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# Prevalence and Microbiological diagnosis of Helicobacter pylori infection in the patients suffering from Acid-peptic Diseases

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***Abstract***
*The link between Helicobacter pylori (H. Pylori) infection and peptic ulceration is now well established: presence of infection is a strong risk factor for later ulcer development and ulcers rarely occur in the absence of H. pylori or the other main risk factor, non-steroidal anti-inflammatory drugs (NSAIDs). The role of H pylori in peptic ulceration in human is presented in this paper. A peptic ulcer is an excoriated area of stomach or intestinal mucosa caused principally by the digestive action of gastric juice. The two most common factors that predispose to ulcers are chronic gastric infection with H. pylori and ingestion of non-steroidal anti-inflammatory drugs. This retrospective analysis of 250 cases of acid peptic diseases in gastro enterology dept, govt Mohan Kumaramangalam medical college, Salem, Tamilnadu, from June 2017 –December 2017*
***Keywords:*** *Helicobacter pylori, peptic ulcer, stomach.*

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## I. Introduction:

***Helicobacter pylori*** was brought to the world attention in 1983 when Warren and Marshall, two Australian investigators, reported isolation of spiral organisms from mucosal biopsy specimens of patients with chronic active gastritis and peptic ulcer disease. Now the organism is accepted as the causative agent of gastritis, peptic ulcer disease, gastric adenocarcinoma and gastric Bcell- lymphoma (MALT lymphoma). More than 50% of the world's population harbor *H. pylori* in their upper gastrointestinal tract. Infection is more common in developing countries than Western countries. Up to 85% of people infected with *H. pylori* never experience symptoms or complications. Individuals infected with *H. pylori* have a 10 to 20% lifetime risk of developing peptic ulcers and a 1 to 2% risk of acquiring stomach cancer. Inflammation of the pyloric antrum is more likely to lead to duodenal ulcers, while inflammation of the corpus (body of the stomach) is more likely to lead to gastric ulcers and gastric carcinoma. However, *H. pylori* possibly plays a role only in the first stage that leads to common chronic inflammation, but not in further stages leading to carcinogenesis. *H. pylori* has been associated with colorectal polyps and colorectal cancer, non-ulcer dyspepsia.

The prevalence of infection varies, in developing countries the infection rates are much higher, compared with those of developed countries*;* the prevalence of infection increases to approximately 60% at the age of 60 or above.. The highest rates of infection are associated with low socio-economic status, crowding, poor sanitation and unclean water supplies.

The mechanisms underlying the altered proliferation by *H. pylori* infection are thought to be caused by an increase in the mucosal content of ammonia, known to be a strong stimulus of cell proliferation, provoked by the bacterium itself. *H. pylori* infection associated hypergastrinoma is also believed to play a role in increasing epithelial cell turnover. H. *pylori* lipopolysaccharide as a virulence factor is responsible for the induction of gastric epithelial cell apoptosis. The organism is transmitted from person to person. within families are closely related. The exact means by which *H. pylori* is transmitted among individuals is uncertain. The first possibility is fecal-oral transmission or any mechanism that allows *H. pylori* enter the stomach of an uninfected host probably is a route of transmission.

Pathogenicity of *H. pylori* may be increased by genes of the *cag* pathogenicity island; about 50–70% of *H. pylori* strains. Following attachment of *H. pylori* to stomach epithelial cells, the type IV secretion system expressed by the *cag* PAI "injects" the inflammation-inducing agent, peptidoglycan, from their own cell walls into the epithelial cells. The injected peptidoglycan is recognized by the cytoplasmic pattern recognition receptor (immune sensor) Nod1, which then stimulates expression of cytokines that promote inflammation. Pathogenic strains of *H. pylori* have been shown to activate the epidermal growth factor receptor (EGFR),

a membrane protein with a TK domain. Activation of the EGFR by *H. pylori* is associated with altered signal transduction and gene expression in host epithelial cells that may contribute to pathogenesis.

**Cancer**

Two related mechanisms by which *H. pylori* could promote cancer are under investigation. One mechanism involves the enhanced production of free radicals near *H. pylori* and an increased rate of host cell mutation. The other proposed mechanism has been called a "perigenetic pathway", and involves enhancement of the transformed host cell phenotype by means of alterations in cell proteins.

