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SPECTRUM OF HELICOBACTER PYLORI GASTRITIS IN DUODENAL ULCER PATIENTS FROM DIFFERENT COUNTRIES. IE Gurer, DY Graham, H El-Zimaity, R.M. Genta, VAMC, Baylor College of Medicine, Houston, Texas

Background: It is commonly accepted that some types of gastritis are more prevalent in certain regions of the world than in others. However, histopathologic studies performed in different areas may not be comparable, because many features of gastritis lack well defined reproducible criteria.

Objectives: To have the same group of pathologists compare type, intensity and topographic distribution of *H. pylori*-gastritis associated with duodenal ulcer in several groups of young patients from different areas of the world.

Methods: Patients aged 20 to 40 with documented duodenal ulcer were recruited in institutions in South Africa, Jordan, Korea, India, and United States. From each patient 6 to 8 biopsy specimens were obtained according to our mapping protocol, sent to our laboratory in Houston where they were stained with the Genta stain. The following features were evaluated using a 6-point visual analog scale: density of *H. pylori*, neutrophils, mononuclear cells, atrophy, and intestinal metaplasia. All specimens with intestinal metaplasia were subsequently stained with an Alcian blue/High Iron Diamine stain.

Results: The prevalence of intestinal metaplasia in these patients is presented in the following table:

| COUNTRY | Number of Subjects | Subjects with Intestinal Metaplasia | % |
|----------------|--------------------|-------------------------------------|----|
| U.S.A. (Texas) | 44 | 11 | 25 |
| South Africa | 22 | 7 | 32 |
| Jordan | 28 | 9 | 32 |
| India | 18 | 5 | 28 |
| Korea | 132 | 76 | 57 |

Conclusions: The results of this study suggest that even an apparently universal manifestation of *H. pylori* infection such as duodenal ulcer may be accompanied by a wide variety of expressions of gastritis. It is also interesting to note that the only group of patients with a significantly higher prevalence of intestinal metaplasia were patients from Korea, a country where the incidence of gastric cancer is much higher than in any of the other four populations included in the study.

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H. PYLORI, GASTRITIS AND SERUM PEPSINOGEN A IN A MALE NON-PATIENT POPULATION. J I Wyatt, T Knight, A Wilson, S Greaves, D Newell, K Hegels, M Corlett, D Forman, J Elder. Dept of Pathology, St James's Hospital, Leeds, UK, and other contributors in the Stoke Stomach Project, Stoke on Trent, UK.

Aim - to explore the prevalence and pattern of gastritis in asymptomatic male volunteers from a UK area with relatively high gastric cancer prevalence.

Methods As part of the 'Stoke Stomach Project' we determined serum pepsinogen A (PGA) levels and *H. pylori* serology in 505 male volunteers age 18-63 yrs. A 10% subsample, representing the range of observed PGA levels underwent endoscopy and biopsy with gastritis graded by the Sydney System.

Results 187/505 (37%) subjects were seropositive for *H. pylori*; the mean PGA was 84.5 ng/ml in the seropositives and 63ng/ml in seronegatives. Of the 29 seropositives endoscoped, 26 had gastritis and 3 had normal histology and were *H. pylori* negative (serological false positives) as were all 25 seronegative subjects. Nine subjects had antrum or antrum predominant gastritis, all with PGA >100ng/ml one with DU. Ten had pangastritis, which was mild in 7 with PGA >90ng/ml (one showed mild corpus atrophy) and moderate in 3 with PGA <80ng/ml, all with some corpus atrophy. Seven had corpus predominant gastritis with corpus atrophy, all with PGA <50ng/ml. Results for serum gastrin, pepsinogen C, and gastric juice pH supported the histological assessment of corpus atrophy.

Conclusions *H. pylori* gastritis is associated with elevated PGA unless this effect is counteracted by corpus atrophy. By extrapolating the subgroup results to the whole group we would estimate about half the 187 seropositives to have gastritis with some corpus atrophy, and about 14% corpus predominant gastritis.

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THE MACH 1 STUDY: OPTIMAL ONE-WEEK TREATMENT FOR H. PYLORI DEFINED?

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We define optimal *Helicobacter pylori* treatment as eradication rate > 90%, easy to take and with few side effects.

