

Original Article

ASSOCIATION OF H PYLORI AND MORPHOLOGICAL CHANGES IN MUCOSA OF ESOPHAGUS AND GASTRIC ANTRUM IN PATIENTS WITH DYSPEPSIA.

Tahira Liaquat, Eyyaz Khaleel and Tahir Bashir

Objective: To find association between H pylori and morphological changes in mucosa of oesophagus and gastric antrum in Patients having dyspepsia.

Methods: One hundred sixty nine patients presenting to outpatient department of Medical units of Lahore General Hospital and services hospital Lahore with symptoms of dyspepsia were included in the study. This study was conducted at Pathology Department of Post Graduate Medical Institute. Endoscopy of all included patients was done in the medical unit, services hospital and endoscopic biopsies from distal esophagus and gastric antrum were taken simultaneously.

Results: Out of 169 patients 31 were H. Pylori positive, 30 (96.8%) cases were with chronic inflammation, 22 (70.9%) cases with neutrophil infiltration, 21 (67.7%) case atrophic changes, 4 (12.9%) cases with metaplasia/dysplasia and 01 (3.2%) case was with malignancy.

Conclusions: Dyspeptic patients with H. pylori infection in esophageal mucosa are prone to develop morphological changes, however, histopathological changes in oesophageal and gastric mucosa of such patients are present in both H. Pylori positive and negative subjects but more in H. Pylori negative cases.

Keywords: pylori, dyspepsia, chronic inflammation, metaplasia.

Introduction

Helicobacter pylori (H. pylori), human pathogen, causes chronic gastritis and has a role in gastric and duodenal ulcer, adenocarcinoma and mucosal associated lymphoid tissue(MALT) lymphoma.¹ It is an important factor in functional dyspepsia and causes gastric carcinoma.² Infection with H. pylori occurs worldwide, varies greatly geographically, exceeding 90% in developing countries compared to 20% to 50% in developed countries.³ The prevalence appears to be inversely proportional to socioeconomic status.⁴

One local study reported that H. pylori infection is 92% in cases of gastrointestinal symptoms of acid peptic disease⁵ (Javed et al, 2010). There is an evidence that the disease outcome may be due to variations in infecting strains.⁶ H. pylori colonization is mostly in the antral mucosa.⁷ Antrum and incisura show atrophic gastritis and intestinal metaplasia with H. pylori infection.⁸ The most favourable environment for bacterial colonization is esophagus.⁹ Inflammation induces other changes like basal hyperplasia and dysplasia.¹⁰ In the oesophagus of healthy patients as well as patients with reflux disease and Barrett's esophagus, H. Pylori have been detected.¹¹ There is paucity of literature regarding the morphological

changes in H.pylori associated gastritis.¹² This study was conducted to find any association of H pylori and morphological changes in mucosa of esophagus and gastric antrum in patients having dyspepsia in local population.

Methods

169 patients presenting to outpatient department of Medical units of Lahore General Hospital and services hospital Lahore with symptoms of dyspepsia were included in the study. This study was conducted at Pathology Department of Post Graduate Medical Institute. Endoscopy of all included patients was done in the medical unit, services hospital and endoscopic biopsies from distal esophagus and gastric antrum were taken simultaneously. Biopsy was placed in jars containing 10% neutral buffered formalin solution¹³. Haematoxylin and Eosin Stain, Immunohistochemical Staining, modified Giemsa Staining were done and Inflammation, activity, H. pylori presence and other mucosal alterations were evaluated semi-quantitatively according to the Sydney system. Atrophic changes and intestinal metaplasia were also determined. Association of H pylori and morphological changes in mucosa of esophagus and gastric antrum in patients having dyspepsia was determined. The data was analyzed by using SPSS 20

(statistical package for social sciences). Quantitative variables like age and size of biopsy etc. were calculated as mean and standard deviation. Qualitative variables like gender, presenting complaint and histopathology were calculated as frequency and percentage. Data was presented in form of tables. Histopathological association with *Helicobacter pylori* was determined by using Chi-square test. P value of ≤ 0.05 was taken as significant.