**AIM:**

- To study the prevalence of peptic ulcer caused by H.pylori.
- To study the predisposing factors of H.pylori induced peptic ulcer.
- Diagnosis of peptic ulcer by various microbial methods.

## II. Materials And Methods

**Inclusion criteria:** All patients presenting with symptoms of peptic ulcer disease or first-degree relatives with gastric cancer, and in certain cases of dyspepsia

**Exclusion criteria:**

1) Previous therapy to eradicate *H. pylori*

2) Patients taking aspirin or non-steroidal anti-inflammatory drugs (NSAIDS) in the past 4 weeks or are on PPI.

**Samples:** 4 antral biopsies and blood sample for antibody

2 biopsy samples in BHI broth 1 in 10% formal saline 1 in urea broth.

**Rapid urease test**: Time taken for color changes from yellow to pink was noted within 4 hrs.

**Gram's staining:** was done on the smears made from crushed biopsy material.

**Culture:** was done on Brucella blood agar and Columbia blood agar with skirrow's antibiotic supplement, for at least 7 days in an anaerobic jar with Gas pack kit. Biopsies can be kept in a medium (*e.g.*, Stuart's transport medium) for 24 at 4 deg. Once isolated, *H. pylori* can be stored frozen at -80 degrees, preferably in broth with 15% to20%glycerol. Blood agar which contain specific antibiotics to inhibit commensal bacteria are used for culture. Cultures should be incubated under microaerobic condition. (85% N2, 10% CO2, 5% O2) at 35 to 37 degrees for 7 days before discarding cultures as negative. Positive identification of *H. pylori* is based on morphological characteristics and positive catalase, oxidase, and urease reactions.

**The enzyme immunoassay(EIA) test** has been the most prevalently used for detecting IgG, with sensitivity and specificity values ranging from 60% to100%.

## III. Results And Discussion

Out of total two hundred fifty patients, ninety-two patients were H. pylori positive giving a prevalence of 36.81% (Table-I). Out of total ninety-two H. pylori positive patients assigned for study, fifty-nine were males and thirty-three were females (Table-II). The minimum age of H. pylori positive patient was 18 years and maximum age was 74 years. The maximum numbers of patients were in the age group of 36-45 years with a mean age of 43.07±11.76 (Table-III). Pain upper abdomen was the most frequent symptom seen in 36 of H. pylori positive patients with epigastric fullness and retrosternal burning, the second and third most common complaints. Rest of the clinical features like belching, vomiting and anorexia were almost of equal frequency in both H. pylori positive as well as negative patients. Multiple complaints were recorded in same patient (Table-IV). Duodenitis and esophagitis were the other common findings documented in 11 and 8 patients, respectively. A single chronic gastric ulcer was noted in two and acute duodenal ulcer in four patients. Overall, inadequate sanitation practices, low social class (32/92), and crowded or high-density living conditions seem to be related to a higher prevalence of H. pylori infection. All the positive patients were given anti-H. pylori treatment.

**Table I : Prevalence of H. pylori.**

| | |
|---|---|
| **Total patients** | 250 |
| **Positive patients** | 92 |
| **Prevalence** | 36.8 |

**Table-II: Number of *H. pylori* positive patients according to sex.**

| Sex | Number of positive patients (n) | Percentage (%) |
|---|---|---|
| Male | 53 | 57.6 |
| Female | 39 | 42.3 |

