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# HELICOBACTER PYLORI: PREVALENCE IN INDIAN CONTEXT

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**ABSTRACT**

*H.pylori* is agram negative bacterium capable of colonizing human stomach, associated with gastritis, gastric ulcer, duodenal ulcer and gastric cancer. The exact mechanism of *H.pylori* transmission is still unclear. On a global scale, prevalence of *H.pylori* was more than 50% and omnipresent in its distribution. Prevalence of *H.pylori* infection in India ranges from 49.94% - 83.30%. The possible mode of transmission is through oral – oral, faecal – oral, gastro to oral. The probable risk factors studied include smoking, alcohol, diet, water borne exposure, density / overcrowding and social factors.



## INTRODUCTION

A gram negative bacterium capable of colonizing human stomach, beneath mucus layer; is small, highly mobile, curved bacillus and requires microaerophilic environment is *Helicobacter pylori* (1). Warren and Marshall described its association with gastritis, gastric ulcer, duodenal ulcer, gastric cancer and other gastroduodenal diseases in 1982 (2,3). Fifty percent of the world population is being infected by *H.pylori* and infection rate is huge in developing countries as against developed countries(4). In developing countries, *H.pylori* infection is more frequent in children below 10 years; childhood infection worsens in more than 80% of adults before they attain 50 years(5). H.plylori infection persists for lifetime once it is acquired, unless it is treated with bactericides (6). Socioeconomic background and living conditions are the key factors which play a vital role in acquiring *H.pylori* infection (7). This review outlines the reservoirs, transmission, risk factors, prevalence of *H.pylori* infection in global and Indian scenario.

### *Reservoir of H.pylori*

Human gut is the most suitable habitat for *H.pylori*. Especially strains which infect human beings do not have any other environmental or significant animal reservoir (8). Several studies indicated the zoonosis transmission, from domestic and commercially reared cats (9,10), milk of cow, sheep and raw goat (11). Recent study conducted by Momtaz *et al* found high sequence homology of *H.pylori* DNA between sheep and human, indicating the sheep might play a role of reservoir (12). Contaminated water may also play a role of vector as indicated by study conducted Queralt *et al*, found 66% of water samples were positive for *H.pylori* (13).

### *Transmission of H.pylori*

In spite of several studies conducted by the researchers the exact mechanism of *H.pylori* transmission is yet to clear. Several studies suggests that the human/person – human/person was the possible mode of transmission is through oral – oral, faecal – oral, gastro to oral (14).

### *Gastro to Oral Transmission*

*H.pylori* was known to be present in the gastric juice of the infected patients (15). Indeed *H.pylori* present in the refluxed gastric juice acts as a vector for the transmission. The prevalence of infection was high in gastroenterologist, endoscopists, nurses and other who were in direct contact with gastric juice (16,17).

Vomiting acts as a vehicle in gastro-oral transmission mode especially in case of epidemic vomiting in children. In acute infection of *H.pylori*, the major clinical manifestation includes vomiting of achlorhydric mucus suggesting the role of vector in transmission (18). The above hypothesis was also supported by a study conducted by Leung at al. Four children were included in the study, had vomiting correlated with gastroenteritis and they were tested serologically to confirm the infection. Among the four children, *H.pylori* was successfully cultured from the vomitus in one child and in two children DNA of *H.pylori* was detected by PCR in vomitus. Surprisingly, seroprevalence of *H.pylori* was negative in 18 months old girl but DNA of *H.pylori* was detected in vomitus after 6 months she was serologically positive indicating the seroconversion for *H.pylori(19)*.

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***Oral – Oral Transmission***

In developed countries, the most common mode of transmission of *H.pylori* was oral to oral as hypothesized by many researchers. Prevalence of *H.pylori* was found to be high in institutionalized populations and within families. But it was not sure whether *H.pylori* was consistent or occasional dweller (20). For the first time Krajden *et al* isolated the DNA from saliva and dental plaques (21).

A new nested-PCR assay was developed by Ismail *et al* to isolate DNA of *H.pylori* in dental plaque samples (22). The authors suggest that new nested PCR was reliable and as sensitive as histology. They also opined that this new assay could be useful to those who were unable to undergo endoscopy. *H.pylori* has been isolated from dental plaques using PCR, LAMP reaction by Amiri *et al*. They found similar positivity results between the two tests (23).

