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Research Article

A PROSPECTIVE, OBSERVATIONAL STUDY ON THE EFFICACY OF DRUG REGIMEN USED FOR *HELICOBACTER PYLORI* ERADICATION IN PEPTIC ULCER DISEASE

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ABSTRACT

Aim of the study: We aimed to study the efficacy of triple drug therapy which is considered as a standard regimen. Objective of the study: To observe the role of pharmacist in preventing the recurrence of H. pylori infection. To assess whether the drug regimen prescribed eradicates the *H. pylori* infection. Results: In a period of 6 months a total of 358 cases were observed. Among them 142 cases are rapid urease test +ve and 216 are rapid urease test – ve cases. Of 142 patients included in the analysis, Peptic ulcer disease was more common in men when compared to women and prevalent in age group of 51-60 years. The body mass index analysis showed that 35.2% of patients were obese. From the personal history of the patients, it was concluded that 32.3% were smokers whereas 31.6% were alcoholic. The medication history analysis showed non-steroidal anti-inflammatory drugs were commonly used by the patients. The diagnostic endoscopic reports show that patients suffer from erosive pan gastritis followed by erosive gastritis. Conclusion: A 14-day triple therapy was found to be completely eradicate *H. pylori* infection. Out of the various demographic details that were taken into consideration age, gender, BMI, smokers, alcoholics, spicy food intake and drug abuse increased symptoms whereas co-morbid illness, sleeping pattern and food interval didn't affect much. Structured patient counselling and follow up had a significant effect which was seen in the form of zero recurrence, 100% medication adherence and improved quality of life.

Key words: *H-Pylori*, amoxicillin, clarithromycin, proton pump inhibitors.

INTRODUCTION

Peptic ulcer disease

Definition

Peptic ulcer disease refers to a group of ulcerative disorders the upper GI tract which is caused due to excessive production of gastric acid and pepsin. Usually effects inner lining of the stomach, duodenum and sometimes the lower oesophagus .[1]

Types of ulcers

- Ulcer in the stomach is known as a Gastric ulcer.
- Ulcer in the duodenum is known as a Duodenal ulcer.
- Ulcer caused by stomach acid moving up into the oesophagus is called as oesophageal ulcer.
- Ulcer which causes mucosal erosions and superficial haemorrhages in patients under extreme psychological stress is called as a stress induced ulcer.
- Ulcer caused due to long term NSAID's use resulting in gastric mucosal erosions is called as drug induced ulcers.
- Ulcer which develops at the gastro jejunal anastomosis typically at the jejunal side due to mucosal erosions is called as a marginal ulcer or stomach ulceration.

H. PYLORI INFECTION

Bacteria

Helicobacter pylori, previously known as Campylobacter pylori, are a Gram-negative, micro aerophilic bacteria found in the stomach. *H. pylori* are a type of bacteria which can invade digestive tract. Long term invasion can cause sores, called ulcers, within the lining of your stomach or the duodenum. [21]

Temperature for growth

The bacteria can survive and grow under the temperature of 30-37^{0}C.[6]

Transmission

H. pylori mode of transmission is poorly understood and is commonly transmitted through the following routes.

1. Faecal – oral route.
2. Oral – oral.
3. Iatrogenic.
4. In developing countries, a combination of untreated water, crowded conditions, and poor hygiene contributes to higher *H. pylori* prevalence. Most people become infected as children, and parents and siblings play a primary role in transmission.[22]

Faecal – oral route

The possible route for transmission of the infection is faecal oral route. The bacteria have been isolated from the faeces of infected children but rarely found in adults may be due to the toxic effect of faeces[23].

Oral – oral route

Due to untreated water in crowded area, poor hygiene and due to contaminated food and water contributes to the spread of infection.[24]

Iatrogenic
The most frequent mode of transmission is iatrogenic in which the tubes or endoscopes that was in contact with gastric mucosa of infected patient is used for another patient.[7]

Spreading of infection
H. pylori enter through oral route, move through the digestive system, and infect the stomach or the duodenum. The bacteria use tail-like flagella to move around and burrow into the stomach lining, which causes inflammation.

Unlike other bacteria, *H. pylori* can survive in the acidic environment of the stomach because they produce urease which neutralizes stomach acid. Urease, which encounters urea to form ammonia, which is harm to human cells. Depending on the infection which occurs in the stomach, can cause overproduction of stomach acid[4].

