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Original Research Article

A study of *Helicobacter pylori* in gastroduodenal diseases

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Keywords

Helicobacter pylori, Biopsy, Isolates, Incidence, Dyspepsia

A B S T R A C T

Hundred patients of both gender between 10–80 years having symptoms of dyspepsia, are subjected to endoscopic examination. The incidence of *Helicobacter pylori* is highest between 41–50 years (78%) with male to female ratio of 2:3.Low socio economic status have a high incidence of *Helicobacter pylori* (65.1%) and (80.9%) in patients having a family history of dyspepsia. The colonization of helicobacter is related to life style i.e. habituation to smoking (76%), addicted to alcohol (36.5%) and beetle nut (3.2%). Higher incidence is in O+ve blood group. Non vegetarians are more for *Helicobacter pylori* infection than vegetarians and patients consuming spicy foods (80.9%) and pickles, chutneys containing excess chillies (90.5%). The biopsy specimens are collected in transport medium containing physiological saline and trimethoprim, processed under aseptic measures within two hours of collection. The standard tests, direct smear, rapid urease and culture were done. *Helicobacter pylori* was declared positive if the bacteria were identified by at least any two of the three diagnostic methods. Direct biopsy smear positive are 63, followed by rapid urease 53 and 49 are culture positive. Out of 49 culture positive cases, 31 are pure isolates of *Helicobacter pylori* and the rest are along with other organisms like *Pseudomonas* species, *Proteus* species and *Klebsiella* species. The colonisation is more common in pylorus (57.2%) than the anterior wall, posterior wall and duodenum. Forty nine isolates of *Helicobacter pylori* were subjected to antibiotic sensitivity testing. The incidence of *Helicobacter pylori* by various studies all over the world and in our country has been well documented and the present study is consistent with other studies all over the world. This study evaluates and is proved beyond doubt that there is a strong association between the presence of *Helicobacter pylori* and acid peptic diseases

Introduction

Helicobacter pylori has been attracting worldwide attention as a possible major feature or cofactor responsible for gastritis and peptic ulcer. Although the presence of bacteria in human stomach has been for a long time, its significance did not gain any importance and remained uncertain. Extensive research since the historical report by Warren (1983) and Marshall (1983) of *campylobacter pylori* now designated as

Helicobacter pylori has proven beyond doubt that *Helicobacter pylori* is the cause of type B gastritis and is an important co factor in the associated gastrodeudenal disease. The discovery of this bacteria changed our concept regarding the micro flora of the stomach, which was thought to be sterile, to a most important pathogen responsible for chronic gastritis and major etiological factor in duodenal ulcer. Thus within a short period, since the report of the bacteria in 1983 over whelming experimental data concerning the bacteria has been published and various studies all over the world have demonstrated the incidence of *Helicobacter pylori* in infected persons. for many years interested was focused on biochemical and humoral factors in the maintenance of mucosal defenses in ulcer patients in ulcer patients but now much attention is paid to the microorganism. Fresh impetus to the search for the etiology of peptic ulcer disease, has opened a new field of investigation by the culture of the microorganism and the demonstration of its association with gastric and duodenal ulcer of all the etiological factors implicated in causation of gastro intestinal ulcerative diseases, which has opened up new vistas in the research of peptic ulcer disease. As the presence of *Helicobacter pylori* is taken as an important prediction for the occurrence and relapse of gastric ulcer (Bayerdoffer, 1993), different laboratory tests have been devised for the isolation and identification of the organism in gastric mucosa in patients of gastric disorders. Therefore, *Helicobacter pylori* can be diagnosed by its isolation, histology, smear microscopic examination, biochemical assays based upon its characteristic metabolic activities such as urease, catalase and oxidase tests and other serological tests. The tests include immunosorbent assay, compliment fixation test and immunoblot. The inter play of cid attack and mucosal defense is modulated by age, blood groups, familial tendency, life style and other physiological considerations, which include acid output. After the initial report by Marshall (1983) several early attempts to isolate the microorganism produced either no growth or low detection rate of *Helicobacter*. Culture of *Helicobacter* may fail because of prolonged transport or storage, over growth by contamination, inhibition of the growth of the organism by polymixin B or nalidixic acid incorporated in the selective culture media. Despite these problems, high incidence rate have been reported by a number of authors. Active research, combining both gastro entomology and microbiology continue to pursue the answer to many aspects of *Helicobacter pylori*. There is much to be learned about *Helicobacter pylori* and its role in the pathogenesis of upper gastrointestinal disease. The different aspects of the bacterium are (i) whether the bacteria is a normal habitat of gastric mucosa (ii) whether it is a part of gastric flora, which causes certain pathogenic changes (iii) the bacterium an aggravating agent taking part in the pathogenesis (iv) is the organism an etiogical factor causing acid peptic disease *Helicobacter pylori* has emerged in the last few years as a bacterium of great interest, and has been described as a micro aerophilic spiral gram negative bacilli (Marshall and Warrner, 1984) that has worldwide distribution with a high prevalence in developing countries and with increasing age. It is closely related to chronic active type (B) astral gastritis and is highly associated with duodenal and gastric ulcers (Borthal *et al.*, 1990) eradication of the infection involves complex combination of drugs. The current treatment is based on triple therapy consisting of bismuth salts, metronidazole and tetracycline. Drugs such as omperazole are also used, but it is found that there is some failure rate to this

treatment due to drug resistant *Helicobacter pylori* Stains. Hence global eradication of this organism will probably rely on therapeutic vaccination and this approach is being actively investigated in different fields. Taking different factors like sex, age, diet, life style, family history and blood groups, this study was undertaken to evaluate the prevalence of *Helicobacter pylori* in patients with dyspeptic symptoms and it sheds light on whether it is a pathogen or not. However, if *Helicobacter pylori* proves to be the cause of common upper gastro intestinal disease, it eradicates and prevention will have a major clinical and economic importance in medical practice.