Method: International multi-centre, double-blind randomised placebo controlled study including 787 patients with proven duodenal ulcer disease either active or in remission. All patients received omeprazole 20mg (O) twice daily in combination with either placebo (P) or two of the following antimicrobials twice daily: Metronidazole 400 mg (M), Amoxicillin 1000 mg (A), Clarithromycin 250 or 500 mg (C250, C500) to eradicate *H. pylori*. Treatments were given one week. *H. pylori* status was assessed by ¹³C-urea breath test (UBT) before and four weeks after cessation of therapy.

Results: The following patients were excluded: 48 *H. pylori* negative and 16 UBT test failures at entry, 12 adverse event discontinuations and 27 miscellaneous.

The eradication rates and 95% CI according to the APT analysis:

| Treatment | No of pat | Eradication | 95% CI |
|-----------|-----------|-------------|--------|
| OAC500 | 106/110 | 96% | 93-100 |
| OMC250 | 105/111 | 95% | 90-99 |
| OMC500 | 106/118 | 90% | 84-95 |
| OAC250 | 93/111 | 84% | 77-91 |
| OAM | 94/119 | 79% | 72-86 |
| OP | 1/115 | 1% | 0-3 |

All treatments were well tolerated. The most common side effects were diarrhea and taste disturbances. Diarrhea seemed to be more related to A and taste disturbances more related to C and M. Only twelve discontinued due to adverse events.

Conclusion: Two of the tested combinations fulfilled our criteria for optimal treatment of *H. pylori*.

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CLARITHROMYCIN (CL) IN COMBINATION WITH OMEPRAZOLE (OM) FOR HEALING OF DUODENAL ULCERS (DU), PREVENTION OF DU RECURRENCE, AND ERADICATION OF H. PYLORI (HP) IN TWO EUROPEAN STUDIES. C. O'Morain, R.P.H. Logan and the Clarithromycin European *H. pylori* Study Group. Meath Hospital, Dublin, Ire, BHURG study, St. Mary's Hospital, London, UK

Patients with HP and DU were enrolled in two well-controlled, randomized, double-blind, multi-center studies. Patients received for two weeks either CL 500 mg TID and OM 40 mg QD or OM 40 mg QD alone; all patients received an additional two weeks of OM (40 mg QD in one study and 20 mg QD in the other). Patients were followed for 6 months. Ulcer status was assessed by endoscopy and HP status was assessed by culture, histology, and ¹³C-UBT at 4-6 weeks post-Rx. 356 patients with DU and HP pretreatment (mean age 47 yrs, mean DU size 10 mm) were enrolled.

| Treatment | DU Healing post-Rx | Hp Eradication at 4-6 weeks post Rx | Ulcer Recurrence at 6 months post-Rx |
|-----------|--------------------|-------------------------------------|--------------------------------------|
| CL+OM | 99%(151/152) | 78% (126/162) | 8% (10/31) |
| OM | 97%(156/161) | 3% (5/171) | 51% (77/150) |

Table includes all patients with both DU and HP pretreatment who had the appropriate post-Rx visit.

5% (5/92) of Hp negative CL+OM patients and 13% (5/39) of Hp positive CL+OM patients had recurrence of ulcer while 53% of Hp positive OM patients had recurrence of ulcer at the end of the 6 months follow-up.

Both CL+OM and OM alone were well tolerated. Only 3% of CL+OM patients 2% of OM patients discontinued Rx due to adverse events.

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DUAL THERAPY OF CLARITHROMYCIN (CL) AND OMEPRAZOLE (OM) FOR TREATMENT OF PATIENTS WITH DUODENAL ULCERS (DU) ASSOCIATED WITH H. PYLORI (HP) INFECTION. R. Hunt, H. Schwartz, D. Fitch, R. Fedorak, F. Al Kawas, N. Vakli. Clarithromycin H. pylori Study Group.