Progression
The process is very slow, taking decades, and may stop at any step because gastric cancers probably require several other factors to develop in addition *H. pylori* infection.
Stage 1 - Normal stomach lining which effects to mucosa.
Stage 2 - Inflammation of the stomach lining can cause chronic gastritis.
Stage 3 - Loss of stomach cells and impaired digestive system can cause atrophic gastritis.
Stage 4 - Transformation of the stomach lining into intestinal metaplasia.
Stage 5 - Beginning stages of stomach cancer can cause dysplasia
Stage 6 - Stomach cancer leads to gastric adenocarcinoma [25]

Epidemiology
In Western countries the percentage of people with *H. pylori* infections roughly matches age (i.e., 20% at age 20, 30% at age 30, 80% at age 80 etc.)[5].

Incidence
Predominant age - 70% of ulcers occur at the age of 25-64 years. Ulcer incidence increases with age.
cases / year - 500,000 new cases/year
Recurrence - 4 million/ Yr

Prevalence
Lifetime prevalence is 5-10% for patients not infected with *H. pylori* and 10-20% if infected. The prevalence based on diagnosed PUD was 0.12-1.50% and that based on hospitalization data was 0.10-0.19%. Majority of studies reported a decrease in incidence or prevalence of PUD over time.[26]

In India, the prevalence of active peptic ulcer was 3.4% and the lifetime prevalence was 8.8%. The duodenal and gastric ulcer ratio was12:1. *H. pylori* was present in 84.6% subjects with peptic ulcer. Peptic ulcer was more prevalent in the elderly. There was low prevalence of peptic ulcer in asymptomatic *H. pylori* infected persons.[27]

Etiology
The main causes of peptic ulcer disease are
- Due to imbalance of digestive fluids in stomach and duodenum.
- An infection with Helicobacter pylori bacteria causes inflammation.
- Long term use of non-steroidal anti-inflammatory drugs (NSAID).
- Smoking
- Alcohol use
- Genetic and Environmental factors
- Psychological stress
- Dietary negligence i.e, Daily intake of spicy foods and caffeine exacerbates symptoms of peptic ulcer disease.
- It also occurs due to low defence mechanism of body or immune system.
- Zollinger-Ellison syndrome
- Crohn's disease
- Liver cirrhosis. [1]

Pathophysiology
H. pylori infection invades the lining of the stomach wall by damaging the mucosal epithelial cells which leads excess secretion of gastric acid production. Since the cells will not be able to sustain the PH of the increased gastric acid levels leading to decreased resistance of mucosa leading to the damage to the lining of cells causing peptic ulcer disease[12].

Pathogenesis of peptic disease
The pathogenesis of peptic ulcer disease involves an imbalance between
- Defensive factors such as mucus-bicarbonate layer, prostaglandins, cellular regeneration, mucosal blood flow.
- Aggravating factors like hydrochloric acid, pepsin, ethanol, bile salts, and drugs). NSAIDs play an important role in the pathogenesis[13].

Mucosal defence mechanism
Mucus bicarbonate layer secreted by surface mucus cells forming a viscous gel over the gastric mucosa. Mucosal defences depend on an adequate blood supply and on formation within the gastric mucosa.

The acid production by parietal cells is done in three secretagogues.
- Gastrin (Endocrine) – produced by G cell in antrum and released into circulation.
- Acetylcholine (neurocrine) – released by vagal terminations.
- Histamine (paracrine) – stored by mast cells and ECL (Enterochromaffin) cells and is released into interstitial fluid[14].

Pathology
The pathology can be divided in three broad categories,
(1) *H. pylori* positive
(2) *H. pylori* negative and non-NSAID associated
(3) NSAID associated[15].

H. pylori causes an inflammatory response in gastric mucosa, with induction of epithelium derived cytokines, predominantly interleukin (IL) 8 and IL 1β.

Influx of neutrophils and macrophages into the gastric mucosa with release of lysosomal enzymes, leukotrienes (LT), and reactive oxygen species hampers mucosal defence and stimulates the immune pathogenetic process of ulcer formation[16].

H. pylori has high urease activity lead to producing ammonia to protect the organism from the acidic gastric environment. Production of alkaline ammonia by bacteria on the surface epithelium and in the glands of the antrum inhibits D cells in the glands from sensing the true level of acidity which leads to inappropriate release of somatostatin and hyper gastrinemia in the stomach[17].

