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RESEARCH ARTICLE

ASSOCIATION OF H. PYLORI INFECTION WITH GASTRODUODENAL DISEASE: A CROSS SECTIONAL STUDY

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ABSTRACT

The 180 patients of both gender between 10 80 years having symptoms of dyspepsia, are subjected to endoscopic examination. The incidence of *Helicobacter pylori* is highest between 41 50 years (72%) with male to female ratio of 2:3.Low socio economic status have a high incidence of *Helicobacter pylori* (65%) and (80%) in patients having a family history of dyspepsia. The colonization of helicobacter is related to life style i.e. habituation to smoking (76%), addicted to alcohol (36.5%) and beetle nut (3%). Higher incidence is in O+ve blood group. Non vegetarians are more for *Helicobacter pylori* infection than vegetarians and patients consuming spicy foods (80%) and pickles, chutneys containing excess chillies (90%). The biopsy specimens are collected in transport medium containing physiological saline and trimethoprim, processed under aseptic measures within two hours of collection. The standard tests, direct smear, rapid urease and culture were done. *Helicobacter pylori* was declared positive if the bacteria were identified by at least any two of the three diagnostic methods. Direct biopsy smear positive are 63, followed by rapid urease 53 and 49 are culture positive. Out of 49 culture positive cases, 31 are pure isolates of *Helicobacter pylori* and the rest are along with other organisms like *Pseudomonas* species, *Proteus* species and *Klebsiella* species. The colonisation is more common in pylorus (57.2%) than the anterior wall, posterior wall and duodenum. Forty nine isolates of *Helicobacter pylori* were subjected to antibiotic sensitivity testing. The incidence of *Helicobacter pylori* by various studies all over the world and in our country has been well documented and the present study is consistent with other studies all over the world. This study evaluates and is proved beyond doubt that there is a strong association between the presence of *Helicobacter pylori* and acid peptic diseases.

INTRODUCTION

Helicobacter pylori (H. pylori) could be a spiral, gram-negative microorganism that inveterately infects over half the world's population, and is presently recognized to play a motive role within the pathological process of inflammation, gastroduodenal lesion, stomachal glandular carcinoma, and mucosa-associated lymphatic tissue (MALT) malignant neoplastic disease (Peek and Blaser, 2002; Suerbaum and Michetti, 2002). Chronic gastritis and peptic ulceration are prevalent in a high magnitude throughout the world (Ghazzawi and Obidat, 2004). Helicobacter pylori inflammation is that the principal reason for chronic active inflammation and has major complications like stomachal glandular carcinoma and mucous membrane associated lymphatic tissue malignant neoplastic disease (Ozbek *et al*., 2010). *H. pylori* sometimes colonize stomachal pits beneath the mucous secretion layer and in close association with gastric epithelial cells. Approximately, 50% of the normal population across the world harbor H. pylori, though only 10-20% of them become symptomatic (Ommunakwe *et al*., 2011; Makola *et al*., 2007). There is an association of *H. pylori* infection with the hygiene related conditions, lifestyle, and economy with annual an incidence rate of *H. pylori* infection ≈ 4-5% in developing nations compared to that of ≈ 0.5% in developed and industrialized countries (Duck *et al*., 2004). There is a high prevalence of *H. pylori* infection in developing countries with up to 80% of the children under the age of 10 years are infected. In India, the prevalence of this infection is 22%, 56%, and 87% in the 0-4 years, 5-9 years and in the 10-19 years age group respectively (Das and Paul *et al*., 2007). There are many other etiological factors such as smoking, nonsteroidal anti-inflammatory drugs (NSAIDS), and reflux of gastric juice (chemical gastritis) that are also implicated to cause chronic gastritis. H. pylori, though is regarded as the primary cause of gastritis, it can act as a synergist in addition to other etiological factors (Parkin *et al*., 2003). Though many studies have already been conducted in the past on the current topic, still there is a paucity of information about *H. pylori* infection prevalence. The present study was undertaken to estimate the prevalence of *H. pylori* infection among patients

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presenting with the dyspepsia and compute its association with gastroduodenal lesions.

MATERIALS AND METHODS

The 260 patients gastric biopsy samples were collected from different hospitals of Kerala and Tamil Nadu includes Madurai (100), Coimbatore (60), Nagercoil (30), Trivandrum (45) and Kolam (25) during the period of one year between all the age groups of both sex with or without upper gastrointestinal endoscopic evidences of gastritis, duodenitis and peptic ulcer and cancer patients were selected for the present study.5ml of transport medium (trimethoprim) in bottles were taken to endoscopy room to collect the biopsy specimens.

