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PREVALENCE OF H. PYLORI

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SUMMARY: Background: Acid peptic disease is a world wide problem among all the age groups and both sexes. Duodenal ulcer is common as compared to gastric ulcer. Its prevalence being 4:1 in USA & UK and 5:1 in Pakistan[1,2,3]. Etiology of peptic ulcer is almost certainly multi-factorial. Basic paradigm for ulcer disease is the imbalance between the digestive activity of acid and pepsin and the protective mechanism in place to resist mucosal digestion. Over the past few years a new line of thought has been evolved after isolating spiral campylobacter like organism from antral biopsy specimens. H pylori is now considered to be an important if not the only causative agent of gastritis and peptic ulcer disease. The dictum; No acid – No ulcer summarized the pathogenesis of peptic ulcer disease but new dictum seems to be; No H.pylori -No ulcer[4,5], as over 90% of Duodenal ulcer and 70% of Gastric ulcer patients are infected byH.Pylori[6]. Aim of the study was to evaluate the prevalence of H.pylori among Duodenal ulcer patients at Faisalabad District and its suburbs. **Study Design:** Descriptive Study. **Period:** From Mar 2008 to Oct 2008. **Materials and Methods:** 50 patients (40 Males, 10 Females) belonging to Faisalabad District and surrounding areas with upper gastrointestinal symptoms of acid peptic disease and endoscopy proved duodenal ulcer were subjected to gastric antral mucosal biopsies for evaluation of the H.Pylori status with the help of unease test and histological examination of biopsy specimen. **Results:** Epigastric pain was the most frequent symptom 90%. (46 out of 50 patients). 92% showed evidence of H. pylori infection. Maximum incidence of H. pylori was recorded in age group IV (46—55 years). Maximum number of patients was skilled workers (35 out of 50) 70%. 80 % of the patients belonged to lower and middle class. Percentage of H.pylori positivity was 89.1 % and 84.34 %. **Conclusions:** Acceptance of contributory role and high prevalence rate of H.pylori instigates us for addition of antimicrobial treatment to the conventional treatment with H2 Blockers and PPIs which is cost effective and alter the course of the disease.

Key words: Duodenal Ulcer, H.pylori, Gastritis, Acid Peptic Disease.

INTRODUCTION

Acid peptic disease is a worldwide problem among all age groups and both sexes. Duodenal ulcer is common as compared to gastric ulcer its prevalence being 4:1 in USA and U.K[1,2] 5:1 in Pakistan[3] and very high prevalence 32:1 in certain areas of India[7]. The etiology of peptic ulcer is almost certainly multi factorial[8].

A basic paradigm for ulcer disease is the imbalance between the digestive activity of acid, pepsin and the protective mechanism in place to resist mucosal digestion[9].

H.pylori is a spiral shaped gram –ve micro aerophilic rod. Its natural habitat is the gastric mucosa but it is also seen in duodenum. H.pylori is one of the world's most common

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bacterial infection, as more than three quarters of the population of developing world are infected from an early age[10], while most individuals are asymptomatic, a significant number of patients develop serious gastrointestinal disease[11].

Ever since its isolation H.pylori has been implicated for acid peptic disease and today it is regarded as an essential factor if not the only causative agent of gastritis and peptic ulcer disease[12].

When human stomach is infected with H.pylori a chronic gastritis always follows. H.pylori induced chronic gastritis is present more in the duodenal ulcer patients[13,14].

The dictum; No acid – No ulcer summarized the thinking concerning pathogenesis of peptic ulcer disease but since the isolation of H.pylori from antral gastric specimens a new line of thought has been evolved and now the new dictum seems to be; No H.pylori – No ulcer[4,5]. Over 90% of Duodenal ulcer and 70% of Gastric ulcer patients are infected with H.pylori[6].

Research has mostly focused on the role of HCl in ulcerogensis since reduction in acid production, either medically or surgically, appears to heal chronic ulceration[14]. Several contributing factors involved in the ulcerogenesis include drugs, psychological and social factors, age, sex, familial and genetic factors, smoking etc.

