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## STUDY OF PREVALENCE OF HELICOBACTER PYLORI IN SYMPTOMATIC PAEDIATRIC PATIENTS ATTENDING OUTPATIENT DEPARTMENT IN A TERTIARY HOSPITAL

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### Abstract

**Background:** The present hospital based cross sectional study was done on 188 pediatric patients attending Outpatient department for various gastrointestinal symptoms. **Material and methods-** The prevalence of Helicobacter pylori was found using Stool Ag card test. Out of these 188 patients, 106 were found positive for Helicobacter pylori by stool antigen card test giving a prevalence of 56.38%. A detailed proforma was filled having information regarding age and sex of the patient, education and occupation of the head of the family, sanitary practices, dietary habits and the patient was also observed for pallor. **Results-** Among a total of 94 males, 56 were positive for Helicobacter pylori (59.6%) and among 94 females, 50 were positive (53.2%). Age wise distribution showed maximum prevalence of Helicobacter pylori in the age group of 15-18 years (73.1%) and minimum in the age group of 3-6 years (29%). A higher prevalence of Helicobacter pylori was found among non-vegetarians (62.5%) and among patients having pallor (63.6%). A prevalence of Helicobacter pylori in upper lower and in lower middle socio-economic group was found to be 56.9% and 52% respectively. **Conclusion-**It was concluded from the study that the prevalence of Helicobacter Pylori is higher in young children of age group 15 to 18 years; especially of low socio-economic status. Thus identification of Helicobacter Pylori infection during childhood is important to prevent the late complications of this disease.

**Keywords:** Helicobacter pylori, stool antigen card test, pallor.

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### Introduction:

Helicobacter pylori, formerly known as campylobacter pylori is a gram negative curved, microphilic and motile organism. It colonizes and grows in human epithelial tissue and mucus. It is a common bacterium infecting about half the world's population <sup>(1)</sup>. There is substantial evidence that it causes chronic gastritis peptic ulcers, duodenal

ulcer and is also involved in development of gastric carcinoma <sup>(2-4)</sup>. H. pylori was identified in 1984 <sup>(5)</sup> and further it was classified as carcinogenic to humans by International Agency for Research on Cancers in next 10years <sup>(6)</sup>. Actual infection rates vary from nation to nation with developing world having higher rates than developed countries <sup>(1, 2)</sup>. The age at which this bacterium is acquired seems

to influence the possible pathologic outcome of the infection. Infections are usually acquired in early childhood in all countries<sup>(7)</sup>. Acquisition at an older age brings different gastric changes more likely to lead to duodenal ulcer<sup>(8)</sup>. High rates of seropositivity in children are found in many developing countries<sup>(9)</sup>. However, in developing countries the prevalence is higher and as much as up to 90% figure has been reported<sup>(10,11)</sup>. Once acquired, *H. pylori* infection generally persists for life, unless treated by specific antimicrobial therapy<sup>(10)</sup>. *Helicobacter Pylori* consists of a large diversity of strains and the genomes of three have been completely sequenced<sup>(12-16)</sup>. The study of *H. pylori* genome is focused to understand the ability of this organism to cause disease. 29% of the loci are in the "Pathogenesis category" of genome database and two of the sequenced strains have an approximately 40 Kb long Cag pathogenicity island which is a common gene sequence responsible for pathogenesis containing over 40 genes. This cag pathogenicity island is usually absent from *Helicobacter Pylori* strains isolated from humans who are carriers of *Helicobacter Pylori* but remain asymptomatic<sup>(17)</sup>. The cag A gene codes for one of the major *Helicobacter pylori* virulence proteins. Bacterial strains that have the 'Cag A' gene are associated with an ability to cause ulcers<sup>(18)</sup>. It is important to state that many individuals who might be harboring the bacterium do not develop clinically apparent disease. Several modes of transmission of *Helicobacter Pylori* are suspected and no single pathway has been clearly identified. It has been demonstrated that housefly has the potential to transmit *Helicobacter Pylori* mechanically<sup>(19)</sup> and thus poor sanitation may potentiate its spread. Person to person contact is considered the most likely transmission route. Another important mode of transmission is iatrogenic in which tubes or endoscopes that have been in contact with gastric mucosa of one individual are used for another