Sample collection: After an overnight fasting of 12 hours, upper gastro intestinal endoscopy was performed in an endoscopy room. The endoscopy was carried out with an Olympus fiber optic endoscope, which is thoroughly cleaned with water and soaked in 2% glutaraldehyde (codex) for 20 minutes, prior to specimen collection. The endoscope is thoroughly cleaned with salvation and normal saline prior to endoscopy done in each and every patient. In the case of normal mucosa and gastritis, as the Helicobacter pylori have patchy distribution, the endoscopic biopsy specimens are taken from the stomachs anterior wall, posterior wall and pylori c astral mucosa. In case of duodenitis, biopsy bits of tissue approximately measuring 1 to 2 mm in diameter is taken from the duodenum and in the case of gastritis or duodenal ulcer biopsy bits are taken around the ulcer region under aseptic measures. The biopsy specimens are collected into a transport media and transported to microbiology laboratory within 2 hours of collection for processing. The transport media used is sterile physiological saline (0.85%) with antibiotic trimethoprim in sterile cotton plugged bijou bottles. Three biopsy bits are collected from each patient, one bit for the microscopic examination, one bit for the rapid urease test and the other for culture and histopathology examination.

Processing of the specimen: All the endoscopic biopsy specimens were processed within 2 hours of the collection because as Helicobacter pylori loses its viability when exposed to atmospheric oxygen for a long time and also to minimize the chances of contamination. Microscopic examination of direct biopsy smear: One biopsy specimen is picked with a sterile forceps and placed on a sterile clean glass slide, with another clean sterile glass slide the specimen is crushed in between the slides. With the crushed biopsy material, smears are made on the slide with a sterile loop. The smears are air dried and heat fixed. The smears are strained by different methods.(i) Gram straining dilutes carbon fuchsin is used as counter strain (iii) Special straining - Giemsa staining, hematoxylin and eosin staining.

Rapid Urease Test: Biopsy specimens were tested for urease activity by using the "HelicotecUT®Plus" test.

Urease Test: The urease test was applied according to MacFaddin (2012). The pure isolates were inoculated heavily on the entire surface of urea agar and stab with the loop wire. The tubes were inoculated at 37°C in the incubator. The formation of purple color was examined after 4 h.

RESULTS AND DISCUSSION

A total of 280 symptomatic patients suffering from acid peptic disease and duodenal ulcer, who have undergone an endoscopic examination is taken for the present study, out of 280 patients studied, 182 patients are positive for Helicobacter pylori. Out of 182 patients, 103 are male, 70 were female, 9 were children. The biopsy specimens of the patients are processed and standard tests done for diagnosis of Helicobacter pylori. The tests are a direct smear, rapid urease, and culture. Out of the 173 patients tested, direct smear positive is rapid urease. Out of 173 HP, positive patient 113 is the age above 40 and in between 45-60. 28 were affected by intestinal cancers, 73 were alcoholic and 4 children were a cancer patient. Out of 100 patients, total number of positive cases is 63, when any two tests are taken Patients of both sexes are taken at random for the study and they are between the age group of 45-60 years, 28 patients are tested out of which 17 are positive with percentage positivity of 70.8 and between the age group 45-60 years, 19 cases are positive with percentage positivity of 68.4. Thirty-four patients tested out of 100 are between the age group of 45-60 years. Out of which 21 are positive for Helicobacter pylori with percentage of positivity 61.8 in the age group, 51 and above, the percentage positivity is about 55. Out of 113 cases, which underwent endoscopic examinations, 58 cases are of normal endoscopic findings, out of which 29 are positive for Helicobacter pylori with the percentage of positivity 50. Seven cases of gastritis out of 11 are positive for Helicobacter pylori with the percentage of positivity 13.6 and 9 out of 9 pylori ulcer cases are positive for Helicobacter pylori with the percentage of positivity 100. Out of 9 cases of duodenitis, 7 are positive for Helicobacter pylori with the percentage of positivity 77.8. Ten out of 10 cases of duodenal ulcer are positive for Helicobacter pylori with 100 percent positivity.

The results of the current study counsel that there's a powerful association of Helicobacter pylori with acid peptic disease and duodenal ulcer. Identification of Helicobacter pylori and its etiopathogenesis has given a powerful reason as the causative agent of the infection. The incidents of Helicobacter pylori is found to be 63 out of 100 patients studied, which is consistent with the study of price *et al*., 1996 (63%). The incidence rates of other works are, Jones *et al*. (2003) (62%), Langerbar *et al*. (1999) (64%), Borromoe *et al*. (2001) (64%), Marshall and Warrner (1984) (58%). Thirty-seven patients out of the 100 were negative for Helicobacter pylori though they suffered from symptoms of dyspepsia. Out of 53 urease positive cases, all are not culture positive, so there are few false positive cases. These are due to urease producing bacteria, like Klebsiella and Proteus (Raisens *et al*., 2002), which may contaminate endoscopy equipment and which are occasionally found in the gastric tissue hypochlorhydria subjects. Rigorous cleaning of endoscopic equipment and the incorporation of an anti microbial agent in the urease bit minimises false positive tests. Forty-nine cultures were positive out of 63 endoscopic biopsy bit studied. All direct smear positive cases were not cultured positive. Therefore the direct smear is the most rapid, simple sensitive test for diagnosis of Helicobacter pylori infection (Personnet *et al*., 2012). Histopathological examination results show Negative histopathological result the lumen is clear there is no bacteria and no inflammatory response (C). The positive histopathological result showed inflammatory cells and therefore the presence of H. pylori. The horizontal arrow indicates the presence of spiral shaped *H. pylori* colonization at the lumen of the gastric tissue, while the vertical arrow showed the inflammation in the tissue (C).

