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A Study On The Prevalence Of HelicobacterPylori Infection In Diabetic Patients.

Dr. Apoorva Dev M[1*], Ms. Habeeb Unnisa[2], Mr. Kamlesh Bari[3], Mr. Mohammad Imran[4], Mr. Jomy Jose T[5]

[1*][M.Pharm, Ph.D from Rajiv Gandhi University of Health Sciences], Associate professor and HOD, Department of Pharmacy Practice, East West College of Pharmacy
Address: Ground floor East West College of Pharmacy NO.63, Near BEL layout ,Magadi road, Vishwaneedham Post, Anjananagar Magadi main road Bengaluru 560091phone no: +91 9739134567, E-mail: apoorvadev.ewcp@gmail.com
[2]Doctor of Pharmacy (Pharm.D) from East West College of Pharmacy under Rajiv Gandhi university of health sciences Address: Door no.1086, upper daira, MG Road Cross Channapatna 562160 Ramnagara district, Karnataka India Phone no: +91 7019985581, E-mail: khanumheena294@gmail.com
[3]Doctor of Pharmacy (Pharm.D) from East West College of Pharmacy under Rajiv Gandhi university of health sciences Address: Kapilvastu, Taulihawa, Kalika tole, Word no. 1 pin code:32800, no:+919450050619 E-mail: kamleshkumar15014@gmail.com
[4]Doctor of Pharmacy (Pharm.D) from East West College of Pharmacy under Rajiv Gandhi university of health sciences Address: Rupandehi Provision No. 5, Maya devi gaupalika ward no.1 Lumbini 32900 Nepal Phone no: +91 9380738057 E-mail: darlingyahoo05@gmail.com
[5]Doctor of Pharmacy (Pharm.D) from East West College of Pharmacy under Rajiv Gandhi university of health sciences Address: Thannickal House , Chalappally P.O, Ezhumattoor, Pathanamthitta, Kerala 689586 India Phone no: +91 8281914837 E-mail: dr.jomyjose@gmail.com

ABSTRACT:
BACKGROUND: Infection with Helicobacter pylori has been recognized as a major public health problem affecting 50% of the world's population. However, the data on the prevalence ofhelicobacter pylori infection in diabetic patients are scanty and contradictory. The aim of the study was to determine the prevalence of helicobacter pylori infection in diabetic patients and toevaluate the glycaemic control.
METHODOLOGY: A prospective study was carried out for 6 months in Gastroenterology Department of Sagar Hospitals Jayanagar-Bengaluru. The study enrolled total 140 participants aged 18 years and above. Rapid urease test was done to detect Helicobacter pylori infection andFBS and PPBS levels were measured to assess blood sugar levels.
RESULTS: H. Pylori infection was detected in 55.7% of the type-2 diabetes mellitus patients and a higher prevalence is seen in the mean age of 52.33=16.58 years. There was a statistically significant association present between pre and post treatment FBS and PPBS levels.
CONCLUSION: Our study shows significantly higher prevalence of Helicobacter Pylori infection in diabetic patients. The results indicate a positive correlation between Helicobacter pylori infection and glycaemic control before and after treatment.

KEY WORDS: Helicobacter Pylori infection, Diabetes mellitus, Rapid Urea Test, Glycaemic control.

INTRODUCTION

Helicobacter pylori, formerly known as Campylobacter pylori, is a type of gram-negative bacterium with a spiral shape. It thrives in environments with low oxygen levels and is commonly found in the stomach. Its helical structure is thought to have evolved to aid its abilityto penetrate the stomach's mucous lining, resulting in infection in humans.The bacterium was first identified in 1982 by the Australian doctors Barry Marshall and Robin Warren for which they were awarded the noble prize in 2005. Helicobacter pylori has been identified as the primary cause of peptic ulcer disease, gastric cancer, and mucosa-associated lymphoid tissue lymphoma (MALT).