Saliva and gastric biopsies were used to detect 16SrRNA of *H.pylori* by Ahmed *et al*. from symptomatic patients. They analysed 400 cases among them 246 were positive by biopsy and 240 were positive by saliva using PCR (24).

Liu *et al* evaluated 246 children from Kindergarten, obtained their dental plaques and analysed for *H.pylori* by nested PCR. They observed 126 samples were positive for *H.pylori* in dental plaques, 70 children with dental caries were also positive for *H.pylori* they reported statistically significant correlation between *H.pylori* and dental caries or hygiene (25). Liu *et al* conducted another study in 443 dyspeptic patients, obtained their dental plaques and biopsy specimens and carried out rapid urease test, histology and nested PCR. *H.pylori* positivity in dental plaque was 59.4% (263/443) and in gastric biopsy it was 61.6% (273/443). They concluded that there might be close relationship between *H.pylori* in the oral cavity and same in stomach or gastric infection, dyspeptic patients (26).

Several evidences were available to prove the above hypothesis, work done by Dane *et al* (27) and Suto *et al*. Suto *et al* concluded that the *H.pylori* was isolated frequently in the oral cavity of subjects with periodontitis, inferring that periodontal pocketing and inflammation might favor the colonization (28).

*Rasmussen et al* from their study concluded that the existence of relationship between gastric infection and the presence of *H.pylori* in the oral microbiota (29). In another study conducted by the same authors inferred that variable cytotoxin genotype cagA and vacA alleles were identified *H.pylori* in the oral cavity and their distribution also varied from saliva and dental plaque indicating the existence of reservoir of *H.pylori*, which might lead to gastric reinfection. They also found the existence of one or more strains of *H.pylori* in saliva, dental plaque, and stomach of the same patient (30). Silva *et al* from their study suggested that saliva and dental plaque might act as transitory reservoir for *H.pylori* (31). Assumpcao *et al* found identical vacA and cagA genotypes in the isolates obtained from gastric biopsy and dental plaques. They concluded that that gastric infection could be correlated with the presence of *H.pylori* in the oral cavity (32).

In contrast, Rossi-Aguiar*et al* collected the samples from saliva, the tongue dorsum and supragingival dental plaque from oral cavity and subgingival dental plaque samples from periodonditis for the detection of *H.pylori* by PCR. *H.pylori* infection was confirmed by rapid urease test and urea breath test. They observed *H.pylori* only in gut but not in any of the samples of oral cavity. They concluded that oral cavity might not be a reservoir for *H.pylori* in patients with epigastric pain syndrome (33). This was in concordant with study done by Oliver *et al*, they performed Histology and PCR to confirm infection from oral cavity and also from gastric biopsy. 84% of the gastric biopsies turned to be positive for *H.pylori* from gastric biopsies and it was not detected in any dental samples suggesting the absence of *H.pylori* in healthy individuals (34). It has been 26 years and researchers are yet to reach any conclusion regarding the role of oral cavity in gastric infection by *H.pylori*.

***Faecal – Oral Transmission***

Dye *et al* showed the presence of *H.pylori* in the distal duodenum causing active chronic gastritis (35) suggesting the passage of the organism through the intestine. Several attempts were made to isolate *H.pylori,* in 1992 Thomas *et al* was first to isolate *H.pylori* from faeces from 1 infected adult and 9 of 23 randomly selected children in Gambian village (36). In 1994, Kelly *et al* isolated *H.pylori* from 12 of 25 *H.pylori*-positive subjects with dyspepsia (37). Parsonneta *et al* found 50% positivity by culturing *H.pylori* from cathartic induced diarrheal stools in infected patients but not from normal stools (38).

Several researchers used PCR to detect DNA of *H.pylori,* Namavar *et al* they obtained samples from oral cavity, esophagus, stomach, bowel, faeces of dyspeptic patients. They isolated DNA from all the clinical specimens and PCR was carried out. *H.pylori* was detected from only one faecal sample (39).

