FREQUENCY OF H-PYLORI INFECTION IN PATIENTS PRESENTING WITH DYSPEPSIA

Atta Muhammad Khan, Musharaf Kamal Khan, Muhammad Abdur Rahman Afridi

ABSTRACT

Objective: To determine the frequency of H-Pylori infection in patients presenting with dyspepsia.

Methodology: This study was conducted at Department of Medicine, Lady Reading Hospital, Peshawar. Study design was cross sectional study in which 137 patients were observed.

Results: In this study mean age was 35 years with standard deviation SD± 1.26. Fifty two percent patients were male and 48% patients were female. The frequency of helicobacter pylori infection was found in 65% patients.

Conclusion: In conclusion, there is high frequency of H. pylori infection among dyspeptic patients in north-eastern. In spite of this, the prevalence of PUD and gastric cancer are low indicating that other factors also play a role in the pathogenesis of these disorders. In view of the large number of patients with endoscopically normal gastric mucosa who had histologic gastritis, it seems prudent to take multiple gastric biopsies to rule out microscopic gastritis, and to look for H. pylori in all dyspeptic patients undergoing endoscopy.

Key Words: dyspepsia, endoscopy, frequency, helicobacter pylori.

INTRODUCTION

Dyspepsia is a syndrome consisting of epigastric pain, burning, fullness, upper abdominal discomfort aggravated by eating, early satiety, nausea, vomiting, abdominal bloating, water brash and belching[1]. It could be either of organic nature (i.e. ulcerative) or functional. Functional Dyspepsia has been defined by Rome III criteria as the presence of one or more chronic dyspepsia symptoms in the absence of any organic, systemic, or metabolic diseases that are likely to explain the symptoms[2]. Functional dyspepsia (FD) is diagnosed if upper gastrointestinal endoscopy does not show structural abnormality explaining these symptoms[1]. Dyspepsia defines a wide spectrum of gastrointestinal disorders that at least sporadically may affect up to 25% of the population[3,4]. Dyspepsia could be due to several causes such as peptic ulcer disease, reflux disease, drugs (especially Non-Steroidal Anti-Inflammatory Drugs, NSAIDs) and idiopathic[4]. Many of the causes of dyspepsia, organic or functional, has strong association with H. Pylori infection[1,2,3,4]. The pathogenesis of FD is unknown however Various pathophysiological mechanisms, such as gastro duodenal dyskinesia, visceral paraesthesia, vagal dysfunction, H. pylori infection, and psychosocial factors, have been proposed to promote the development of FD. H. pylori infection is the sole cause of FD that can be successfully eliminated by well-established medical interventions[5].

Department of Medicine, M.T.I, LRH, Peshawar

....

Address for correspondence:
Dr. Atta Muhammad Khan
Assistant Professor
Department of Medicine, M.T.I, LRH, Peshawar
Cell: 0301-8955154
E-mail: attamuhammadkhan@yahoo.com

Helicobacter Pylori is closely associated with chronic gastritis, peptic ulcers and gastric adeno-carcinoma and lymphoma and is one of the most common infectious diseases worldwide[6,7]. The risk of acquiring H-Pylori infection is related to socioeconomic status and living conditions. Factors such like density of housing, overcrowding, number of siblings, sharing bed, and lack of running water have all been linked to a higher acquisition of infection, however, a decline in prevalence of infection appears to parallel economic improvement[8].

Helicobacter pylori, a gram-negativemicroaerophilic bacterium found on the luminal surface of the gastric epithelium, was first isolated by Warren and Marshall in 1983. It induces chronic inflammation of the underlying mucosa. The infection is usually contracted in the first few years of life and tends to persist indefinitely unless treated[6]. H-Pylori status can be determined by whole blood test, enzyme linked immunosorbant assay (ELISA), stool for H-Pylori antigen and carbon 13 urea breath test[3].

Many studies have been conducted to document the prevalence of H.Pylori in dyspeptic patients, of these one study showed 50% prevalence of Helicobacter Pylori in dyspeptic patients[6] while other study showed 40% to 60% prevalence[5]. Another study done in Pakistan revealed that 65% of their patients with the symptoms of dyspepsia had Helicobacter Pylori infection[8]. Studies from the African continent have yielded a prevalence rate between 60 and 71%[7]. A study from Nigerian adults reported prevalence of H-Pylori infection of 81%[9] while other studies reported prevalence of 35% to 87%[10,11,12].

The rationale of the study is that the controversial statistics around the world and very little data in our setup make H-pylori a new challenge for the medical

practitioner in our setup. Keeping in mind the importance of timely diagnosis and management, we plan to study the status of H-Pylori in patients with dyspepsia presenting to our hospital settings. Once we know the exact incidence in this part of the world we will be able to compel some guidelines and its timely treatment. We can improve the quality of life of such patients as well as reduce the incidence of gastric carcinoma in such patients and thus reducing the economic burdon on health care system associated with this problem. Also the results will be disseminated with public health and other health improving agencies.

