

Helicobacter pylori management in primary care

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Helicobacter pylori is a Gram-negative bacterium that chronically infects the stomach of more than 50% of the human population, and represents the major cause of gastric cancer, gastric lymphoma, gastric autoimmunity and peptic ulcer diseases [1–4]. The International Agency for Research on Cancer classifies *H. pylori* as a human carcinogen for distal gastric cancer. Eradicating the bacterium, in high-risk populations, reduces the incidence of gastric cancer [5]. Likewise, antibiotic treatment leads to the regression of gastric MALT lymphoma [2]. *H. pylori* also contributes to other conditions, such as vitamin B12 and iron deficiencies, idiopathic thrombocytic purpura, and growth retardation in children [6].

Current guidelines indicate that the eradication of *H. pylori* infection is considered mandatory in patients with peptic ulcer and gastric malignancies, such as gastric adenocarcinoma and MALT lymphoma [6, 7]. Furthermore, it is recommended in patients with non-ulcer dyspepsia, especially in those with the evidence of macroscopic or microscopic mucosal abnormalities (erosions, intestinal metaplasia, atrophy), naïve non-steroidal anti-inflammatory drugs (NSAIDs) users, chronic NSAIDs users, first-degree relatives of gastric cancer patients as well as in unexplained

iron deficiency anaemia, and idiopathic thrombocytopenic purpura. Low-dose aspirin (ASA) therapy is widely used in primary care because of the proved efficacy in both primary and secondary prevention of cardiovascular events [8]. A synergistic interaction between *H. pylori* infection and NSAIDs has been extensively documented although the benefits of *H. pylori* eradication in NSAIDs users are conflicting [6, 9–11]. *H. pylori* has been shown to increase, by almost seven times, the risk of upper gastrointestinal complications in chronic NSAIDs users [12, 13]. The relationship between *H. pylori* infection and NSAIDs in gastroduodenal pathology is complex. Since both NSAIDs and *H. pylori* can cause peptic ulcers, *H. pylori* eradication can only be expected to prevent the recurrence of *H. pylori* ulcers, and while it may also reduce the incidence of ulcers among those with both *H. pylori* and NSAID use, the effects will vary depending on the proportion with real *H. pylori* ulcers in the population studied [6].

Zullo et al. [14] designed a very interesting study (reported in the current issue) to assess the management of *H. pylori* infection in a very large cohort of chronic NSAID users in primary care clinical settings. *H. pylori* was being used only in a minority (less than 20%) of primary care patients receiving chronic NSAID therapy. *H. pylori* was eventually cured in two-third of the infected cases. The low alertness towards such *H. pylori* infection in these patients suggests a need for prompt implementation of current guidelines. Furthermore, the results obtained by Zullo et al. [14], other large meta-analysis studies, strongly support the concept that patients requiring long-term NSAIDs/ASA therapy should be tested and cured of the infection [9, 14, 15] because the cure of *H. pylori* infection contributes to the reduction of potential life-threatening gastrointestinal critical events (such as gastroduodenal bleeding) in primary care unstable patients.

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