**H pylori eradication and GERD**

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**ARTICLE**

**ARTICLE SUMMARY**
Ninety-eight patients with healed duodenal ulcer (DU) and proven Helicobacter pylori gastritis were prospectively, randomly assigned to receive bismuth plus metronidazole plus amoxicillin; bismuth plus metronidazole plus placebo; or metronidazole plus two placebos, and were followed every three months for one year with a detailed systematic gastrointestinal symptom inventory and upper gastrointestinal endoscopic evaluation. Eighty-seven patients had analyzable results, which were retrospectively evaluated for symptoms of gastroesophageal reflux disease (GERD) and endoscopic evidence of reflux esophagitis with respect to whether H pylori had been eradicated. Sixty-three patients had successful eradication of H pylori, while 24 did not. Those whose H pylori was eradicated had a higher incidence of GERD symptoms and/or esophagitis (37% compared with 13%, P=0.04), GERD symptoms alone (29% compared with 8%, P=0.04) and esophagitis alone (21% compared with 4%, P=0.10).

**COMMENTARY**
This retrospective analysis of prospectively collected data was undertaken in response to a publication that raised concern and controversy among gastroenterologists regarding the potential adverse consequences of H pylori eradication (1). Some studies have confirmed a protective role for H pylori in GERD (2-4), while others have not (5-8). Carlo Fallone and colleagues have demonstrated that GERD occurs in predisposed individuals when the protective effect of H pylori is eliminated, and that this effect is independent of pathogenic factors such as cagA status and host factors such as dietary change or weight gain.

This paper reviewed the potential mechanisms by which H pylori may protect against GERD. First, healing of corpus gastritis may increase acid secretion from subnormal to normal; however, most patients with DU have predominantly antral gastritis and acid hypersecretion. Second, ammonia produced by H pylori’s urease activity could neutralize gastric acidity – an effect that would be lost with bacterial eradication. Third, because gastrin increases lower esophageal sphincter tone, posteradication correction of H pylori-induced hypergastrinemia may result in increased gastroesophageal reflux.

Many patients treated for H pylori present with GERD. Unfortunately, primary care physicians seem to be eradicating H pylori, not only in patients with DU or uninvestigated dyspepsia, but also in patients with normal upper gastrointestinal bariam contrast studies or even in patients initially presenting with GERD. In the latter group, I sense that some patients experience a worsening of their GERD symptoms and are often more difficult to treat.

The authors also pointed out the inverse relationship between H pylori prevalence and adenocarcinoma of the esophagus, a disease known to be related to GERD. Moreover, there are other concerns over indiscriminate eradication of H pylori, such as the increase in prevalence of antibiotic resistance. Long term studies weighing the benefits and risks of H pylori eradication in large populations with and without DU are needed.

**REFERENCE**