METHODOLOGY

This study was conducted at Department of Medicine, Lady Reading Hospital Peshawar. Study design was cross sectional study and the duration of the study was six months (from December 2016 to May 2017) in which a total of 137 patients were observed by using WHO sample size calculator according to following assumptions. Confidence level 95%, population proportion65%[8] absolute precision 8%. Non-probability consecutive sampling technique was used for sample collection. More over all the patients of either sex with signs and symptoms of dyspepsia and patients of any age between 18-60 years were included. While patients with history of alcoholism, history of drugs intake (NSAIDS, Steroids) without gastric protection, history of chronic renal failure, Pregnancy (confirmed through history) were excluded from the study.

Data was collected on a specially designed Proforma (annexure 1). Patients with signs and symptoms of dyspepsia presenting through Department of Medicine, LRH were enrolled in the study. After collecting baseline information about the patient, record of signs, symptoms, history and presenting complaints was made. Once the appropriate patient found then informed written consent was taken from the patients. Renal functional tests and other baseline tests were done at hospital laboratory. This was detected by examination of stool for H.PyloriAntigen. (stool antigen test for H.Pylori). Stool sample was sent to the specified laboratory for detection of H-pylori antigen. H-pylori results were reported by consultant pathologist having minimum of five years experience. Single pathologist and strictly exclusion criteria was followed, so that to avoid any confounders and making the study results unbiased.

Data was entered in computer using SPSS version 11 (a statistical software) for analysis. Descriptive statistics like mean ± SD was used for numerical variables like age. Frequencies and percentages were recorded for categorical variables like sex, and H-Pylori status (positive/negative). H-Pylori was stratified between age, and gender to see the effect modifiers. Post stratification chi square test was applied in which P value ≤0.05 was considered as significant value. Results was described and also presented in the form of tables and graphs.

RESULTS

In this study (16%) patients were less than 20 years. (29%) patients were in age group ranged from 21-30 years, (19%) patients were in age ranged from 31-40 years, (13%) patients were in age ranged from 41-50 years and (23%) patients were above 50 years. Mean age was 35 years with standard deviation SD± 1.26. Fifty two percent patients were male and (48%)

Table 1: Age distribution (n= 137)

Age	Frequency	Percentage
< 20years	22	16%
21-30 years	40	29%
31-40 years	26	19%
41-50 years	18	13%
Above 50 years	31	23%
Total	137	100%

Mean age was 35 years with SD ±1.26

Table 2: Gender distribution (n= 137)

Gender	Frequency	Percentage
Male	71	52%
Female	66	48%
Total	137	100%

Table 3: Frequency of Helicobacter Pylori infection (n= 137)

Helicobacter Pylori Infection	Frequency	Percentage
Yes	89	65%
No	48	35%
Total	137	100%

Table 4: Association of Helicobacter Pylori infection with age distribution (n= 137)

Helicobacter Pyori Infection	< 20 years	21- 30 years	31- 40 years	41- 50 years	>50 years	Total
Yes	14	30	16	9	20	89
No	8	10	10	9	11	48
Total	22	40	26	18	31	137

Chi square test was applied in which P value was 0.631

Table 5: Association of Helicobacter Pylori infection with gender distribution (n= 137)

Helicobacter Pylori Infection	Male	Female	Total
Yes	49	40	89
No	22	26	48
Total	71	66	137

Chi square test was applied in which P value was 0.577 patients were female. The incidence of helicobacter pylori infection was found to be (65%) in our setup. More over helicobacter pylori infection was stratified with age as in 89 cases of helicobacter pylori infection, 14 patients were in age less than 20 years, 30 patients were in age range 21-30 years, 16 patients were in age range 31-40 years,9 patients were in age range 41-50 years, 20 patients were in more than 50 years. Similarly helicobacter pylori infection was stratified with gender as in 89 cases of helicobacter pylori infection, 49 patients were male and 40 patients were female.