It's estimated that approximately 50% of the global population is affected by helicobacter pylori infection. Stomach cancer is the third leading cause of cancer death worldwide, after lungand liver cancer. It has been recognized that 77% of the world's non-cardiac gastric cancer is attributable to helicobacter pylori infection.

After entering the host stomach, helicobacter pylori utilizes its urease activity to neutralize the hostile acidic condition at the beginning of infection. Flagella-mediated motility is then required for helicobacter pylori to move toward host gastric epithelium cells, followed by specific interactions between bacterial adhesion with host cell receptors, which thus leads to successful colonization and persistent infection. Finally, helicobacter pylori releases several effector proteins/toxins, including cytotoxin-associated gene A (CagA), and vacuolating cytotoxin A (VacA), causing host tissue damage

The probable routes of transmission of helicobacter pylori infection are fecal-oral, oral-oral, and intra-familial, thus rendering the risk factors for helicobacter pylori infection is strongly linked to dietary habits and personal cleanliness.

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The acquisition of helicobacter pylori infection does not seem to be influenced by seasonal factors, and it is equally prevalent among both genders. Since there no significant animal or environment source for human strains of helicobacter pylori, it's most likely transmitted from person to person. Many individuals carry helicobacter pylori infection without experiencing any symptoms. [2] while other persons may experience an ache or burning pain in your stomach (abdomen), Stomach pain that may be worse when your stomach is empty, Nausea, Loss of appetite, Frequent burping, Bloating, Unintentional weight loss.

Peptic ulcer is a common health problem which is formed as a result of inflammation caused by helicobacter Pylori infection. An "ulcer" in an open sore. Peptic Lems are sores that develop in the lining of the stomach, lower oesophagus, or small intestine. In other words, Peptic ulcer disease is a condition in which painful sores or ulcers develop in the lining of the stomach known as gastric ulcer or in the first part of the small intestine known as duodenal ulcer. Normally, a thick layer of mucus protects the stomach lining from the effect of its digestive juices. But some etiological factors reduce this protective layer, allowing stomach acid to damage the tissue.[3]

Other Complications associated with helicobacter pylori infection include:

Ulcers- Helicobacter pylori can damage the protective lining of the stomach and small intestine. This can allow stomach acid to create an open sore (ulcer). About 10% of people with helicobacter pylori infection will develop an ulcer.

Inflammation of the stomach lining- Helicobacter pylori infection can affect the stomach, causing irritation and swelling (gastritis).

Stomach cancer - Helicobacter pylori infection is a strong risk factor for certain types of stomach cancer.

Individuals with type 2 diabetes mellitus have shown a higher prevalence of helicobacter pylori infection compared to non-diabetic individuals. It is also well known that infections in diabetic patients occur frequently and tend to be severe, due to the impairment of the immune status. It has also been suggested that delayed gastric emptying (gastroparesis diabeticorum) may lead to bacterial overgrowth in the upper gastrointestinal tract.[29]

Helicobacter pylori infection is associated with the pathogenesis of Type 2 diabetes mellitus, which is associated with a general activation of the innate immune system, and a chronic, cytokine-mediated state of low-grade inflammation. The host immune response to helicobacter pylori infection is complex and involves up regulation of several pro-inflammatory cytokines, such as C-reactive protein (CRP), interleukin 6 (IL-6), and tumour necrosis factor- α (TNF-α), which are implicated in insulin resistance and the development of diabetes. Thus, a potential relationship between helicobacter pylori infection and diabetes is highly suspected.[31]

Helicobacter pylori-induced gastritis can potentially affect the secretion of gastric-related hormones such as leptin and ghrelin, as well as gastrin and somatostatin, which may influence a predisposition to diabetes. Gastrin increases food-related and glucose-stimulated insulin release,and somatostatin regulates pancreatic insulin secretion and inhibits insulin release. Patients with helicobacter pylori infection could therefore have altered insulin release, as they have elevated basal and stimulated serum concentrations of gastrin and decreased somatostatin.