Results

Out of 169 cases of dyspeptic patients, 110 (65.1%) are males and 59 (34.9%) are females. So M:F ratio is 1.9:1. Out of 169 cases, 31 (18.3%) cases are H. Pyloric positive with males and females 21 (12.4%) and 10 (5.9%) respectively and 138 cases are without H. Pylori infection with males and females 89 (52.7%) and 49 (29%) respectively. Out of 169 cases, 75 (44.4%) cases have no neutrophil infiltration activity while 94 (55.6%) cases with neutrophil activity include mild (n=61), moderate (n=25) and severe (n=8) activity. Among 94 cases with neutrophil infiltration, 72 (42.6%) cases are H. Pylori negative and 22 (13.1%) cases have H. Pylori positive. The difference between these cases is highly significant (P=0.004) statistically (**Table 4**).

Table-1: Age distribution.

Age (in years)	All subjects (n=169)	H. Pylori+ve (n=31)	H. Pylori-ve (n=138)
Mean± SD	39.9±10.8	38.1±10.1	40.4±11.0
Ranges	14 - 65	15 - 64	14 - 65
Total	169	31	138

Table-2: Gender distribution.

Gender	No of patients (n)	Percentage
Male	31	18.3%
Female	138	81.7%
Total	139	100%

Table-3: Distribution of Subjects according to H. Pylori infection.

H.Pylori Infection	No of patients (n)	Percentage
Yes	31	18.3%
No	138	81.7%
Total	139	100%

Table-4: Morphological changes.

Morphology	H.Pylori+ve Cases (n=31)	H.Pylori-ve Cases (n=138)
Neutrophil activity	22	72
Chronic inflammation	22	119
Atrophy	21	30
Metaplasia	04	35
Malignancy	01	13

Out of 169 cases, 20 (11.8%) cases have no inflammation while 149 (88.2%) cases have mild (n=84/49.7%), moderate (n=58/34.3%) and severe (n=7/4.1%) type of chronic inflammation. In 149 cases with chronic inflammation, n=30 (17.6%) cases are H. Pylori positive and n=119 (70.4%) with H. Pylori negative. The difference between these are highly significant (p=0.004) statistically. Out of 31 cases with atrophy, n=21 (67.7%) cases with atrophic changes are H pylori positive and n=30 cases are without H pylori. Out of 169 cases, n=130 (76.9%) cases have no metaplasia/dysplasia and among 39 patients with metaplasia, n=4 (2.4%) cases are H. Pylori positive and n=35 (20.7%) cases are H. Pylori negative and difference is highly significant (p=0.003) statistically. Out of 169 cases, 155 (91.7%) cases have no Malignancy while 14 (8.3%) cases are with malignancy. In 14 cases of gastro esophageal Malignancy (12 gastric, 2 esophageal), 01 (0.6%) case with gastric malignancy is H. Pylori Positive while 13 (7.7%) cases are H. Pylori negative and the difference is highly significant statistically.

Discussion

In this study, 110(65%) are males & 59(34.9%) are females and M:F ratio is 1.9:1. Regarding H. Pylori infection, 31(18.3%) cases are H. Pylori +ve and out of 31 cases, 21(12.4%) are males & 10(5.9%) are females. The number of H. pylori positive males in one study was 13 (18.6%) i.e. greater than the number of H. pylori positive women which was 9 (12.9%). This is in accordance with the data of Persson et al (2010)¹⁴ where H. pylori positive male patients were 29% and females 26%. This research work showed n=31(18.3%) cases of positive H. pylori infection by immunostaining. Yamaoka Y et al (2006)¹⁵ found H.pylori immunopositivity to be 68% while in the study of Afzal et al (2006)¹⁶ this was 70%.

Chronic inflammation in our study is present in n=149(88.2%) cases. Out of 149 cases, n=30(17.6%) cases are with H. Pylori +ve infection and n=119 (70.4%) are without H. Pylori infection. Lymphocytes are not present in the normal oesophageal/gastric mucosa, so their presence in biopsies is evidence of chronic inflammation (Mackiorkowska et al 2003)¹⁷ Nwokediuko and Okafur (2007)¹⁸ found chronic inflammation in 66.7% patients. In this study, Neutrophil infiltration activity is present in 94(65.6%) cases while 22(23.4%) cases are H. Pylori +ve and 72 (76.6%) are with H. Pylori ve. Contreras M et al(2012)¹⁹ showed 38(86%) cases with neutrophil infiltration activity having H. Pylori +ve.