**Figure no: 1 – Distribution of patients based on sex.**

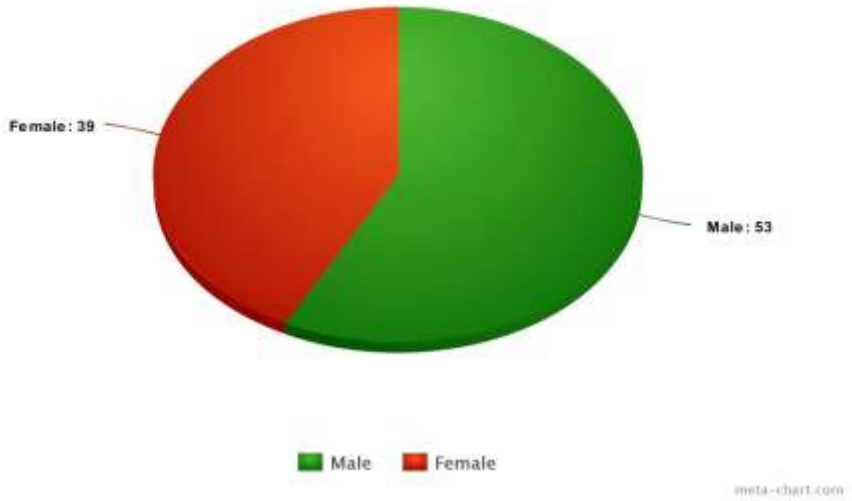


**Table-III: Number of *H. pylori* positive patients according to age group.**

| Age group | Total no(n) | Percentage (%) |
|---|---|---|
| 18 - 25 | 5 | 5.43 |
| 26-35 | 14 | 15.2 |
| 36-45 | 40 | 43.5 |
| 46-55 | 22 | 24 |
| 56-65 | 8 | 8.7 |
| 66-75 | 3 | 3.3 |

**Figure no:2 Distribution of patients based on age group.**

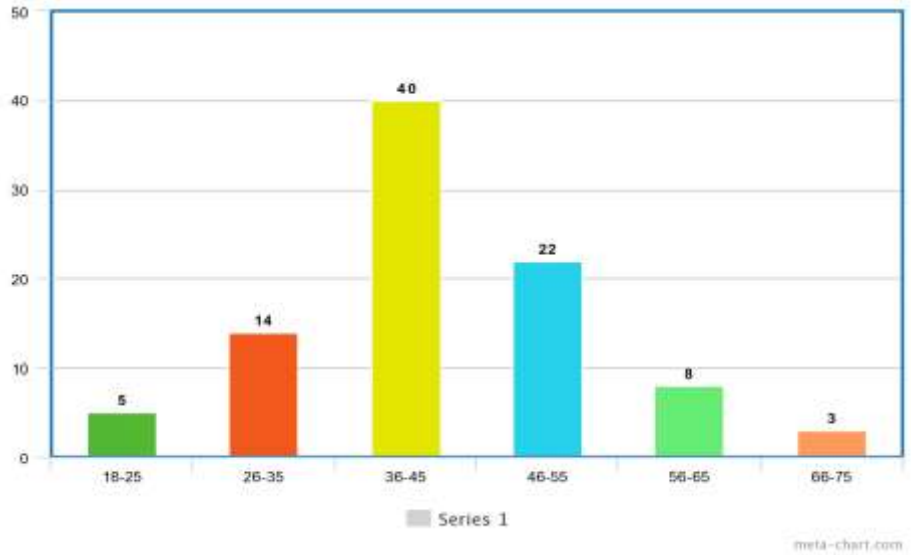


**Table-IV: Symptom profile in *H. pylori* positive patients.**

| Symptoms | Total cases(n) | Percentage (%) |
|---|---|---|
| Pain upper abdomen | 36 | 39.1 |
| Retrosternal burning | 10 | 10.8 |
| Belching | 14 | 15.2 |
| Fullness after meals | 17 | 18.5 |
| Vomiting | 7 | 7.6 |
| anorexia | 8 | 8.69 |

## IV. Conclusion

Prevalence is high in developing countries (90%), whereas in industrialized countries the figure is lower (50%) and is decreasing. Childhood is the critical period for infection, and transmission most probably occurs from person to person. patients infected at an early age are likely to develop more intense inflammation that may be followed by atrophic gastritis with a higher subsequent risk of gastric ulcer, gastric cancer, or both. Acquisition at an older age brings different gastric changes more likely to lead to duodenal ulcer. The infection rate is generally higher and begins at an earlier age in developing as compared to developed countries, indicating an important role of socio-economic milieu in its transmission.

For detection of *H pylori* infection in patients with dyspepsia, an indirect test such as serology along with a direct one such as urease test might be more useful, as such a combination will detect the organisms as well as the antibodies. Recently cumulative evidence strongly suggest that *H. pylori* infection alters the kinetic pattern of gastric epithelium. Rising antibiotic resistance increases the need to search for new therapeutic strategies *H. pylori* infection is very common in the south Indian population. A high prevalence is seen in all gastroduodenal diseases and more than half the population without any abdominal symptoms was colonized by the bacterium.