Materials and Methods

One hundred patients with acid peptic disease attending the gastroenterology department of govt. general hospital as out patients as well as inpatients were taken for the present study. The patients between the age groups of 10–70 years of both sex with or without upper gastrointestinal endoscopic evidences of gastritis, doudenitis and peptic ulcer were selected for the present study.5ml of transport medium (trimethoprim) in bijou bottles were taken to endoscopy room to collect the biopsy specimens. Sample collection- after an overnight fasting of 12 hours, upper gastro intestinal endoscopy was performed in an endoscopy room. The endoscopy was carried out with an Olympus fibre optic endoscope, which is thoroughly cleaned with water and soaked in 2% glutaraldehyde (cidex) for 20 minutes, prior to specimen collection. The endoscope is thoroughly cleaned with salavon and normal saline prior to endoscopy done in each and every patient. In the case of normal mucosa and gastritis as the *Helicobacter pylori* has patchy distribution, the endoscopic biopsy specimens are taken from the stomachs anterior wall, posterior wall and pylori c astral mucosa. In case of duodenitis, biopsy bits of tissue approximately measuring 1 to 2 mm in diameter is taken from the duodenum and in the case of gastritis or duodenal ulcer biopsy bits are taken around the ulcer region under aseptic measures. The biopsy specimens are collected into a transport media and transported to microbiology laboratory within 2 hours of collection for processing. The transport media used is sterile physiological saline (0.85%) with antibiotic trimethoprim in sterile cotton plugged bijou bottles. Three biopsy bits are collected from each patient, one bit for the microscopic examination, one bit for the rapid urease test and the other for culture and histopathology examination.

Processing of the specimen: All the endoscopic biopsy specimens were processed within 2 hours of collection because as *Helicobacter pylori* loses its viability when exposed to atmospheric oxygen for a long time and also to minimise the chances of contamination.

Microscopic examination of direct biopsy smear: One biopsy specimen is picked with a sterile forceps and placed on a sterile clean glass slide, with another clean sterile glass slide the specimen is crushed in between the slides. With the crushed biopsy material, smears are made on the slide with a sterile loop. The smears are air dried and heat fixed. The smears are strained by different methods. (i) Simple straining of the smear with dilute carbol fuchsin. (ii) Gram straining dilute carbol fuchsin is used as counter strain (iii) Special straining - Giemsa straining, haematoxylin and eosin straining.

The direct smears are screened for any spiral or curved shaped pink coloured bacilli for half an hour. Rapid urease test is done. Inoculation into the plates (i) Blood agar media, (ii) Chocolate agar media, (iii) Skirrows media. One set of inoculated plates

are kept in a candle jar. Another set of plates kept in a plastic bag along with, MacConkey plate inoculated with *Escherichia coli* and the bag is sealed airtight. Both the candle jar and the plastic bag are kept in the incubator at 37°C. For humidity a bowl of water is kept in the incubator. The plates are examined after 48 hours for any growth. From the suspected colonies hanging drop, gram straining is done. Biochemical tests done to confirm the growth of *Helicobacter pylori* i.e., oxidase test, catalyse test, hydrogen sulphide test and glucose fermentation test. Anti biotic sensitivity testing was done by Kirby Bauer's method commercially available antibiotic disks, ampicillin, amoxycillin, chloramphenicol, streptomycin, norfloxacin, tetracycline, erythromycin, gentamycin, contrimaxazole, penicillin, furazolidine, and nitrofurantoin are applied in the chocolate agar plate, streaked with the organism. The plate is incubated for 48 hours at 37 c. After 48 hours, the zone of clearance, around the antibiotic disk is observed. The drugs to which the organism is sensitive and to those it is resistant are noted down.

Results and Discussion

A total of 100 symptomatic patients suffering from acid peptic disease and duodenal ulcer, who have undergone endoscopic examination are taken for the present study, out of 100 patients studied, 63 patients are positive for *Helicobacter pylori* and 37 are negative for *Helicobacter pylori* (Table 1). The biopsy specimens of the patients are processed and standard tests done for diagnosis of *Helicobacter pylori* are shown in Table 2. The tests are direct smear, rapid urease and culture. Out of the 100 patients tested, direct smear positive are 63, rapid urease positive are 53 and 49 are culture positive. The analysis is of different bacteriological parameters are done by permutations and combinations. Out of the standard tests if any two tests are, it is taken as positive for *Helicobacter pylori*, which is called as Gold standard. Out of 100 patients, total number of positive cases are 63, when any two tests are taken (urease, culture, and smear the total number of positive cases are 35, smear and culture positive are 12, smear and urease positive are 14, urease and culture positive are 2 (Table 3). Patients of both sexes are taken at random for the study and they are between the age group of 10–80 years. Between the age group of 41–50 years, 24 patients are tested out of which 17 are positive with percentage positivity of 70.8 and between the age group 21–30 years, 19 cases are positive with percentage positivity of 68.4. Thirty four patients tested out of 100 are between the age group of 31–40 years. Out of which 21 are positive for *Helicobacter pylori* with percentage of positivity 61.8 in the age group, 51 and above, the percentage positivity is about 55 and in the age group of 10–20 years the lowest percentage of about 33 is noted (Table 4). Sex wise distribution of isolation of *Helicobacter pylori* is shown in Table 5. 40 patients out of 69 male patients are positive for *Helicobacter pylori* with percentage positivity of 57.8.

Out of 31 female patients, 23 are positive for *Helicobacter pylori* with percentage positivity of 74.2. The patients under study belong to both and middle groups of socio-economic status. Out of 100 patients studied, 83 patients below to low socio-economic status and 17 belong to middle socio-economic status. Fifty four out of 83 belonging to low - socio- economic status are positive for *Helicobacter pylori* with percentage positivity of 65% 9 out of 17 belonging to middle socio economic status are positive for *Helicobacter pylori* with percentage positivity of 52.9 (Table 6).