Mucus, increase in serum pepsining A, increased gastrin production. Gastroduodenal inflammation (gastritis and duodenitis) as a result of H.pylori infection, disrupts the mucosal architecture and defence mechanism leading to the development of ulceration. This causal realation is supported by the fact that eradication of H.pylori is associated with resolution of acute gastritis[15].

For many years treatment of duodenal ulcer has been based on Shwartz's dictum "no acid – no ulcer" and where as this is remarkably successful with 90-100% duodenal ulcer healing within 2 months of antisecretory treatment[16], there is a disappointingly high rate of ulcer recurrence on stopping treatment. It is now clear that antisecretory drugs do not change the natural history of the underlying ulcer disease. The natural history can be changed, however, by eradication of H.pylori which decreases the incidence of ulcer recurrence[17,18]. Various studies have shown that eradication of H.pylori prevents ulcer relapse and rebleeding in both duodenal and gastric ulcer improves ulcer healing and normalizes histology and acid secretion from chronic gastritis[19].

The above information explosion on H. pylori's role in ulcerogenesis and effect of its eradication on the natural history of ulcer has revolutionized the ulcer treatment which now consists of an H2 receptor antagonist or a proton pump inhibitor combined with antimicrobial combination regimens"[20,21,22,23,24,25,26]. The duration of treatment to eradicate H.pylori has become progressively shorter. Since the first reports in which 6 week, combination therapy was given have been replaced. The current consensus suggests that 2 week treatment is sufficient[22].

OBJECTIVE

Purpose of this study was to evaluate the prevalence of H. pylori among duodenal ulcer patients in Faisalabad District and its suburbs. Our knowledge of the role of H. pylori in peptic ulcer disease makes it possible to formulate new strategies for treatment which not only reduces the duration but also the cost of treatment. This is very important from economic point of view for a developing country like ours.

MATERIALS AND METHODS

This study was conducted at Allied Hospital, Faisalabad, which is 1100 bedded teaching hospital affiliated with Punjab Medical College, Faisalabad. The endoscopy unit and pathology department' were involved in the study for endoscopy and histological evaluation respectively.

Patients

The study included a total of 50 patients (40 males, 10 Females: mean age 43 – years; range 15 – 65) with upper gastrointestinal symptoms of acid peptic disease and endoscopy proved duodenal ulcer. These patients

belonged to Faisalabad district and surrounding areas having a population over 3 million. As Allied Hospital is the only major tertiary care center in this region so this study gave an idea about the prevalence of H. pylori in duodenal ulcer disease in this region.

These patients were subjected to gastric antral mucosal biopsies for evaluation of the Helicobacter pylori status with the help of urease test and histological examination of the biopsy specimens.

Inclusion criteria

- Male and female patients aged between 15 – 65 years.
- Patients with endoscopically established duodenal ulcer.
- Patient of duodenal ulcer having recurrences.

Exclusion criteria

- Anatomical or mechanical abnormality of oesophagous or stomach.
- Patients who have undergone major gastro - duodenal surgery.
- Active upper G. I. bleed.
- History of NSAID intake.
- Patients with systemic diseases.
- Alcoholic.

Endoscopy

Endoscopy was done at the start of the study using the Olympus GIF, Q – 40 gastroscope to confirm the diagnosis of duodenal ulcer and to take two antral mucosal biopsies. The oesophagous, stomach and duodenum were thoroughly examined and findings noted. Two biopsy specimens were taken from gastric antrum of those patients who had a duodenal ulcer. One specimen was put in Helicourease kit and other was placed in 10 % Formalin and sent to pathology departmet for histological examination.

