WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.453
Volume 13, Issue 11, 1125-1131. Research Article ISSN 2277–7105

PREVALENCE OF H.PYLORI AMONG GASTRITIS PATIENTS IN GASTROENTEROLOGY DEPARTMENT IN A TERTIARY CARE HOSPITAL

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Article Received on
11 April 2024,

Revised on 01 May 2024,
Accepted on 21 May 2024

DOI: 10.20959/wjpr202411-32447

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ABSTRACT

Project title: Prevalence of H.pylori among gastritis patients in gastroenterology department in a tertiary care hospital. **Background:** In developing countries Helicobacter pylori infection is a major health ailment. The infection is related to high morbidity and mortality ranging from chronic gastritis to gastric malignancies. The prevalance of H. pylori infection greatly varies from region to region and country to country. H.pylori gastritis is the principle cause of chronic active gastritis and has major complications such as gastric adenocarcinoma and mucosa-associated lymphoid tissue lymphoma. **Aim:** The aim of the study is to determine the prevalence of H.pylori among gastritis patients in gastroenterology department in a tertiary care hospital. **Materials and Methods:** A retrospective cross-sectional study was conducted over a period of six months in a tertiary care hospital. A total of 100 gastritis patients were included in the study. Case records were retrospectively reviewed for demographic details, laboratory data, management and outcomes. Data analysis was conducted by using SPSS and Microsoft Excel 2019. **Result:** The prevalence of H.pylori was found to be 18. Gender and age has no significant effect on infection thus we conclude that infection does not depend on gender and age of the patient. While considering the association of symptoms and presence of H. pylori, there is no significant association between them. There is no significant association with the presence of H. pylori and risk factors. Out of 100

samples 54 patients were of pan gastritis and 46 patients were of antral gastritis. **Conclusion:** The study provides an insight into the prevalance of H.pylori.

KEYWORDS: H.pylori- Helicobacter pylori, CagA- Cytotoxin associated gene A, IL- Interleukin, ROS- Reactive oxygen species, GI- Gastrointestine.

INTRODUCTION

The term "gastritis" was first used in 1728 by the German Physician, George Ernst Stahl to describe the inflammation of the inner lining of the stomach- now known to be secondary to mucosal injury (ie, cell damage and regeneration).[1]

H. pylori transmission by oral to oral and feco- oral are the most common routes.[2] Crowded living conditions, poor sanitation, poor personal hygiene and a poor water supply are associated with higher rates of infection. The H. pylori uses its flagella to burrow in the stomach lining and reaches the epithelial cells, underneath which is less acidic.[3] The H. pylori also survives in the highly acidic environment of the stomach by producing urease which converts the urea in the stomach to carbon di oxide and ammonia which acts as a buffer. This ammonia is toxic to human cells. The urease is also required for maintaining chronic infection.[4]

H.pylori has flagella that enable it to move and help it to penetrate the mucous layer so that it comes into contact with the gastric epithelial cells. H pylori produces inflammation by activating a number of toxins and enzymes that activate IL-8, which attracts polymorphs and monocytes that leads to acute gastritis. The lymphocytes and other mononuclear cells are activated by Antigen presenting cells that lead to chronic gastritis. After the primary exposure to H pylori the infection is established within a few weeks.[5]

It produces inflammation through the production of a number of toxins and enzymes. The intense inflammation can result in loss of gastric glands that are responsible for the production of acid. This is referred as atrophic gastritis. As a result gastric acid production drops. The virulence genotypes of the microbe is an important determinant for the severity of the gastritis and the formation of intestinal metaplasia, the transformation of gastric epithelium. This transformation can leads to gastric cancer.[5]

The H. pylori affects the gastric mucosa by these slowly progressing steps:

Stage 1: Normal stomach lining (Mucosa)

Stage 2: Inflammation of the stomach lining (Chronic gastritis)

Stage 3: Loss of stomach cells and impaired digestive system (Atrophic gastritis)

Stage 4: Transformation of the stomach lining (Intestinal metaplasia)

Stage 5: Beginning stages of stomach cancer (Dysplasia)

Stage 6: Stomach cancer (Gastric adenocarcinoma.[3]

MATERIALS AND METHODS

Study design: Retrospective cross-sectional study

Study setting: SH Medical Centre, Kottayam

Study duration: 6-month duration study

Sample size: 100 patients

Sampling technique: Convenience sampling technique

Sample size determination

100 cases will be collected from Gastroenterology department

$N= Z^2 (p\times q)/E^2$

Where, Z = 1.96 (95%)

p = 0.85, q = 1-p = 0.15, E = 0.07

$N = 1.96^2(0.85 \times 0.15)/ 0.07^2$

= 99.6 = 100

Criteria for patient selection

Inclusion criteria

1. Adults and geriatrics
2. Patients with gastritis
3. Both male and female

Exclusion criteria

1. The pediatric patients

RESULT

The total number of participants in our study was 100 patients.

Prevalence of h. pylori

Table 1: Prevalence of H.pylori.

H. Pylori	Frequency (n=100)	Prevalence
Yes	18	18
No	82	0

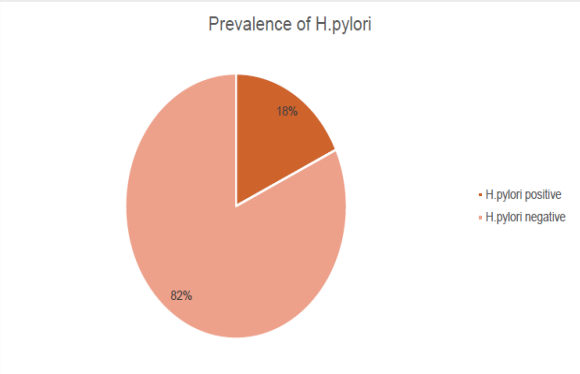

Figure 1: Prevalence of H.pylori.