Urease catalyzes production of ammonia, when in large concentrations lead to formation of toxic complexes such as ammonium chloride which along with bacterial phospholipases A

and C impair the phospholipid-rich layer in the mucosa that maintains mucosal hydration and integrity of the gastric epithelial barrier.

Metaplasia is an essential prerequisite for *H. pylori* colonization of duodenal epithelium, because colonization is specific and exclusive to gastric epithelial cells. After colonization of islands of duodenal gastric metaplasia, the inflamed duodenal mucosa becomes more susceptible to peptic acid attack and ulceration[18].

Ulcers can exist in the absence of *H. pylori* infection and non-NSAID group. Zollinger-Ellison syndrome, truly idiopathic ulcers, Cushing's ulcer, high-dose upper abdominal radiotherapy, fall in this category[19].

NSAID induced ulcers

Prostaglandins are important for mucosal integrity. Cyclo-oxygenase such as COX 1 and COX 2 inhibitors, COX 2 is supposed to cause gastric ulcer. Neutrophils invades mucosa which liberates oxygen free radicals, proteases release which leads to reducing capillary blood flow. The role of nitric oxide (NO) and hydrogen sulphide (H_2S), in maintaining integrity of gastric mucosa is well-known. NO and H_2S increase blood flow to mucosa, stimulate mucus secretion, and inhibit Neutrophil adherence. NSAIDs, inhibit NO and H_2S[20].

Stress effecting peptic ulcer disease

Stress leads to alteration in the behaviour as well as in physiology in the body of humans.

Altered behaviour leads to increased loss of appetite and reduced or disturbance in sleep. It has increased the social history of individuals for smoking, drinking alcohol and increased intake of NSAID's due to stress related headache[21]. This leads to decreased mucosal defences and leading to duodenal ulcers. Secondly on the altered physiology, the following occurs:

1. Decreased blood flow
2. Increased acid secretion
3. Increased gastric motility and decreased duodenal motility
4. Decreased immune defences.

The above with a *H. pylori* infection causes damage to epithelial mucosal cells in the stomach lining and will spread to the duodenum causing duodenal ulcers[22].

Signs and symptoms

- Abdominal pain and Epi -gastric pain associated with heart burn
- Nausea, vomiting, diarrhoea, anorexia mostly seen in gastric ulcer when compared to duodenal ulcer.
- Bloating**,** Cramping, Weight loss**,** Nocturnal pain
- Presence/absence of Melena / Hematemesis
- For duodenal ulcer – the abdominal pain starts after 1-3 hrs of intake of food
- Gastric ulcer – pain occurs right after having food as food may accumulate, precipitate and exacerbates pain
- Ulcer complications may include upper GI bleeding, perforation, penetration to adjacent organs i.e, pancreas, biliary tract, liver and gastric outlet obstruction[23].

Diagnosis

- Physical examination – Epigastric tenderness between umbilicus and xiphoid process.
- Haemoglobin, haematocrit and stool tests are done to detect for bleeding.

The diagnostics methods are broadly classified as:

i. Invasive method &
ii. Non-invasive method.

Invasive method

Invasive methods of investigation include gastric biopsy taken at endoscopy for Rapid Urease Test (RUT), bacterial culture, histology or the polymerase chain reaction. PUD diagnosis depends on visualising ulcer either by upper GI radiography or by endoscopy.

Non-invasive method

Serology

Infection with *H. pylori* elicits a systemic IgG antibody response that can be detected for diagnostic purposes. Anti-H.pylori antibodies can be detected by using ELISA or western blot. These tests are rapid and simple to perform, and less expensive than tests requiring endoscopic biopsies. Serological test is very sensitive and can detect fraction of bacteria which may be undetectable by biopsy or Urea breath test-based methods[24].