H. Pylori status

H. Pylori status was determined by rapid urease test and histological examination

RESULTS

A total of 50 patients with endoscopically proved duodenal ulcer were included in the study. Demographic data of these patients and diagnostic modalities recorded are as follows. (Table-I)

Table-I. Demographic Data (N=50)	
M/F	40/10 (4:1)
Age range (mean age) years	15-65 years (43 years)
Smokers/ non smokers	14/36
Tobacco chewers	6(12%) patients
Alcoholics	1(2%) patients
Duration of symptoms	
< 6 months	2 (4%) patients
6 months - 1 years	15 (30%) patients
1-5 years	25 (50%) patients
> 5 years	8 (16%) patients
Ulcer size	
0.5-1cm	35 (70%) patients
> 1cm	15 (30%) patients

H. Pylori positivity

Out of 50 patients, 46 showed evidence of H. pylori infection so prevalence of H. pylori recorded was 92%. (Table-II)

Table-II.		
Total Patients	**H. Pylori +ve Patients**	**Prevalence of H.P**
50	46	92%

Distribution of H. Pylori positivity according to sex

Regarding H. pylori positivity 37 out of 40 (92.5%) males were H.pylori +ve and 3 out of 40 (7.5%) were H. pylori –ve. In case of females 9 out of 10 patients (90%) were H. pylori +ve so positivity was 92.5:90% i.e. 1.03:1. (Table-III)

Incidence of H. Pylori passivity according to age

Maximum incidence of H. pylori was 100% recorded in age group IV (46 – 55 years) and minimum incidence was 66.66% in age group I (25 35 years)- . (Table-IV)

Distribution of patients according to age

Age of the patients ranged from 15 – 65 years (Mean age 43 years). Majority of the patients were in 3rd – 5th decades of life. Maximum number of patients were in age group IV (46 – 55 years) – 36% next was age group III (36 – 45 years) – 26%, then age group II (26 -35 years) age group V (56 -65 years) – 16 % each and age group 1 (15 – 25 years) – 6%. (Table-V)

Table-III.

Sex	No. of patients	H. pylori+ve	H. pylori-ve
Male	40	37 (92.5%)	3(7.5%)
Female	10	9 (90%)	1(10%)

Table-IV.

Group	Age range Years	No. of Pts	H.P. +ve n-%age		H.P. -ve n-%age	
I.	15-25	03	02	66.66	1	33.33
II.	26-35	08	07	87.50	1	12.50
III.	36-45	13	12	92.31	1	7.69
IV.	46-55	18	18	100	0	0.00
V.	56-65	08	07	87.5	1	12.5

Table-V.

Group	Age range (Years)	Male Pts.	Female Pts.	Total Pts.	%age of Pts.
I.	15-20	02	01	03	06
II.	26-35	07	01	08	16
III.	36-45	10	03	13	26
IV.	46-55	15	03	18	36
V.	56-65	06	02	08	16

Socioeconomic status of patients

Association of H.pylori with the socio-economic status of the patients was also evaluated, taking into account the educational and economical status of patients. It showed that 80% of the patients belonged to lower and middle class. (Table-VI)

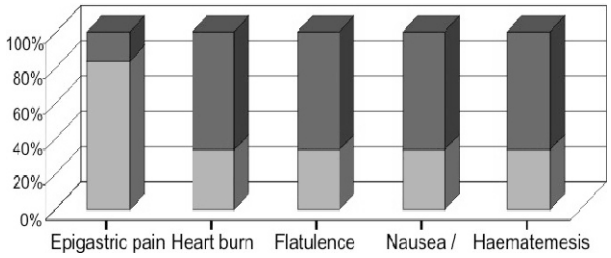
Table-VI (N=50)

Socio-Econimic Status	No. of Patients	%age
Lower class	25	50
Middle class	15	30
Upper class	10	20

Symptoms

Frequency of symptoms was also recorded. Epigastric pain was the most frequent symptom (90%) followed by vomiting 36%, flatulence 30%, Heart burn 16% and haematemesis/ 4%. (Fig. 1)

