Editorial

Gastric atrophy as an important risk factor for gastric cancer development after Helicobacter pylori eradication

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Helicobacter pylori (*H. pylori*) infection plays an important role in gastric cancer development (1). Several studies have demonstrated the possibility of gastric cancer prevention by the *H. pylori* eradication (2,3). However, after successful *H. pylori* eradication, gastric cancers sometimes develop (4,5). Since little has been reported on the risk factors for primary gastric cancer after *H. pylori* eradication, Toyoshima et al conducted a retrospective, endoscopy-based, long-term, large-cohort study to clarify the risk factors for gastric cancer following *H. pylori* eradication (6). The author performed 10 years surveillance esophagogastroduodenoscopy of 1,232 cancer-free patients after successful *H. pylori* eradication. Gastric cancer developed in 15 patients (17 lesions) after successful *H. pylori* eradication. The cumulative incidence rates were 1.0% at 2 years, 2.6% at 5 years, and 6.8% at 10 years, respectively. Gastric cancers developed after *H. pylori* eradication were characterized as intestinal type histopathology, within the mucosal layer, and <20 mm in diameter. Multivariate analysis identified higher grade of endoscopic gastric atrophy at *H. pylori* eradication (hazard ratio 1.77; 95% confidence interval, 1.12–2.78; P=0.01) as the only independently associated parameter.

It is well known that the pathological state of gastritis, such as severe gastric mucosal atrophy or intestinal metaplasia is closely associated with the risk of gastric cancer among *H. pylori* infected patients (1). Recent studies have also suggested that the severe gastric atrophy at *H. pylori* eradication is associated with increased risk of metachronous gastric cancer (7). Our group has reported that severe gastric atrophy and intestinal metaplasia remained in the adjacent mucosa of the gastric cancer developed after *H. pylori* eradication (8). The effect of *H. pylori* eradication in preventing gastric cancer can be attributed to the improvement of chronic inflammation or atrophy. However, such effect would be limited in patients with baseline severe atrophic gastritis because gastric atrophy may not improve after *H. pylori* eradication.

The strength of the current study is that the author demonstrated a risk factor for gastric cancer after *H. pylori* eradication among larger cohort of cancer free patients. The author also demonstrated characteristics of gastric cancers developed after *H. pylori* eradication as tiny intramucosal cancer with intestinal type histopathology, which is in line with several other studies (4,5). Current result have provided useful information for many endoscopists to perform more appropriate clinical implementation reflecting individual risk for gastric cancer after *H. pylori* eradication.

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