Li *et al* used PCR to detect *H.pylori* in 88 gastric biopsy, 85 saliva, and 71 fecal specimens from 88 patients. *H.pylori* infection was confirmed in 71 of 88 patients by culture and histology. The PCR assay was able to detect *H.pylori* DNA in the faeces from 15 out of 61 patients (25%) with proven *H.pylori* infection and one among 10 patients without gastric *H.pylori* infection (40).

Falsafi *et al* applied stool PCR assay for the diagnosis of *H.pylori* infection in children. They found an association between higher density of *H.pylori* in histology and positivity by stool PCR (41).

In a study by Smith *et al* from Nigeria, collected 97 stool samples from dyspeptic patients and the results were compared with urea breath test. Out of 97 patients 38 were positive for 16SrRNA, 20 were positive for glmM genes, 47 out of 97 were positive by UBT (42).

In contradiction, Zwet *et al* (43) did not find positivity by stool PCR analysis. Presence of *H.pylori* in stool indicates the possible mode of transmission, but the viability of *H.pylori* cannot be garunteeded.

***Prevalence***

On a global scale, prevalence of *H.pylori* was more than 50% and omnipresent in its distribution (4). For *H.pylori* infection, gender discrimination was not found which infects both men and women (44). The rate of *H.pylori* infection is high in developing countries in comparison with developed countries this could be attributed to the poor hygiene, lack of proper sanitary requirements (45). Even in developed and developing

countries the rate of infection varies within and among the geographic locations (46). Adults acquiring *H.pylori* infection is rare suggesting the infection might have acquired in childhood (47).

***Worldwide prevalence***

Though the prevalence of *H.pylori* infection was 50% on global scale but huge geographical variations exists. In a study conducted by Sethi *et al* from Canada, the prevalence of *H.pylori* infection was found to be 37.9%, based on histologic findings (48). In contrast a study conducted from rural Mexico, the sero-prevalence was found to be 52.2% (49).Study from Eastern Europe revealed 39.8% prevalence in Cyprotic patients and PCR was the tool used for the diagnosis of infection (50).

In Western Europe, two different studies provided minimal and maximal sero-prevalence of *H.pylori* infection. The minimal sero-prevalence was 31.7% from Netherlands (51) whereas maximal was 84.2% from Portuguese (52).

The prevalence of *H.pylori* infection in Africa was found to be high; in Nigeria, the prevalence in dyspeptic patients was found to 93.6% by serology and 80% by histology (53). In Morocco, it was 75.5% by histology (45), 65.7% sero-prevalence in Gondar, Ethiopia (55).

In Asia, the prevalence of *H.pylori* infection scales from 54% to 76%(5). In China the prevalence of *H.pylori* infection among the healthy individuals was found to be 63.4% (56). The prevalence in Bhutan was obtained from the two different studies conducted among volunteers and dyspeptic patients, it was 73.4% (57) and 86%(58) respectively. In asymptomatic dyspeptic patients of Kazakstan, the sero-prevalence was 65.7% (59). A cross-sectional nationwide multicentre study survey revealed the sero-prevalence of 54.4% in Korea (60). Among the healthy individuals of Saudi Arabia the sero-prevalence was 28.3% (61).

***Prevalence in India***

Prevalence of *H.pylori* infection in India ranges from 49.94% - 83.30% (62). It varies among different age groups – 22% prevalence in the age group of 0-4 years; 87% in 10-19 years; 88% in adults (63).

Pandya *et al* collected 855 gastroduodenal biopsies and cultured on various selective media for *H.pylori* from the symptomatic patients of Patel College of Paramedical Science and Technology. They could isolate 125 out of 855, among 125 only 80 were obtained in pure form, indicating the prevalence of 64% (64). At Gujarat Adani Institute of Medical science Bhuj, a study was carried out to know the role of *H.pylori* in children with recurring abdominal pain. Each kid was screened for IgG antibodies specific for *H.pylori* by quantitative ELISA. It was positive in 8 out of 20 kids - 64% (65).

Jammu Kashmir the prevalence was 70% (66). In a study conducted by Gupta *et al* in Punjab for a period of two years, they screened 200 patients by obtaining antral biopsies for rapid urease test with symptoms of dyspepsia who were attending Sri Guru Ram Das Institute of Medical Sciences and Research, Amritsar. The overall positivity was 80.5% (161/200) (67).