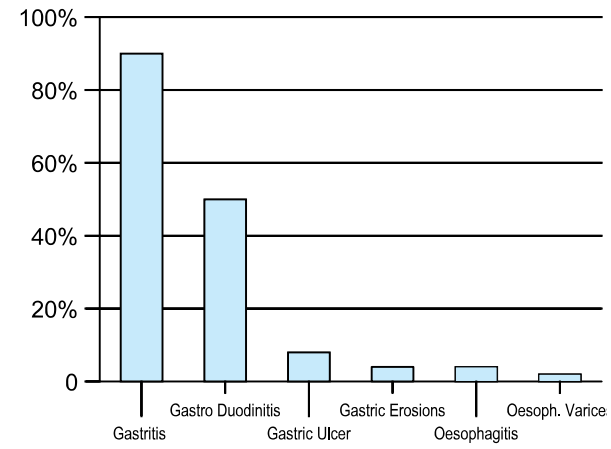
Fig-1



Endoscopic findings

Coexistant endoscopic findings showed 95% incidence of gastritis and 50% incidence of gastroduodinitis. Gastric ulcer was found in 8% Gastric erosions in 4%, oesophagitis in 4% and oesophageal varices in 2% of cases. (Fig 2)

Fig-2.



Histological evidence of chronic atrophic gastritis in H. pylori +ve patients was very high i.e. 95. 65%.

Table-VII.

Chronic atrophic gastritis	Presents		Absent	
	n	%age	n	%age
	44	95.65	2	4.35
Total No. of H. pylori +ve patients=46				

DISCUSSION

Recent discovery of Helicobacter pylori as a cause of ulcer and gastritis has aroused interest in the etiopathology of peptic ulcer disease and dyspepsia. Prevalence of the H. pylori induced type B gastritis reaches upto 100% in duodenal ulcer disease[28].

Studies have shown that eradication of H. pylori infection is associated with healing of this type of gastritis and marked reduction in the rate of recurrence of duodenal ulcers.

Since the discovery of H. pylori several studies and exhaustive investigations have been undertaken to study the association and possible causative role of the organism in duodenal ulcer. This study is another attempt in this respect (though at a smaller scale). The results of this study are more or less consistant with those observed in previously conducted studies. The reported prevalence of antral H. pylori in patients with active duodenal ulcer ranges from 70-100%[29].

According to the present study the prevalence of H. pylori in duodenal ulcer patients belonging to Faisalabad District is 92% (46 out of 50 patients showed antral H. pylori colonization). This result is comparable with various previous studies which have recorded a consistently high H. pylori colonization rate in patients with duodenal ulcer – usually over 90% as shown in the following table VIII:-

The reason for variation in prevalence i.e. 70 – 100% could be due to ethnic differences, low sensitivity of diagnostic tests and age group of patients. High prevalence rate of H. pylori in this study may be due to the age factor (as majority of patients were in 4th & 5th decades of life at which age H. pylori infection is generally high),

Table-VIII		
Study	**Year of study**	**Prevalence of H. pylori %age**
Sassoon levi et al	1989	86.27
P. H. Ketlaris et al	1992	100
P. J. Hu et al	1995	98.97
Shortland W	1992	73
Hosking et al	-	-
Lai. K. H. et al	1991	70
Abdul Fateh	-	-
Qureshi et al	1997	92
Nanivadekar et al	1990	91.6
This study	1997	92

economic status of patients which predisposes to the spread of H.pylori infection, and selection of high sensitivity diagnostic tests i.e. urease test and histology.

Regarding the sex, this study showed predominance of male patients over female patients of duodenal ulcer, (M/F ratio – 4:1) which is comparable to other studies showing predominance of male gender. Lam S. K, and Org G.B. documented a M/F ration of 4:1. A study conducted by W. Ahmed et al in 1990[3] revealed a ratio of 5.7:1. In the literature it is reported that there is lower incidence of duodenal ulcer in young women until the onset of menopause and this led to the idea that somehow female hormones protect against the development of duodenal ulcer[30] .In addition higher rate of smoking habits and stressful life may also be contributory factors.