DISCUSSION

Since H. pylori was first cultured by Warren and Marshall in 1983, much has been learned about its clinical aspects and its epidemiology. Knowledge of the epidemiology of this infection comes mainly from prevalence studies. Investigation of the incidence of H. pylori infection has been limited due to difficulties in identifying the case at the onset. In general, H. pylori infection is more frequent in developing countries than in developed nations. In developed countries, H. pylori infection is acquired at fairly constant rate of 2-6% per year with prevalence 20-40% in adults.[13]

Our study shows that the prevalence rate of 65% for H. pylori was relatively high. The finding is consistent with other similar studies in Africa.[14] Jemilohun et al[15] in Ibadan, Nigeria, reported the common abnormalities at endoscopy which favorably compares with the findings in this study i.e. prevalence rate of 60.5% and 18.6% respectively for gastritis and duodenitis. Gastric ulcer (GU) was recorded in 9.3% of the patients, 8.1% had oesophagitis, while 3.5% and 2.3% of them had gastric cancer and duodenal ulcer (DU), respectively. In a similar study in the United States, a H. pylori prevalence rate of 93% was reported among symptomatic East African Immigrants.[16] This finding is consistent with a similar hospital based study conducted by Klusters JG et al[17] in which anti-H. pyloriIgG rate was reported to be 71% and 51%, respectively, among symptomatic and asymptomatic patients. In addition, the commonest identifiable lesion at endoscopy in this study was gastritis which had a frequency of 42.2%. This is comparable to the frequency of 60% obtained in a previous study conducted in north-eastern Nigeria[18].

Our study showed that despite the high prevalence of H. pylori infection in this part of Nigeria, the prevalence of serious gastroduodenal pathologies (GU, DU and gastric cancer) was low as these lesions were documented in only 14.7% of all the patients. This is consistent with findings of previous studies conducted in the same region[18]. This indicates that other factors, possibly genetic and dietary, also play a role in the pathogenesis of these disorders in this environment.

In a large Canadian trial of 1,040 dyspeptic patients in primary care conducted between 1999 and 2001, ulcer disease due to H. pylori was uncommon, with a prevalence of 5.3%, whereas oesophagitis was present in 43%[19]. In another recent study, conducted in many countries, 2,741 dyspeptic patients without alarm symptoms underwent endoscopy[20]. The most common findings were reflux oesophagitis with erosions (15%). Peptic ulcer disease was uncommon: gastric ulcers were found in 2.7% and duodenal ulcers in 2.3% of the subjects. The prevalence of upper gastrointestinal malignancy was 0.22%[21]. The low prevalence of these diseases reflects the changing epidemiology of H. pylori infection in the developed countries. In 1980s, before the widespread eradication of H. pylori was instituted, peptic ulcer disease was very prevalent in Western countries[18]. In the United Kingdom, peptic ulcer disease was found in 18% of cases, and an additional 12% had evidence of pylori-duodenal disease, such as scarring. In a study of dyspeptic patients undergoing endoscopy in Sweden in the late 1980s, peptic ulcer disease was present in 13% of patients[21].

More over it is a chronic annoying disorder affecting commonly the general population. Because of repeated sick report by the patients having dyspepsia, a lot of money is spent on diagnostic evaluation and therapeutic management of this condition. It causes absence of persons from work or duty because of disturbing symptoms. There is strong association of Helicobacter pylori with dyspepsia, as this organism is known to cause serious complications like peptic ulcer disease, gastric carcinoma and MALT lymphoma. Treatment of nonulcer dyspepsia especially is in the form of HP (Helicobacter pylori) eradication therapy, but the response to therapy and the cure rate remains controversial. So while talking of dyspepsia and Helicobacter pylori many major issues arise in one's mind.

Majority of the patients of dyspepsia having normal endoscopy, were having Helicobacter pylori infection on histopathology, a cost effective approach would be to start eradication therapy without testing for HP. However if facilities are available one can go for non-invasive testing for HP first before starting treatment especially in younger patients. The urea breath test is indicated for the initial diagnosis of the infection and for follow-up of eradication therapy at an interval of four weeks to avoid false negative results. HP serologic testing although have sensitivity and specificity similar to those of the urea breath test, inconsistent results have been reported. PCR for HP DNA is a sensitive

method for the diagnosis of HP infection and its use as a diagnostic tool along with histology increases the detection rate of HP infection. Stool antigen tests for HP provide an alternative to the urea breath test, with a sensitivity of 89 to 98 percent and a specificity of over 90 percent.[21] Stool tests are suitable for follow-up of infection, provided that an eight-week interval is allowed after therapy. Endoscopy should be done in patients with alarm symptoms like weight loss, haemetemesis and malaena; in those having persistent symptoms in spite of treatment; where symptoms recur after completion of therapy and in elderly patients presenting with new onset dyspepsia.

CONCLUSION

In conclusion, there is high frequency of H. pylori infection among dyspeptic patients in north-eastern. In spite of this, the prevalence of PUD and gastric cancer are low indicating that other factors also play a role in the pathogenesis of these disorders. In view of the large number of patients with endoscopically normal gastric mucosa who had histologic gastritis, it seems prudent to take multiple gastric biopsies to rule out microscopic gastritis, and to look for H. pylori in all dyspeptic patients undergoing endoscopy.

