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Association between Type 2 Diabetes Mellitus and *Helicobacter pylori* Infection.

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ABSTRACT

To determine the frequency of Helicobacter pylori infection in diabetic and non-diabetic patients and to compare the frequency of H. pylori infection in both groups. Case control studyt his hospital based study was conducted on 100 subjects and divided into two groups: group A – Type 2 diabetics and group B – non-diabetics, consisting of 40 and 60 patients respectively. All patients > 30 years of age, both gender & with history of dyspepsia, epigastric pain, or bloating for more than a month. They were screened for the presence of H. pylori infection by studying the H&E and Giemsa stained sections of their gastric biopsies. 72.5% of diabetics showed positive *H.pylori* colonization whereas non diabetic group showed only 38.3% positivity. Odds ratio, Chi square and p value are calculated. Diabetic patients were more prone and are at risk to acquire H. pylori infection. There is a well-established association between H. pylori infection and gastric adenocarcinoma gastric MALT lymphomas. A short course of antibiotic therapy can eradicate H. pylori and reduce the incidence of these complications significantly. Hence, all gastric biopsies sent from patients with dyspeptic symptoms must be screened for presence of H. pylori.

Keywords: *Helicobacter pylori,* diabetes, gastric adenocarcinoma, antibiotic therapy.

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INTRODUCTION

Helicobacter pylori is a gram negative spiral shaped bacterium that resides in the gastric mucosa. It secretes an enzyme that raises the pH, de-gels the protective mucin coat & reaches the gastric epithelial cells to cause ulcers. It may survive undetected for decades, then manifest with gastritis or dreaded complications – gastric adenocarcinoma and gastric MALT lymphomas. *H. pylori* affecting approx. 50% of the world population [1], has a chronic smodering effect on us. End number of studies are going on, to state type 2 DM, as a risk factor for acquiring *H. pylori* infection.

OBJECTIVE

To determine the frequency of *H. pylori* infection among diabetics and nondiabetics (controls) & to compare the same.

MATERIALS & METHODS

Study type: Case-control study, Study period: june 2014 – june 2015,dept. of pathology, SBMCH.

Inclusion criteria: Endoscopic gastric biopsies & gastrectomy specimens received from SBMCH & other hospitals who were of,

1. >25 yrs of age,
2. either gender
3. C/O dyspepsia, bloating, epigastric symptoms>1 month
4. K/C/O DM for approximately 5 yrs with c/o dyspepsia, bloating, epigastric symptoms > 1 month

Exclusion criteria:

1. Type 1 diabetes
2. Persons already on steroids, immunosuppressive therapy, *H.pylori* eradication therapy. The individuals who come under inclusion criteria were screened for *H. pylori* infection by studying the H&E and Giemsa stained sections of their gastric biopsies.

RESULTS

Table 1

	H.pylori Positive	H.pylori Negative
Diabetic group	29	11
Non-diabetic group	23	37

Frequency of H.pylori (+) among diabetics (29/40) = 72.5%
Frequency of H.pylori (+) among non-diabetics (23/60) = 38.3%
Odds ratio = 4.24
Chi-square = 11.77
p value < 0.05, statistically significant. [2-7],[8-10]

DISCUSSION

In patients with type 2 diabetes mellitus, there is reduction in cellular and humoral immunity[11], reduced GI motility & acid secretion and certain chemical changes occur in gastric mucosa due to altered glucose metabolism[12]. All these factors play a major role for the increased risk of pathogen colonization in diabetics.

Infection with *H.pylori* leads to local and systemic diffusion of pro-inflammatory cytokines like CRP, IL-6, IL-1β and TNF-α, which are implicated in the pathogenesis of type 2 DM. hsCRP has become the main focus

of investigation as a risk factor for type 2 DM. Regardingly, 11 prospective studies have been carried out .Among them,7 studies showed a positive association with DM[16-22] and 4 studies showed a negative correlation[23-26]. IL-6 modifies adipocyte glucose and lipid metabolism & alters the body weight[27,28]. In adipose tissue, TNF-α play a critical mechanism by which fat cells induce peripheral insulin resistance[29].