Contrary to the result of this study M/F ratio is different in developed countries like United States where it is reported to be 1:1 by Kurata JH et al, 1985[31] and United Kingdom where it is reported to be 2:1 by coggon D et al, 1981[2]. These wide geographical differences in the sex ratios support environmental factor theory and the changing habits of females in the developing vs developed countries.

Regarding H.pylori positivity male gender showed only a marginal predominance i.e. 1.03:1 which is comparable with 1.1:1 documented by another local study conducted by shahana Urooj Kazmi et al in 1996[32] similarly LJ. Murray et al reported a M/F ratio of 52.6% vs 48.5% in1995. It is generally concluded that H.pylori infection seems to be equally common among men and women[33] and this observation coincides with the results of this study.

Distribution of patients according to age has revealed that majority of patients in the present study were in their 3rd – 5th decades of life. This figure is consistent with that reported in the literature. In 1996 M. Hobsley and A.M. Tunio[34] has reported high prevalence of H.pylori infection in age group 31 – 50 years. Similarly majority of patients included in a study by Boixeda D et al belonged to 4th and 5th decades. In addition the prevalence of H.pylori showed an increase with the increasing age as is documented by Clearfield 1991. Difference in the prevalence with age suggests two possibilities. Either risk factors for infection in adults differ from those acting during childhood or most infections may be acquired before childhood and the observed increase in seroprevalence with age could be predominantly a cohort effect.

The environmental and social factors play an important role in the spread of H.pylori infection in our community as well as in other developing countries with similar conditions. This high rate of infection worries us to improve the living conditions.

Main presenting symptom of the patients was epigastic pain (90%) followed by vomiting 36%. This is comparable with studies of W. Ahmed et al, 1990[3] (85% and 61% respectively) and J. I. Kazi et al, 1990[35] (83.64% and 40.4% respectively).

Diagnostic tests used to assess the H.pylori status showed a positivity comparable with that documented in the literature. Urease test was positive in 89.1% of patients (41 out of 46 Patients). This percentage is in

between a 79.9% positivity shown in a study by shortland W, Hosking W, Hosking et al, 1992 and 96.5% positivity reported by Serrano N et al, 1995,[36].

Histological evidence of H.pylori was recorded in 86.34% of patients. It is generally observed that histology yields quite variable results e.g reported histological evidence of H.pylori is 50%, 60% and 87.2% in studies conducted by R.Fabre et al, 1993,[37] Shahana Urooj, Kazmi et al 1996[32] and P.J Hu et al, 1995 respectively. This variation may be due to improper collection of mucosal biopsy specimen, improper or delayed transport of the specimen to the concerned laboratory and above all the technique used and capability of the laboratory staff. In this study we gave special attention regarding collection and transport of the specimens and the concerned pathology department was well organized and skilled, so histological examination yielded reasonable results.

Histological evidence of chronic atrophic gastritis (type B gastritis) was recorded in 95.65% of H.pylori +ve patients. This finding is consistant with the generally accepted version that when human stomach is infected with H.pylori, chronic gastritis almost always follows. Ghazala Haq et al, 1991,[38] documented a 96% evidence of gastritis while P.H Ketaris et al, 1992,[39] has documented gastritis in 89.7% of cases.

So the results of this study confirm the strong correlation between the presence of H.pylori in the gastric antrum and histoloigcally proven gastritis.

CONCLUSION

The study revealed Helicobacter pylori positivity in92% patients of duodenal ulcer in Faisalabad region.

Prevalence of H.pylori increases with age and maximum number of H.pylori +ve patients were in 3rd – 5th decades.
Male to female ratio was 1.03:1.
Urease test based H.pylori positivity was 89.1%.
Histology based H.pylori positivity was 84.34%.

The acceptance of contributory role and a high prevalence rate (92%) of H.pylori instigates us to formulate new strategies for the treatment of peptic (duodenal) ulcer disease i.e. addition of antimicrobial treatment to the conventional treatment with H2 antagoinsts and PPI. This would be cost effective and reduce the duration of treatment by altering the course of the disease.

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