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Original article

Helicobacter pylori eradication improves acid reflux and esophageal motility in patients with Gastroesophageal Reflux Disease and antral gastritis

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SUMMARY

Introduction: The relationship between Helicobacter pylori (Hp) gastritis, the most common infection worldwide and Gastroesophageal Reflux Disease (GERD), a major health problem in Western Countries, remains controversial. AIM: To investigate the association between Hp infection and GERD and the impact of Hp eradication on esophageal acid exposure and esophageal motility in Hp-positive patients with GERD. Methods: Twenty seven Hp-positive (group I) and 20 Hp-negative (group II) patients with GERD underwent endoscopy-biopsy, esophageal manometry and 24-hour pH-metry. All group I patients received eradication treatment and six months later they were re-evaluated with 24-hour pH-metry, esophageal manometry and endoscopy-biopsy. Results: There were no significant differences between the two groups regarding sex, age, grade of esophagitis, manometric and pH-metry findings. All Hp-positive patients had antrum predominant gastritis. In all group I patients' eradication of Hp was successful. Gastritis and esophagitis were healed in all patients. The mean Lower Oesophageal Sphincter Pressure (LOSP) showed a significant increase of 11.7 mmHg before and 12.48 mmHg after eradication ($p<0.04$). A significant decrease in DeMeester score was observed (mean score 62.92 before versus 41.88 after eradication ($p<0.01$). Conclusions: 1. In patients with GERD the presence of Hp has no impact on manometric and pH-metry findings. 2 The eradication of Hp infection results in increase in LOSP with a consequent decrease in esophageal acid exposure.

Keywords: Helicobacter pylori, Gastroesophageal Reflux Disease, antral gastritis.

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The relationship of Helicobacter pylori infection and Gastroesophageal Reflux Disease (GERD) has unclear for many years.[1] Several mechanisms have suggested a protective or an aggressive role while other investigators proposed an independent coincidence of the two conditions.[2] The next rising problem is the impact of *H* pylori eradication in patients with GERD, which is the purpose if this study.[3,4]

Aim: The aim of this study was to investigate the association between Hp infection and GERD and the impact of Helicobacter pylori eradication on oesophageal motility and on the oesophageal acid exposure in patients with GERD.

PATIENTS

From March 1999 to April 2008, 47 consecutive patients (M: 30, F: 17), with a mean age of 39,89±13,55 (range 18-70 years) presenting GERD symptomatology for at least six months were enrolled in the study. Exclusion criteria were past history of peptic ulcer disease, pre-

Abbreviations:

GERD: Gastroesophageal reflux disease
Hp: Helicobacter pylori
LOSP: Lower Oesophageal Sphincter Pressure

vious gastric surgery, stricture, Barrett's oesophagus and motility disorders of the oesophagus.

METHODS

All patients underwent endoscopy-biopsy, esophageal manometry and 24-hour pH-metry.The score of esophagitis was calculated using the modified Savary-Miller system. Two biopsy specimens were obtained from the antrum and two from the corpus for histology and CLO-test. The results of the later allowed us to seperate the patients into two groups: Group I, twenty-seven Hp-positive patients and Group II 20 Hp negative patients.

Manometry was performed using an 8 channel perfusion catheter (Synectics Medical) and pH -metry (Synectics Medical) with a single antimony electrode and computer analysis software programme. The results are displayed as the DeMeester score.

All group I patients received a 7 days eradication treatment with rabeprazole 20mg, amoxicillin 1gr and clarithomycin 500mg, twice a day, the treatment being given for Hp eradication only and not in order for the PPI's to reduce acid secretion.

Six months later all Group II patients were re-evaluated by endoscopy-biopsy as well as pH-metry and manometry. Comparisons were made between the two groups [baseline measurements] as well as within the Group II before and after eradication of Hp. Statistical analysis was performed by the ANOVA test and the SPSS for Windows package (version 11.0, SPSS, Chicago, IL).

RESULTS

No significant differences were found between the two groups regarding sex, age, grade of oesophagitis, manometric and pH-metry findings. Pre-treatment endoscopy revealed reflux oesophagitis in 17 of 27 and in 12 of 20 patients, in Groups I and II, respectively. The LOSP was 11.78±4.68 mmHg in Hp-positive [Group I] and 12.10±4.61 mmHg in Hp-negative patients, while the median DeMeester score was 63,60 and 62,90, respectively. This finding led as to consider that the Hp status itself had no impact on manometric and pH-metry findings in patients with GERD.

All Hp-positive patients [Group I] had antrum predominant gastritis. The mean LOSP showed a significant increase after eradication of 11.7±4.75 mmHg before and 12.48±4,68) mmHg after treatment (p<0.04) (Figure 1). A significant decrease in DeMeester score was also prominent in the same time periods, respectively (mean score 62.92±12.75 before versus 41.88±13.75 after eradication (p<0.01) (Figure 2). In other words, Hp eradication resulted in a statistical significant increase in LOSP with a consequent decrease in oesophageal acid exposure.

DISCUSSION

Despite the large number of published studies the pathophysiological interrelation between GERD and Hp remains controversial. There is a great heterogeneity

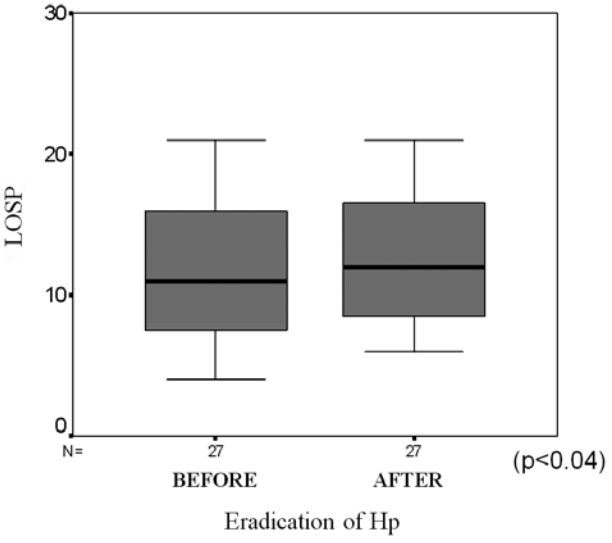

Figure 1. Lower oesophageal sphincter pressure before and after Hp eradication.