Eradication of *H. pylori* with dual therapy (proton pump inhibitor plus antibiotic) is an effective, safe and simple regimen to prevent ulcer recurrence. **AIM:** The results of a well-controlled, randomized, double-blind, multi-center study to assess CL+OM versus OM and CL monotherapy in DU healing, recurrence, and Hp eradication are reported. **METHODS:** Patients with DU (>5mm) and Hp were enrolled and received either 1) CL 500 mg TID and OM 40 mg QD (2 weeks) followed by OM 20 mg QD (2 weeks), 2) CL 500 mg TID for 2 weeks, or 3) OM 40 mg QD (2 weeks) followed by OM 20 mg QD (2 weeks). Repeat endoscopy was done at the end of Rx, at 4-6 weeks, 3 and 6 months post-Rx or at symptomatic recurrence. DU status was assessed by endoscopy and Hp status was assessed by antral and corpus culture, histology, and by ¹³C-UBT. **RESULTS:** 219 patients (mean age 47 yrs, mean DU size 9 mm) were enrolled.

| Treatment | DU Healing post-Rx | Hp Eradication at 3 months post Rx | Ulcer Recurrence at 6 months post Rx | |
|-----------|--------------------|------------------------------------|--------------------------------------|-------------|
| | | | Hp - | Hp+ |
| CL+OM | 94% (60/63) | 72% (41/57) | 6% (2/35) | 57% (12/21) |
| OM | 88% (62/70) | 0% (0/44) | 0% (0/1) | 74% (42/57) |
| CL | 71% (49/69) | 40% (19/48) | 12% (2/17) | 36% (10/26) |

Table includes all patients with both DU and HP pretreatment who had the appropriate post-Rx visits.

CL+OM, OM alone, and CL alone were well tolerated. Only 2% of CL+OM patients, no OM patients, and 2% of the CL patients discontinued treatment due to adverse events. **CONCLUSION:** Dual therapy with CL+OM is an effective and safe treatment for cure of Hp and prevention of DU recurrence.

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RANITIDINE BISMUTH CITRATE PLUS CLARITHROMYCIN IS EFFECTIVE IN THE ERADICATION OF HELICOBACTER PYLORI AND PREVENTION OF DUODENAL ULCER RELAPSE. W.L. Peterson, S.J. Sontag, A.A. Ciociola, D.L. Sykes, D.J. McSorley, D.D. Webb and the H. pylori Ulcer Group. VA Medical Center, Dallas TX, VA Medical Center, Hines IL, Glaxo Inc., RTP NC.

Ranitidine bismuth citrate is a novel compound with antisecretory, cytoprotective and antihelicobacter properties. The purpose of this factorial-designed, multi center, randomized, double-blind, placebo-controlled study was to compare the combination of ranitidine bismuth citrate (RBC) and clarithromycin (CLAR) to RBC alone, and CLAR alone, in the eradication of *Helicobacter pylori* (Hp), healing of the duodenal ulcer (DU), and prevention of DU relapse. **Methods:** 136 Hp infected patients with an active DU were treated with either RBC 400mg b.i.d. for four weeks plus CLAR 500mg t.i.d. for two weeks, RBC 400mg b.i.d. for four weeks plus placebo (PLC) t.i.d. for two weeks, PLC b.i.d. for four weeks plus CLAR 500mg t.i.d. for two weeks, or PLC b.i.d. for four weeks plus PLC t.i.d. for two weeks. Only patients with baseline positive culture, or positive histology and rapid urease test (CLO) were considered Hp infected. Only patients with healed ulcers at the end of the four week treatment period were eligible to be entered into the six months post-treatment observation period, i.e., 84 of 136 patients. Of those 84 patients, 76 had an endoscopy performed at 1, 3 and 6 months post-treatment and were evaluated for ulcer relapse. Patients whose ulcers were unhealed at the end of the four-week treatment period or did not wish to continue were dropped from the study, and given alternative therapy, i.e., 52 of 136 patients. Hp was considered eradicated if at least two of three tests performed at four weeks post-treatment were negative. Any positive test classified the patient as infected. A single negative test classified the patient as unvaluable. A total of 68 patients were evaluable for Hp eradication.

Results:

| Treatment | Hp Eradication % (No. patients) | 6 Month Ulcer Relapse % (95CI) |
|----------------|---------------------------------|--------------------------------|
| RBC+CLAR | 82% (14/17)* | 20% (2.2-37.8) |
| RBC 400mg bid | 0% (0/20) | 84% (67.9-100) |
| CLAR 500mg tid | 36% (8/22) | 41% (21.4-60.6) |
| PLC | 0% (0/9) | 87 (62.9-111) |

*p < 0.009 as compared to others

Conclusion: RBC 400mg b.i.d. when taken with CLAR 500mg t.i.d. is highly effective therapy for eradication of Hp and ulcer disease.