In this study, grading of morphological variables was done according to the Upgraded Sydney System.²⁰ All our patients had chronic inflammatory mono-nuclear infiltrate. This was mild (grade I) in 61 (36.1%) cases, moderate (grade 2) in 25 (14.8%) cases and marked (grade 3) in 08 (4.7%). Chronic inflammation eventually progresses to atrophy of mucosal glands. The onset of atrophy is related to the duration of infection, strain of the organism, dietary factors and host immunity.²¹ Atrophy in this study group is present in 51(30.2%) cases and 21(12.4%) cases are with H. Pylori +ve while 30(17.6%) without H. Pylori infection. In one study, atrophy was seen in only one (1.43%) patient whereas Parasenthi et al (2011)²² found atrophy in 22.1% patients. Metaplasia/ Dysplasia in our study is present in 39(23.1%) cases in which 04(2.4%) cases having H. Pylori +ve and 35(20.7%) cases are without H. pylori infection. Contreras M et al (2012)¹⁹. Malignancy in this study is present in 14(8.3%) cases and one (0.6%) case shows malignancy with H. Pylori +ve and 13(7.7%) showed no H. Pylori

infection in mucosa of esophagus of dyspeptic patients. Morphological study of the variables incorporated in the Upgraded Sydney System allows pre-cancerous changes like atrophy and intestinal metaplasia to be picked up early. Thus appropriate treatment can be instituted.²³

Conclusion

Dyspeptic patients with H. pylori infection in esophageal mucosa are prone to develop morphological changes, however, histopathological changes in oesophageal and gastric mucosa of such patients are present in both H. Pylori positive and negative subjects but more in H. Pylori negative cases. No significant association was found among H. pylori infection, Barrett's oesophagus and oesophageal carcinoma.

*Department of Medicine
Ganga Ram Hospital, Lahore
www.esculapio.pk*

References

- Shrestha S, Paudel P, Pradhan GB, Shrestha L, Bhattachan CL. Prevalence study of H. pylori infection in dyspeptic patients coming to Nepal Medical College Teaching Hospital, Jorpati, Kathmandu. Nepal Med Coll J. 2012 Sep; 14(3):229-33.
- Mahmood S & Hamid A. 2010. Comparison between Invasive and Noninvasive Tests In Diagnosis Of Helicobacter Pylori Infection. Pak. J. Biol. Sci, 13, 509-512.
- Frenck Jr RW, Clemens J. Helicobacter in the developing world. Microbes Infect 2003; 5:70513.
- Domínguez-Bello MG, Beker B, Guelrud M, Vivas J, Peraza S, Pe' rez ME, et al. Short report: socioeconomic and seasonal variations of Helicobacter pylori infection in patients in Venezuela. Am J Trop Med Hyg 2002; 66:4951
- Javed M, Amin K, Muhammad. 2010. Prevalence of H. Pylori. Professional Med J, 17, 431-439.
- Nizami S Q, Bhutta Z A, Weaver L. 2005. Helicobacter Pylori Colonization In Infants In A Periurban Community In Karachi, Pakistan. J PedGastroenterol, 41, 191-194.
- Rugge M, Mario F, Cassaro M. 2007. Pathology Of The Gastric Antrum And Body Associated With Helicobacter Pylori Infection In Non Ulcerous Patients: Is The Bacterium A Promoter Of Intestinal Metaplasia? Histopathology, 22, 9-16.
- Xia H H X, Kalantar J S, Talley NJ . 2000. Antral-Type Mucosa In The Gastric Incisura, Body, And Fundus (Antralization): A Link Between Helicobacter Pylori Infection And Intestinal Metaplasia&Quest. Am J Gastroenterol, 95,114-121.
- Herbella F A & Patti M G. 2010. Gastroesophageal Reflux Disease: From Pathophysiology To Treatment. World J ou Gastroenterology: 16, 3745.
- Jang J, Lee S, Jung Y et al. 2003. Malgun (Clear) Cell Change In Helicobacter Pylori Gastritis Reflects Epithelial Genomic Damage And Repair. The Am Jpathol, 162, 1203-1211.
- Pei Z, Yang L, Peek RM . 2005. Bacterial biota in reflux esophagitis and Barrett's esophagus. World J Gastroenterol 11:727783.
- Parvez Mujawar,1 Dhiraj B. Nikumbh,2 Kishor H. Suryawanshi,3 Poonam S. Pagare,4 and Akshay Surana5. Helicobacter pylori Associated Gastritis in Northern Maharashtra, India: A Histopathological Study of Gastric Mucosal BiopsiesJ Clin Diagn Res. 2015 Jun; 9(6): EC04EC06.
- Guenther T, Hackelsberger A, Kuester D et al. 2007. Reflux Esophagitis Or Helicobacter Infection? Diagnostic Value of The Inflammatory Pattern In Metaplastic Mucosa At The Squamocolumnar Junction. Pathology-Research And Practice, 203, 831-837.
- Persson C, Canedo P, Machado J et al. 2010. 'Polymorphisms in inflammatory response genes and their association with gastric ca-