The patients under study have different dietary habits out of 100 patients, 11 are vegetarian and 89 are non vegetarians. Six out of 11 vegetarians are positive for *Helicobacter pylori* with percentage positivity of 54.5, 57 out of 89 non vegetarians are positive for *Helicobacter pylori* with percentage positivity of 64. Both vegetarians and non vegetarians, who are positive for *Helicobacter pylori* consume spicy food, pickles and chutneys with chilies. Out of 63 positive cases 51 are positive for *Helicobacter pylori* who consume spicy food with percentage positivity of 80.9. Fifty seven patients consuming excess use of chilies are positive for *Helicobacter pylori* with percentage positivity of 90.5 (Table 7 and 8). The life style of patients who are positive for *Helicobacter pylori* having a family history of dyspepsia and who are addicted to either alcohol, smoking or beetle nut is shown in Table 9. 51 patients having family history of acid poetic disease are positive for *Helicobacter pylori* with percentage positivity of 80.9. Among the patients positivity for *Helicobacter pylori*, 23 patients are addicted to alcohol to alcohol with percentage positivity of 36.5 and 48 are smokers with percentage positivity of *Helicobacter pylori* is 76.2. Two patients out of 63 patients are positive for *Helicobacter pylori*, who are addicted to beetle nut. Twenty seven patients out of 63 positive patients for *Helicobacter pylori* belong to O+ve blood group with percentage positivity of 42.8. The percentage positivity of B+ve blood group is 12.7 and A+ve blood group 9.5 as shown in Table 10. Out of 100 specimens cultured, 31 isolates are *Helicobacter pylori* alone with *pseudomonas* aeruginosa, 6 isolates of *Helicobacter pylori* with *Proteus mirabilis*, 3 isolates of *Helicobacter pylori* with *Candida albicans* and 6 isolates of *Helicobacter pylori* with *Klebsiella pneumoniae*. The other organisms isolated alone are *Proteus* mirabilis 9, *Pseudomonas* arugenosa 18, *Candida albicans* 7, Klebsiella pneumoniae 3, 15 cases out of 100 are negative for culture (Table 11). In 36 out of 63 positive for *Helicobacter pylori*, the biopsy bits taken are from the pylorus with percentage positivity of 57.2. The other sites of biopsy are taken from posterior wall with percentage positivity of 9.5 anterior wall with percentage positivity of 12.7 and duodenum with percentage positivity of 20.6 (Table 12). Correlation of endoscopic findings in incidence of *Helicobacter pylori* is shown in Table 13. Out of 63 isolates positive for *Helicobacter pylori*, 29 are of normal endoscopic findings with percentage positivity 46. Other endoscopic findings among the *Helicobacter pylori* positive cases are 7 cases of gastritis with percentage of positivity 11.1 and 9 positive cases of pylori ulcer with percentage of positivity 14.3. Seven cases of duodenitis are positive for *Helicobacter pylori* with percentage positivity 11.1 and 10 cases of duodenal ulcer with percentage of positivity 15.9. One positive case of esophagitis out of 63 is positive for *Helicobacter pylori* with percentage of positivity 1.6. Incidence of *Helicobacter pylori* in various gastric disorders is shown in Table 14.

Out of 100 cases, who underwent endoscopic examinations, 58 cases are of normal endoscopic findings, out of which 29 are positive for *Helicobacter pylori* with percentage of positivity 50. Seven cases of gastritis out of 11 are positive for *Helicobacter pylori* with percentage of positivity 13.6 and 9 out of 9 pylori ulcer cases are positive for *Helicobacter pylori* with percentage of positivity 100. Out of 9 cases of duodenitis, 7 are positive for *Helicobacter pylori* with percentage of positivity 77.8. Ten out of 10 cases of duodenal ulcer are positive for *Helicobacter*

pylori with 100 percent positivity. Drugs sensitivity testing was done for 49 isolates. All isolates are 100% sensitive to chloramphenical, Tetracycline, ciprofloxion, cefaxone and pefloxacin. Forty eight isolates out of 49 are sensitive to netilmicin with 48 percent positivity followed by norfloxacin (91.9), streptomycin (67.3), amoxycillin (46.9), erythromycin (44.9), ampicillin (38.8) and gentamycin (8.2), in the descending order are shown in Table 15. All isolates of *Helicobacter pylori* are resistant to Nalidixic acid. Forty six isolates of *Helicobacter pylori* are resistant to gentamycin with percentage positivity of 93.9. Resistance pattern of other drugs in the descending order is observed as follows. ampicillin (59.2), erythromycin (57.2), amoxycillin (53.1), streptomycin (12.3), norfloxacin (10.2) and netilmicin (2) (Table16). Study of *Helicobacter pylori* in gastric disorders by various studies, all over the world and in our country and the prevalence of *Helicobacter pylori* in our study is shown in the Table 17.

The results of the present study suggest that there is a strong association of *Helicobacter pylori* with acid peptic disease and duodenal ulcer. Identification of *Helicobacter pylori* and its etiopathogenesis has given strong reason as the causative agent of the infection. The incidents of *Helicobacter pylori* is found to be 63 out of 100 patients studied, which is consistent with study of price *et al.*, 1985 (63%). The incidence rates of other works are, Jones *et al.* (1984) (62%), Langerbar *et al.* (1984) (64%), Borromoe *et al.* (1987) (64%), Marshall and Warrner (1984) (58%) and Goodwin *et al.* (1985) (58%). Thirty seven patients out of the 100 were negative for *Helicobacter pylori* though they suffered from symptoms of dyspepsia. The reason could be that (i) Colonisation of the bacilli is patchy in distribution, (ii) Scanty colonisation of the organism, (iii) The patient is on drugs like H2 receptor antagonist cimetidine at the time of endoscopy which has affected the results as the patients are taken at random. Therefore 37% of patients with non ulcer dyspepsia are negative for *Helicobacter pylori* which is consistent with the results of greenberg and bank (1990) (37%).

Helicobacter pylori infection is found common in elderly age group (41–50) with percentage positivity of 70.8. The observation is similar to the studies of Yadley *et al.* (1988), who reported that prevalence of *Helicobacter pylori* in dyspeptic subjects was highest in 3rd decade with 70% which is similar to study. The incidences of *Helicobacter pylori* are found 2:3 in male and female patients in our study. The majority of patients in the present study found to have O+ blood group which correlates well with studies the entire world. The interplay of acid affect and mucosal defense is modulated by lifestyle, consisting of habituation to alcohol, smoking, consuming of beetle nut and excessive intake of spicy food, pickles and chutneys with excess chilies. These factors probably explain the excess acid output in the stomach and attack on the mucosal surface. Therefore there is high frequency of colonization of *Helicobacter pylori* in the damaged gastric mucosa. *Helicobacter pylori* is more common in low socio economic status than the middle and higher socio economic status in developing countries like ours; this could be so because of middle land higher socio economic groups are less expensive surrounding environment and tend to live rather protected lives in homes with the good sanitation. Persons having a history of any one member of the family suffering from dyspepsia are more prone for *Helicobacter pylori* infection as seen in this study and are also suggested by Mytchel *et al.* (1987), that

transition is within the family contact. In all 100 cases of duodenal ulcers and gastric ulcers, diagnosed endoscopically there is colonisation of *Helicobacter pylori* in our study, which is consistent with study of Tytgel,1988.In some normal endoscopic findings though there is colonization of *Helicobacter pylori*, there is no ulceration due to different strains of *Helicobacter pylori* . In some cases though there are ulcers found endoscopically, there is no colonisation of *Bacilli*, because the ulcers formed are due to some other unrelated factors (i) Non steroidal anti inflammatory drugs, (ii) Smoking, (iii) Acid hyper secretion.