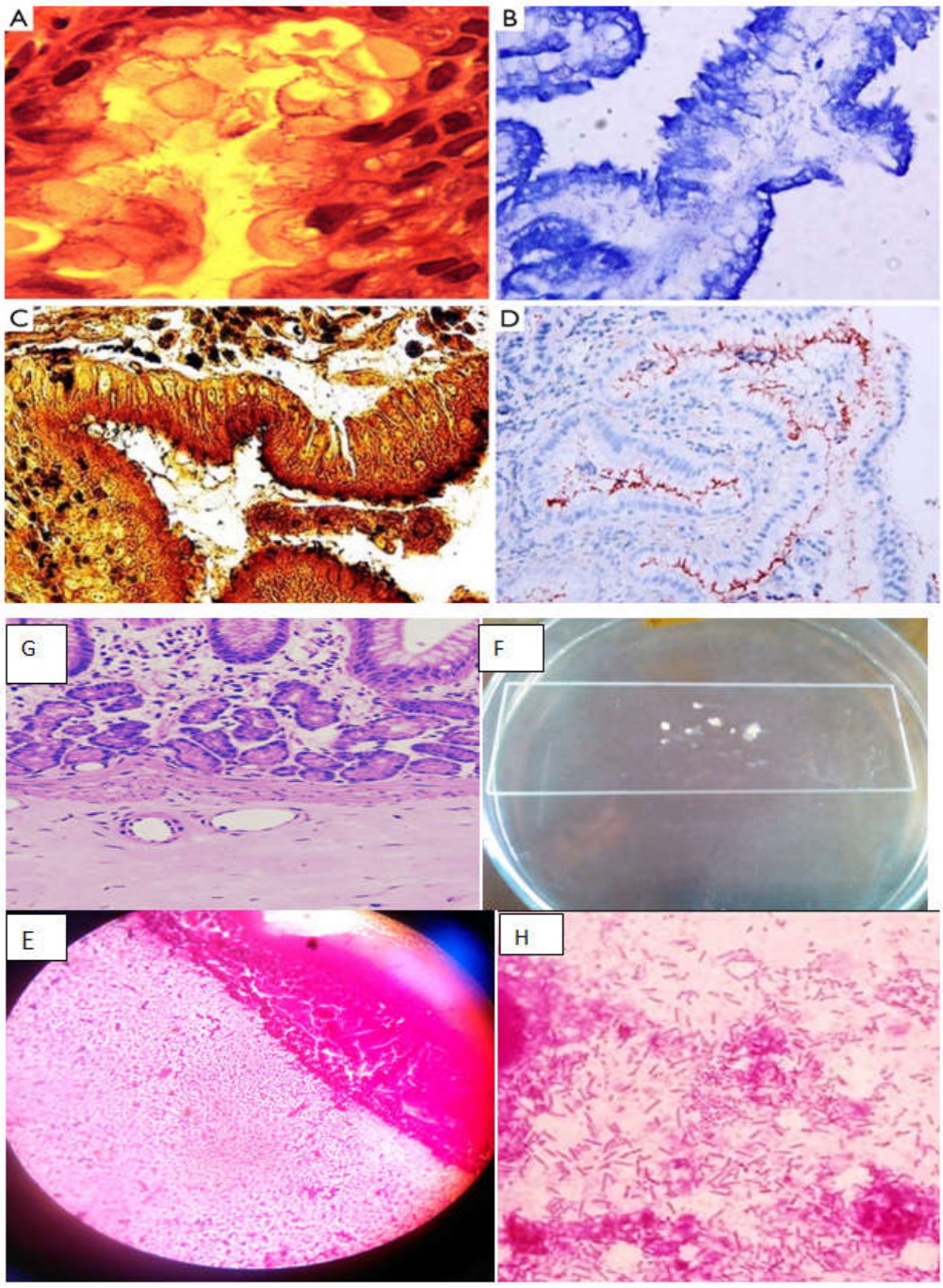

Figure 1. The spiral-shaped *Helicobacter pylori* organisms are present in (A) hematoxylin and eosin stain (×1,000), (B) Giemsa stain (×400), (c,D,E,G and H) denotes the microscopic examination, (F) denotes the crushed tissue for inoculation

The reason for duodenal ulcers in *H. pylori* infection is, *H. pylori* infection blocks normal physiological inhibitory mechanisms from the antrum to both gastric cells and to parietal cell region, resulting in increased gastric release and impaired inhibition of gastric acid secretion, which will probably lead to an increased duodenal acid load as a general prerequisite for the development of duodenal ulcer disease. It additionally shows a correlation between the severity of redness and therefore the presence of *H, pylori* area unit in agreement with antecedently revealed studies (Buck, 1990; Rothstein *et al*., 1990). Our findings of comparable sensitivity for culture and histological examination and lower sensitivity for enzyme testing and direct Gram-stained smears for the detection of *H. pylori* differ from previously published results in some studies using more intensive methods, such as grinding and rinsing the biopsy tissue or obtaining a second biopsy specimen from the gastric fundus.

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