Urea breath test

The urea breath test is the preferred as a non-endoscopic method to know the level of H-pylori eradication

The 13C-labeled urea which is hydrolyzed by the bacterial urease enzyme with the formation of 13CO2, which is detected in the exhaled breath. An advantage of the Urea breath test is to detect active infection.[8]

Treatment

Non- pharmacological treatment

- Avoid smoking.
- Avoid alcohol use.
- Avoid stress.
- Avoid Spicy foods.
- Avoid foods that cause indigestion and increase the symptoms.
- Avoid Citrus fruits and dairy products like chocolates.
- Avoid skipping of meals.
- Avoid use of drugs especially Aspirin and promote Acetaminophen use.
- Avoid corticosteroid use.
- NSAID and corticosteroid should not be used for 6 weeks (healing time) when therapy is initiated

Pharmacological treatment

Medical therapy shall include the following:

- PPI – Proton Pump Inhibitors {Pantoprazole, Rabeprazole, Lansoprazole, Esomeprazole, Ilaprazole}
- Antibiotics include Triple drug regimen {Amoxicillin/Metronidazole + Clarithromycin + PPI}.
- Quadruple drug regimen {PPI + Metronidazole + Tetracycline + Bismuth sub salicylate}
- In some cases, to reduce symptoms prostaglandin analogue prescribed i.e, Misoprostol.
- Mucosal protectants – Sucralfate.
- To reduce acidity caused by histamine stimulated acid production – H2 receptor antagonists prescribed {Ranitidine, Cimetidine, Famotidine, Nizatidine.
- First-line eradication therapy is Triple drug regimen which includes a proton pump inhibitor (PPI)–based, three-drug regimen containing two antibiotics, usually Clarithromycin and Amoxicillin, reserving metronidazole for back-up therapy (e.g., Clarithromycin – metronidazole in penicillin-allergic patients).

- The PPI (Pantoprazole, Rabeprazole, Esomeprazole, Illaprazole, Omeprazole) should be taken 30 to 60 minutes before a meal along with the two antibiotics. Although an initial 7-day course provides minimally acceptable eradication rates, longer treatment periods (10 to 14 days) are associated with higher eradication rates.
- **Quadruple therapy** along with PPI such as bismuth subsalicylate, metronidazole and Tetracycline achieves similar eradication rates as PPI based triple therapy and permits shorter treatment duration. However, this regimen is often recommended as second-line treatment when a Clarithromycin–Amoxicillin regimen is used initially. All medications except the PPI should be taken with meals and at bedtime.
- Quinolone-based therapy is one of the most used third-line regimen. Levofloxacin have been widely used as *H. pylori* rescue therapy. A randomized study revealed that the eradication rate of sitafloxacin -based triple regimen was better than that of levofloxacin-based triple regimen. Clarithromycin - resistant strains when compared to the standard lansoprazole, Amoxicillin, and Clarithromycin triple regimen.[11]
- **Second-line empiric treatment should be followed:**

Use antibiotics that were not included in the initial regimen, include antibiotics that do not have resistance problems, Use a drug that has a topical effect and Can be extended to 14 days.

Thus, if a PPI–Amoxicillin–Clarithromycin regimen fails, therapy should be instituted with a PPI, bismuth subsalicylate, metronidazole, and Tetracycline for 14 days.

Maintenance therapy with a PPI or H_2RA (Ranitidine, Famotidine, Cimetidine) is recommended. For treatment of NSAID-induced ulcers, non-selective NSAIDs should be discontinued if an active ulcer is confirmed, treat with a PPI, H_2 RA, or sucralfate. Co therapy with a PPI or misoprostol or switching to a selective cyclooxygenase-2 (COX-2) Celecoxib, etoricoxib inhibitor is recommended for patients at risk of developing an ulcer-related complication. HP-positive patients should receive eradication therapy. In *H-pylori* negative patients have higher PPI doses e.g., omeprazole 40 mg/day heal the majority of ulcers. Continuous PPI treatment is often necessary to maintain healing.

High-dose PPI and Amoxicillin dual therapy is a useful option as an alternative third-line treatment regimen. (Patients with penicillin allergy should be rechecked for allergy test) Or the dose and duration of the regimen is increased from 7 days to 14 days

METHODOLOGY

Study design: The study is prospective and observational.

Inclusion criteria
- Inpatients and outpatients of hospital.
- Patients above 20 years of age.
- Patients who are willing to give their consent.
- Patients who are NSAID's abusers.

Exclusion criteria
- Patients who are not willing to give consent form.
- Patients under the age of 20 are excluded.
- Patients who are allergic and hypersensitive to drugs.
- Pregnant woman, breast feeding, childbearing age
- Uncontrolled diabetes mellitus or hypertension.
- History of malignancy, surgery of oesophagectomy, gastrectomy and hereditary diseases such as galactose intolerance, lactase deficiency and glucose- galactose malabsorption

Method of data collection
- Data collection
- Patient questionnaire/interview

Duration of the study
The study was conducted for a period of 6 months.