World Health Organization (WHO) defines diabetes mellitus as a chronic metabolic disorder characterized by elevated blood glucose levels (hyperglycaemia) resulting from defects in insulin secretion, insulin action, or both. It encompasses several types of diabetes, including Type 1, Type 2, and gestational diabetes, each with distinct causes and risk factors. Diabetes can lead to various complications and requires ongoing management to control blood sugar levels and minimize health risks.[10]

Diabetes is fast gaining the status of a potential epidemic in India with more than 62 million diabetic individuals currently diagnosed with the disease. In 2000, India (31.7 million) topped the world with the highest number of people with diabetes mellitus followed by China (20.8 million) with the United States (17.7 million) in second and third place respectively.

The first widely accepted classification was published by the WHO in 1980. Two major classesof diabetes mellitus were proposed: Insulin dependent diabetes mellitus (Type I) and Non- insulin dependent diabetes mellitus (Type II). Other types as well as gestational diabetes were also included.The exact cause of type 1 diabetes is unknown. Where as Type 2 diabetes is mainly the result of two problems:

- Cells in muscle, fat and the liver become resistant to insulin as a result, the cells don'ttake in enough sugar.
- The pancreas can't make enough insulin to keep blood sugar levels within a healthyrange.

Exactly why this happens is not known. Being overweight and inactive are key contributingfactors.[13]

Gestational diabetes is diabetes found for the first time when a woman is pregnant. Women who are overweight, have had gestational diabetes before or have a strong family history of diabetes are at a higher risk of developing gestational diabetes. Untreated gestational diabetes may causeproblems to the baby. Both the mother and the baby are at increased risk for Type II diabetes forthe rest of their lives.

Microvascular Complications of Diabetes:

Diabetic retinopathy is a diabetes complication that affects eyes. It's caused by damage to theblood vessels of the light-sensitive tissue at the back of the eye (retina). At first, diabetic retinopathy might cause no symptoms or only mild vision

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problems. But it can lead to blindness. The condition can develop in anyone who has type 1 or type 2 diabetes.
Diabetic nephropathy is the leading cause of renal failure in the United States. It is defined byproteinuria of > 500 mg in 24 hours in the setting of diabetes, but this is preceded by lower degrees of proteinuria, called "microalbuminuria' . Microalbuminuria" is defined as albuminexcretion of 30–299 mg/24 hours. Without intervention, diabetic patients with microalbuminuria typically progress to proteinuria and overt diabetic nephropathy. This progression occurs in both type 1 and type 2 diabetes. As many as 7% of patients with type 2 diabetes may already have microalbuminuria at the time they are diagnosed with diabetes.[23] Diabetic nephropathy affects the kidneys' ability to do their usual work of removing waste products and extra fluid from your body.
Diabetic neuropathy is a type of nerve damage that can occur if you have diabetes. High blood sugar (glucose) can injure nerves throughout the body. Diabetic neuropathy most often damagesnerves in the legs and feet.

METHOD

[IRB approval date 17-04-2023]
A prospective study was conducted for six months in the Gastroenterology Department of Sagar Hospitals Jayanagar-Bengaluru. Patient aged 18 years and above who recently underwentendoscopy with positive Rapid Urease Test (RUT) were included in the study. Exclusion criteria include Patients who are recently treated with Antibiotics for any infection or disease control(10 days prior/on going) as it may give false results, Patients who are taking Anti- coagulant drugs due to increased risk of bleeding, Pregnant and lactating women.

Consent was taken from all the patients included in the study for publication of the collected information.
Data was collected from the patient's case note, laboratory reports, treatment chart and patient interaction. Our study also aimed educating patient's/patient's attender about the disease and the treatment by providing them with leaflets that includes complete information about what is helicobacter Pylori infection, symptoms, causes, diagnosis, treatment, role of treatment including diet and lifestyle modification which enhances patient knowledge about the disease and the treatment. The leaflet was validated by expert panel members [Doctor's and Chief Dietician of Sagar Hospitals] both Kannada and English version.