Out of 100 cases studied, 63 are direct smear positive for *Helicobacter pylori*. Rapid urease test is done with one of the biopsy bit and it is found that out of 100 cases 53 are positive, a similar observation was made by Mecnutley in 1986. In this study there are 13 cases which are direct smear positive, rapid urease negative and culture positive. A similar observation of 13 cases is reported by Wyelt *et al.* (1988).

Out of 53 urease positive cases, all are not culture positive, so there are few false positive cases. These are due to urease producing bacteria, like *Klebsiella* and *Proteus* (Raisens *et al.*, 1988), which may contaminate endoscopy equipment and which are occasionally found in the gastric tissue hypochlorhydria subjects. Rigorous cleaning of endoscopic equipment and the incorporation of an anti microbial agent in the urease bit, minimises false positive tests. Forty nine cultures were positive out of 63 endoscopic biopsy bit studied. All direct smear positive cases were not culture positive. Therefore the direct smear is the most rapid, simple sensitive test for diagnosis of *Helicobacter pylori* infection (Personnet *et al.*, 1985).

Though urease test is simple and rapid it gives some false positive results as shown in the study . Culture is the most confirmative test for *Helicobacter pylori* as found in the present study. Culture is also a standard method as it not only identifies the organisms precisely but also provides *Helicobacter pylori* for serotyping and gives drug sensitivity pattern of the infecting strains. Hence a combination of culture with straining along with urease provides the best sensitivity for both diagnosis and new methods of evaluation (Goodwin and Marshell, 1984). Drug sensitivity is done for all positive isolates of *Helicobacter pylori*. In this study we found 100% sensitivity to chlorampheniocol, tetracycline, ciprofloxacin, pefloxacin followed by above 90% sensitivity to netilmicin and norfloxacin which is almost similar to the studies found in the literature.

All the isolates are 100% resistant to nalidixic acid (Goodwin *et al.*, 1989),which concurs with the observation made in the present study, but the ampicillin, amoxycillin and gentamycin are also found to be more resistant. In the present study, 63% positivity was found which is similar to the observation made by many studies in India and in other countries. *Helicobacter pylori* is a gram negative, spiral microaerophilic organism which is oxidase positive, catalase positive and urease positive. Traditionally, the pathophysiology of acid peptic diseases and duodenal ulcer is related to excess secretion of gastric acid and most current medical and surgical approaches to treatment are aimed at reducing acid output. But the current study approves that gastric and peptic ulceration as an infectious etiology. Hence there is a close association between gastric disease and the bacterium and also there is colonization of *Helicobacter pylori* in non ulcer dyspepsia (George *et al., 2000*).

Table.1 Prevalence of *Helicobacter pylori*

Endoscopic study Total no.	*Helicobacter pylori* Positive	*Helicobacter pylori* Negative
100	63	37

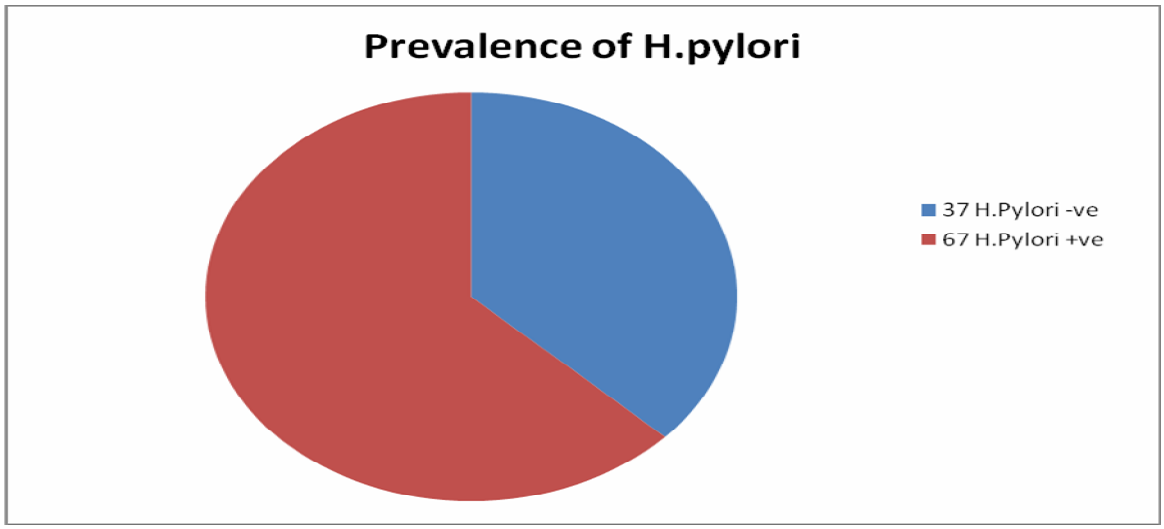

Table.2 Positive by standard tests

TOTAL NO. OF CASES	DIRECT SMEAR	RAPID UREASE	CULTURE
100	63	53	49

Table.3 Analysis of bacteriological parameters for diagnosis of *Helicobacter pylori*

TOTAL NO. OF CASES	SMEAR + UREASE+ +VE CULTURE	SMEAR+ CULTURE +VE	SMEAR + UREASE +VE	UREASE + CULTURE +VE	TOTAL POSITIVE
100	35	12	14	2	63*