Ethical Approval: Institutional ethical clearance number for this study IEC/MCSC/PROT/2019/007.

RESULTS

Table 1: Distribution of patients based on rapid urease test

Rapid Urease Test	No Of Patients	Total no. of patients
RUT +ve	142 (40%)	358
RUT -ve	216 (60%)	

Table 2: Gender wise distribution of *H. pylori* infected patients

Gender	Male (%)	Female (%)	Total no. of patients
No. of patients	88 (62%)	54 (38%)	142

Table 3: Distributions of patients based on number of in and outpatients

Department	Inpatient (IP)	Outpatient (OP)	Total no. of patients
No. of patients	20 (14%)	122 (86%)	142

Table 4: Grouping of patients based on age

Gender	20 - 30	31 - 40	41 - 50	51 - 60	61 - 70	71 – 80
Male	9 (10.23%)	13 (14.77%)	23 (26.14%)	35 (39.77%)	8 (9.09%)	0
Female	3 (5.56%)	9 (16.67%)	12 (22.22%)	22 (40.74%)	6 (11.11%)	2 (3.70%)

No of males = 88, No of females = 54

Table 5: Classification of cases based on BMI

BMI	Males	Females
> 30 (Obese)	30	20
25 - 29.9(Overweight)	27	13
18.5 - 24.9(Normal)	20	14
< 18.5(Underweight)	11	7

N = 142, M = 88, F = 54

Table 6: Grouping patients based on personal history

Abuse	Both Alcoholic and Smoker	No Abuse	Only Smoker	Only Alcoholic
No. of patients	30	27	16	15

N = 88 Males

Table 7: Distribution of cases based on dietary intake

Diet	Spicy foods	Caffeine/Tea	Citrus foods	Beverages
No. of patients	113 (79.5%)	109 (76.7%)	66 (46.4%)	57 (40.1%)

Table 8: Distribution of patients based on food intake patterns

Food	Overeating	Frequent meals	Skipping meals	Normal
No. of patients	43 (30%)	41 (29%)	32 (23%)	26 (18%)

Table 9: Medical history of patients with drug abuse

Medications	NSAID Abuse	NSAID Induced Ulcer	Corticosteroid Abuse	Corticosteroid Induced Ulcer	None
No. of patients	19 (12%)	12 (8%)	7 (4%)	4 (3%)	116 (73%)

Table 10: Classification of cases based on clinical manifestations of the patients

Complaints	No. of patients
Abdominal pain	127
Epigastric pain	102
Heart burn BF	89
Stress	86
Loss of appetite	47
Nausea & vomiting	47
Melena	34
Weight loss	28
Heart burn AF	22
Hematemesis	18
Loose Stools	17
Constipation	16

Table 11: Co-morbid illness observed in PUD patients

Co-morbidity	No. of patients
HTN	81
DM	53
Fatty liver	25
Anaemia	22
Hypothyroid	17
CLD	13
Portal HTN	11
Cholelithiasis	11
Asthma	9
CKD	8
RA	5
UTI	5
Spondylosis	1

Table 12: Distribution of the study sample based on type of ulcer

Type of ulcer	No. of patients
Gastric ulcer	103 (73%)
Duodenal ulcer	39 (27%)

Table 13: Distribution of patients based on choice of drug combinations

Drug	Triple drug + Probiotics	Only Probiotics	Only Triple drug
No. of patients	41 (31%)	9 (7%)	81 (62%)

Table 14: Choice of antibiotics prescribed to PUD patients

Drug	Amoxicillin Triple drug	Metronidazole Triple drug	Rifamixin	Quadruple drug	None
No. of patients	113 (79%)	14 (10%)	4 (3%)	0	11 (8%)

Table 15: Distribution of in and outpatients based on choice of PPI and H_2RA

Drug	Esomeprazole	Pantoprazole	Rabeprazole	Ranitidine
No. of Inpatients	0	20 (14.08%)	0	1
No. of Outpatients	113 (79.5%)	84 (59.1%)	15 (10.5%)	0

Table 16: Symptomatic relief medications given to PUD patients

Drug	PPI	Sucralfate	Tramadol + Acetaminophen	Ondansetron	Probiotics	Lactulose
No. of patients	142 (100%)	113 (79.57%)	82 (57.7%)	47 (33.09%)	41 (28.8%)	27 (19.01%)