- Design validity was done using BALD criteria.
- Content validity using Lawshe's content validity ratio.
- Readability validity using Flesch's Reading Ease Score.

RESULTS:

A prospective study was carried out on all adult patients aged above 18 years who visited Out- Patient (OP) and In-patient (IP) Gastroenterology Department of Sagar Hospitals Jayanagar- Bengaluru. The study specifically targeted individuals diagnosed with helicobacter Pylori infection in Diabetes patients. The study aimed to gather valuable information about the current Prevalence of helicobacter Pylori infection in diabetic patient, changes in glycaemic control before and after helicobacter Pylori infection treatment.

Table 1 : Distribution of patients according to gender [N= 140]

Gender	Frequency	Percentage
Male	78	55.7
Female	62	44.3
Total	140	100

The above table 1 shows the frequency and percentage of male and female patients included in this study.
The current study included total 140 patients out of which 62 were females and 78 were males contributing to 44.3% females and 55.7% males. The study shows males predominated over females in the prevalence of helicobacter Pylori infection. Our study reveals a higher prevalence of helicobacter Pylori infection in males compare to females, attributing to reasons like smoking addiction which was more in males than females, which may affect the occurrence of helicobacter pylori infection. Long-term smoking alters the normal physiology of the gastrointestinal tract and affects the factors that protect or heal the lining, including secretion of mucus, blood flow, and production of bicarbonate, which could contribute to its effect on the susceptibility to helicobacter pylori infection however the role of gender as a risk factor is still in debate.

Table 2 : Distribution of patients according to age group [N= 140]

Age group (years)	Frequency	Percentage
<29	18	12.9
30-59	71	50.7
≥60	51	36.4

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Total	140	100

The above table 2 shows the frequency and percentage of age group included in this study with Total Mean±SD: 52.33±16.58 years, Range: 18-82 years
Table 2 represents the age distribution of the given population, in our study patients were divided into three groups based on age as 18-29 years, 30-59 years ,60 and above. The study suggest 12.9% of patients belongs to the age group of 18-29 years, 50.7% of patients belongs to the age group of 30-59 years, and 36.4% f patients belongs to the age group 60 years and above. Higher prevalence of helicobacter Pylori infection was found in patients aged between 30-59 years with total mean±SD: 52.33±16.58 years.

Table 3 : Distribution of patients according to social history [N=140]

Social history	Frequency	Percentage
Non-Smoker	89	63.6
Smoker	51	36.4
Total	140	100

The above table 3 shows the frequency and percentage of social history [smoking] of the patients included in this study
Table 3 shows 63.6% of patients were non-smoker and 36.4% of patients were smoker. Since smoking is associated with increased risk of developing diabetes through influencing factors such as secretion of hormones cortisol,catecholamines and growth hormone that may lead to insulin resistance therefore, it has a major impact on glycaemic control.

Table 4 : Distribution of patients according to Glycaemic control [N=140]

HbA1C	Frequency	Percentage
<5.6 (Normal)	15	10.7
5.7-6.4 (Pre-diabetic)	45	32.1
≥ 6.5 (Diabetic)	80	57.1
Total	140	100

The above table 4 shows the frequency and percentage of HbA1C levels of the patients included in this study.
Table 4 represents distribution of subjects according to glycaemic control using HbA1C test. The study enrolled total 140 patients out of which of 10.7% had their HbA1C level under control(normal) ,32.1% had their HbA1C level partially controlled(pre-diabetic) and 57.1% had their HbA1C levels uncontrolled (diabetic patients) Present study indicates a higher prevalence of helicobacter pylori infection in diabetic patients.

