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Editorial

Helicobacter pylori and Gastroesophageal Reflux Disease: A complex relationship

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Helicobacter pylori (Hp) has been shown to be one of the etiological factors for peptic ulcer disease. On the other hand Gastroesophageal Reflux Disease (GERD) is a common disease of multifactorial etiology. The role of Hp in the pathogenesis of GERD remains controversial. There is a great heterogeneity among studies, resulting in different conclusions. There are data suggesting a protective role of Hp in GERD, other data suggest an aggravating role and many studies support a mere co-existence of the two conditions.[1]

Epidemiological studies showed that the prevelance of GERD is lower in countries with high incidence of Hp infection, suggesting that Hp gastritis may protect from GERD.[2]

The first such published study a few years ago found an exacerbation of GERD after eradication of Hp in patients with peptic ulcer disease.[3] More recent studies came to the same conclusion: the eradication of Hp results in development of clinical GERD.[4,5]

On the other hand many studies support the beneficial effect of Hp eradication on GERD symptomatology, recurrence, and disease symptom-free interval.[6,7,8]

It is also known that the effect of proton pump inhibitors on intragastric pH rise is stronger in Hp positive patients.[9]

We have no conclusive data that Hp infection has any direct or indirect effect on reflux mechanisms. So the possible way by whitch Hp may impact the pathogenesis of GERD is the alteration of volume, gastric acid secretion and acidity.

Hp infection can lead to different types of gastritis (antrum predominant, chronic corpus gastritis or pangastritis). The type and the localization of gastritis may have great importance and may be related to the age of contamination with the Hp.The earlier the contamination and corpus gastritis, the earlier the appearance of the atrophy. If contamination occurs in middle age the gastritis is limited to the antrum. The antrum predominant gastritis is the most common type in Western Countries. In East Asian countries Hp-gastritis is primarily located in the corpus-pangastritis. The antrum Hp gastritis, via several mechanisms,leads to increased acid secretion and the eradication of Hp may decrease the acid production. In contrast, in East Asian patients the eradication of Hp sometimes leads to an increase in oesophageal acid exposure. The price for the possible protection from GERD is that atrophy and intestinal metaplasia of gastric mucosa may increase the risk of gastric cancer.

There are very few studies about the effect of Hp eradication on pH-metry and manometry in patients with GERD, with conflicting results.[10,11]The recent study 'Helicobacter pylori eradication improves acid reflux and esophageal motility in patients with GERD and antral gastritis'[12] showed that the eradication of Hp improves acid reflux well as esophageal motility in patients with GERD. However the beneficial results of Hp eradication in this study are most likely due to the type of gastritis of the patients (antrum predominant), the most common type in Western Countries. In case of atrophic corpus gastritis or atrophic pangastritis the results may be totally different. In addition, the follow up time was only six months and perhaps the results might have been different if the follow up was longer.

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It is evident that the relationship pf Hp infection and GERD is indeed a complex one and it requires further study.

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