A total of 358 cases have been collected in our study. Out of which 142 (40%) were RUT positive and 216 (60%) were RUT negative patients. (Table 1)

Out of total 142 prescriptions assessed 62% were male and 38% were female patients. In this analysis there was a predominance of *H. pylori* in male patients of about 24% when compared to females. (Table 2)

About 142 patients were included in the study of which 122 were Outpatients and 20 were Inpatients. (Table 3)

From the data collected and analysed we have found that the incidence of PUD increases with increasing age. The average age group of affected individuals was found to be 51-60 years of which males were 39.77% and females were 40.74%. (Table 4)

By the above results we can say that most of the patients had BMI >30 and are categorized as Obese (males 30, females 20). (Table 5)

The patients were grouped based on their personal history which included smoking, alcohol consumption and no abuse. Out of 88 males 52.2% of patients were smokers, 51.1% of patients were alcoholic and out of these 88 males only 30.6% patients had no abuse. (Table 6)

The dietary intake information was collected. The information includes the type of food being consumed, amount of caffeine/tea consumed per day, intake of beverages and citrus fruits. 79.5% of the patients have high intake of spicy food in their daily diet followed by 76.7% of the patients have frequent intake caffeine, tea and 40.1% had beverages frequently. (Table 7)

It was observed that 18% of the patients consumed food in normal pattern (3 times a day), overeating was found to be in 30% patients (eating excess food per meal), 29% of the patients used to have frequent meals and skipping of meals was observed in 23% patients (usually skipped Breakfast). (Table 8)

The medical history of patients with drug abuse has been collected and analysed which concludes that 12% of the patients were NSAID abusers and 4% were corticosteroid abusers. Based on the abuse of such drugs, 8% of the patients were reported with NSAID induced ulcer, 3% of patients with corticosteroid induced ulcer and 73% of patients had no drug abuse. (Table 9)

The cases have been classified based on the clinical manifestations. Majority of patient's i.e., 89.4% have complained of abdominal pain followed by 71.83% of epi-gastric pain, 62.6% of heartburn before food, 60.56% of stress and 11.2% of constipation. (Table 10)

The data for the co-morbid illness of the patients were collected and observed which concluded that 57.04% of the patients were hypertensive patients, 37.32% were diabetic, 17.6% were suffering from fatty liver, 15.49% were anaemic, 0.70% suffered from Spondylosis and the other co-morbid illness stated were minimum and limited. (Table 11)

The cases have been classified based on the type of ulcer and the 73% of patients have been diagnosed with Gastric Ulcer and 27% with duodenal ulcer. (Table 12)

The combinations of drugs given to the patients are 62% of patients were prescribed alone with triple drug regimen. 7% of patients were prescribed with probiotics and remaining 31% of patients were given both triple drug therapy and probiotics in combination. (Table 13)

The antibiotics were prescribed for the complete eradication of *H. pylori* infection. In our study 79% of patients were prescribed with amoxicillin triple drug therapy, 10% of patients were prescribed with metronidazole triple drug therapy (given for those who had allergy with amoxicillin) and 3% of patients were prescribed with other choice of antibiotics. (Table 14)

The highly recommended drug in our study for reduction in acid production is proton pump inhibitors. The PPI's prescribed were 79.57% of the outpatients were prescribed with esomeprazole. 14.08% of pantoprazole were prescribed for in patients and 59.1% were prescribed for outpatients. 10.5% of outpatients were prescribed with rabeprazole drug. And the use of H_2 Receptor antagonists was very minimal. From this result we can conclude that esomeprazole is more effective when compared to other proton pump inhibitors. (Table 15)

The drugs given were: 100% of patients were prescribed with proton pump inhibitors, 57.7% of patients were prescribed with combination of tramadol and acetaminophen, 79.57% of patients were prescribed with sucralfate, 28.8% of patients were prescribed with probiotics for antibiotic induced diarrhoea, 33.09% of patients were prescribed with ondansetron, 19.01% of patients were prescribed with lactulose for constipated patients. (Table 16)

DISCUSSION

In our 6 months study 358 patients were observed in Hyderabad multi-speciality hospital, 40% were rapid urease test +ve and 60% were rapid urease test –ve. Out of the 40% of rapid urease test +ve patients 73% of the patients were diagnosed with gastric ulcer and 27% were diagnosed with duodenal ulcer.

