

## EDITORIAL

*H. PYLORI*: DOES CO-MORBIDITY AFFECT THE PREVALENCE?

*Helicobacter pylori* is a spiral bacterium which resides in the lumen of the stomach. It was rediscovered by Warren and Marshall in 1983. Since then there have been major changes in the understanding and treatment of peptic ulcer disease.

The bacterium is most often found in the antrum of the stomach, residing on the surface and at the foveolar regions of the gastric mucosa, close to intercellular junctions. The presence of these bacteria provokes an intense polymorpho neutrophil infiltration.

With persisting infection, acute gastritis becomes chronic with an increase in lymphocytes and plasma cells in the lamina propria. Chronic gastritis may involve the antrum or may spread to the whole of the stomach, a pattern more commonly seen in developing countries.

Alone, or in combination with genetic or other environmental factors, some individuals may progress to atrophic gastritis with loss of glandular component of the gastric mucosa. It is worth noting that, on a world scale, a far greater number of cases of gastric atrophy are as a result of this infective process than those caused by autoimmune gastritis (previously, labeled type A, associated with pernicious anaemia).

There is a marked difference in the prevalence of *H. pylori* between developed and developing countries. In the developed world with high socio-economic status, the prevalence of *H. pylori* is low ranging between 15% and 54% in the general population(1).

In the developing world with low socioeconomic status, the prevalence of *H. pylori* is high ranging from around 70% in Asia and south America upto 85% in Africa. Infection in these countries occurs at an early age(1,2).

*H. pylori* is associated with a number of diseases causing dyspepsia in both developed and developing countries. In developing countries *H. pylori* is found in 95% of duodenal ulcers, 67-85% of gastric ulcers and there is an attributable risk to gastric cancer in 50% of cases(3). In Kenya, all cases of peptic ulcer had evidence of *H. pylori*, while dyspeptic patients with normal mucosa on endoscopy had *H. pylori* in 80.5% of cases. The evidence of *H. pylori* in gastric cancer cases was very low, 25% of cases(3). It is now well established that infection with *H. pylori* is the most important cause of peptic ulcer disease(4,5) and that successful eradication of this organism markedly reduces the rate of ulcer relapse.

The prevalence of *H. pylori* in other conditions have also been described. The prevalence of *H. pylori* was found to be low in inflammatory bowel disease compared to controls. The prevalence was even lower in Crohn's disease than in ulcerative colitis(6). It has been found that the prevalence of *H. pylori* in HIV/AIDS patients is lower than in HIV negative patients. Marano *et al.* (7) in America found that the prevalence of *H. pylori* in AIDS patients

with histologic chronic active gastritis was much lower than the prevalence previously reported for HIV-negative patients with similar pathology. Vaira *et al* (8) from Italy also found that *H. pylori* prevalence in HIV-1 positive residents was lower than in HIV-1-negative residents. However, Battan *et al*(9) did not find a difference in the prevalence of *H. pylori* between AIDS/ARC patients and age matched controls. This led them to postulate that cell-mediated deficiency does not appear to increase the risk of infection with *H. pylori*. Fabris *et al* (10) from Italy looked at the immune status further. They found that the prevalence of *H. pylori* was low in cases of CD4 cell counts less than  $100 \times 10^6/L$  and high in cases of CD4 cell counts of  $200 \times 10^6/L$ . Thus, cell-mediated immune deficiency does not appear to increase the risk of infection with *H. pylori*.

Most of the work on this subject has been reported from the developed world. Ali Mohamed *et al* (11) in this issue of the journal reports their findings of *H. pylori* in HIV-1 positive patients from Kenya. They found that cell-mediated immune deficiency does not appear to increase the risk of infection with *H. pylori*.

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