LETTERS TO THE EDITOR EDİTÖRE MEKTUP *Helicobacter pylori* seropositivity in patients with reflux esophagitis

Reflü özofajitte Helikobakter pilori seropozitivitesi

To the Editor

To date, the relationship between gastroesophageal reflux disorder (GERD) and Helicobacter pylori (HP) infection remains unclear, as the results of various reports have conflicted (1-2). In most reports, the presence of HP infection has been reported to be preventive against GERD by way of hypochlorhydria induced during chronic infection with this bacterium (3-4). Nonetheless, some reports have challenged this theory and even presented contrary results (5). We aimed to clarify the subject in our patients with the diagnosis of GERD.

Seventy-two patients (group 1) with symptomatic reflux disease and endoscopic diagnosis of reflux esophagitis were included into the study. None of the patients had previous HP eradication and no one had systemic disease. Moreover, they did not use any drug with potential effects on GI motility. The male to female ratio was 51/21 and the mean age was 45.6+12.6 (16-77 years). The controls were 121 asymptomatic healthy people (group 2) without a previous history of HP eradication. HP se-

ropositivity was investigated by ELISA using HP specific kit to determine serum anti HP Ig G. The control group had a mean age of 40+13.06 (15-76 years) and the male to female ratio was 84/37. There was no statistical difference in age or sex distribution in either group (p>0.05). HP seropositivity rates were 80.6% in group 1 and 66.7% in group 2. The difference was statistically significant (p<0.05).

Our result challenges the classical theory of protection induced by HP in GERD. On the other hand, we do not know whether HP can induce gastroesophageal reflux by its effects on gastric motility (6). Moreover, esophageal motility disturbance has been reported in patients with HP infection. From this point of view, esophageal clearance decreases and reflux esophagitis risk increases in HP-infected subjects (7). At least according to our study, we believe that the presence of GERD is not a handicap in planning eradication of this bacterium.

REFERENCES

- Nogueira-de Rojas JR, Jimenez-Gonzalez A, Cervantes-Solis C. Helicobacter pylori gastric infection is a protection factor for gastroesophageal reflux disease. Rev Gastroenterol Mex 2002; 67: 22-7.
- 2. Wu JC, Lai AC, Wong SK, et al. Dysfunction of oesophageal motility in Helicobacter pylori-infected patients with reflux oesophagitis. Aliment Pharmacol Ther 2001;15: 1913-9.

Address for correspondence: Ufuk SAMSAR

Manavgat Sok. Yılmazevler Koop. Al-Blok No: 9 Kozlu, Zonguldak, Turkey

- 3. Beil W, Birkholz C, Wagner S, et al. Interaction of Helicobacter pylori and its fatty acids with parietal cells and gastric H/K-ATPase. Gut 1994; 35: 1176-80.
- 4. Cave DR, Vargas M. Effect of a Campylobacter pylori protein on acid secretion by parietal cells. Lancet 1989; 22; 2: 187-9.

Manuscriptreceived: 11.11.2003 Accepted: 18.11.2003

- 5. Werdmuller BFM, Loffeld RJ. Helicobacter pylori infection has no role in pathogenesis of reflux esophagitis. Dig Dis Sci 1997; 42: 103-5.
- 6. Miyaji H, Azuma T, Ito S, et al. The effect of Helicobacter pylori eradication therapy on gastric antral myoelectrical activity and gastric emptying in patients with non-ulcer dyspepsia. Aliment Pharmacol Ther 1999; 13: 1303-9.
- Wu JC, Lai AC, Wong SK, et al. Dysfunction of oesophageal motility in Helicobacter pylori-infected patients with reflux oesophagitis. Aliment Pharmacol Ther 2001; 15: 1913-

Ufuk SAMSAR¹, Yücel ÜSTÜNDAĞ², Selim AYDEMİR², İshak ÖZER TEKİN³, Erdem KOÇAK¹

Karaelmas University, Department of Internal Medicine¹, Gastroenterology Unif and Department of Immunology³, Zonguldak