PREVALENCE OF HELICOBACTER PYLORI IN DIFFERENT SURGICAL DISEASES: A PRELIMINARY REPORT

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ABSTRACT

The aim of this study was to explore the prevalence of Helicobacter pylori infection in different surgical diseases in patients with six different cancer types. We analyzed sixty consecutive patients with malignancy (gastric cancer, pancreatic cancer, hepatocellular cancer, intestinal cancer, colon cancer, rectal cancer). Detection of specific IgA and IgG antibodies to Helicobacter pylori in human serum was determined by Enzygnost Anti-Helicobacter pylori II/IgA (IgG) using ELISA processor (Siemens, Germany). This study confirmed statistically significant association between Helicobacter pylori seropositivity and all types of cancer included in the study. All patients had elevated levels of Helicobacter pylori IgA and IgG antibodies. Patients with examined cancer types that underwent abdominal surgery exhibited a strong antibody reaction to Helicobacter pylori.

INTRODUCTION

Helicobacter pylori is the commonest chronic infection in humans. About half of the population in the developed countries carries this bacteria and nearly 90% are adults (1). The prevalence of H. pylori varies widely. More than 80% of adults are infected in Japan and South America (2). In European countries 30-60% of middle aged has serological evidence of infection (3). Epidemiological evidence suggests that many people acquire the infection in childhood. Social deprivation household crowding and number of siblings are the important risk factors. The prevalence of infection increases with age, although this may be largely a cohort effect (4). The mode of transmission is unknown but proposed routes include oral-oral and faecal-oral. Helicobacter pylori have been implicated as having a pathological role in a variety of gastrointestinal diseases. The International Agency for Research on cancer has categorized Helicobacter pylori as a group I carcinogen (5). Small number of studies confirmed H. pylori as an important factor linked to the development of malignancy.

The aim of this study was to explore the prevalence of H. pylori infection in different surgical diseases in patients with six different cancer types.
PATIENTS, MATERIALS AND METHODS
This study reports preliminary results of the large prospective, cohort study carried out in the Clinical Center of Serbia. We analyzed 60 consecutive patients with malignancy (gastric cancer, pancreatic cancer, hepatocellular cancer, intestinal cancer, colon cancer, rectal cancer) who underwent surgical treatment at the Department of Emergency Surgery of Clinical Centre of Serbia, between September 2007 and September 2009. Patient data consisted of age, gender, diagnosis and Helicobacter pylori status. Preoperative evaluation was done in all patients. In each group we analyzed the presence H. pylori infection. The blood samples were taken for testing Helicobacter pylori prior to surgery. Commercial test Enzygnost Anti-Helicobacter pylori II/IgA (IgG) (Simens, Germany) was used for the qualitative detection of H. pylori -specific human IgA and IgG antibodies in the serum. Quantification in Enzygnost-units (U/mL) is achieved by a single-point procedure.

STATISTICAL ANALYSIS
Date were presented as mean ± standard deviations or as percentages of total values. Difference was considered statistically significant if p value was less than 0.05. All the available data were analyzed by computer program (SPSS, Chicago, IL, USA).

RESULTS
Mean age in all patients groups was 60±12 years. The study included 36 men and 24 women. The prevalences of specific IgA and IgG antibodies to Helicobacter pylori in patient’s serum according to cancer type are given in Table 1. The most frequent cancer type were rectal and hepatocellular. All patients, regardless the cancer type, had elevated H. pylori IgA and IgG antibodies levels.

Table 1. Prevalence of Helicobacter pylori in different types of cancer

<table>
<thead>
<tr>
<th>Cancer type</th>
<th>Number of patients (%)</th>
<th>IgG positive</th>
<th>IgA positive</th>
</tr>
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<tbody>
<tr>
<td>Gastric cancer</td>
<td>6 (10.0%)</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Pancreatic cancer</td>
<td>8 (13.3%)</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Hepatocellular cancer</td>
<td>14 (23.3%)</td>
<td>14</td>
<td>14</td>
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<tr>
<td>Intestinal cancer</td>
<td>7 (11.7%)</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Colon cancer</td>
<td>11 (18.3%)</td>
<td>11</td>
<td>11</td>
</tr>
<tr>
<td>Rectal cancer</td>
<td>14 (23.4%)</td>
<td>14</td>
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</tr>
</tbody>
</table>

DISCUSSION
The main finding of this study was that patients with examined cancer types that underwent abdominal surgery exhibited a strong antibody reaction to Helicobacter pylori. All of them were positive of H. pylori infection.

H. pylori has been implicated as a carcinogenic factor for non-cardia gastric cancer and causes a persistent inflammatory-proliferative state that evolves from chronic superficial gastritis to precancerous atrophic gastritis, metaplasia, and dysplasia (6). Prospective epidemiological studies have shown that H. pylori positive patients may be indicated in high risk populations of non-cardia gastric cancer development (7,8). Most studies of Asian populations have found a positive association between H. pylori seropositivity and cardia cancer, whereas most studies of Western
populations have found no association or an inverse association (8, 9). Meta-analysis of Huand and coworkers confirmed the relationship between H. pylori seropositivity and gastric cancer (10).

Pancreatic cancer is among the most fatal cancers worldwide and one for which few preventable risk factors has been established. Gastric carriage of H. pylori, particularly cytotoxin-associated gene-A-positive (CagA+) strains, is a risk factor for peptic ulcer disease and gastric cancer and may have a similar etiologic relationship with pancreatic cancer. Two previously published studies (11,12) examined H. pylori and pancreatic cancer. The first study found greater H. pylori seropositivity among 92 subjects with pancreatic cancer (65%) than among 62 control subjects (45%; control subjects had either colorectal cancer or no disease) and showed a similar significant doubling of risk. The other study of Stolzenberg-Solomon and coworkers supported a possible role for H. pylori carriage in the development of exocrine pancreatic cancer.

However, the possibility that Helicobacter pylori is an initiator of colorectal neoplasia (13, 14) is a subject of debate. Most associations between neoplastic colorectal lesions (adenomas and carcinomas) and H. pylori are based on studies correlating these lesions with H. pylori seropositivity (15-18) or indirect evidence such as increased gastrin or CagA+ levels (19,20). Other studies have failed to demonstrate this association based on seropositivity (21-23); indeed it has been suggested that H. pylori does not colonize rectal mucosa (24).

Although our preliminary results suggests the association of H. pylori positivity and different cancers, further studies are necessary.

Bibliography