

## Early Development of Reflux Esophagitis after Successful Helicobacter Pylori Eradication in Superficial Gastritis

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### ABSTRACT

The relationship between gastroesophageal reflux disease (GERD) and Helicobacter pylori (H. pylori) eradication is still debated. Recently, we had a patient of GERD who had developed it shortly after H. pylori eradication therapy. A 72-year-old man was diagnosed by endoscopy as suffering from severe superficial gastritis in the stomach body. A rapid urease test showed H. pylori infection. He was then started on proton pump inhibitor (PPI) based therapy for two weeks eradicating H. pylori. After completion of H. pylori eradication, he complained of a heart-burn sensation. Follow-up endoscopy showed reflux esophagitis, of grade B according to the Los Angeles classification. Since the patient had developed GERD after completion of the triple therapy, this suggests that H. pylori eradication must have triggered the development of de novo GERD after a short period of time.

**Keywords:** GERD, Helicobacter pylori, PPI

### INTRODUCTION

Gastroesophageal reflux disease (GERD) is a complex disorder, involving gastric acid coming up from the stomach into the esophagus. Helicobacter pylori (H. pylori) is the key pathogenetic factor in gastritis, peptic ulcers, stomach cancer and mucosa-associated lymphoid tissue (MALT) lymphoma.<sup>1</sup> The pathogenesis of reflux esophagitis is not fully understood.<sup>2,3</sup> In addition, the relationship between GERD and H. pylori eradication is still debated. Here, we present a patient with obvious GERD which developed 14 days after proton pump inhibitor (PPI) based H. pylori eradication.

### CASE REPORT

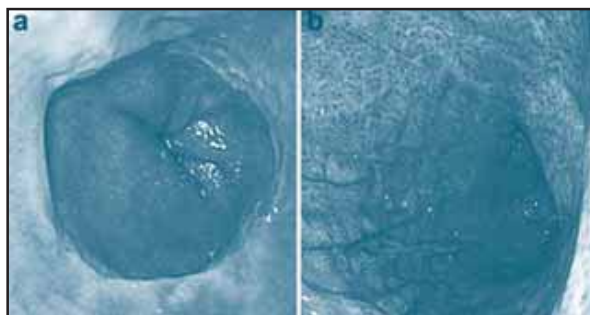
A 72-year-old man presented with progressive dysphagia, heart burn and regurgitation. The symptoms developed two weeks after completion of H. pylori

eradication. He had no history of weight loss and his body mass index was 22.5 kg/m<sup>2</sup>. He was a non-alcoholic and had never smoked. His physical examination revealed no specific findings. He had undergone an endoscopy as a part of regular medical check-up. The upper gastrointestinal endoscopy showed the typical findings of severe superficial gastritis in the stomach body and fundus, but normal findings in the esophagus (Figure 1). H. pylori infection was detected by the rapid urease test (CLO test; Kimberly-Clark, USA) and biopsy specimens obtained from the greater curvature of the body and antrum showed gastritis and presence of H. pylori. Then, H. pylori eradication therapy was initiated

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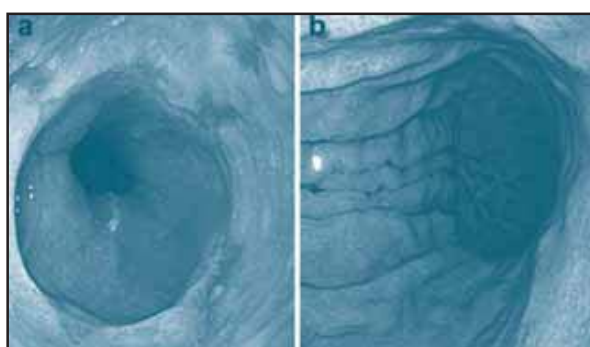
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after the patient was given a complete explanation of the therapy. *H. pylori* eradication therapy consisted of omeprazole 20 mg, amoxicillin 1,000 mg and clarithromycin 500 mg, all of which were administered twice daily for 14 days.