Saha *et al* from New Delhi, recruited 50 dyspeptic patients who visited Guru Teg Bahadur Hospital and their stool samples were collected for the quantitative detection of antigen by ELISA. 30 cases were positive for the infection, hence the prevalence of *H, pylori* infection was 60% (68). A collaborative work was carried out by Rastogi *et al* from GSVM Medical College, Kanpur in Uttar Pradesh with KGMU, Lucknow during 2012 – 2013. This study included 208 cases that were asymptomatic for gastroduodenal diseases, their stool samples were taken and analysed for stool antigen using HpSA test. Out of 298, 92 (44.23%) were positive for *H.pylori* infection (69).

A serological cross sectional study indicated the prevalence of 27.5% in Assam. In the study 3ml of blood samples were collected from 80 patients with gastroduodenal diseases for the detection of IgG antibody by ELISA. 22/80 (27.5%) cases were positive for *H.pylori* infection by ELISA (70).

Histological biopsy, urease test and serological tests were done for the detection of *H.pylori* infection in dyspeptic patients of Odisha. Authors found a prevalence of 58.8% in their study (71). A comparative study of invasive and non-invasive methods was carried out in Bhopal, Madhya Pradesh. They evaluated several diagnostic methods like culture, histology, Rapid urease test, urea breath test and serology. Biopsy samples were obtained from 92 patients and blood samples from 57 patients. 55 cases were positive either by culture or by any other three tests, resulting in the prevalence of 59.7% (72). In Imphal, Manipur a cross sectional study was conducted to know the prevalence of *H.pylori* infection in gastroduodenal diseases and antibiogram pattern. This study consisted of 60 patients with gastroduodenal diseases attending the OPD of Regional Institute of Medical Sciences. They observed the prevalence of 43.3% in their study (73).

***Prevalence in South India***

The prevalence in South India was 80% (74,75). A hospital based cross sectional study was done on patients attending outpatient department with symptoms of upper gastrointestinal tract in ACS Medical College and Hospital, Chennai. They involved 400 patients to check for the qualitative detection of antibodies for all isotypes (IgG,IgM and IgA) by immune – chromatographic in human blood. Out 400 patients 117 (29.5%) were positive for *H.pylori* infection (76).

Molecular detection of *H.pylori* infection in gastric biopsy and also from dental plaques was done by Bharath *et al* in Bhimavaram, Andra Pradesh. They studied 56 dyspeptic patients by PCR and Rapid urease test and they found prevalence of 70.5% who were positive for both the tests (77). A total of 264 children were included in a study conducted by Rajaram *et al* in Tirupathi Andhra Pradesh. The study was intended to know the seroprevalence of *H.pylori* in children attending the paediatric department of S.V.R.R.G.G Hospital. Blood samples were collected from children in the age group of 2-12 years and detected for IgG antibody by ELISA. The overall seroprevalence was 44.31% (78).

Adlekha *et al* carried out a study in Ernakulum, Kerala to the obtain information regarding *H.pylori* prevalence. They obtained biopsies for histology and RUT, infection was considered as positive only when both the tests were positive. Out of 530, 329 were positive for *H.pylori* infection and thus the prevalence was 62.0% (79). Paul *et al* from south Kerala conducted a study to know the prevalence of *H.pylori* infection among dyspeptic patients. They obtained serum samples for HP – IgG – ELISA, biopsy specimens for RUT and culture. The study included 205 cases among them 89(35.6%) were

seropositive, 30(12%) were positive for RUT. Their conclusion was low seroprevalence of *H.pylori* infection in south Kerala (80). In Karnataka; a total of 100 patients were included in a study carried out by Pradeep *et al.* They picked up four biopsies from each patient to carry out RUT (2), histopathology (2), they also obtained blood for IgG estimation by ELISA. They got 59% prevalence in their study (81).

A single centred study was implemented to obtain knowledge regarding the prevalence of *H.pylori* infection in Shivamogga. RUT and Histology were done for the 100 patients with symptoms of APD. 59% prevalence was obtained from APD patients (82).