Table 5 : Association between social history and Glycaemic control [N=140]

HbA1C	Non-smoker (n=89)	Smoker (n=51)	*p* value*
5.7-6.4 (Pre-diabetic)	27	18	0.08
≥ 6.5(Diabetic)	50	30	

The above table 5 shows the association social history and glycaemic control of thepatients included in this study.
The above table 5 shows the association social history and glycaemic control of the patients included in this study. Our study did not find any significant association between smoking and glycaemic control in the study population. p -value 0.08) which indicates smoking is not responsible for elevated blood glucose levels in diabetic patients with helicobacter Pylori infection.

Table 6: Before and after treatment fasting blood sugar levels [N=140]

Normal		Pre-Diabetic		Diabetic		Total
Before	**After**	**Before**	**After**	**Before**	**After**	
18	22	24	28	98	90	**140**

The above table 6 shows before and after treatment fasting blood sugar levels innormal, pre-diabetic and diabetic patients included in this study.

Table 6 represents before and after treatment FBS levels. The study enrolled total 140 patients showed 12.9 % patients had their FBS levels under control before treatment and 12.1% patients had their FBS levels under control after treatment, 17.1% patients had their FBS levels partially controlled before treatment and 10.7% patients had their FBS levels partially controlled after treatment ,70% patients had their FBS levels uncontrolled before treatment and 68%

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patients had their FBS levels uncontrolled after treatment. When comparison made between before and after treatment in the FBS levels our study showed statistically significant association Therefore, This study findings suggest there a significant impact of helicobacter Pylori infection treatment on FBS levels. Indicating an improvement in glycaemic control after the course of treatment period.

Table 7 : Before and after treatment Post prandial blood sugar levels [N=140]

Normal		Pre-Diabetic		Diabetic		**Total**
Before	**After**	**Before**	**After**	**Before**	**After**	
15	24	26	27	99	89	**140**

The above table 7 shows before and after treatment post prandial blood sugar levels in normal, pre-diabetic and diabetic patients included in this study.

Table 7 shows study enrolled total 140 patients showed 10.7 % patients had their PPBS levels under controlled before treatment and 10% patients had their PPBS levels under controlled after treatment,18.6% patients had their PPBS level partially controlled before treatment and 12% patients had their PPBS partially controlled after treatment, 70.7% patients had their PPBS uncontrolled before treatment and 63.5% patients had their PPBS uncontrolled. Our study showed statistically positive significant association before and after treatment in PPBS levels . Therefore, our study findings suggest there a significant impact of helicobacter Pylori infection treatment on FBS and PPBS levels indicating an improvement in glycaemic levels following the course of treatment regimen.

DISCUSSION

Helicobacter pylori is a common bacterial infection, and it is estimated that around 50% of the world's population may be infected with it. The mode of transmission is not entirely clear, but itis believed to spread through contaminated food, water, and close person-to-person contact.

The findings provide insights on prevalence of helicobacter Pylori infection along with glycaemic control following pre and post helicobacter Pylori infection treatment .The study focused on several factors for prevalence of helicobacter Pylori infection such as Age, and gender, Markers of glycaemic control include glycated haemoglobin (HbA1c), postprandial blood sugar (PPBS), and fasting blood sugar (FBS). The duration of the trial lasted for six months, where total 140 patients were included. Regardless of gender and diabetic status.

According to this study findings males predominated over females in the prevalence of helicobacter Pylori infection contributing to 55.7% males and 44.3 % females attributing to reasons like in our study smoking addiction were more in males than females, which may affectthe occurrence of helicobacter pylori infection. In this study greater proportion of subjects who met the inclusion criteria are aged 30-59 This was due to poor hygiene, using outhouse toilets and drinking water contaminated water.whereas contrast findings were found in other studies conducted by Ali K Jumaa and Sarkis K Stark showed 28.4% males and 38.3% females were infected with H. pylori infection.