REFERENCES

1. Kamalapron P, Sobhonslidsuk A, Jatchavala J, Atisook K, Rattanasiri S, Pramoolsinsap C. Factor predisposing to peptic ulcer disease in asymptomatic cirrhotic patients. Alimentary Pharmacology & Therapeutics. 2005;21(12):1459-65.

2. Auroux J, lamarque D, Roudot TF, Deforges L, Chaumette MT, Richardet JP etal. Gastroduodenal ulcer and erosions are related to portal hypertensive gastropathy and recent alcohol intake in cirrhotic patients. Dig Dis Sci. 2003;48(6):1118-23.

3. Shahin WA, Abdel-Baset EZ, Nassar AK, Atta MM, Kabil SM, Murray JA, Low incidence of Helicobacter pylori infection in patients with duodenal ulcer and chronic liver disease. Scand J Gastroenterol. 2001;36(5):479-84.

4. Pellicano R, Leone N, Berrutti M, Cutufia MA, Fiorentino M, Rizzetto M, Ponzetto A, et al. Helicobacter pylori seroprevalence in hepatitis C virus positive patients with cirrhosis. J Hepatol. 2000;33:648-50.

5. Ponzetto A, Pellicano R, Leone N, Berrutti M, Turrini F, Rizzetto M. Helicobacter pylori seroprevalence in cirrhotic patients with hepatitis B virus infection. Neth J Med. 2000;56:206-10.

6. Kim DJ, Kim HY, Kim SJ, Hahn TO, Jang MK, Baik GH, et al. Helicobacter pylori Infection and Peptic Ulcer Disease in Patients with Liver Cirrhosis. Korean J Intern Med. 2008;23(1):16-21.

7. Bilal A, Fazal MO, Shaheen M, Qurashe FS, Ali G, Iqbal MI. Frequency of peptic ulcer in patients having decompensated cirrhosis of liver. A.P.M.C 2008;2(1):35-40.

8. Fallah MA, Prakash C, Edmundowicz S. Acute gastrointestinal bleeding. Med Clin NorthAm 2000;84(5):1183–208.

9. Jiranek JC, Kozarek RA. A cost-effective approach to the patient with peptic ulcer bleeding.SurgClin North Am 1996;76(1):83–103.

10. Boonpongmanee S, Fleischer DE, Pezzulo JC. The frequency of peptic ulcer disease asa cause of upper-GI bleeding is exaggerated. GastrointestEndosc 2004;59(7):788–94.

11. Palmer K. Acute upper gastrointestinal haemorrhage. Br Med Bull 2007;83:307–24.

12. Kaplan RC, Heckbert SR, Koepsell TD. Risk factors for gastrointestinal bleedingamong older patients. Cardiovascular Health Study Investigators. J Am GeriatrSoc2001;49(2):126–33.

13. Pounder LE, Ng D. The prevalence of Helicobacter pylori infection in different countries. Aliment. Pharmacol. Ther. 2005;2:33-9.

14. Baako BN, Darko R. Incidence of Helicobacter pylori infection in Ghanaian patients with dyspeptic symptoms referred for upper gastrointestinal endoscopy. West Afr J Med. 1996;15(4):223-27.

15. Jemilohun AC, Otegbayo JA, Ola SO, Oluwasola OA, Akere A. Prevalence of Helicobacter pylori among Nigerian patients with dyspepsia in Ibadan. Pan Afri Med J 2010;6:18.

16. Suerbaum S, Michetti P. Helicobacter pylori infection. N. Eng. J. Med. 2002;347:1175-86.

17. Klusters JG, Van vliet AH, Kuipers EJ. Pathogenesis of Helicobacter pylori infection. Clin. Microbial. Rev. 2005;19(3):499-90

18. Mustapha SK, Ajayi NA, Nggada HA, Pindiga UH, Bolori MT et al. Endoscopic findings and the frequency of Helicobacter pylori among dyspeptic patients in North-Eastern Nigeria. The Internet Journal of Gastroenterology. 2007;6(1).

19. Thomson AB, Barkun AN, Armstrong D, et al. The prevalence of clinically significant endoscopic findings in primary care patients with uninvestigated dyspepsia: the Canadian Adult Dyspepsia Empiric Treatment-Prompt Endoscopy (CADET-PE) study. Aliment. Pharmacol. Ther. 2003;17:1481-91.jf

20. Vakil N, Talley N, van Zanten SV, et al. STARS 1 Group. Cost of detecting malignant lesions by endoscopy in 2741 primary care dyspeptic patients without alarm symptoms. Clin. Gastroenterol. Hepatol. 2009;7:756-61.

21. Kagevi I, Löfsetd S, Persson LG. Endoscopic findings and diagnoses in unselected dyspeptic patients at a primary health care centre. Scand. J. Gastroenterol.1989;24:145-50.