patient<sup>(20)</sup>. Occupationally acquired infections have also been reported especially among, endoscopists and gastroenterologists<sup>(10,20-22)</sup>. Another possible route is faecal and *Helicobacter pylori* has been isolated from faeces of infected young children<sup>(10,21)</sup>. Studies have been done to investigate the association between the seroprevalence of *Helicobacter Pylori* and Hepatitis A virus<sup>(23-28)</sup>. Faeces contaminated water may be a good source of infection. Consumption of uncooked vegetables irrigated with water contaminated with untreated sewage was associated with *Helicobacter Pylori* seropositivity<sup>(29)</sup>. The municipal water supply has greater chances of spreading *Helicobacter Pylori* infection as compared to private water supply<sup>(30)</sup>. Sporadic isolates have been found from dental plaques and saliva<sup>(31,32)</sup>. Various socio-economic conditions comprising of high density crowding, poor sanitary practices family's income, educational level and occupation have been held responsible in spreading of pathogens<sup>(33-35)</sup>. In developing countries, factors related to community and religion might be also important<sup>(36)</sup>. Early detection of *Helicobacter Pylori* population and its eradication may result in a significant improvement in severity of dyspeptic symptoms. It is important to find out *Helicobacter Pylori* prevalence and identify high risk population so that treatment strategies can be appropriately planned. Hence the present cross sectional hospital based study was done on patients attending OPD for various gastro-intestinal disorders and the prevalence of was estimated *Helicobacter Pylori*.

### **Material and methods:**

The study was conducted in the department of Paediatrics at GSVM Medical College, Kanpur in collaboration with department of Physiology at KGMU, Lucknow during 2012-2013. A total of 188 patients of both the sexes attending OPD for gastrointestinal disorders were screened for

*Helicobacter pylori*. Written informed consent was taken from parents of all the patients after explaining them the nature and purpose of study. Ethical clearance was taken prior to the study from the ethical committee. Patients who had taken proton pump inhibitors or antibiotic for a month prior to study were excluded from the study. Patients stool sample was collected in an airtight container and the stool assay was performed using Immunocard STAT HpSA test. (Standard diagnostics Inc). The test device and the sample were brought to room temperature prior to testing. The test device was laid on a flat dry surface and about three drops of the prepared sample were poured into the sample well. Interpretation was done after fifteen minutes. A positive result was shown by two colour bands, one at test band and another at control band. Negative test result showed only control band. HpSA test is a non-invasive and an accurate test especially useful for screening of asymptomatic subjects. Pallor was seen in the lower palpebral conjunctiva. Data were analysed by the chi-square test to compare the association between different variables and *Helicobacter pylori* positive rates. A value of  $P < 0.05$  was considered statistically significant. The calculations were done using the software package SPSS 16.0.

**Results:**

Out of total 188 patients, 106 patients were *Helicobacter pylori* positive by Immunocard STAT HpSA test, giving a hospital based prevalence of 56.38%. Out of total 94 males, 56 were positive for *Helicobacter pylori* (59.6%) whereas out of 94 females 50 were positive (53.2%), (Table 1).

The prevalence was estimated in different age groups. The maximum number of positive patients was found in the age group of 15-18 years (73.1%) and the minimum prevalence was in the age group of 3-6 years (29%), (Table 1). For socio economic status, the groups were classified according to

modified Kuppuswamy scale for urban families. Out of total 160 patients belonging to upper lower group 91 were positive for *Helicobacter pylori* (56.9%), 25 belonging to lower middle socioeconomic group, 13 were positive (52%) and only two children of upper middle group were found positive, (Table 1).

Out of total 188 patients, 88 were non-vegetarians with 65 positive for *Helicobacter pylori* (62.5 %) and 100 were vegetarians with 51 positive (51%) (Table 2).

Pallor was present in 66 patients with 42 positive (63.6 %) and absent in 122 patients with 64 positive for *Helicobacter pylori* (52.5%), (Table 2).