*GOLD STANDARD: ANY TWO TESTS TAKEN AS POSITIVE

Analysis of bacteriological parameters for diagnosis of *H. pylori*

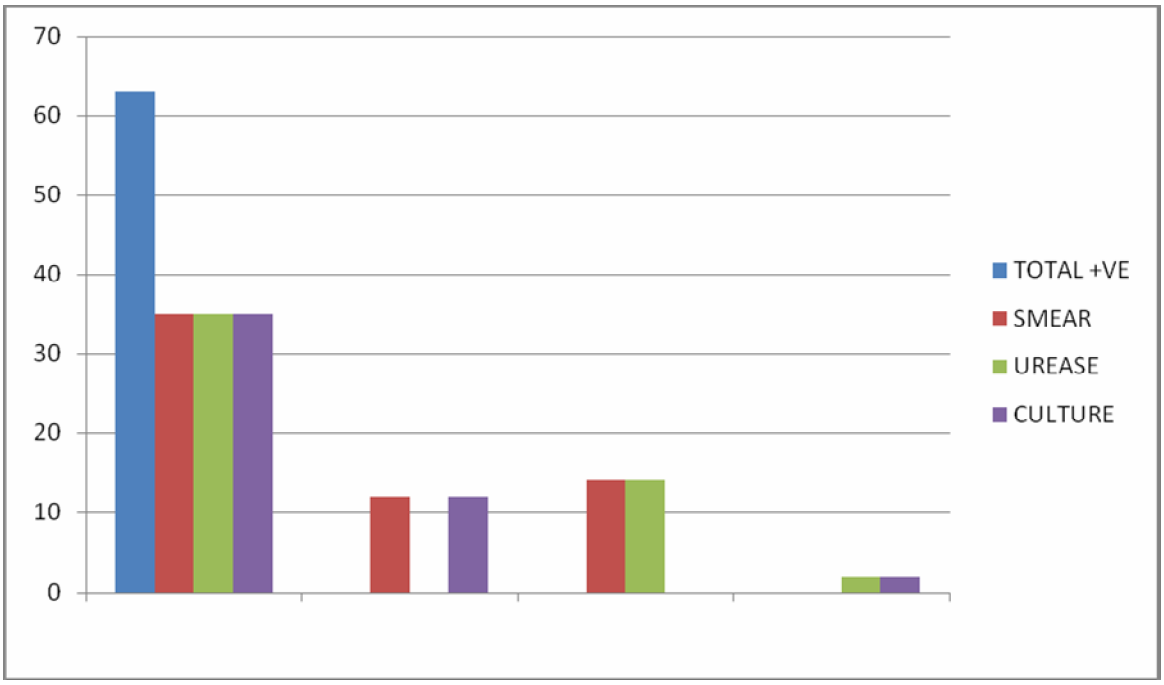

Table.4 Age wise incidence *Helicobacter pylori*

Age groups	Total number of patients	Number of patients positive for *H. pylori*	Percentage of positivity
10–20	3	1	33.3
21–30	19	13	68.4
31–40	34	21	61.8
41–50	24	17	70.8
51–60	11	6	54.5
ABOVE 60	9	5	55.6
TOTAL	100	63	100%

Age wise incidence of *H. pylori*

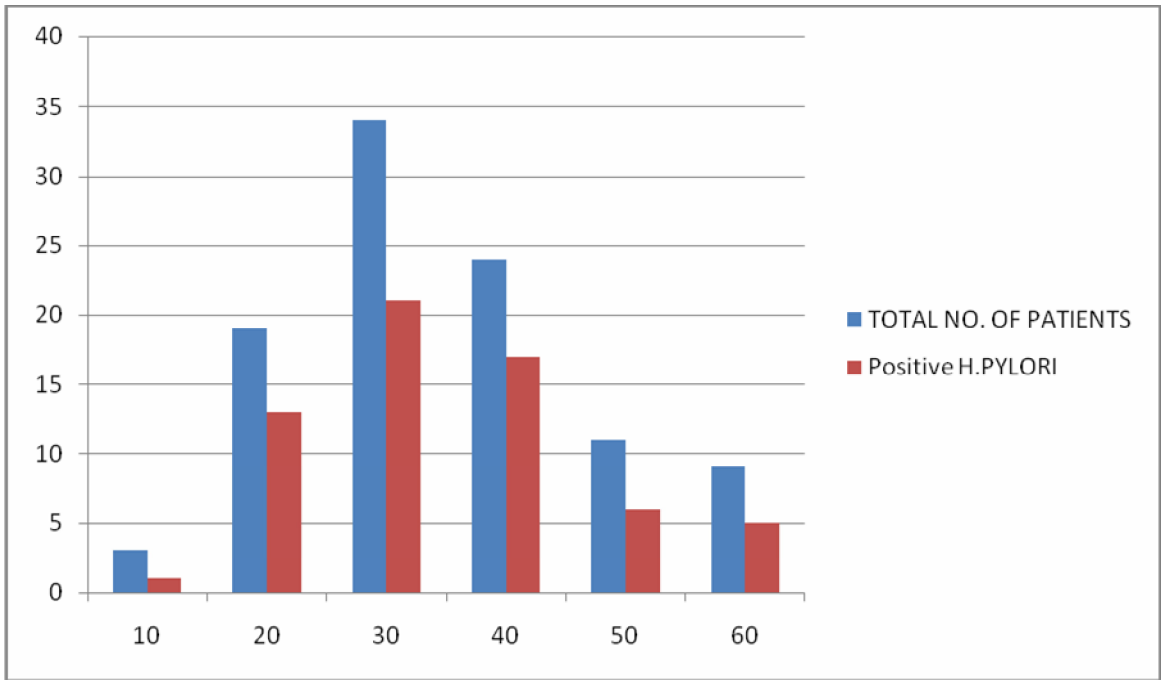

Table.5 Sex wise distribution of *Helicobacter pylori*

SEX	TOTAL NO. OF PATIENTS	NO. OF PATIENTS POSITIVE FOR H.PYLORI	PERCENTAGE OF POSITIVITY
MALE	69	40	57.8
FEMALE	31	23	74.2
TOTAL	100	63	

Table.6 Incidence of *H. pylori* in different socio economic groups

SOCIO ECONOMIC STATUS	TOTAL NO.	POSITIVE FOR H.PYLORI	PERCENTAGE POSITIVITY
LOW	83	54	65.1
MIDDLE	17	9	52.9
HIGH	NIL	NIL	NIL
TOTAL	100	63	

Table.7 Isolation of *H. pylori* in relation to dietary habits

TOTAL TESTED	VEGETARIAN			NON VEGITARIAN		
	TOTAL NO. PERCANTAGE	POSITIVE FOR H.PYLORI	POSTIVITY	TOTAL NO. PERCENTAGE	POSITIVE FOR H.PYLORI	POSITIVITY
100	11	6	54.5	89	57	64

Table.8 Relation of positivity of *H. pylori* of the consumption of spices and chilies

NO. OF H.PYLORI	SPICY FOOD CONSUMPTION		PICKLES & CHUTNIES WITH EXCESS CHILLES	
	TOTAL NO. PERCENTAGE	POSITIVITY	TOTAL NO.	PERCENTAGE POSITIVITY
63	51	80.9	57	90.5