In a study conducted by Shetty *et al* from Manipal University, the study comprised of 38 patients with the symptoms of gastritis. They attained antral biopsies for RUT, HPE and Culture. The rate of positivity in various tests were as follows RUT- 36.9%, HPE - 42.1% and Culture – 39 (83).

Using $^{14}$ C UBT a study was implemented by Swaroop *et al* to know the prevalence of *H.pylori* infection in 209 individuals. 37.7% prevalence was found from their study from Dakshina Kannada district(84). 58% prevalence was noted in Mukka, Mangalore (85).

***Risk factors***

Several authors while conducting the prevalence studies in various geographic areas included a questionnaire designed to analyse the risk factors involved in *H.pylori* infection. Fisher's exact test, Chi square test, Student's t test, Crude and adjusted relative risks and Odds ratio were the common tests used to determine the statistical significance.

***Smoking***

The possible association between *H.pylori* infection and smoking has been assessed by several authors. Several studies which supports smoking as a significant risk factors includes; Lin eta al found an association between frequency of *H.pylori* seropositivity with current smoking habits (86); Woodward *et al* concluded that the independent relationship with smoking suggested a possible second source of spread of infection in future (45); Bateson also confirmed strong association of *H.pylori* infection with cigarette smoking (87).

Ogihara *et al* conducted a study to find the relationship between *H.pylori* infection and smoking habits. They found negative association of smoking with *H.pylori* seropositivity. However, *H.pylori* seropositivity risk was decreased linearly with cigarette consumption per day and dose dependent negative association might increase in the acidity in the stomach because of smoking (88).

Few authors did not find any significant association: Khalifa *et al* performed urea breath test to confirm the infection of *H.pylori* in non-ulcer dyspeptic patients and their smoking status was noted. They observed 43% prevalence and they did not find statistically significant differences between smokers and non-smokers (89); Eurogast study group concluded from their study that there was no effect of smoking on the prevalence of *H.pylori* infection after adjusting for the other risk factors (90); Rosenstock *et al* observed no association with smoking habits and *H.pylori* infection (91); Shinchi *et al* demonstrated that smoking was not to related to *H.pylori* infection (92).

***Alcohol***

Considerable number of epidemiological studies failed to show statistically significant association with *H.pylori* and alcohol consumption. Eurogast study group conducted a cross sectional study to know the seroprevalence, risk factors associated with *H.pylori* infection from 3149 asymptomatic patients over 17 geographically defined populations. They observed the seroprevalence of infection was higher in the older age group of 55-94, (62.4%) than in the younger age group of 25-34 (34.9%) and no effect of alcohol consumption on *H.pylori* infection (90). Murray *et al* examined the determinants of *H.pylori* infection in a developed country. In their study there was no demonstrable association between *H.pylori* infections and intake of alcohol (93). Shinchi *et al* in a cross-sectional study of 566 men aged 50-55 years and demonstrated that the alcohol intake was not related to *H.pylori* infection (92).

Few studies suggest alcohol consumption reduces risk of *H.pylori* infection: Rosenstock *et al* observed association of *H.pylori* infection with weekly alcohol intake >or = 6 drinks. They inferred that drinking wine reduces the rate of *H.pylori* infection in Danish adults (91); Ogihara *et al* showed a negative association of *H.pylori* seropositivity with the volume of alcohol consumed. Concluded that drinking was negatively and dose-dependently associated with *H.pylori* seropositivity (88); Brenner *et al* carried out a cross sectional study to assess the association of smoking, alcohol and coffee consumption to active *Helicobacter pylori* infection. Their results suggested a protective effect of alcohol consumption against active infection with *H.pylori* (94)*;* in a another study Brenner *et al* reports suggested that moderate alcohol consumption may facilitate spontaneous elimination of *H.pylori* infection among adults (95).