**Table 1: Helicobacter pylori positive patients according to socio-demographic characteristics**

Characteristics	Total no. of subjects	Subjects positive for Helicobacter pylori No. (%)	P-value
<b>Sex</b>			
Males	94	56 (59.6%)	>0.05
Female	94	50 (53.2%)	
<b>Age group (years)</b>			
3-6	31	9 (29 %)	<0.05
7-10	65	34 (52.3 %)	
11-14	66	44 (66.7 %)	
15-18	26	19 (73.1%)	
<b>Socioeconomic status</b>			
Upper lower	160	91(56.9 %)	>0.05
Lower middle	25	13(52 %)	
Upper middle	3	2(66.7 %)	

**Table 2:- Helicobacter pylori positive patients according to type of diet and anemia**

Characteristics	Total no. of subjects	Subjects positive for Helicobacter pylori No. (%)	P-value
<b>Type of diet</b>			
Vegetarian	100	51(51%)	>0.05
Non-Vegetarian	88	55(62.5%)	
<b>Pallor</b>			
Present	66	42(63.6 %)	>0.05
Absent	122	64(52.5 %)	

## **Discussion:**

The prevalence of *Helicobacter pylori* infection varies worldwide, but higher colonization rates have been seen in developing countries, compared to developed countries. This study was carried to find out the prevalence of *Helicobacter pylori* among patients attending OPD for symptoms of gastrointestinal disorders. These patients were screened for *Helicobacter pylori* by Immunocard STAT HpSA test. In a study from South India, 105 children were screened for *Helicobacter pylori* and its prevalence rate varied from 44% to 46 %<sup>(37)</sup>. The overall prevalence recorded in our study was 56.38% which is higher in comparison to the above study. This can be explained by the fact that prevalence of *Helicobacter pylori* varies widely by geographic area, age, race, ethnicity and Socio-economic status. In our study, age wise distribution showed maximum prevalence in the age group of 15-18 years (73.1%) and minimum in the age group of 3-6 years (29%). A study from Hyderabad has shown that *Helicobacter pylori* infection increases with age with 60% at 3-10 years, 50% at 11-15 and 84% by 16-20 years of age<sup>(38)</sup>. In a study from Mumbai, it has been shown that the prevalence of IgG antibody was 22%, 56% and 87% in 0-4, 5-9 and 10-19 years age group respectively in 340 subjects<sup>(39)</sup>. Another similar study from Bangalore has detected *Helicobacter pylori* infection in 82% of 50 children of 6-18 years of age by 13 Carbon urea breath test<sup>(40)</sup>. Kang et al in a report from South India found 57% of subjects between 6 months to 4 years positive for IgG antibodies for *Helicobacter pylori*<sup>(41)</sup>. Sharma et al too reported 50% seropositivity for *Helicobacter pylori* in children below 10 years of age<sup>(42)</sup>. In a study from Chennai in an urban upper class population, a 21.1% prevalence rate was seen in individuals between 12-20 years of age<sup>(43)</sup>. In the present study, among *Helicobacter pylori* positive patients 59.6% were males and 53.2% were females. Although there is a slightly greater male preponderance but the difference between the genders was not significant which is similar in the study from South India<sup>(37)</sup>. In the present study, out of 66 patients having pallor 42 patients were positive for *Helicobacter pylori* (63.6%). *Helicobacter pylori* colonization appears

to impair iron uptake and increase iron loss. Regarding the possible role of *Helicobacter pylori* in iron deficiency anemia, a recent metaanalysis indicated that the infection is associated with depleted iron deposits. The mechanism by which *Helicobacter pylori* induces this alteration is not clear but it appears to involve GI blood loss, diminished iron absorption from diet and increased consumption of iron by the bacteria<sup>(44)</sup>. A study from Bangladesh has shown the prevalence of iron deficiency anemia with decrease in hemoglobin and serum ferritin was significantly higher in *Helicobacter pylori* infected patients<sup>(45)</sup>. The prevalence of *Helicobacter pylori* in our study was found to be higher in low socioeconomic groups being 56.9% in upper lower and 52% in lower middle groups. This is consistent with previous studies which have demonstrated that the prevalence of *Helicobacter pylori* as well as gastritis is more frequent in those who come from large families, have poor hygiene, low standards of living, poor sanitation practices and overcrowded living conditions<sup>(46, 47, 48)</sup>. Socioeconomic status is not restricted to income and social class but also considers other factors such as living standards, urbanization and educational level<sup>(49)</sup>. A prevalence of 51% was seen in vegetarians and 62.5% in non-vegetarian group which was though higher in non-vegetarians but was not significant ( $p > 0.05$ ) which supports the fact that it is probably the food prepared under unhygienic conditions which plays a role in transmission of *Helicobacter pylori* in developing countries and not the type of food consumed<sup>(50)</sup>.

