, N.R. Williams 1, P.G. Johnson 2, R.J. Dickinson 3, 1 Papworth Hospital, Cambridge, UK; 2 BSIA, Belford, UK; 3 Hitchingbrooke Hospital, Huntingdon, UK

A link between H. pylori seropositivity and coronary heart disease via plasma fibrinogen has been suggested, although this has been disputed. Fibrinogen (and Factor VII) were measured in non-smoking men who recently had a myocardial infarction (MI: n = 35) and healthy controls (n = 27) with known H. pylori status (assessed by carbon-13 urea breath test, BSIA, UK). Results are presented in the Table.

<table>
<thead>
<tr>
<th>H. pylori</th>
<th>Fibrinogen (g/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>20 (2.68-3.8)</td>
</tr>
<tr>
<td>Positive</td>
<td>3.07 (2.83-3.30)</td>
</tr>
<tr>
<td>Post-MI</td>
<td>3.04 (2.82-3.26)</td>
</tr>
<tr>
<td>Post-MI</td>
<td>3.26 (2.64-3.89)</td>
</tr>
</tbody>
</table>

Conclusions: HP infection is significantly lower in patients with RE than in controls. The pathogenesis of RE may be related to low prevalence of atrophic gastritis (preservation of acid secretion) in this patient group.

1C:27 LOW PREVALENCE OF HELICOBACTER PYLORI INFECTION IN PATIENTS WITH REFLUX ESOPHAGITIS
M. Mihara, K. Haruna, T. Kamada, K. Kiyohira, T. Goto, M. Sumii, S. Tanaka, M. Yoshikura, K. Sumii, G. Kajiyama. First Dept. of Internal Medicine, Hiroshima University School of Medicine, Hiroshima, Japan

Recent studies have shown the close association of H. pylori (HP) infection with gastritis or peptic ulcer diseases. There is little information concerning the relationship HP infection and reflux esophagitis (RE). The aim of this study is to investigate the prevalence of HP in patients with RE, along with histologic gastritis. Methods: The study population consisted of 70 patients with RE and 70 age- and sex-matched controls who have no localization lesions in the upper GI tract. HP infection was evaluated by Giemsa stain on antral and fundic biopsies and serum IgG antibody. Gastritis and atrophy were scored as follows: normal = 0, mild = 1, moderate = 2 and severe = 3. Serum pepsinogens (PGs) were determined by RIA.

Results: Mean ± SE, p < 0.05, ** p < 0.01

Conclusions: HP infection is significantly lower in patients with RE than in controls. The pathogenesis of RE may be related to low prevalence of atrophic gastritis (preservation of acid secretion) in this patient group.

1C:28 PREVALENCE OF H. PYLORI (HP) INFECTION IN INFLAMMATORY BOWEL DISEASES (IBD). A CONTROLLED STUDY

Objectives: To evaluate HP prevalence in patients (p.) with IBD, submitted to several therapeutic regimens. Material & Methods: 50 p. were studied prospectively. 26 with Crohn Disease (CD), median age = 44.82 ± 15.72; 24 with Ulcerative Colitis (UC), median age = 44.52 ± 12.84. Results were compared to those obtained in 50 healthy volunteers studied as control group (CG). These groups were matched by age (p < 0.0042 between CD and UC groups and p < 0.05 between the two pathological groups and CG. HP was evaluated as follows: 1) Urea 14C Breath Test (n = 43: p > 0.50 CG); 2) Urea & Histology of gastric mucosal in diseptic p. (n = 11). Statistical analysis was carried out by χ², Kendall Coefficient of Concordance and Spearman Rank Order Correlation. Results: Prevalence of HP was as follows: IBD group = 58% (CD = 42% and UC = 75% with p = 0.01) and CG = 62%. A significant decrease of Hp+ prevalence was verified in CD p. treated with Metronidazol (MTN) (40%, p < 0.02) and in those submitted to surgery 19%, p = 0.008, also dominantly (87.5% of operated p.) included in the CD population. Duration, extension, clinical activity or other therapies (sulphasalazine, mesalazine, steroids and azathioprine) were not correlated to the HP status in the IBD group. Conclusions: 1. Prevalence of Hp infection was similar in IBD population and control group. A significant decrease was shown in CD when compared to UC group. 2. Low prevalence of Hp+ in the CD group was associated with previous surgery and with present or previous MTN therapy. Almost
all patients submitted to surgery had medical intractability after being submitted to intensive drug treatment.

