

Prevalence of histology proven *Helicobacter pylori* infection in a cohort of Sri Lankan patients with gastric carcinoma

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Helicobacter pylori (*H. pylori*), first described by Warren and Marshal in 1983 have been implicated in the pathogenesis of several diseases. Although peptic ulcer disease is the most studied association with *H. pylori* infection, this bacterium is seemingly involved in the pathogenesis of several extra gastric diseases. At present, many of these associations remain largely uncertain, and the debate to confirm or refute causality related to these associations is still open. In contrast the association of *H. pylori* and the development of gastric carcinoma is more definite [1].

Although there have been several reports of the prevalence of *H. pylori* among different upper gastro intestinal diseases [2,6] there are no reports published to date on the prevalence of *H. pylori* among Sri Lankan patients with gastric carcinoma [2-6].

In this study we describe the prevalence of *H. pylori* infection among 56 newly diagnosed patients with gastric adenocarcinoma at the Gastroenterology Unit in the National Hospital of Sri Lanka from 1 January 2007 to 31 December 2010. Of the 56 patients 43 (76%) were male. The average age at diagnosis was 61.9 years (range 44-84 years). The majority of tumours were located at the fundus and cardia (20/56), followed by the body (13/56), antrum (2/56), lesser curvature (19/56), and greater curvature (2/56). A minimum of four gastric biopsies were collected from each patient into 10% formalin. Haematoxylin and Eosin and modified Giemsa staining techniques were used to detect *H. pylori* infection and the slides were examined by an experienced histopathologist. Of the 56 gastric carcinomas, 12 (21%) tested positive for *H. pylori* and 44 (78%) were negative.

The prevalence of *H. pylori* infection on gastric biopsies has been variable. In an extensive review of gastric cancer and *H. pylori*, the EUROGAST Study Group determined that the presence of *H. pylori* conferred an approximately 6-fold risk of gastric cancer, accounting for about half of all gastric cancers [3]. Cross-sectional studies reveal infection rates between 50% to 100% in gastric adenocarcinoma [1, 4, 5].

The lower prevalence rates seen among our patients could be due to several reasons. Firstly it could be due to the method of detection. We used histology and the modified Giemsa staining technique to detect *H. pylori*. Although, the modified Giemsa has equal sensitivity when compared with immune histochemistry, which is the agreed gold standard for histology, when compared with PCR, histology

has relatively low specificity and sensitivity [6]. We were unable to use the PCR technique due to its high cost. Secondly, the low prevalence rates seen in our study could also mirror the low prevalence rates of *H. pylori* (49%) in our general population with upper gastro intestinal diseases which previous studies have shown [7]. Thirdly it could be due to an actual lesser association of *H. pylori* with gastric carcinomas. Finally, ingredients used in our cooking which have been shown to have bactericidal and anti-adhesive properties against *H. pylori* such as turmeric, cumin, ginger and chilli might be an explanation for our low detection rates [8].

In conclusion, although the prevalence of *H. pylori* in gastric adenocarcinoma has been high in some studies conducted in other regions, a lower prevalence was seen amongst our patients. Further studies with more sensitive and specific diagnostic techniques would be needed to confirm our findings.

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