## **Conclusion:**

The present study revealed substantial prevalence of *Helicobacter pylori* in symptomatic children with males more affected and maximum prevalence in the age group of 15-18 years. The prevalence is higher in low socioeconomic classes with poor sanitation practices and unhygienic water supply. A higher prevalence of *Helicobacter pylori* seen in subjects having pallor may be contributed to poor iron absorption in these patients. Similarly, a higher prevalence of *Helicobacter pylori* was noticed among non-vegetarians which may be the

contributing factor in the development of peptic ulcer and gastric cancer in patients harboring *Helicobacter pylori*. In conclusion, there is a very high prevalence of *Helicobacter pylori* among children which acquire the infection early in life which increases slowly and steadily peaking in young adults. Identification of the population harboring *Helicobacter Pylori* is essential at an early age it still remains a challenge for clinicians.

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### **References:**

1. Pounder RE, Ng D. "The prevalence of *Helicobacter pylori* infection in different countries" *Aliment Pharmacol. Ther. a.* 1995; (Suppl 2): 33
2. Labigne A; de Reuse H. Determinants of *Helicobacter pylori* pathogenicity. *Infectious Agents and Diseases*; 1996; 5:191- 202.
3. McColl KEL. *Helicobacter pylori*, clinical aspects. *Journal of Infection* 1997; 34: 7 - 13.
4. Riegg SJ, Dunn BE, Blaser MJ, Microbiology and pathogenesis of *Helicobacter pylori*. *Infections of the gastrointestinal tract.* New York. Raven Press. 1995; 535 - 550.
5. Marshall BJ, Warren JR: Unidentified curved bacilli in the stomach of patients with gastritis and ulceration. *Lancet* 1984;1:1311 – 1315
6. International Agency for Research on Cancers. Mono-graphs on the evaluation of carcinogenic risks to humans. Geneva World Health Organisation. 1994: 61.
7. Kusters JG, Van Vliet AH, Kuipers. EJ Pathogenesis of *Helicobacter pylori* infection". *Clinical Microbiol Rev* 2006 19(3): 449 – 90.
8. Brown LM. *Helicobacter pylori* epidemiology and routes of transmission" *Epidemiol Rev* 2000 22(2): 283 – 97.
9. Blecker U, Vandenplas Y. Ethnic differences in *Helicobacter pylori* infection. *Eur J Paediatric* 1993; 152:176-6.
10. Dunn BE, Cohen H, Blaser MJ, *Helicobacter pylori*. *Clinical Microbiology Reviews* 1997, 10: 720 -741.
11. Bardhan PK. Epidemiological features of *Helicobacter pylori* infection in developing countries. *Clinical Infectious Disease.* 1997, 25: 973 - 978.
12. Tomb JF, White O, Kerlavage AR, Clayton RA, Sutton GG, Fleischmann RD. The complete genome sequence of the gastric pathogen *Helicobacter pylori*" August 1997: *Nature* 388 (6642): 539 – 47.