### 1C:29 DOES *HELICOBACTER PYLORI* AFFECT THE BLOOD AMMONIA LEVEL OF HEMODIALYSIS PATIENTS?

Z. Fireman, D. Coscas, A. Sternberg, Y. Wagner, H. Zonder, Hillel Yaffe Medical Center, Hadera, Israel

Hyperammonemia, alone, causes mental state changes and, in combination with other substances, can lead to a catatonic state. Helicobacter pylori (HP) through its urease activity produces ammonia from urea. The hypothesis is that urea, which is found in large quantities in the gastric lumen of uremic patients, is transformed into ammonia, by the HP urease activity which is transmitted to the blood, contributing to mental state changes in these patients.

The blood ammonia levels of HP positive uremic patients were compared with those of HP negative patients. Sixteen patients on chronic hemodialysis were evaluated. The examinations were carried out prior to the hemodialysis sessions. Nine patients were found to be HP(+) and seven HP(−).

<table>
<thead>
<tr>
<th>No. of pts</th>
<th>Stomach</th>
<th>Blood</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>urea (µg/dL)</td>
<td>ammonia (µg/dL)</td>
</tr>
<tr>
<td>HP+</td>
<td>9</td>
<td>33.1</td>
</tr>
<tr>
<td>HP−</td>
<td>7</td>
<td>26.5</td>
</tr>
<tr>
<td>P value</td>
<td>0.499</td>
<td>0.5375</td>
</tr>
</tbody>
</table>

**Conclusion:**
1. No difference was found between the blood ammonia levels in HP positive and HP negative patients.
2. Although ammonia is elevated in the gastric lumen of both HP(+) and HP(−) patients, its blood level is normal.
3. Hyperammonemia is probably not a contributing factor to mental changes in uremic patients.
4. There is no clear-cut explanation for the elevated gastric ammonia level in HP negative patients.

### 1C:30 HIGH PREVALENCE OF *HELICOBACTER PYLORI* INFECTION IN CORONARY HEART DISEASE DEMONSTRATED BY THE 13C-UREA BREATH TEST

C. Martín de Argila, D. Boixeda, A. Fuertes, R. Cantón, M. Barba, C. Arocena, J.P. Gisbert. García Plaza, "Ramón y Cajal" Hospital, Madrid, Spain

Previous studies have reported an association between coronary heart disease (CHD) and *H. pylori* infection, but all of them have used serological tests to confirm infection.

**Aim:** To determine the prevalence of *H. pylori* infection in a large group of patients with CHD and its relationship with different conventional CHD risk factors.

**Materials and Methods:** One hundred and twelve consecutive patients (95 males and 17 females; mean age: 59.1 ± 11.9 yrs) were studied with documented CHD admitted at the Coronary Care Unit in our Hospital. Patients with previous history of peptic ulcer disease or digestive conditions were excluded. Information was inquired on the presence of conventional risk factors for cardiovascular disease (diabetes, hyperlipidemia, smoking, and arterial hypertension). Eighty-three healthy persons (24 males and 59 females; mean age: 51.5 ± 10.7 yrs) comprised the control group. IgG antibodies to *H. pylori* were measured in all persons by means of a serological ELISA method (Helico-G, Porton, Cambridge, UK). All persons with CHD underwent also a 13C-Urea breath test (13C-UBT) to study *H. pylori* infection.

**Results:** Ninety-one (81.3%) of 112 patients with CHD had a positive serology for *H. pylori* (> 10 U/ml) compared with 53 (63.8%) persons out of the 83 in the control group (p < 0.01). In 90/112 (80.4%) of patients with CHD the 13C-UBT was positive (ΔCO2 > 5/1000). No association was observed between *H. pylori* infection and the different risk factors for CHD.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Yes H. pylori +</th>
<th>H. pylori −</th>
<th>No H. pylori +</th>
<th>H. pylori −</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking</td>
<td>43 (82.7)</td>
<td>9 (17.3)</td>
<td>47 (78.3)</td>
<td>13 (21.7)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>33 (76.7)</td>
<td>10 (23.3)</td>
<td>57 (82.6)</td>
<td>12 (17.4)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>19 (88.6)</td>
<td>2 (11.4)</td>
<td>71 (79.8)</td>
<td>18 (20.2)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>40 (76.9)</td>
<td>12 (23)</td>
<td>50 (83.3)</td>
<td>10 (16.7)</td>
</tr>
</tbody>
</table>

*p > 0.05 in all comparisons between the different groups.

**Conclusions:** Patients with CHD had a high prevalence of *H. pylori* infection, significantly higher than that observed among healthy persons. The absence of an association between conventional risk factors for CHD and *H. pylori* infection suggests an independent action of *H. pylori* in its possible involvement in CHD.