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EFFECT OF OMEPRAZOLE AND CLARITHROMYCIN PLUS TINIDAZOLE ON THE ERADICATION OF HELICOBACTER PYLORI AND THE RECURRENCE OF DUODENAL ULCER.

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Although it is presently recognized that *Helicobacter pylori* (*H.p.*) infection is the main acquired factor in the pathogenesis of duodenal ulcer (DU) disease and that DU patients must be treated with antimicrobials in order to greatly reduce, or even abolish, the risk of recurrence, a consensus agreement for the optimal therapy (greatest efficacy with least untoward effects and lowest cost) has yet to be accomplished. We have recently reported (Gastroenterology, 1993; 104:A40) that in patients with *H.p.* gastritis, a short term and low dose therapy with clarithromycin (C.), omeprazole (O.) and tinidazole (T.) is highly effective for long term eradication and that absence of side effects and good compliance are likely to be major determinants of effectiveness.

Aim: in the present study we aimed to investigate the effect of combining one week administration of low dose C. plus T. with a conventional four week healing treatment with O. on the eradication of *H.p.* in DU patients and on the rate of recurrence of duodenal ulcer.

Methods: 171 patients (115 males; 56 females, mean age \pm SE 53.9 \pm 1.0 yrs) with active DU and *H.p.* infection received a 4 weeks administration of O. 20 mg u.l.d. and, during the first week, a combination antimicrobial treatment with C. 250 mg b.i.d. plus T. 500 mg b.i.d.. *H.p.* infection, as well as eradication or relapse, was established by urease test, histology and ¹³C-urea breath test (58 pts) or brush cytology (115 pts). Upper GI endoscopy with antral biopsies and brush cytology or ¹³C-urea breath test were performed prior to treatment and at month 1, 3, 6 and 12 after treatment withdrawal. Drug tolerability was evaluated by patient interview and compliance by pill counting.

Results: All, but one single patient who complained of nausea and vomiting, tolerated well and completed the treatment, and took more than 90% of the prescribed medication. At month 1 after the end of treatment, ulcers were healed in 167 patients and *H.p.* was eradicated in 158 (94.6%). Follow-up evaluations performed at month 3 (114 pts), 6 (74 pts) and 12 (48 pts) showed persistent eradication and no ulcer recurrence in all cases.

Conclusions: One week administration of clarithromycin 250 mg b.i.d. plus tinidazole 500 mg b.i.d. combined with four weeks administration of omeprazole 20 mg u.l.d., is highly effective for long term eradication of *H.p.* infection also in DU patients, and, in this group of patients, was able to fully abolish ulcer recurrence.

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THE TREATMENT OF DUODENAL ULCER WITH GR122311X (RANITIDINE BISMUTH CITRATE) AND CLARITHROMYCIN. K.D. Bardhan¹, C. Dallaire², H. Eisold³, A.E. Duggan⁴ ¹Rotherham District General Hospital, UK. ²St Françoise d'Assise Hospital, Quebec, Canada. ³Mössingen, Germany. ⁴Glaxo Research and Development Limited, UK.

Introduction: This double-blind, randomised, multicentre study compared the efficacy and safety of GR122311X (GR) 400mg bd monotherapy for 28 days (GR400) to treatment with GR 400mg bd or 800mg bd in co-prescription with clarithromycin 250mg qds for 14 days, followed by 14 days of GR 400mg bd monotherapy (GR400+CLAR and GR800+CLAR, respectively) in duodenal ulcer (DU) treatment. **Patients and Methods:** 232 patients with active DU (215 with confirmed *Helicobacter pylori* (*H.p.*) infection) entered the study. Patients with healed ulcers were followed for up to 6 months on no therapy with endoscopy at 1, 3 and 6 months. The primary efficacy assessment, overall success rate, was defined as the proportion of patients whose ulcers were healed and who remained ulcer-free during follow-up and was estimated by life-table analysis. *H.p.* was assessed by ¹³C-urea breath test (UBT) and antral and corpus CLOtest™ at each endoscopy. In a modified Intent-to-Treat analysis of patients with both CLOtest and UBT results, *H.p.* eradication was assumed if all tests were negative (4 biopsies and excess $\delta^{13}C_{O2} \leq 5$ per mil) >4 weeks after the end of treatment.