The prevalence of duodenal ulcer is high in south India and the high prevalence of *H. pylori* infection might suggest an important role in its pathogenesis. More population-based studies are required to evaluate its role in other gastrointestinal disorders in this region.

## Reference


[1]. "Helicobacter pylori - Chapter 3 - 2016 Yellow Book | Travelers' Health | CDC". wwwnc.cdc.gov. 9 June 2015. Retrieved 25 April 2017.

[2]. Amieva, Manuel; Peek, Richard M. (2016). "Pathobiology of Helicobacter pylori–Induced Gastric Cancer". Gastroenterology. 150 (1): 64–78. doi:10.1053/j.gastro.2015.09.004.

[3]. Blaser MJ (2006). "Who are we? Indigenous microbes and the ecology of human diseases" (PDF). EMBO Reports. 7 (10): 956–60. doi:10.1038/sj.embor.7400812.

[4]. Yamaoka, Yoshio (2008). Helicobacter pylori: Molecular Genetics and Cellular Biology. Caister Academic.

[5]. Brown LM (2000). "Helicobacter pylori: epidemiology and routes of transmission" (PDF). Epidemiol Rev. 22 (2): 283–97. doi:10.1093/oxfordjournals.epirev.a018040.

[6]. Bytzer P, Dahlerup JF, Eriksen JR, Jarbøl DE, Rosenstock S, Wildt S (April 2011). "Diagnosis and treatment of Helicobacter pylori infection". Dan Med Bull. 58 (4): C4271. Archived from the original on 5 January 2014. Retrieved 7 August 2013.

[7]. Chang, A. H.; Parsonnet, J. (2010). "Role of Bacteria in Oncogenesis". Clinical Microbiology Reviews. 23 (4): 837–857. doi:10.1128/CMR.00012-10. ISSN 0893-8512.

[8]. Kusters JG, van Vliet AH, Kuipers EJ (July 2006). "Pathogenesis of Helicobacter pylori Infection". Clinical Microbiol Rev. 19 (3): 449–90. doi:10.1128/CMR.00054-05.

[9]. Suerbaum S, Michetti P (October 2002). "Helicobacter pylori infection". N. Engl. J. Med. 347 (15): 1175–86. doi:10.1056/NEJMra020542.

[10]. Fuccio L, Zagari RM, Eusebi LH, Laterza L, Cennamo V, Ceroni L, Grilli D, Bazzoli F (2009). "Meta-analysis: can Helicobacter pylori eradication treatment reduce the risk for gastric cancer?".

[11]. Wu Q, Yang ZP, Xu P, Gao LC, Fan DM (2013). "Association between Helicobacter pylori infection and the risk of colorectal neoplasia: a systematic review and meta-analysis". Colorectal Dis. 15 (7): e352–64. doi:10.1111/codi.12284.

[12]. Saccà, SC; Vagge, A; Pulliero, A; Izzotti, A (December 2014). "Helicobacter pylori infection and eye diseases: a systematic review.". Medicine. 93

[13]. Olson JW, Maier RJ (November 2002). "Molecular hydrogen as an energy source for Helicobacter pylori". Science. 298 (5599): 1788–90. doi:10.1126/science.1077123.

[14]. Stark RM, Gerwig GJ, Pitman RS, Potts LF, Williams NA, Greenman J, Weinzweig IP, Hirst TR, Millar MR (February 1999). "Biofilm formation by Helicobacter pylori". Lett Appl Microbiol. 28 (2): 121–6. doi:10.1046/j.1365-2672.1999.00481.x.

[15]. Chan WY, Hui PK, Leung KM, Chow J, Kwok F, Ng CS (October 1994). "Coccoid forms of Helicobacter pylori in the human stomach". Am J Clin Pathol. 102 (4): 503–7.

[16]. Josenhans C, Eaton KA, Thevenot T, Suerbaum S (August 2000). "Switching of Flagellar Motility in Helicobacter pylori by Reversible Length Variation of a Short Homopolymeric Sequence Repeat in fliP, a Gene Encoding a Basal Body Protein". Infect Immun. 68.

[17]. Rust M, Schweinitzer T, Josenhans C (2008). "Helicobacter Flagella, Motility and Chemotaxis". In Yamaoka Y. Helicobacter pylori: Molecular Genetics and Cellular Biology. Caister Academic Press.


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