- ncer: A Huge systematic review and meta-analyses', *Am j epidemiol*, 370.
15. Yamaoka, Y., Ojo, O., Fujimoto, S., Odenbreit, S., Haas, R., Gutierrez, O., El-Zimaity, H. M., Reddy, R., Arnqvist, A. and Graham, D. Y. 2006. 'Helicobacter pylori outer membrane proteins and gastroduodenal disease', *Gut*, 55(6), 775-781.
 16. Afzal S, Ahmad M, Mubarik A. 2006. Morphological spectrum of gastic lesions-endoscopic biopsy findings. *Pakistan Armed Forces medical journal*, 87, 91-113.
 17. Maciorkowska E, Kaczmariskii M, Kemona A. 2003. Comparative evaluation of gastric mucosa morphological changes in children and adults with H. pylori +ve. *Ann Academiae Aedcae*, 48:100-104.
 18. Nwodiuko SC and Okafur OC. 2007. Gastric mucosa in non-ulcer dyspepsia: A histopathological study of Nigerian patiens. *The Internet J Gastroenterol*, ISSN:1528-8323.
 19. Contreras M, Salazar V, Alexandra M. 2012. High frequency of Helicobacter pylori in the esophageal mucosa of dyspeptic patients and its possible association with histopathological alterations. *Int. J Infect Dis* 1016/j.ijid.Q22012.01.007.
 20. Zhang YL, Lai ZS, Zhou DY. 2000. Supra angular biopsy is more reliable for atrophy recognition: analysis of 1598 cases for gastric mucosal histological examination. *World J.Gastroenterol*. 6(6):893-897
 21. Owen DA. 2003. Gastric and Carditis. *Mod.Pathol*, 16(4):325-341.
 22. Parasenthi C, Parasenthi NL, Manikiran SS. 2011. Focus on current trends in the treatment of H. pylori infection: an update. *J.Pharm. SCI.Rev. and Res.* 9(2):42-51.
 23. Rubio CA and Befrits R. 2004. Gastric intestinal metaplasia. *J.Clin.Pathol*, 57(8):894-895.

Medical News

EXERCISE MAY PREVENT HEART ATTACKS IN OTHERWISE HEALTHY PEOPLE

New research published in the *European Heart Journal* suggests that even people with no signs of cardiovascular disease should exercise to prevent a heart attack. Cardiorespiratory fitness can be a predictor of future problems, warn the researchers. Even fit and healthy people should exercise regularly to keep heart disease at bay. Heart disease remains the leading cause of death among men and women in the United States, responsible for the deaths of around 610,000 people each year. Coronary artery disease is the most common form of heart disease, which often results in a heart attack. However, even healthy people might be at risk of a heart attack, new research points out. Even if someone has no signs of cardiovascular problems, low cardiorespiratory fitness may predict future heart disease. For this reason, healthy individuals should exercise regularly to keep heart disease at bay.