This study also evaluated the association between social history and glycaemic control but there was no association found, further glycaemic control was evaluated using HbA1C, FBS and PPBS. The study enrolled total 140 patients out of which of 10.7% had their HbA1C level under control(normal) ,32.1% had their HbA1C level partially controlled(pre-diabetic) and 57.1% had their HbA1C levels uncontrolled (diabetic patients) present study indicates a higher prevalence of helicobacter pylori infection in diabetic patients. Other studies conducted by Fariba Keramat, Seyyed Hamid Hashemi, Amir Majlesi, Shahram Haddadinejad, Alireza Monsef Esfehani, Jalal Poorolajal findings includes 79 DM patients with mean age 51.20±11.06years, 70 control with mean age 48.49±15.23 years respectively, P=0.114.

The study enrolled total 140 patients out of which of 10.7% had their HbA1C level under control(normal) ,32.1% had their HbA1C level partially controlled(pre-diabetic) and 57.1% hadtheir HbA1C levels uncontrolled (diabetic patients) Present study indicates a higher prevalence of helicobacter pylori infection in diabetic patients. Similar findings were found in the study conducted by Chiman Hameed Saeed showed significantly high prevalence of helicobacter pylori infection in diabetic patients especially in those with fair control of blood glucose as compared to non-DM individuals. Routine screening of diabetic patients for Helicobacter pylori is recommended.

In our study a comparison was made between pre and post treatment FBS and PPBS control after better understand the glycaemic control. Findings include 12.9 % patients had their FBS levels under control before treatment and 12.1% patients had their FBS levels under control after treatment, 17.1% patients had their FBS levels partially controlled before treatment and 10.7% patients had their FBS levels partially controlled after treatment ,70% patients had their FBS levels uncontrolled before treatment and 68% patients had their FBS levels uncontrolled after treatment. The differences in postprandial blood sugar (PPBS) levels were observed in thisstudy, with 10.7% of patients having controlled PPBS levels before treatment compared to 10% after treatment. Additionally, 18.6% of patients had partially controlled PPBS levels before treatment, decreasing to 12% after treatment. The majority, 70.7%, had uncontrolled PPBS before treatment, which decreased to 63.5% after treatment. The study revealed a statistically significant association between PPBS levels before and after treatment, suggesting a notable impact of helicobacter Pylori infection treatment on PPBS levels. This indicates an improvement in glycaemic levels following the prescribed treatment regimen. Notably, both

fasting blood sugar (FBS) and PPBS levels showed a positive association in this study before and after helicobacter Pylori infection treatment.
Our study also has few limitations which include study's sample size was relatively small. Also The study did not differentiate between newly diagnosed and long-term diabetic patients.
Further Elucidating the molecular and cellular mechanisms underlying the interaction between helicobacter Pylori and diabetes is required also focusing on how the infection may influence insulin sensitivity.

CONCLUSION

The study was designed to assess the prevalence of H. pylori infection in diabetic patients shows significantly higher prevalence Helicobacter Pylori infection in diabetic patients with 55.7% for glycaemic control the study utilized HbA1C, FBS and PPBS levels. When comparison was made between before and after treatment in the FBS and PPBS levels our study showed statistically significant association Therefore,our study findings suggest there a significant impact of Helicobacter Pylori infection treatment on FBS levels and PPBS levels indicating an improvement in glycaemic control after the course of treatment with triple therapy [amoxicillin 750mg+ clarithromycin 500mg + esomeprazole 40mg].

STATISTICAL ANALYSIS

Statistical analyses were performed using IBM SPSS Statistics for Windows, Version 25.0. Armonk, NY: IBM corp. Results on continuous measurements were presented on mean SD. Normality of the data was assessed using Shapiro Wilk test. Inferential statistics like chi-square test was used to check difference between the groups. The significance of level adopted was 5%.

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CONFLICTS OF INTEREST

There are no conflicts of interest.

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