# **Original Research Paper**



## **Pathology**

# A HISTOPATHOLOGICAL STUDY OF ASSOCIATION HELICOBACTER PYLORI INFECTION WITH CARCINOMA STOMACH IN JLNMCH, BHAGALPUR

Dr. Ankur	Senior resident Department of pathology Jawaharlal Nehru Medical College, Bhagalpur, Bihar	
Dr. Mritunjay	Associate professor Department of surgery Jawaharlal Nehru Medical College,	
Kumar	Bhagalpur, Bihar	

Aim: The aim of the study is to see the correlation of carcinoma stomach with infection by Helicobacter pylori by means of histopathology. Methods: In this study, 80 malignant looking lesions biopsies were taken by endoscopy. The study was done between April 2016 to March 2017 in department of pathology, JLNMCH, Bhagalpur. Thorough histo pathological analysis were done with H & E stained slides and special stains for the detection on Helicobacter pylori- i.e. Giemsa, Triple stain. Results: The study showed that in 48 biopsies were malignant and 32 biopsies were not associated with malignancies. H. Pylori infection was seen association in 25 cases of malignant biopsies and 14 cases of non malignant biopsies. Males were more affected than females. Average age of presentation of malignancy was 55 years. Most common presentation was dysphagia. Most common carcinoma associated with h pylori infection was adenocarcinoma of stomach. Conclusion- It was shown in this study that H.pylori infection is the major risk factor for the development of Gastric carcinoma.

## **KEYWORDS**: Helicobacter pylori, malignant biopsies, adenocarcinoma, histopathology

#### Introduction-

Humans are the only host for H.pylori, which is found in stomach, and in duodenum, oesophagus and rectum on areas of metaplastic gastric epithelium. Other helicobacter species have been isolated from the animals. Animal models of Helicobacter infection have been developed due to shared characteristics of other Helicobacters like H.mustelae and H. felis with H.pylori. H.pylori exists the world over and its prevalence in the population increases with age. In developed countries, prevalence increases about 1% per year of age where it is rare in children, and reaches 70% in the seventh decade. In developing countries, more than 50% children acquire the infection by the age of 10 years, and more than 80% of the population gets infected by the age of 20 years. In asymptomatic individuals prevalence of H.pylori infection varies from 31%-84%. H.pylori infection is chronic and once acquired remains life long, unless eradicated by antibiotics given for some other conditions. Humoral and tissue immune response by the host is usually not sufficient to clear the infection. Though the mode of transmission is not yet well established, most probably it takes place by oral-oral or faeco-oral route and important risk factors are socioeconomic status and age. Overcrowding, poor socio-economic status and poor hygiene are associated with high infection rate. Reinfection rate after eradication is quite high in developing countries due to the above mentioned risk factors. Colonisation of H.Pylori occurs by producing urease and gastric acid inhibitory protein. It can colonise only in gastric type epithelium and cannot stay anywhere else in the GI tract in absence of gastric mucosa. Metaplasia, which is present in more than 90% of patients of duodenal ulcer, occurs by replacing the columnar cells, normally covering the duodenal villi, by gastric type epithelium. Adhesion of H.pylori to the gastric epithelium occurs by tissue specific proteins. Colonisation of the duodenal bulb by H.pylori leads to mucosal inflammation which makes it vulnerable to attack by acid or pepsin or bile resulting into ulceration, however, factors leading to gastric metaplasia in the duodenal bulb are not known. Stimulation of the immune system of H.pylori contributes to host damage and it evades the immunological clearance. Gastric cancer is the third most common cause of cancer-related death in the world. It is now wellestablished that Helicobacter pylori infection predispose individuals toward gastric adenocarcinoma later in life. It has since been classified as a class I carcinogen by the World Health Organization. Research suggests that the oncogenic effects of Helicobacter pylori can occur through a variety of mechanisms, including the indirect inflammatory effects of Helicobacter pylori on the gastric mucosa and the direct epigenetic effects of Helicobacter pylori on individual cells. Whilst infected with Helicobacter pylori, a combination of environmental and host-dependent factors determines the likelihood of developing gastric cancer. Controversy remains regarding the effects of eradication of Helicobacter pylori on the prevention of further progression of gastric lesions and the possibility for regression of atrophic gastritis.

