Helicobacter pylori testing in a population of Korean patients with pernicious anemia

TO THE EDITOR: Pernicious anemia is a megaloblastic anemia caused by intrinsic factor deficiency, secondary to autoimmune destruction of the acid- and pepsin-secreting portion of the gastric mucosa. Kaptan *et al.* [1] suggested *Helicobacter pylori* (*H. pylori*) as a causative agent in the development of adult cobalamin deficiency, where upper gastrointestinal endoscopy revealed *H. pylori* infection in 77 (56%) of 138 patients with cobalamin deficiency. It has also been suggested that pernicious anemia may onset many years before clinical cobalamin deficiency, *via* an autoimmune process likely triggered by *H. pylori* [2]. In addition, restoration of hemoglobin after *H. pylori* eradication in patients with pernicious anemia has been observed in some Hispanic patients [3]. However, a causative role of *H. pylori* in pernicious anemia was not observed in other studies [4, 5], such that causation remains controversial.

We reviewed *H. pylori* infection status and its clinical implications in patients with pernicious anemia. Medical records were obtained for all patients diagnosed with pernicious anemia between 2002 and 2017 at the Chungnam National University Hospital, Daejeon, Korea. We performed Giemsa staining of gastric body and antrum tissue prepared from paraffin blocks, reviewed the results of *H. pylori* testing, and analyzed clinical data. Of the 70 patients who were diagnosed with pernicious anemia during the study period, 40 were included in the analysis. The median age was 64 yr (range, 33–81 yr), and the male/female ratio was 1.2. Autoimmune disorders were found in eight patients (20.0%). Antibody to intrinsic factor was detected in 24 of 37 patients (64.9%), and antibody to parietal cells in 18 of 39 patients (46.2%). Of the 38 patients who underwent gastroscopic examination, 22 (59.5%) revealed intestinal metaplasia in addition to chronic gastritis in the body. Positive results for various *H. pylori* tests included: one of four patients (25.0%) for immunoglobulin G (IgG) antibodies to *H. pylori*, four of eight patients (50.0%) in the urea breath test, and one of seven patients (14.3%) in the CLO test. Additionally, 3 of 38 patients (7.9%) showed positive Giemsa staining. Nine of forty patients (22.5%) tested positive in at least one *H. pylori* test, which was a similar proportion to that in the general Korean population [6]. Compared to the *H. pylori*-negative patients, *H. pylori*-positive patients showed higher mean corpuscular volume (119.3±9.5 vs. 102.3±7.9 fL, $P<0.001$) and less frequent autoimmune disorders (0% vs. 25.8%, $P<0.001$). The implications of this finding are not clear. No other differences were observed between the two groups in terms of symptoms, laboratory and histologic findings, and responses to treatment.

We report that the proportion of *H. pylori*-positive cases among Korean patients with pernicious anemia did not differ from that of among the general population, and that clinical features did not differ between the *H. pylori*-positive and -negative groups. These results indicate that *H. pylori* infection did not play a major role in the development or progression of pernicious anemia. Pernicious anemia is the most common cause of vitamin B12 deficiency, and one of the most prevalent autoimmune diseases in Western countries. Although pernicious anemia affects virtually all racial and ethnic groups, it is relatively uncommon in East Asia. The results of the present study are in line with a previous Japanese report [4]. Taken together, it is suggested that the role of *H. pylori* in the development of pernicious anemia differs among ethnic groups, and by region.

Ik-Chan Song, Myung-Won Lee, Seung-Woo Baek, Hyewon Ryu, Yoon-Seok Choi, Deog-Yeon Jo
Division of Hematolog/Oncology, Department of Internal Medicine, College of Medicine, Chungnam National University, Daejeon, Korea

Correspondence to: Deog-Yeon Jo
Division of Hematology/Oncology, Department of Internal Medicine, Chungnam National University Hospital, 282 Munwha-ro, Jung-gu, Daejeon 35015, Korea
E-mail: deogyeon@cnu.ac.kr

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No potential conflicts of interest relevant to this article were reported.

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