LETTER TO THE EDITOR

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Association between *Helicobacter pylori* and Gastric Cancers

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To the Editor,

Gastric cancer is the third most common cause of cancer-related deaths in both sexes worldwide. *Helicobacter pylori* infection is the greatest known risk factor for the development of gastric cancer. *H. pylori* can cause chronic active gastritis and atrophic gastritis by producing persistent acute-on-chronic inflammation. Therefore, screening and treatment of *H. pylori* is an important strategy for preventing gastric cancer in high-risk populations, particularly among Japanese and Korean populations.[1] We recently read the paper by Kim *et al*.,[2] who evaluated the clinicopathologic features of *H. pylori* infection-negative gastric cancer (HPIN-GC) compared with *H. pylori* infection-positive gastric cancer (HPIP-GC). Based on their results, the authors concluded that the prevalence of HPIN-GC was extremely low, and its clinicopathologic characteristics were similar to that of HPIP-GC. However, we believe that certain additional points should be discussed.

First, patients with gastric cancer were divided into two groups according to the *H. pylori* status in the study. One hundred sixty-four patients with past infection (by anti-*H. pylori* IgG positivity) or an eradication history of *H. pylori* were included as the HPIP-GC group. However, patients with current *H. pylori* infection have a higher risk of developing gastric cancer compared with patients with past infection or an eradication history of *H. pylori* because the eradication of *H. pylori* reduces the risk of gastric cancer. Several clinical studies have reported that the successful treatment of *H. pylori* decreases the risk of developing gastric cancer by approximately 3 fold.[3] Therefore, we suggest that patients with gastric cancer should be categorized into three groups: patients with current, past, and negative *H. pylori* infection. Therefore, Kim *et al*.[2] could also evaluate the effect of *H. pylori* eradication on gastric cancer in their study.

Second, Kim *et al.*[2] have defined current *H. pylori* infections according to the results from at least one of the following tests: histological, rapid urease, and culture. However, the American College of Gastroenterology guidelines for the management of *H. pylori* infection recommends using at least two different tests to diagnose of *H. pylori* except culture.[4] Therefore, this issue might be a reason for the small number of patients with HPIN-GC compared with previous studies.

Third, the concentrations of serum pepsinogen (PG) I and II were measured in fasting serum samples, and patients with PG I/II ratio ≤3 were considered to have atrophic gastritis. Importantly, the serum levels of PG cannot provide accurately identifications if proton pump inhibitors are used.[5] However, information on the use of these drugs is not available in this study. Finally, infection with CagA-positive *H. pylori* strain has been associated with a higher risk of developing gastric cancer because it is a more virulent strain.[1] However, Kim *et al*.[2] did not assess genetic analyses for *H. pylori*.

Therefore, we conclude that the study by Kim *et al*.[2] should be rearranged to account for the above-mentioned suggestions before interpretations are provided. This could provide the readers of the journal with clearer information regarding the relationship between *H. pylori* infection and gastric cancer.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

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