Results (Intent-to-Treat Analysis)

| | GR400 | GR400 + CLAR | GR800 + CLAR |
|--|-------|--------------|--------------|
|--|-------|--------------|--------------|

- | | | | |
|---|-----|------|------|
| • Numbers of patients with DU | 82 | 75 | 75 |
| • Overall success rates after 6m follow-up (life-table estimates) | 51% | 89%* | 87%* |
| • Healing rates at 4wk (LOCF)† | 83% | 89% | 93% |
| • Relapse rates after 6m (LOCF)† | 40% | 6%* | 9%* |
| • <i>H. pylori</i> eradication (observed) | 2% | 94%* | 84%* |
| • Patients with any adverse event | 29% | 28% | 25% |

*p<0.001 for comparison of monotherapy with each co-prescription regimen.

†LOCF = last observation carried forward

Conclusion The dual therapy was well tolerated and effective in healing of DU, eradication of *H. pylori* and prevention of DU recurrence for up to 6 months. It had significant advantages over monotherapy.

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AMOXICILLIN PLUS OMEPRAZOLE VERSUS TRIPLE THERAPY FOR THE ERADICATION OF *H. PYLORI* AND HEALING OF DUODENAL ULCERS. JY Sung, TKW Ling, R Suen, VKS Leung, EKW Ng, SCS Chung. Depts of Medicine and Surgery, Chinese University of Hong Kong. Hong Kong.

We have previously shown that one-week triple therapy (Bismuth, Tetracycline and Metronidazole) is effective in eradicating *H. pylori* (HP) infection and healing duodenal ulcers (DU) (Lancet 1994;343:513-7) but the side effects of triple therapy were significant. Other studies have shown that amoxicillin plus omeprazole is equally effective with fewer side effects.

Objective: To compare the anti-Helicobacter effect of one-week triple therapy (BTM) versus amoxicillin/omeprazole (Amox/Omp) in the treatment of HP associated DUs.

Patients & Method: Patients with endoscopically proven DU and HP infection confirmed by smear, culture and CLO test of biopsies were recruited. Patients were randomized to receive EITHER bismuth subcitrate 120mg, tetracycline 500mg and metronidazole 400mg all taken 4 times daily for one week (BTM) OR omeprazole 20mg and amoxicillin 1g twice daily for 2 weeks (Amox/Omp). Six weeks after randomization, endoscopy was repeated to document ulcer healing and eradication of HP. Biopsies were taken both in the antrum and the body of the stomach. HP eradication was defined as absence of the organism in BOTH locations.

Results:

| | BTM | Amox/Omp | P |
|----------------------------------|------------|------------|-------|
| Total No. of Patients | 46 | 42 | - |
| Patients defaulted followup | 4 | 3 | - |
| Patients violated protocol | 5 | 2 | - |
| Ulcer healing at 6 weeks (%) | 33/37 (89) | 31/37(84) | 0.49 |
| <i>H. pylori</i> eradication (%) | 32/37 (86) | 27/37 (73) | 0.15 |
| Side-effect (%) | 30/37 (81) | 17/37 (46) | <0.01 |

Conclusion: Amoxicillin plus omeprazole therapy for two weeks have achieved an ulcer healing effect and HP eradication rate comparable to the one-week therapy but with much fewer side effects.

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LONG-TERM EFFECT OF ANTI-HELICOBACTER PYLORI THERAPY ON GASTRIC MALT LYMPHOMA. HISTOLOGICAL AND MOLECULAR EVALUATION OF 15 CASES.

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Histological regression of gastric low grade MALT lymphoma (ML) after eradication of *Helicobacter pylori* (*H. pylori*) infection has been reported. To assess the long-term efficacy of the antibiotic therapy, fifteen patients (7 females, age 34-76) with a diagnosis of gastric ML stage IE associated to *H. pylori* infection underwent anti-*H. pylori* therapy and bioptic follow up for 11-44 months (mean follow up 23 months). At each sampling, histological evaluation and PCR for immunoglobulin heavy chain gene rearrangement were performed.

H. pylori was eradicated in 14 cases and histological remission was found in 13 cases 2 to 4 months after the eradicating therapy. All the 13 cases with histological regression of lymphoma are free of disease and reinfection 8-30 months after eradication of *H. pylori* (mean 18 months). Monoclonality was demonstrated in 10 of the 13 cases with histological remission. Disappearance of PCR detected amplification bands following eradication of *H. pylori* was demonstrated in 6 cases and was synchronous to histologic remission in 4 of them whereas monoclonality persisted for 9 and 24 months in absence of histological evidence of lymphoma in the remaining 2. Monoclonality persists 13-27 months after eradication and histological remission in 4 cases.