Old age is a well-known risk factor for peptic ulcer disease in all other studies. This was also found to be the risk factor for PUD in our study which was seen in the age group of 51-60 years.

In our study, majority of patients were men with 62% and female were 38% which is not consistent with the results of Hyo Jun Ahn et.al, 40% were male and 60% were female.

Recently Boylan and colleagues examined the association of peptic ulcer disease with BMI in a prospective cohort study called the 'Health professionals' follow up study and they found that BMI was a risk factor of peptic ulcer disease and the same was observed in our study.

The patient's diet information was collected, and they were high on intake of spicy foods followed by intake of caffeine and tea. From the personal history of the patients (88), it was concluded

that 34.9% are both alcoholic and smokers, 32.3% were smokers whereas 31.6% were alcoholic.

In our study, the medical history of patients with drug abuse showed 45% were NSAID abusers out of which 29% were reported with NSAID induced ulcer and 17% were corticosteroid abusers out of which 9% of patients were reported to have corticosteroid induced ulcers. These are considered as drug induced gastritis. In the study of Shilpa AH, estimated that 38% cases caused gastritis because of drug abuse[19].

In our study, 73% were diagnosed with gastric ulcer and 27% were diagnosed with duodenal ulcer. Gastric ulcers affected more in the age group of 20-60 years and duodenal ulcers were affected after age of 60 years. A study was done on peptic ulcer in the year 2014 which analysed and concluded that stomach ulcers tend to occur after the age of 60 and they affect more women than men. Duodenal ulcers usually appear between ages 30 and 50 and is more common in men than women.

In our study, triple drug regimen was given for 14 days and showed 100% effectiveness to the patients affected with *H. pylori* infection. And esomeprazole was the most effective proton pump inhibitor. In the paper entitled "Comparison between single dose esomeprazole and pantoprazole based triple therapy on the effectiveness for helicobacter pylori eradication in Taiwanese population" H.Y Shih et.al, show a higher eradication rate in esomeprazole containing triple therapy than pantoprazole containing triple therapy[28].

Evidence indicates a strong association between *H. pylori* infection and dyspeptic symptoms gastric ulcer, duodenal ulcer and development of gastric cancer hence treating *H. pylori* infection is essential to reduce the risk of development of complications. According to current guidelines triple combination therapy is considered as a standard regimen for the first line Anti *H. pylori* treatment. Studies suggested eradication rates achieved by first line treatment with PPI, Amoxicillin and clarithromycin have decreased to 70-85%, due to increase in antibiotic resistance but in our study, there was 100% eradication rate. We observed that patients with higher education responded significantly better to Anti HP treatment which is attributed through improved quality of life and zero recurrence of ulcers.

In addition, the physician emphasized the influence of alcohol intake, smoking and importance of reducing stress levels in patients. Patient education on preventive measures and treatment compliance resulted in better treatment outcome.

Despite of our important findings the study had the limitation that the patient undergoing endoscopy for HP related symptoms constituted a minority of infected population but is still a representative of the population. Another limitation of our study is that during the follow up period not all the patients came up for the endoscopic procedure after completion of treatment.

CONCLUSION

Our study focussed on the efficacy of drug regimen used for Helicobacter pylori eradication of Peptic Ulcer Disease. Males were at more risk than females which could possibly be due to cigarette smoking, alcohol consumption and high intake of spicy foods on a daily basis. The age group that was affected the most was 51-60 years in both males and females. Among the various demographic details that were taken into consideration are age, gender, BMI, smoking, alcoholism, spicy food intake and drug abuse did not affect much. The medication history when considered was found to be that most of the patients are abusers of drugs like NSAIDs (Aspirin, Ibuprofen) and corticosteroids (Prednisolone) and those that has exacerbated the symptoms for PUD. The endoscopic reports concluded that 73% of the patients were diagnosed with Gastric ulcer and 27% with duodenal ulcer. In *H. pylori* infected patients with PUD, 14 days triple therapy which is a combination of amoxicillin, clarithromycin and esomeprazole were prescribed in accordance to standard guidelines. Triple drug therapy for *H. pylori* eradication showed 100% eradication rate and zero recurrence or relapse which was confirmed through Urea Breath Test. Hence quadruple drug treatment was not prescribed to any of the patients. Structured patient counselling and follow up had a significant effect which was seen in the form of zero recurrence, 100% medication adherence and improved quality of life.

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