Isolation of *H. pylori* in relation to dietary habits

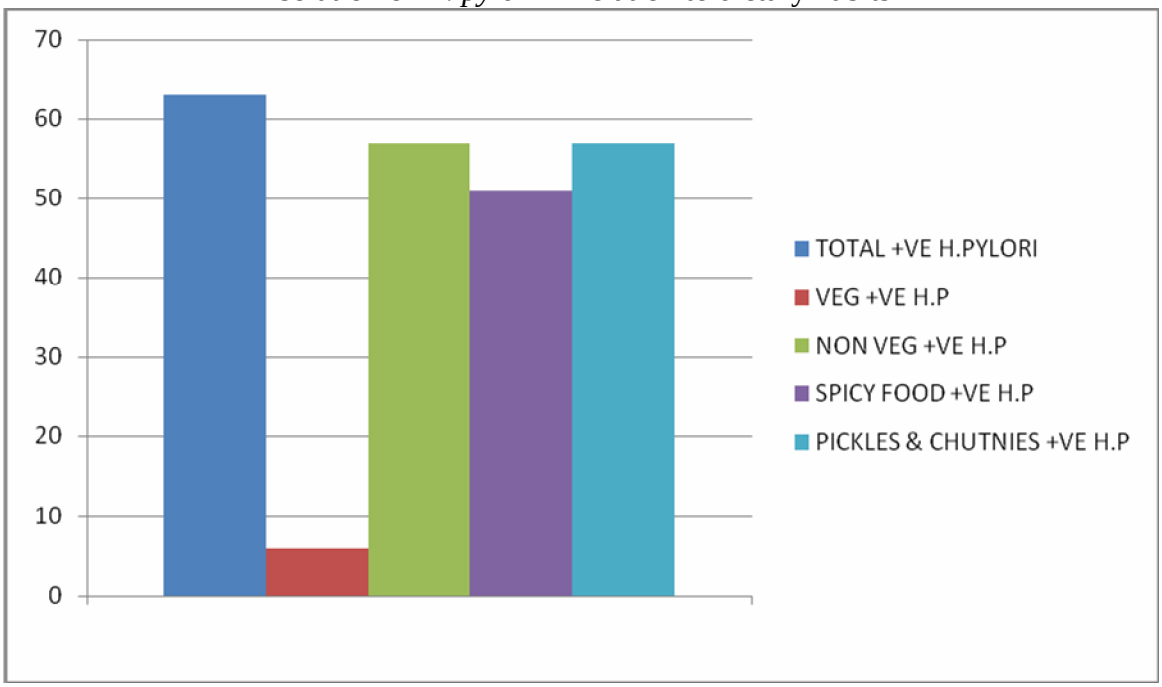

Table.9 Life style and familial tendency in isolation of *H. pylori*

TOTAL POSITIVE CASES	FAMILY HISTORY				ALCOHOL				SMOKING				BETTLE NUT			
	POSITIVE NEGATIVE				POSITIVE NEGATIVE				POSITIVE NEGATIVE				POSITIVE NEGATIVE			
	TOTAL NO.	%	TOTAL NO.	%	TOTAL NO. %	%		TOTAL NO.	TOTAL NO. %	%		TOTAL NO.	TOTAL NO.	%	TOTAL NO.	%
63	51	80.9	12	19	23	36.9	40	63.5	48	76.2	15	23.8	2	3.2	61	96.8

Life style & familial tendency in isolation of *H. pylori*

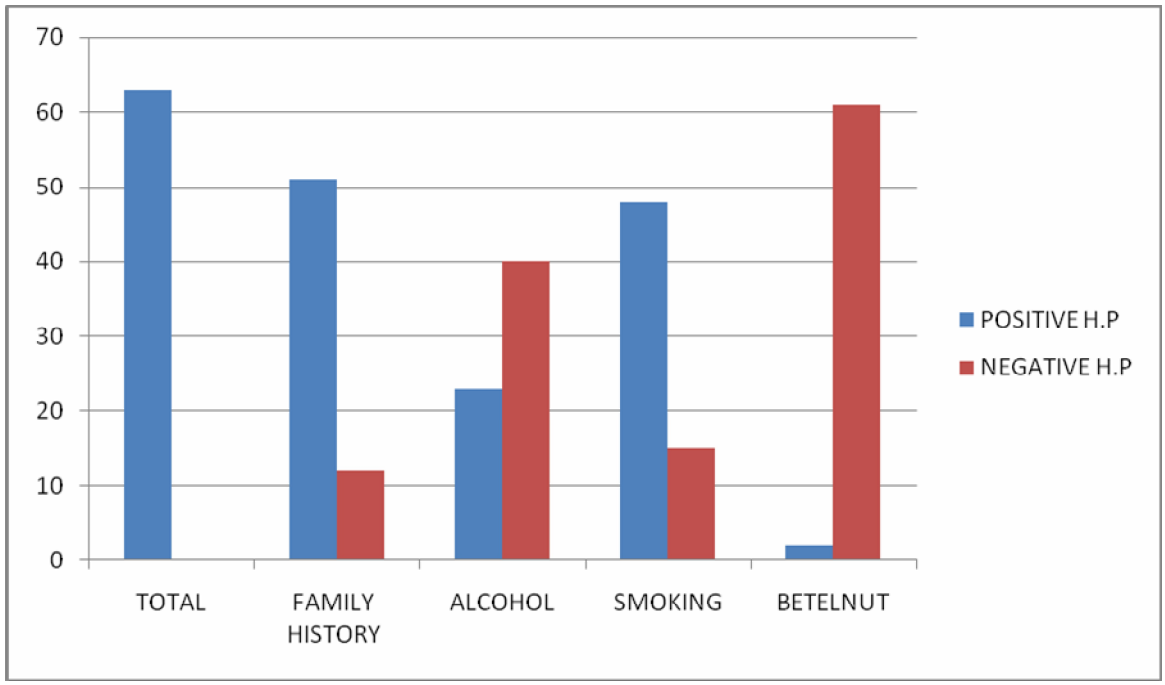

Table.10 Isolation of h.pylori in relation of different blood groups

H.PYLORI POSITIVE	A +VE	B +VE	AB +VE	O +VE	BLOOD GROUP NOT KNOWN
63	6 (9.5%)	8 (12.7%)	--	27 (42.8%)	22

Table.11 Incidence of isolation of other bacteria and *H. pylori*

H.PYLORI ALONE	31
H.PYLORI with Pseudomonas aeruginosa	3
H.PYLORI with proteus Mirabilis	6
H.PYLORI with Candida albicans	3
H.PYLORI with Klebsiella pneumoniae	6
Proteus Mirabilis	9
Pseudomonas aeruginosa	18
Candida Albicans	7
Klebsiella Pneumoniae	3
Culture Negative	15
TOTAL	100

Table.12 Relation of *H. pylori* isolation to biopsy site

BIOPSY SITE	NO. OF POSITIVE CASES	PERCENTAGE POSITIVITY
PYLORUS	36	57.2
POSTERIOR WALL	6	9.5
ANTERIOR WALL	8	12.7
DUODENUM	13	20.6
TOTAL	63	100%

