Is the Presence or Absence of *Helicobacter pylori* in Gastric Mucosa a Greater Risk?

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**Abstract**

*Helicobacter pylori* is a unique organism which is pathogenic for stomach - duodenum (chronic gastritis, duodenal ulcer, gastric ulcer, gastric malignancy, mucosa-associated lymphoid tissue (MALT) lymphoma) and protective for oesophagus (Barrett’s oesophagus, oesophageal adenocarcinoma) at the same time in an individual. For prevention of diseases, the necessity of presence of some bacteria throughout the gastrointestinal lumen needs to be emphasized. The concept - only good *Helicobacter pylori* is a dead *Helicobacter pylori*, is dangerous and humans should learn to live in harmony with a few bacteria throughout the gastrointestinal tract.

© INTRODUCTION

Since *Helicobacter pylori* discovery in 1983, its relationship to diseases such as chronic gastritis, duodenal ulcer, gastric ulcer, gastric malignancy, mucosa associated lymphoid tissue (MALT) lymphoma has been well described. However, the increasing prevalence of diseases, such as Barrett’s oesophagus, oesophageal adenocarcinoma at lower end of oesophagus, resulting from the absence of *Helicobacter pylori* in gastric mucosa, is not widely appreciated.

In gastrointestinal lumen of humans, some bacteria are pathogenic (Salmonella, Shigella) while others are protective (Lactobacillus, Bifidobacteria). *Helicobacter pylori* (Hp) is a unique organism which can be both pathogenic and protective (simultaneously) in an individual.

Humans are the only major reservoir of Hp and about half the world’s population is infected with it. Hp infection of gastric mucosa usually persists for several decades and invariably causes chronic gastritis (CG). In some patients, Hp causes duodenal ulcer (DU), gastric ulcer (GU), gastric adenocarcinoma (GAC) mucosa associated lymphoid tissue (MALT) lymphoma and has occasionally been blamed for non-ulcer dyspepsia (NUD). In contrast, Hp provides protection against development of Barrett’s oesophagus (BO) and oesophageal adenocarcinoma (OAC). The aim of this article is to describe and compare the risk of different diseases resulting from the presence or absence of Hp in gastric mucosa of humans.

**Duodenal ulcer**

Hp infection has 10% lifetime risk of peptic ulcer. In some patients with Hp infection, predominantly restricted to antrum, DU occurs. Antral gastritis causes damage to the delta cells (in the antrum) secreting hormone somatostatin which normally inhibits gastrin secretion, resulting in hypergastrinaemia. Gastrin is a trophic hormone and causes an increase in parietal cell mass (PCM) and acid-pepsin output. Increased acid load in the duodenum causes gastric metaplasia in the duodenum in which Hp colonizes and causes DU.

In male DU patients from North America, Europe, Australia, maximum acid output (MAO) is 37.5 mEq/hr (double than in normal individuals) while it is only 25 mEq/hr in patients from Mumbai (India). This is due to the presence of Hp in body mucosa of stomach for a few years only (prior to DU), in patients from western countries in contrast to Hp presence for several decades in patients from India, due to exposure to Hp in childhood. Since MAO values are lower in DU patients from India, gastric metaplasia in duodenum is uncommon in them.

Hp is present in gastric mucosa of 80-90% of DU patients and its recurrence is reduced from 90% to 10% per year on eradication of Hp. Hp infection in antrum increases the risk of DU by 3-6 folds (Odds Ratio (OR) =3-6).  

**Gastric ulcer, Chronic Gastritis, Gastric Carcinoma**

Hp preferentially colonizes in antrum initially and later extends to a varying extent in the body mucosa causing superficial or atrophic gastritis. This results in
Gastrooesophageal reflux disease (GERD)

Due to improved sanitation during last four decades in developed countries, the prevalence of Hp infection and associated CG, has declined appreciably. Since CG lowers MAO, reduced Hp colonization of the gastric mucosa will result in higher MAO in the population. Higher MAO increases the damage of GERD, as the concentration and the volume of reflux, together with the duration of acid-pepsin exposure determines the damage of oesophageal mucosa. In absence of Hp infection in gastric mucosa, the risk of OAC is increased up to 40 folds in patients with GERD.