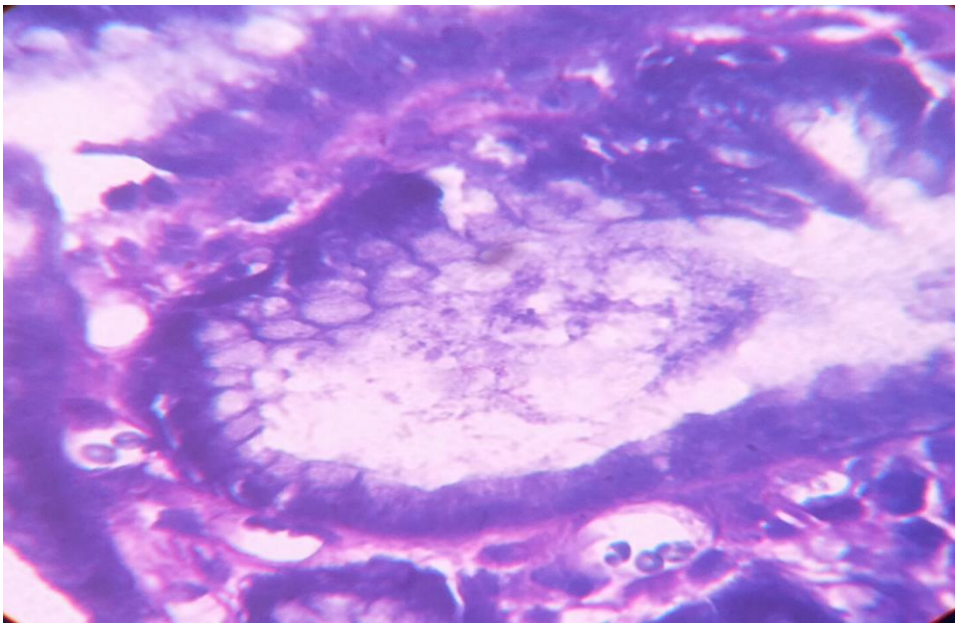

Figure 1: *Helicobacter pylori* colonization in the superficial glands of gastric mucosa

Insulin producing β cells of pancreas are susceptible to damage by the inflammation & oxidative stress caused by H.pylori infection[30]. Cytokines like IL-1β, TNF-α, IFN-γ, vacuolating cytotoxin of certain H. pylori strains inhibit insulin secretion & induces apoptosis of beta cells[31,32]. H.pylori induced gastritis alters secretion of insulin regulating hormones like gastrin and stomatostatin**[**33], increases leptin and reduces ghrelin secretion which are the key hormones of energy homeostasis. This promotes obesity, reduces insulin sensitivity and alters glucose homeostasis[34,35].

The production of free radicals near the site of H.pylori infection increases the rate of host cell mutation. Increased local production of TNFα, IL-6 alters gastric epithelial cell adhesion and leads to dispersion & migration of mutated epithelial cell without the need for any further mutations in tumor suppressor genes. The vacuolating cytotoxin (vac A) and certain strains of *H.pylori* harbouring the cytotoxin associated gene (cag A) produces greater tissue damage and is directly toxic to gastric epithelial cells. They send strong signals to the immune system that an invasion is underway.[36,37]

CONCLUSION

Diabetic patients are more prone and are at risk to acquire *H. pylori* infection. The presence of *H.pylori* infection increases the risk of developing gastric adenocarcinoma by 6 fold & is strongly implicated in causing gastric B cell MALT lymphomas. A course of 'triple therapy' for 10-14 days can eradicate *H. pylori* and reduce the incidence of its dreaded complications significantly. Hence, all gastric biopsies sent from patients with dyspeptic symptoms must be screened for presence of *H. pylori*.

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