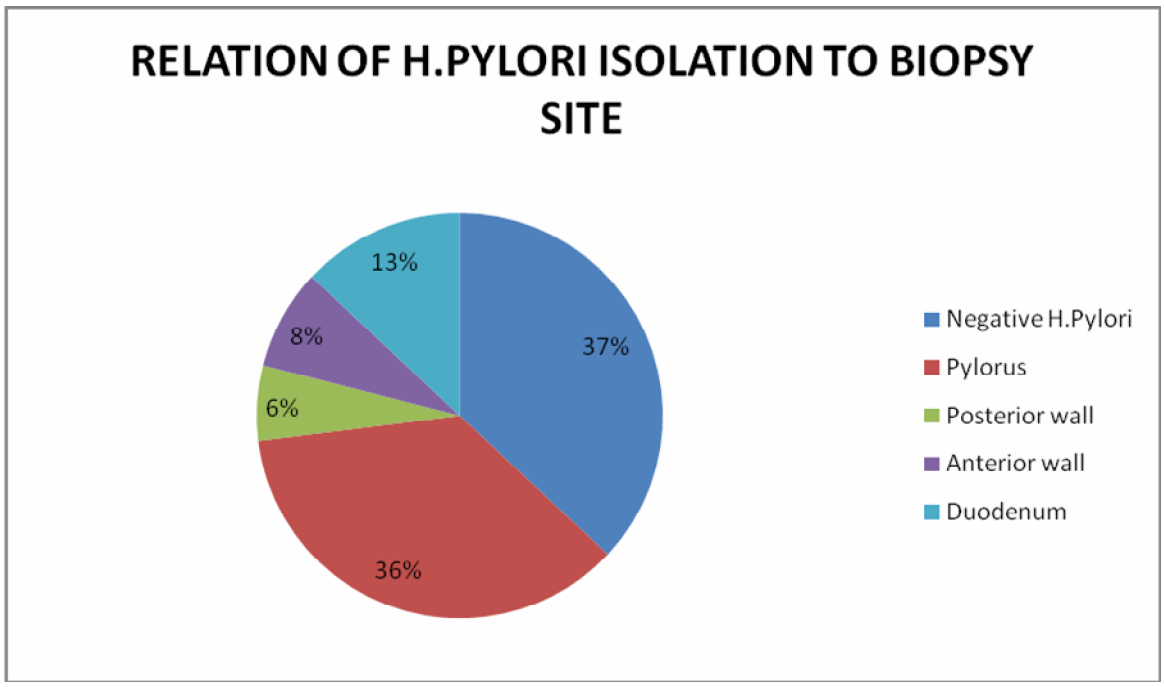

Table.13 Correlation of endoscopic findings with incidence of *H. pylori*

ENDOSCOPY FINDINGS	H.P POSITIVE OUT OF TOTAL ENDOSCOPIES	PERCENTAGE POSITIVITY
NORMAL	29	46.0
GASTRITIS	7	11.1
PYLORI C ULCER	9	14.3
DUODENITIS	7	11.1
DUODENAL ULCER	10	15.9
ESEOPHGITIS	1	1.6
TOTAL	63	100

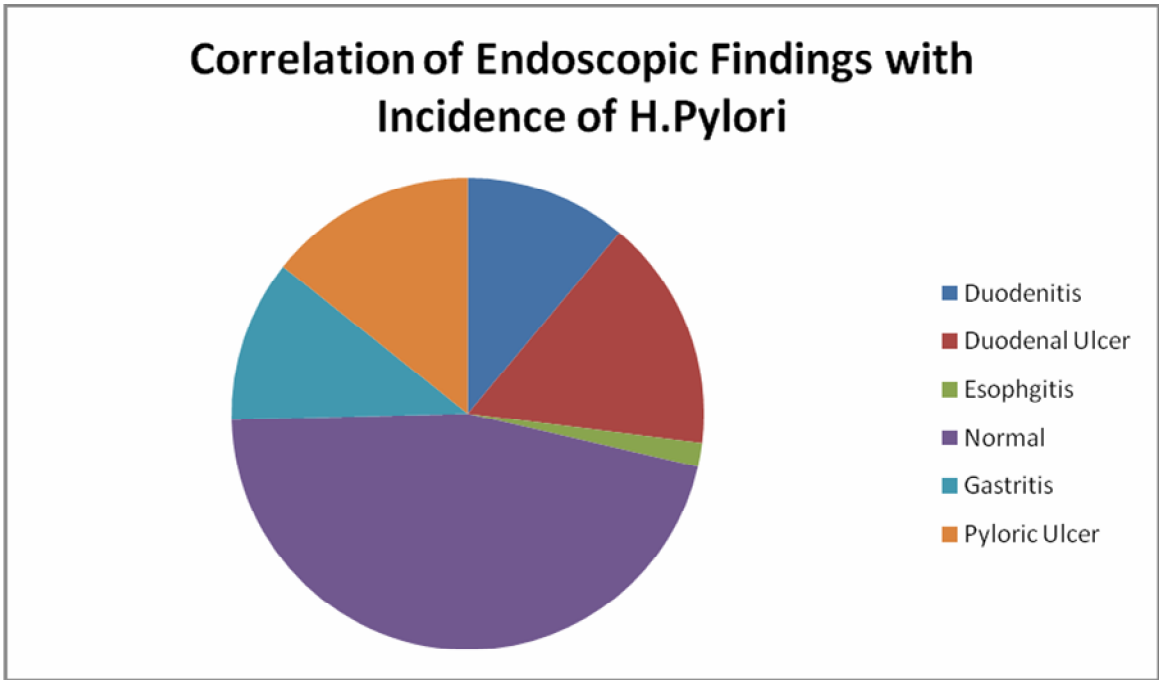

Table.14 Incidence of *H. pylori* in various gastric disorders

ENDOSCOPY FINDINGS	NO.TESTED	H.PYLORI POSITIVE	PERCENTAGE POSITIVITY
NORMAL	58	29	50
GASTRITIS	11	7	13.6
PYLORI CULCER	9	9	100
DUODENITIS	9	7	77.8
DUODENAL ULCER	10	10	100
ESEOPHGITIS	1	1	100
VARICES	2	--	0%
TOTAL	100		