***Diet***

Begue *et al* demonstrated the role of dietary risk factors associated with the transmission of *H.pylori.* They found high prevalence of infection was associated with increased consumption of food from street vendors, and low prevalence was observed in children who consumed fruits and didn't have food from street vendors. No association was found with consumption of fish, chicken, beef, beans, vegetbles, rice, cheese, milk, and unboiled water. These observations support the role of food prepared under unhygienic conditions as a probable mechanism of transmission of *H.pylori* in developing countries (96). Mard *et al* demonstrated an association between *H.pylori* infection with mean daily intakes of sausages and burgers. They concluded that some dietary aspects which might play a role in the severity of infection include consumption of fast foods and low intake of fresh vegetables (97). AlKalbani *et al* carried out a pilot cross sectional study which included 100 patients; they showed a strong correlation between the frequency of soft drink consumption and the risk of *H.pylori* infection (98). Mhaskar *et al* assessed the risk factors involved in *H.pylori* infection and peptic ulcer disease. Intake of meat, fish were found to be risk factors for *H.pylori* infection. On the other hand, intake of chili pepper and concurrent parasite infestations were appeared to play protective role in *H.pylori* infection (99). Kaba *et al* investigated the prevalence and risk factors associated with *H.pylori* infection among the gastritis students. Their observations suggested that the several factors were responsible for the exacerbation of the infection – 1)

consumption of protein rich food, 2) khat chewing, 3) coffee and tea consumption and 4) skipping meals (100)

***Water borne exposure***

Klein *et al* observed that those children who had consumed external water source were three times more likely to be infected than those who had consumed internal water sources. In children whose homes were supplied with municipal waters were 12 times more likely to be infected than from those families who were supplied water from community wells however, all families were from high income background. They concluded that municipal water was apparently found as a source of infection (101). In a study, Goodman *et al* observed multiple factors responsible for the transmission of *H.pylori* infection, viz 1) swimming in rivers, streams or pools, 2) consumption of stream water, 3) children who frequently used raw vegetables (102). Hopkins *et al* noticed that vegetable grown in water contaminated by sewage water and consumption of raw vegetables might act as an important factor for the transmission of *H.pylori* (103). Mhaskar *et al* found an association between *H.pylori* infections and drinking nonfiltered and nonboiled water (99). Kampczyk *et al* also concluded that drinking contaminated water from well was a risk in acquisition *H.pylori* infection (104).These were few studies which pose contaminated water as a risk factor for *H.pylori* infection.

***Density/overcrowding***

Several studies assessed the role overcrowding as a risk factor in *H.pylori* infection. McCallion *et al* found the importance of household living conditions in the acquisition of *H.pylori infection*. They also confirmed household crowding and parents sharing their bed with kids were risk factors for infection (105). Peach *et al* showed status of *H.pylori* was significantly associated with those who were working in public contact for many years, living in a house with more six numbers during childhood (106). Sibship size (more than 4 v/s 1), parental history of gastroduodenal diseases were the risk factors associated with *H.pylori* infection as suggested by KiKuchi *et al* (107). According to Goodman *et al*, the number of persons who lived in the house, especially if the more number of children in the house of greater importance than the number of adults were the risk factors associated with *H.pylori* infection (102). Garaham *et al* also related that living in crowded conditions was responsible for *H.pylori* infection (108).

***Social factors***

Globally, many of the researchers have been correlating *H.pylori* infection with social factors. SES based occupation (usually based on the Registrars General's classification of Occupation (109) I-V that separated jobs into professional, managerial, skilled, semiskilled, and unskilled occupations), education and income were most commonly used measures for social factors (20).
Murray *et al* observed significant association between *H.pylori* and socio class. They referred the Registrars General's classification of Occupation and grouped into manual (III M, IV and V) and non-manual (I, II and IIINM) classes. There was a statistical significant trend across the social classes; with the lowest rate of infection in class I and highest in class V (109). In a study conducted by Lin *et al* compared income levels with that of *H.pylori*, in their study low income levels was associated with frequency of *H.pylori* infection (86). Replogle *et al* compared educational level with *H.pylori* infection; it was observed that lower level of education was associated with higher prevalence of *H.pylori* infection (110). Eurogast study group demonstrated differences in rate of *H.pylori* infection in subjects with higher education had considerably lower rate of infection compared with subjects with education up to secondary level (46.9%) or those with primary education only (185-90). The results were in concordant with the studies done by Peach *et al* (106), Rothenbacher *et al* (111) and Torres *et al* (112).

***Others***

Kikuchi *et al*. in Japan stated that risk of *H.pylori* infection was high if the parents or siblings had history of gastric disorders (107). The hypothesis was supported by the reports of Gasbarrini *et al* (113).

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