Barrett’s oesophagus

Long term increased acid exposure of esophagus leads to columnar cell replacement, more resistant to acid-peptic damage; specialized intestinal metaplasia follows and BO is diagnosed. BO increases the risk of OAC by 40-50 folds, (OR= 40-50). Prevalence of BO is 8% in patients with GERD and is 10 times more common than in normal population. 10 – 15 % of the patients with GERD have short segment BE while 3 – 5 % have long segment BE. In India prevalence of BO is low (2.6 % in patients with NUD) compared to patients from developed countries, as the MAO value is significantly lower amongst Indians.

Oesophageal Adenocarcinoma

OAC arises from the sequential pathologic changes of low and HGD in BO. Development of HGD in BO poses high risk of development of OAC. In patients with BO the risk of dysplasia increases 3.3% per year. Average time taken for progression of low and HGD to develop OAC is approximately 4 and 3.5 years respectively. Ten percent of patients with HGD develop OAC every year and 40% of patients with HGD harbor OAC at the time of diagnosis. Presence of Hp in gastric mucosa significantly reduces the risk of development of OAC (OR = 0.3-0.5). Incidence of OAC has increased 4 folds in white western population during the last three decades. OAC comprised 15% of all oesophageal cancers in 1970-80, but currently contributes 60% of oesophageal cancers in Unites States. This dramatic rise in OAC corresponds to the decline in prevalence of Hp and increase in GERD in developed countries. The role of obesity (if any), due to high calorie and fat intake diet of western population, in further worsening of GERD, is not assessed. In India, Hp and CG still remains widely prevalent and hence BO and OAC are uncommon. Hp is pathogenic in diseases such as DU, GU, CG, GAC, MALT lymphoma. In contrast, its absence in gastric mucosa increases the severity of GERD and the prevalence of BO and OAC. Eighty percent of patients with Hp infection are asymptomatic and 20% suffer from symptomatic diseases. Eradication of Hp is always recommended in DU, GU, MALT lymphoma; its
eradication in other conditions (CG, NUD) is not desirable, as exposure of multiple antibiotics to a large population may cause appreciable side-effects and significant drug resistance. Furthermore, eradication of Hp in asymptomatic subjects is not beneficial as Hp has an important protective role against development of BO and OAC.

The slogan of the last century - only good Hp is a dead Hp, is an example of failure to appreciate the beneficial role of Hp against serious diseases such as BO and OAC. Eradicating Hp wherever detected (CG, NUD), may perhaps lead to a further increase of BO and OAC. Pseudomembranous colitis, Crohn's disease, OAC are examples of the disharmony, in symbiotic relationship between humans and microbes (or helminth), in intestinal lumen. To prevent serious diseases, humans should learn to live in harmony with some protective bacteria required through out the gastrointestinal tract.

REFERENCES


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**Announcement**

MAPICON 2005

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21st, 22nd, 23rd October, Nagpur.

The highlight of the Conference include following:

The Conference building is aesthetically designed to have an excellent acoustics.

More than 1000 delegates are expected to attend.

Nationally and Internationally acclaimed faculties shall address the delegates using the state-of-the-art audio-visual.

Parallel scientific sessions and Buzzer Sessions.

Many pharma companies have agreed to display their products and services.

The following is the delegate fees.

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<th>Category</th>
<th>Upto 30/9/2005</th>
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Accommodation, transport and sight seeing shall be arranged on request.

Accompanying persons shall have fun time at the venue itself.

For further details, please contact: Dr. SM Patil, Organising Secretary, 2nd floor, Yugdharma Complex, Ramdaspeth, Nagpur 400 010.

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