13. "Genome information for the *H. pylori* 26695 and J99 Strains Institute Pasteur 2002 Retrieved 200809-01.
14. *H. pylori* 26695 complete genome" National center for Biotechnology information, Retrieved 2008 - 09 - 1.
15. "H pylori J 99 Complete genome" National Center for Biotechnology information Retrieved 2008 -09-01.
16. Oh JO, Kling- Backhed H, Giannakis M, Xu J, Fulton RS, Fulton LA et al. The complete genome sequence of a chronic atrophic gastritis *H. pylori* strain: Evolution during disease progression". *Proc Nat. Acad Sci U.S.A.* June 2006: 103(26): 9999-10004.
17. Baldwin DN, Shepherd B, Kraemer P, Hall MK, Sycuro LK, Pinto-Santini DM et al. Identification of *H. pylori* genes that contribute to stomach." Colonization. *II Infect Immun* 2007; 75(2): 1005 - 16. Ooi: 10. 1128/IALO1176-06 PMC 1828534, PMID 17101654.
16. Broutet N, Marais A, Larnouliatte H, Mascarel A, Samoyean R, Salamon R et al. Cag A status and Eradication Treatment. Outcome of Anti *H. pylori* Triple Therapies in patients with nonulcer dyspepsia. *J Clin Microbiol.* 2001; 39(4): 1319 – 22.
17. Grubel P Hoffman JS, Chong FK, Burstein NA Mepam C, Cave DR. Vector Potential of houseflies (*Musca domestica*) for *Helicobacter pylori*. *Journal of Clinical Microbiology* 1997; 35: 1300 -3.
18. Akamatsu T, Tabata K, Hironaga M ,Kawakami H, Yyeda M. Transmission of *Helicobacter pylori* infection via flexible fiberoptic endoscopy, *American Journal of infection control.* 1996; 24: 396 - 401.
19. Lin SK, Lambert JR, Schembri MA, Nicholson L, Korman MG. *Helicobacter pylori* prevalence in endoscopy and medical staff. *Journal of Gastroenterology and Hepatology* 1994; 9: 319 - 324.
20. Chong J, Marshall BJ, Barkin JS, McCallum RW, Reiner DK., Hoffman SR et al. Occupational exposure to *H. pylori* for the endoscopy professional: a sera epidemiological study. *American Journal of Gastroenterology* 1994; 89:1987-1992.
21. Rudi J, Toppe H, Marx N, Zuna I, Theilmann L, Stremmel W et al. Risk of infection with *Helicobacter pylori* and hepatitis and virus in different groups of hospital workers. *American journal of Gastroenterology* 1997; 92:258-262.
22. Sathar MA, Gowws E, Simjee AE ,Mayat AM. Sero-epidemiological study of *Helicobacter pylori* infection in south African children transaction of the royal college of medicine and hygiene 1997;42:258-262.
23. Handt LK, Fox JG, Dewhirst FE, Fraser GJ, Paster BJ, Yan LL. *Helicobacter pylori* isolated from the domestic cat: public health implications infection and immunity 1994;612:2367-2374.
24. Fox JG. Non-human reservoir of *Helicobacter pylori*

