

ASSOCIATION OF HELICOBACTER PYLORI WITH ACID PEPTIC DISEASE IN KARACHI

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ABSTRACT

The prevalence of H. pylori infection in 200 dyspeptic patients undergoing upper GI endoscopy was investigated by histology and rapid urease test. H. pylori was associated with 86% cases of chronic gastritis, 84.6% cases of duodenal ulcers and 78.5% of gastric ulcers. None of the 15 histologically normal gastric biopsies showed H. pylori (JPMA 40 ; 240, 1990).

INTRODUCTION

Gastritis and peptic ulcer disease are common problems in dyspeptic patients of Karachi^{1,2}. The aetiology, however, is poorly understood and treatment often unsatisfactory. Ever since its isolation³. Helicobacter pylori (H. pylori, previously called Campylobacter pylori) has been implicated for acid peptic diseases. The purpose of this prospective study was to investigate the prevalence of H. pylori infection among dyspeptic patients in Karachi.

PATIENTS AND METHODS

Association of H. pylori infection was investigated in 200 patients with dyspepsia and upper abdominal pain referred for upper gastrointestinal endoscopy. Patients requiring emergency endoscopy, or with previous gastric surgery or malignancy were excluded. The upper gastrointestinal endoscopy was performed with usual procedural preparation. Endoscopic findings were recorded and multiple antral biopsies were taken routinely within 5cm of pylorus if no endoscopic gastric lesion was found and from the site of lesion if gastritis was seen and also from adjoining intact mucosa in cases of gastric/duodenal ulcers. Two antral biopsy fragments were placed in 10% formalin for histology and one in 2% Christensen urea broth for detection of urease, the results of which were read at maximum of 4 hours⁴. Four micron thick sections of formalin fixed paraffin embedded tissue were cut and stained by H&E for histological diagnosis and acridine orange stain for detection of H. pylori in the tissues. Presenting symptoms of the patients were also recorded.

RESULTS

Two hundred cases (152 males and 48 females) between the ages of 15 — 70 years were investigated for associated H. pylori infection. Of the total, 159 (79.5%) showed histologic evidence of H. pylori along with gastritis, while 26 showed isolated gastritis but no H. pylori and 15 showed no histologic gastritis or H. pylori infection. Rapid urease test was positive in 116 out of 159 cases. The main presenting features of 159 infected patients were epigastric pain (133), heartburn (80), flatulence (71), nausea/vomiting (70) and anorexia (58). Of 159 H. pylori infected cases, 66% showed active and 34%

inactive gastritis, while of 26 cases with isolated gastritis, 23% showed active and 77% inactive gastritis ($P < 0.001$). There was no significant difference in *H. pylori* infection in cases with superficial or atrophic gastritis. Of 185 histologic proven cases of gastritis, *H. pylori* infection was present in 159 (86.4%), while 44 of 52 cases (84.6%) with duodenal ulcer and 11 of 14 cases (78.5%) with gastric ulcer showed *H. pylori* infection.

DISCUSSION

The present study showed a high prevalence (79.5%) of *H. pylori* infection in dyspeptic patients of Karachi. The bacteria was only found in the stomach of these patients and not in any of the 50 duodenal biopsies taken, out of which 41 showed changes of duodenitis. The fact points to the particular predilection of *H. pylori* for gastric mucosal epithelium. A close relation was seen with gastritis and peptic ulceration. *H. pylori* was first isolated by Marshall in Australia but it soon attracted interest from pathologists and gastroenterologists from all over the world, most of them produced results which are comparable with that of ours⁵⁻⁷. The initial work showed that *H. pylori* infection is universal and has high association with inflammatory and ulcerative disease of stomach. However, the primary pathogenic role of the bacteria and the cause and effect relationship was difficult to prove, and few reports made the issue more controversial. Johnston et al⁸ found *H. pylori* in one of the eleven histologically normal antral biopsies. Price et al⁹ found the bacilli in one out of 12 normal biopsies. Ho et al¹⁰ found *C. pylori* in 14% of those with normal gastric mucosa. He also showed that cases of gastritis treated with prostaglandin E. or placebo, the bacteria remained positive and persisted in the same density as before treatment even in those cases where gastritis showed improvement. In our present series of 200 patients, *H. pylori* was not found in any of the 15 patients whose gastric biopsies showed no significant changes on histology. The controversy began to resolve as evidence for *H. pylori* as a primary pathogen in causing gastritis started accumulating rapidly. Healing of gastritis after eradication of the bacilli by bismuth¹¹ and even with antibiotics such as Amoxicillin¹² and Erythromycin¹³ have been achieved. Two reports of self inoculation of the *H. pylori* have been published, both resulted in gastritis which healed spontaneously in one of the volunteers while proceeded into a chronic form in the other, the more usual form seen with *H. pylori* infection. Our recent study¹⁴ of cellular and humoral immune responses not only provides an evidence for *H. pylori* as a primary pathogen but also explains the plausible mechanism by which changes of gastritis evolves after *H. pylori* infection. We¹⁵ have also very recently described the ultrastructural changes associated with *H. pylori* infection and also very clearly demonstrated the penetration of the bacilli into the gastric surface epithelial cells. The evidence so far gathered has quite clearly convinced the pathologists that the new bacillus is a pathogen responsible for causing gastritis. However the clinical importance of the infection has still to be elucidated, since many of the workers have demonstrated *H. pylori* infection in asymptomatic patients also. Although the infection is difficult to eradicate and relapses are common, still the clinical importance of the infection cannot be evaluated without well programmed double blind, placebo control clinical trials aimed to eradicate the bacilli from the stomach and see the effect on endoscopic, histological and symptom status of the patient. Moreover long term follow up of these patients might furnish some important information about the relapses of the infection, symptomatology and pathology.

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