**Figure 1.** Endoscopic findings (a) normal gastroesophageal junction (b) superficial gastritis in the stomach body

He developed symptoms of progressive dysphagia, heart burn and regurgitation two weeks after completion of *H. pylori* eradication. There was no history of any other medications after *H. pylori* eradication and before the appearance of his symptoms. Follow-up endoscopy along with a CLO test was repeated due to the symptoms, but the CLO test was negative for *H. pylori*. Endoscopy showed that severe superficial gastritis of the body and fundus was completely cured. However, reflux esophagitis had developed in the previously healthy esophagus. The upper endoscopy showed multiple linear erosions from the gastroesophageal junction to the middle portion of the esophagus. His disease status was categorized as grade B esophagitis according to the Los Angeles classification (Figure 2).



**Figure 2.** Endoscopic findings six weeks after completion of *H. pylori* eradication (a) gastroesophageal junction showing newly-formed, multiple, linear erosions (b) gastric body showing healed superficial gastritis

## DISCUSSION

Gastroesophageal reflux disease is a common clinical condition. GERD symptoms occur in 25 % - 40 % of the general population. The relationship between GERD and *H. pylori* infection varies by geographic location.<sup>1</sup> It is well known that *H. pylori* eradication cures peptic ulcers and prevents their recurrence.<sup>4</sup> Recently, there have been reports that showed that there is no relationship between GERD and *H. pylori*.<sup>5,6</sup> However, it is still controversial as to whether there is a significant relationship between *H. pylori* eradication and GERD.

There have been some reports on de novo development or exacerbation of GERD after *H. pylori* eradication.<sup>7</sup> *H. pylori* seems to play a protective role against GERD, mainly via the development of atrophic gastritis causing decreased gastric acid secretion and the effect of ammonia secreted by *H. pylori* on gastric acid.<sup>8</sup> Others have suggested that there is no significant association between de novo development of GERD and *H. pylori* eradication.<sup>5</sup> Furthermore, Schwizer et al. demonstrated the beneficial effects of *H. pylori* eradication on GERD symptoms.<sup>9</sup>

Previous studies, however, have reported a relationship between GERD and *H. pylori* after long periods of time. However, in our case, the symptoms of GERD occurred two weeks after completion of *H. pylori* eradication. This was a very short time interval considering that the patient had undergone PPI based *H. pylori* eradication. Additionally, alcohol and obesity are known risk factors for GERD.<sup>10</sup> Alcohol reduces lower esophageal sphincter pressure, and obesity elevates abdominal pressure. Both lead to reflux of gastric contents into the esophagus. However, the lifestyle factors of our patient had not changed during or immediately after the triple therapy.

Several mechanisms could be involved in de novo development of GERD shortly after *H. pylori* eradication. As one potential mechanism, inflammation at the gastric corpus could prevent the severity of GERD. In such cases, *H. pylori* infection could result in decreased hydrochloric acid secretion in diffuse gastritis and gastric atrophy, damaging parietal cells located in the gastric corpus.<sup>11-13</sup> After eradication of *H. pylori*, chronic gastritis in the corpus is cured and the parietal cells are able to secrete gastric acid<sup>14</sup>, so that treated individuals demonstrate normal gastric acid production capacity and become susceptible to harmful gastric acid reflux, leading to the development of GERD.

Another possible mechanism of the development of GERD is an alternation of gastroesophageal motility with *H. pylori* infection. Jensen DM et al. postulated the role of gastrin in gastroesophageal motility, which increases lower esophageal sphincter pressure.<sup>15</sup>

Therefore, reductions in gastrin level, as observed with *H. pylori* eradication, could reduce lower esophageal sphincter pressure and consequentially facilitate GERD.

To the best of our knowledge, a few cases of de novo development of GERD shortly after *H. pylori* eradication have been reported. Although the relationship between *H. pylori* eradication and GERD remains to be elucidated, this case suggests that *H. pylori* eradication may trigger

the development of de novo GERD after a short period of time.

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