

Detection of *Helicobacter pylori* by Histology in Biopsy Specimens of Patients with Gastric Ulcers in a Selected Group of Patients from Teaching Hospital, Peradeniya

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In the medical literature, about 60% to 100% of benign gastric ulcers have been reported to be associated with *Helicobacter pylori* infection. However, in clinical practice such association is often not observed by histology.

The aim of this study was to assess the detection rate of *H. pylori* infection by histology in clinical biopsy specimens of patients detected to have benign gastric ulcers in teaching Hospital Peradeniya.

The study included 31 cases endoscopically detected to have gastric ulcers. Ulcers were biopsied as routine clinical specimens and the rest of the gastric mucosa was not biopsied. None of the cases had prior anti *H. pylori* therapy in this institution. However, most had been treated with proton pump inhibitors. Malignant ulcers were excluded. Histological assessment was performed using haematoxylin and eosin stain and toluidine blue stain. Following histological parameters were studied and graded according to the Sydney system: *H. pylori* organisms, acute and chronic inflammation, atrophy and dysplasia. Fourty six cases with endoscopically detected other benign gastric abnormalities and 57 cases with endoscopically normal gastric mucosa were also assessed similarly. All cases were age and sex matched.

H. pylori detection rate among gastric ulcers was 3/31 (9.7%). The *H. pylori* density was Sydney grade 2 in all. Of the 28 cases without *H. pylori*, 21(75%) did not have significant inflammation (no inflammation or Sydney grade 1 chronic inflammation); 4 (14.3%) had Sydney grade 2 and none had grade 3 chronic inflammation; 2 (7.1%) had active chronic gastritis; 1 (3.6%) had chronic atrophic gastritis. *H. pylori* detection rate in cases with other gastric abnormality group was 6/46 (13%) and in endoscopically normal group 2/57 (3.5%). The differences were not statistically significant ($p>0.05$). The *H. pylori* density in these 8 cases ranged from Sydney grade 1 to 2.

The *H. pylori* detection rate by histology in the gastric ulcer group appears to be low in the present study group. This detection rate appears to be more or less similar to non ulcer groups. *H. pylori* density is relatively low in all cases. Histology is a relatively specific but less sensitive method of detecting *H. pylori*. The density of *H. pylori* organisms and their distribution affect the histological detection of the organism. Inter-observer variation is high when the *H. pylori* density is low. Treatment with proton pump inhibitors is known to induce migration of the organisms from the antrum to body. Therefore, sampling of the antrum only, may give rise to false negative results. *H. pylori* infection is associated with significant mucosal inflammation. Therefore, the 21 cases without significant background inflammation are less likely to have undetected *H. pylori* infection. Since histology is a main mode of detection of *H. pylori* gastritis in Sri Lanka, it is important to investigate further whether or not histology under estimate the *H. pylori* infection in the biopsy specimens and if so the contributory cause/s.