The eradication of *H. pylori* induced a quick and persistent histological remission in 93% of cases. The molecular regression of ML seems to be much slower than the histological one.

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LONG-TERM CLINICAL COURSE OF ULCER DISEASE AND INCIDENCE OF REFLUX ESOPHAGITIS IN A LARGE COHORT OF DUODENAL ULCER PATIENTS FOLLOWED AFTER ERADICATION OF HELICOBACTER PYLORI. JLabenz, B.Tillenburg, U.Peitz, M.Sollböhrmer, M.Stolte, G.Börsch. Department of Internal Medicine and Gastroenterology, Elisabeth Hospital Essen, Germany

Aim: The aim of the present study was to evaluate the long-term clinical course of formerly relapsing duodenal ulcer disease after cure of *H. pylori* infection.

Methods: 203 patients with endoscopically proven relapsing duodenal ulcer disease and without endoscopically visible signs of reflux esophagitis at the time of *H. pylori* eradication were prospectively followed for 1 to 5 years. During the follow-up without any antiulcer drugs, patients were reinvestigated clinically and endoscopically in one-year intervals and when dyspeptic symptoms recurred. During each endoscopy, the *H. pylori* status was assessed by means of an urease test, culture and histology.

Results: During follow-up of 346 patient years, 6 ulcer relapses were detected (rate: 1.7% per patient year; 95%-CI: 1%-4%), which were associated either with *H. pylori* recurrence (n=1), intake of Aspirin or NSAIDs (n=4), or cryptogenic liver cirrhosis (n=1). A total of 4 patients had *H. pylori* recrudescence or reinfection, respectively, during the first year after treatment and none relapsed later (rate: 1,2% per patient year; 95%-CI: 0%-3%). Twenty patients (rate: 9,9%; 95%-CI: 6%-15%) developed an endoscopically proven reflux esophagitis, which was mild (grade I or II) in 19 patients and severe in one female patient with stenosis of the duodenal bulb.

Conclusion: *H. pylori* eradication cures duodenal ulcer disease in the long-term. In addition, eradication of *H. pylori* is a stable phenomenon at least during the first five years after treatment. In patients with recurring dyspeptic symptoms, reflux disease should be considered.

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SERUM RESPONSE TO HELICOBACTER PYLORI IN PRIMARY B-CELL GASTRIC LYMPHOMA.

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Background: It has been demonstrated that the acquisition of organized lymphoid tissue (MALT) in the stomach is almost always associated with *H. pylori* (HP). The aims of this study were to assess the serological responses to HP and the type I strain-specific CagA protein in patients with gastric B cell MALT lymphoma.

Methods: 48 untreated cases (median age 59 y, range 28-76) of gastric MALT lymphoma were studied. The presence of HP was confirmed by a modified Giemsa stain. Serum positivity for HP IgG and IgA was determined by ELISA. Serum CagA IgG antibodies were assayed by ELISA using a recombinant fragment of CagA.

Results:

| | HP IgG | | HP IgA | | CagA IgG | | |
|--------------|--------|----|--------|----|----------|----|----|
| | + | - | + | - | + | - | |
| Histo HP | + | 33 | 4 | 10 | 27 | 19 | 17 |
| | - | 8 | 3 | 3 | 8 | 3 | 9 |
| Serum Hp IgG | + | | | 17 | 23 | 20 | 21 |
| | - | | | 4 | 8 | 2 | 5 |

37/48 (77%) biopsies were histologically positive for HP. 41/48 (85%) were seropositive for HP IgG. Two patients IgG seronegative were positive for IgA giving an overall seropositivity of 90%. 8/11 (73%) histologically negative patients were seropositive for HP IgG. 3/48 patients were neither histologically, nor serologically HP positive (age 59, 66, 69 years). 20/41 (49%) IgG positive sera recognised CagA.

Conclusions: This study demonstrates a high frequency of seropositivity for HP in patients with gastric MALT lymphoma (90%) which is greater than that found in the background population (50-60%). The CagA seropositivity of patients with gastric MALT lymphoma appears to be similar to that in those with HP associated chronic gastritis suggesting no particular association between MALT lymphoma and infection with either type I or type II strains of HP.