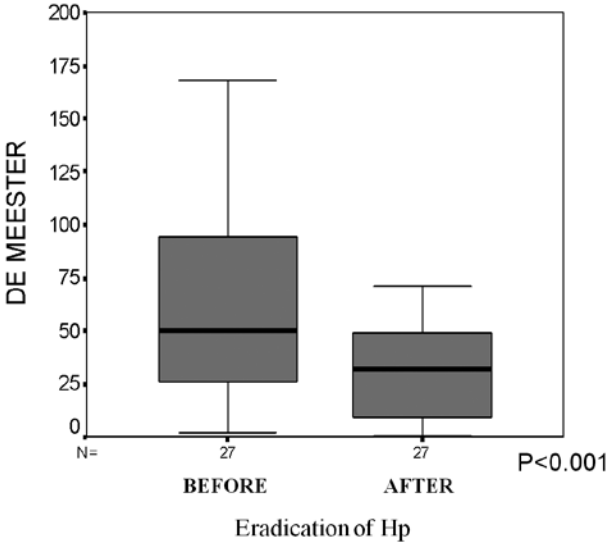

Figure 2. DeMeester score before and after Hp eradication.

among studies resulting in different conclusions. There are data suggesting a protecting role of Hp in GERD, other data suggest an aggravating role and many studies support a mere co-existance of the two conditions.

Award et al[5] studied 37 patients with GERD (78% Hp-positive) using ambulatory 24-hour oesophageal pH-metry and manometry. There was no difference in the manometric findings between Hp-positive and Hp-negative groups but the Hp-positive patients had a lower acid exposure tendency (DeMeester score). This may suggest a protective role of Hp in the pathogenesis Of GERD.

Manes et al[6] studied 50 patients with GERD (24 Hp-positive and 26 Hp-negative) using oesophageal manometry and 24-hour oesophageal pH-metry. They concluded that the presence of Hp has no impact on esophageal motility, LOSP, or gastroesophageal reflux.

In our study no statistical difference was found between Hp–positive and Hp- negative patients, regarding the manometric findings, acid exposure and severity of esophagitis. It is possible that the chronic presence of the bacterium leads to a type of 'adoption' or the two conditions are independent.

There are many studies about the effect of Hp eradication on the development or exacerbation of GERD. Most of them include patients with dyspepsia, ulcer as well as asymptomatic patients (Hp–positive).

Labenz J and Blum[7] in the first of these studies published, found an exacerbation of GERD after eradication of Hp in patients with peptic ulcer disease. More recent that studies[8-10] came to the same conclusion; that is that the eradication of Hp results in development of clinical GERD.

On the other hand many studies support the positive effect of Hp eradication on GERD symptomatology , recurrence, and disease free interval.

Loren and Sugg in a great meta- analysis of 8 prospective studies concluded that Hp eradication does not worsen pro-existing GERD but improves GERD symptoms and increases disease free intervals.[11]

Finally some authors support that Hp eradication has no impact on GERD symptoms, suggesting a mere co-existance of the two conditions.[12,13] However there are a few published studies about the possible relationship of Hp eradication on measurable parameters of GERD, such as acid exposure and motility.

Verma et al[14] found no significant correlation between all motility indexes and acid exposure of the oesophagus in patients with GERD before and after eradication of Hp. Similarly, Guliter and Kandilci[15] studied 18 patients with GERD and Hp gastritis using 24-hour pH-metry and static oesophageal manometry. After successful eradiacation of Hp no influence was found in the pH and manometric findings but only a slight improvement of clinical symptomatology.

In East Asian countries, where Hp-gastritis is primarily located in the corpus, the eradication of Hp sometimes leads to an increase in oesophageal acid exposure and a consequent worsening of the clinical picture. Wu et al[16] studied the oesophageal acid exposure in 14 patients with GERD and Hp-gastritis. Twenty-six weeks after eradication of Hp, three of the patients presented with worsening of the heartburn index.

In our study, in patients with GERD and antral gastritis, a significant increase in LOSP and a decrease of DeMeester score was found after Hp eradication. One possible reason for that may be the normalization of hypergastrinaemia after eradication of Hp, given that gastrin decreases LOSP. Furthermore the increase of LOSP, in combination with the decrease of acid volume due to the healing of gastritis may lead to the improvement of oesophageal acid exposure. However the beneficial results of Hp eradication in this study are most likely due to the type of gastritis of our patients (antrum predominant active**)**, the most common type in Western Countries. In cases of atrophic corpus gastritis the results may be totally different. The type and the localization of the gastritis may have great importance and may be the result of the age of contamination with the Hp. The earlier the contamination and corpus gastritis the earlier and the appearance of the atrophy. If contamination occurs in middle age the gastritis is limited to the antrum.

In conclusion our study suggests that the presene of Hp has no relationship to the pH-metry values and manometric findings in patients with GERD. In case of Hp infection the bacterium should be eradicated not only to prevent peptic ulcer and gastric cancer but also because it may have a beneficial effect on esophageal function.

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