- .Alimentary Pharmacology and Therapeutics 1995;9(suppl.2):93-103.
25. Webb PM, Knight T, Elder JB, Newell DG, Forman D. Is helicobacter pylori transmitted from cats to humans? *Helicobacter* 1996; 1:79-81.
  26. Furuta T, Kamata T, Takashima M, Futami H, Arai H, Hanai H. Study of transmission routes of H.pylori in relation to seroprevalence of hepatitis and virus. *Journal of clinical Microbiology* 1997, 35:1891-3.
  27. Hopkins RJ, Vial PA, Ferreccio C, Ovalle J, Prado P, Sotomayor V et al. Seroprevalence of helicobacter pylori in Chile: vegetables may serve as one route of transmission *Journal of infectious Disease* 1993; 168:222-6.
  28. Klein PD, Opekun AR, Smith EO, Graham DY, Gaillour A. Water source as risk factor for helicobacter pylori infection in Peruvian children *Gastrointestinal Physiology working group. Lancet* 1991, 337:1503-6.
  29. Namavar F, Roosendaal R, Kuipers EJ. Presence of helicobacter pylori in the oral cavity, oesophagus, stomach and faeces of patients with gastritis *European Journal of clinical Microbiology and infectious Disease*, 1995,14: 234-237.
  30. Megraud F. Transmission of helicobacter pylori. foecal oral route *Alimentary Pharmacology and Therapeutics*. 1995,9(suppl-2):85-91
  31. Peach HG, Pearce DC, Farish SJ. Helicobacter pylori infection in an Australia regional city: prevalence and risk factor. *Medical Journal of Australia*. 1997, 167:310-3.
  32. Malaty HM, Paykov V, Bykova O, Ross A, Graham DP, Anneger JF et al. Helicobacter pylori & socio-economic factors in Russia, *Helicobacter*. 1996,1:82-7.
  33. Rothenbacher D, Bode G, Winz T, Berg G, Adler G, Brenner H. H. pylori in outpatients of a general practitioner. Prevalence and determinants of current infection. *Epidemiology and infection* 1997, 119:151-7.
  34. Lindkvist P, Enquesslassie F, Asrat D, Muhe L, Nilsson I, Giesecke J. Risk factor for infection with H.pylori-a study of children in rural Ethiopia *Scandinavian Journal of Infectious Disease* 1998,30:371-6.
  35. Kate V, Ananthakrishnan N, Ratnakar C, Badrinath S. Anti-H.pylori IgG sero prevalence rates in asymptomatic children and adults from South India. *Indian J Med Microbiol* 2001; 19:20-5.
  36. Graham DY, Adam E, Reddy GT, Agarwal JP, Agarwal R, Evans DJ. Seroepidemiology of Helicobacter pylori infection in India. Comparison of developing and developed countries. *Dig Dis Sci* 1991; 36:1084-8.
  37. Gill HH, Majumdar P, Shankaran K, Desai HG. Age related prevalence of Helicobacter pylori antibodies in Indian subjects. *Indian J Gastroenterol* 1994; 13: 92-4.
  38. Dore SP, Krupadas S, Borgonha S, Kurpad AV. The 13 C urea breath test to access H. pylori infection in school children. *Natl Med J India* 1997; 10: 57-60.
  39. Kang G, Rajan DP, Patra S, Chacko A, Mathan MM. Use of serology, the urease test and histology in diagnosis of helicobacter pylori infection in symptomatic and asymptomatic Indians. *Indian J Med Res* 1999; 110:86-90.
  40. Sharma S, Dhole TN, Prasad KN, Ayyagari A. Evaluation of 66kDa directed IgG response for detection of helicobacter pylori infection. *Indian J Med Microbiol* 1996;14:17-21.
  41. Alaganantham TP, Pai M, Vaidehi T, Thomas J. Seroprevalence of helicobacter pylori infection in an urban, upper class population in Chennai. *Indian J Gastroenterol* 1999;18:66-8.
  42. Bermejo F, Garcia – Lopez S. A guide to diagnosis of iron deficiency and IDA in digestive diseases. *World J Gastroenterol* 2009; 15 (37): 4638 - 43.
  43. Sultana S, Sarker SA, Sattar S, Ahmed T, Fuchs GJ, Davidsson L et al. Serum ferritin, Hemoglobin, Soluble Transferrin Receptor and Helicobacter pylori infection in periurban community children in Bangladesh. Paper Presented at 8th CCDM. Scientific Session 5 - Helicobacter pylori 024 (109) [ www.icddr.org]
  44. Graham DY, Malaty HM, Evans DG, Evans DJ, Klein PD, Adam E. Epidemiology of Helicobacter pylori infection in asymptomatic population in the United States. Effect of age race, and socioeconomic status *Gastroenterology* 1991; 100: 1495 - 1501.
  45. Edwards FC, Goghill NF. Aetiological factors in chronic atrophic gastritis. *Br Med J* 1966; 2:1409 - 1415.
  46. Massarrat S, Paidlik A, Pittner P, Schmitz - Moorman P, Wurbs M. The role of certain habits and various diseases in the occurrence of gastritis. *Hepatogastroenterology* 1983; 30:249.
  47. Khalifa MM, Sharaf RR, Aziz RK. Helicobacter pylori: a poor man's gut pathogen? *Gut pathogens* 2010, 2:2
  48. Rodolfo E. Begue, Jose L. Gonzales Herman Correa - Gracian, Si. Chin Tang. Dietary risk factors associated in the transmission of Helicobacter pylori in Lima, Peru. *AM J. Trop. Med. Hyg.* 1998; 59 (4): 637.

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