The prevalence of H pylori was found to be 18%, therefore it is evident from the study that H pylori is infrequent and of low consequence.

Gender distribution

Table 2: Percentage distribution according to gender.

Sex	Frequency (n=100)	Percentage
Females	56	56%
Males	44	44%

Table 3: Association of H pylori and gender.

Sex	H. Pylori(+)	H. Pylori (-)	Total	x^2 Test	P Value
Female	12	44	56	x^2=1.014	p=0.314
Male	6	38	34		

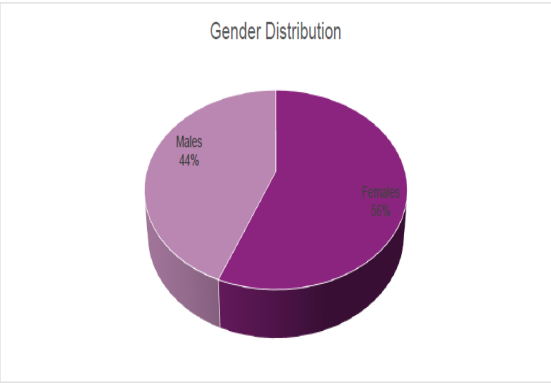

Figure 2: Gender distribution.

Gender has no significant effect on infection thus we conclude that infection does not depend on sex of the patient.

Age distribution

Table 4: Percentage distribution according to age.

Age		Frequency(n=100)	Percentage
Young Adults	18-39years	40	40%
Middle agedadults	40-59	32	32%
Old adults	>60years	28	28%

Table 5: Association of H. pylori and age.

Age	H. Pylori (+)	H. Pylori (-)	Total	x^2 TEST	P Value
18-39years	8	48	56	x^2=0.310	P =0.578
40-59	4	12	16		
>60years	6	22	28		

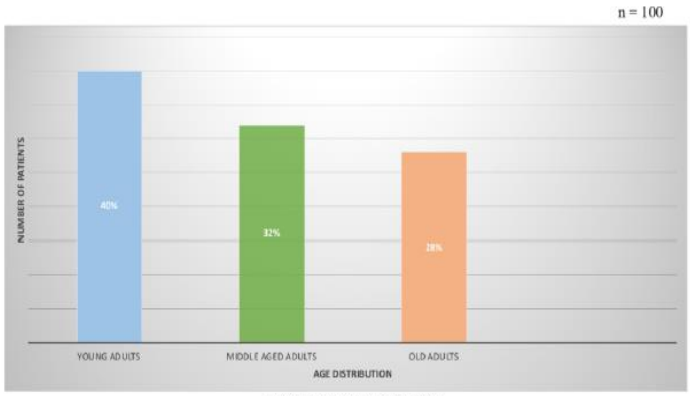

FIGURE 3: DISTRIBUTIONOF AGE

Age has no significant effect on infection thus we conclude that the infection does not depend onthe age of the patient.

Association of symptom and h pylori

Table 7: Association of symptoms and H.pylori.

Symptoms	H. Pylori(+)	H. Pylori(-)	Total	x^2 TEST	P value
Vomiting (yes)	10	37	47	x^2=0.645	P=0.422
vomiting(No)	8	45	53		
Nausea(Yes)	4	16	20	x^2=0.068	P=0.795
Nausea(no)	14	66	80		
Abdominal Pain(yes)	17	66	83	x^2=2.038	P=0.153
Abdominal Pain(no)	1	16	17		
Regurgitation(yes)	2	3	5	x^2=1.726	P=0.189

Regurgitation(no)	16	79	95		
Diarrhoea(yes)	6	17	23	x^2=1.323	P=0.250
Diarrhoea(no)	12	65	77		

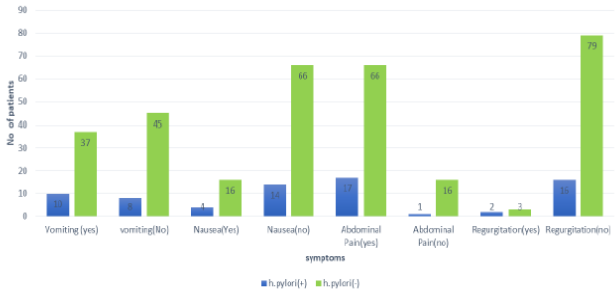

FIGURE 8.5: DISTRIBUTION OF SYMPTOMS

There is no significant association between symptoms and H. Pylori.

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CONCLUSION

Helicobacter pylori infection is known to be among the most common human infections worldwide; approximately 50% of the world's population is infected with *H. pylori* while most of the infected individuals are asymptomatic; it acts as a cofactor to produce gastrointestinal (GI) disorders (dyspepsia) in a small but significant minority. *H. pylori* gastritis is the principal cause of chronic active gastritis and has major complications such as gastric adenocarcinoma and mucosa-associated lymphoid tissue lymphoma. The purpose of our study was to analyse the prevalence of H. pylori among gastritis patient. The prevalence of Helicobacter pylori was analyzed among gastritis patients. The prevalence of H. pylori among 100 gastritis patients were 18.The age at which more prevalence of H pylori were found in young adults and middle-aged adults

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