#### Material and Methods

In this study, 80 malignant looking lesions biopsies were taken by endoscopy. The study was done between April 2016 to March 2017 in department of pathology, JLNMCH, Bhagalpur. Thorough histo pathological analysis were done with H & E stained slides and special stains for the detection on Helicobacter pylori- i.e. Giemsa, Triple stain. Both male and female patients were taken into study with age group between 10 yrs to 70 yrs.

Inclusion Criteria: All endoscopic biopsies of the gastric region. Exclusion Criteria: All lesions of the mouth and pharynx and oesophagus.

Procedure- Endoscopies were performed in department of surgery, JLNMCH, Bhaglapur, using upper GI endoscope. Biopsies were taken from the suspected malignant lesions. The biopsy specimen was kept in 10% formalin for fixation. Five micron thick sections were cut perpendicular to this surface and four to five sections were prepared on each slide. Each section was stained with H and E and studied microscopically. Adequacy of biopsy was assessed. An attempt was made to diagnose the lesion on gross visualisation during endoscopy and to correlate them histopathologically. Special stains were done whenever required

### Results

The study showed that in 48 biopsies were malignant and 32 biopsies were not associated with malignancies.

	Biopsies
Number of Malignant	48
Number of Benign	32
Total	80

H. Pylori infection was seen association in 25 cases of malignant biopsies and 14 cases of non malignant biopsies.

	Biopsies	H. Pylori infection Association cases
Number of Malignant	48	25
Number of Benign	32	14
Total	80	39

Males were more affected than females.

	Number of patients
Males	55
Females	25
Total	80

Average age of presentation of malignancy was 55 years. Most common presentation was dysphagia.

Most common carcinoma associated with h pylori infection was adenocarcinoma of stomach.

#### DISCUSSION:

The use of endosopy and endoscopic biopsies have led us to find many associations with carcinoma stomach. In this study we have shown that H. Pylori infection is almost associated with 50% of the biopsies and more than 50% associations are malignant. In this study majority of cases were of male gender. The reason for gender ratio favouring males could be of the fact that males are exposed to more risk factors than females. Out of 80 cases studied, 68 cases diagnosed endoscopically as gastric carcinoma correlated with those of histopathological diagnosis as Adenocarcinoma. Our study showed good correlation in the cases of carcinoma. Findings were similar to the studies conducted by Hecker et al, Sharma S. et al. In our study the average age group affected was 55 years which was similar with other studies. Incidence of carcinoma increased with increase of age similar to the study done by Sharma S et al.

#### Conclusion

The aetiology of gastric cancer is complex and multifactorial, involving environmental and host related factors as well as genetic and epigenetic alterations. H. pylori infection is a one of the cause for gastric cancer. It was shown in this study that H.pylori infection is the major risk factor for the development of Gastric carcinoma.

#### References

- Megraud F. Epidemiology of Helicobacter pylori infection: where are we in 1995? Eur J Gastroenterol Hepatol (1995; 7:292-5) Mendal MA, Goggin PM, Molineaus N et al. Childhood living conditions and Helicobacter pylori seropositivity in adult life. Lancet (1992; 332: 896-7)
- 2.
- Noach LA, Rolf TM, Bosma NB et al. Gastric metaplasia and Helicobacter pylori infection. Gut 1993; 34: 1510-14 Ofman JJ, Shaheen NJ, Desai AA, et al. The quality of care in Barrett's esophagus:
- Endoscopist and pathologist practices. Am J Gastroenterol (2001;96(3):876-881)
  Dacosta RS, Wilson BC, Marcon NE. New optical technologies for earlier endoscopic diagnosis of premalignant gastrointestinal lesions. J Gastroenterol Hepatol (2002;17:S85-S106)
- McBroom HM, Ramsay AD. The clinicopathological meeting: a means of auditing diagnostic performance. Am J Surg Pathol (1993; 17(1):75-80)
  Shennak MM, Tarawneh MS, Al-Sheikh TM. Upper gastrointestinal diseases i
- symptomatic Jordanians: a prospective endoscopic study. Ann Saudi Med (1997;17(4):471-474)
- Paymaster JC, Sanghvi LD, Gangadharan P. Cancer of gastrointestinal tract in western India. Cancer (1968;21(2):279-288)
  Sharma S, Makaju R, Dhakal R, et al. Correlation between endoscopic and
- histopathological findings in gastric lesions. Kathmandu Univ Med J (2015;51(3):216-
- Hecker R, Fitch R, Rowland R. The value of endoscopy and biopsy in the diagnosis of gastric carcinoma. Med J Aust (1975;2(12):472-474)