Incidence of *H. pylori* in various gastric disorders

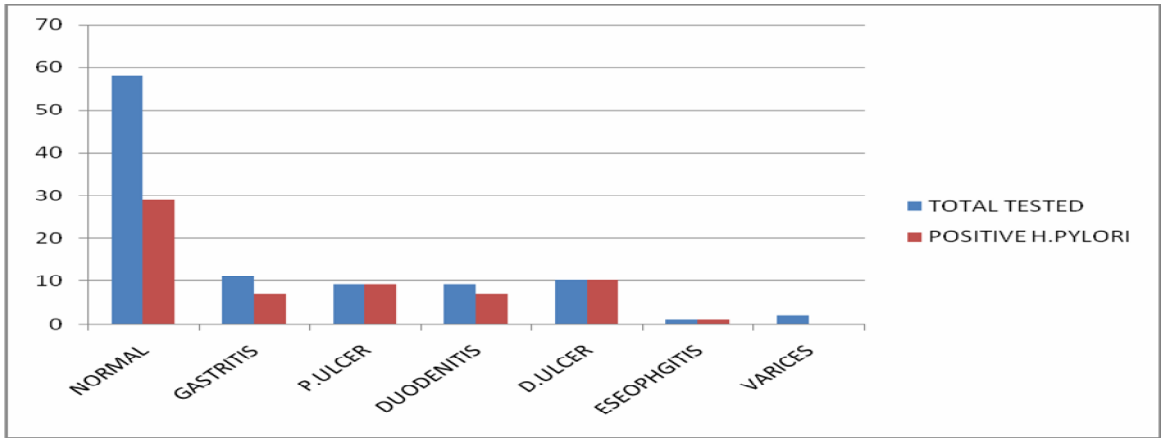

Table.15 Sensitivity pattern of *H. pylori*

DRUG	NO.OF SENSITIVE ISOLATES	PERCENTAGE POSITIVITY
CHILORAMPHENICOL	49	100%
STREPTOMYCIN	33	67.3%
NORFLOXACIN	45	91.9%
TETRACYCLIN	49	100%
ERYTHROMYCIN	22	44.9%
CIPROFLOXACIN	49	100%
NETILMICIN	48	98%
CEFAXONA	49	100%
PEFLOXACIN	49	100%
AMPICILLIN	19	38.8%
GENTAMYCIN	4	8.2%
NALIDIXIC ACID	NIL	0%

Sensitivity pattern of *H. pylori*

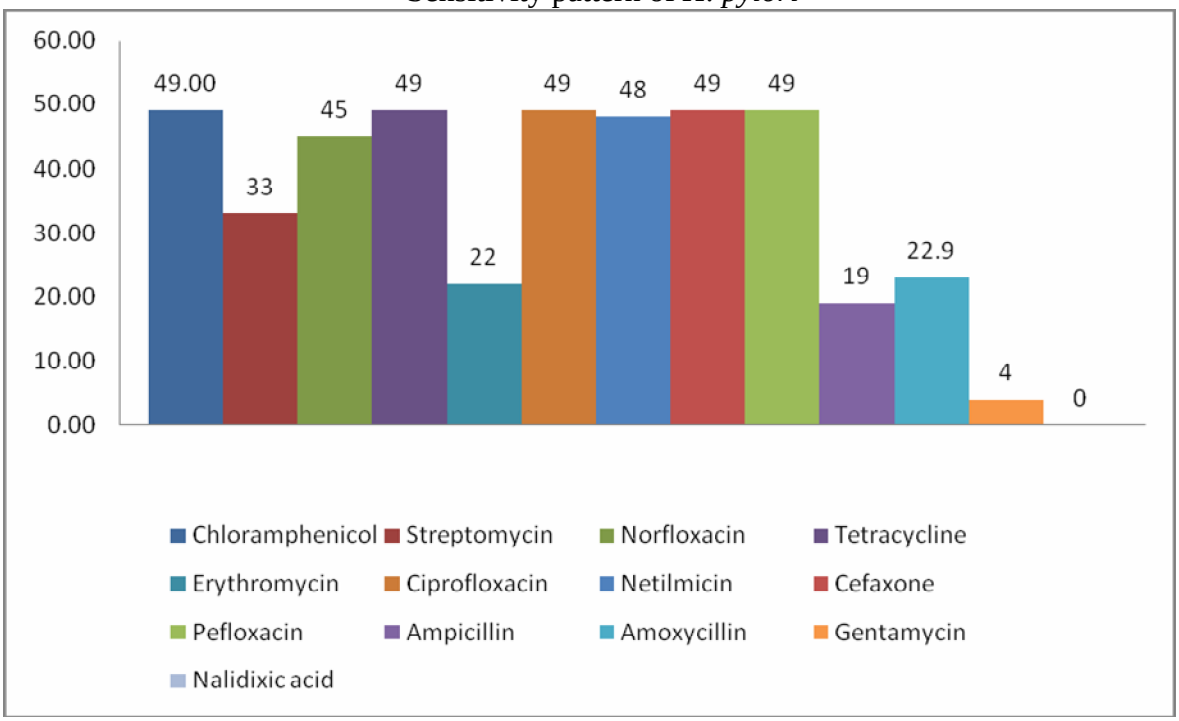

Table.16 Resistance pattern of *H. pylori*

DRUG	NO.OF RESISTANT ISOLATES	PERCENTAGE POSITIVITY
AMOICILLIN	29	59.2%
AMOXYCILLIN	26	53.1%
GENTAMYCIN	46	93.9%
NALIDIXIC ACID	49	100%
ERYTHROMYCIN	28	57.2%
STREPTOMYCIN	6	12.3%
NORFLOXACIN	5	10.2%
NETILMICIN	1	2%

Table.17 incidence of *H. pylori* by various studies

AUTHOR	YEAR OF STUDY	NUMBER OF CASE STUDIED	POSITIVE CASES	PERCENTAGE POSITIVITY
Marshall and Warrner	1984	96	56	58
Jones *et al.*	1984	50	31	62
Langerberg *et al.*	1984	50	32	64
Mc.Nutly *et al.*	1984	83	47	57
Good Win *et al.*	1985	103	60	58
Price *et al.*	1985	51	32	63
Pinkcard *et al.*	1986	80	48	80
Morris *et al.*	1986	382	174	46
Borromeo *et al.*	1987	80	51	64
Hazell *et al.*	1987	376	218	58
Taylor *et al.*	1987	57	21	41
Marshall *et al.*	1987	141	79	56
Tnowell *et al.*	1987	102	57	56
Anderson *et al.*	1987	153	71	46
Nanivadekar (Bombay)	1990	24	22	92
Shabnam (Chandigarh)	1991	100	43	43
Sumeet Sinha	1995	50	41	82
Vijay Dermateja	1996	170	51	30
D.Nair *et al.* (Delhi)	1997	114	76	66.6
Present study	1997	100	63	63

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