Relationship Between Helicobacter Pylori Infection and GERD in Patients under 18 Years Old

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Abstract: The relationship between Helicobacter pylori and gastroesophageal reflux disease (GERD) has been controversial in published literature in the past decade. The aim of this study was to evaluate potential relationships between H. pylori infection and GERD in pediatric patients. Data of 114 patients entered our study. The results of Giemsa staining on biopsy specimens revealed only 5 patients (4.4%) had H. pylori infection and other 109 (95.6%) had no infection. The association between GERD and H. pylori is not very clear in the literature, but our results showed a low prevalence of this bacteria among GERD patients and supported the idea that suggests it has a protective role for developing GERD symptoms. The previous studies suggested that GERD was more prevalent in the younger and male patients.

Key words: Gastroesophageal Reflux · Helicobacter pylori · Endoscopy

INTRODUCTION

In 1983, Warren and Marshall reported an association between the presence of H. pylori in the gastric mucosa and antral gastritis in adults [1]. H. pylori is a gram-negative, spiral, flagellated bacterium that colonizes the gastric mucosa in the infected host. H. pylori infection has been implicated in the development of gastritis and peptic ulcer disease, as well as chronic atrophic gastritis [2]. H. pylori infection increases the risk of gastric malignancies such as adenocarcinoma and mucosal-associated lymphoid tissue [MALT] lymphoma [3-11]. It is well known that H. pylori infection is acquired in childhood, often before the age of 5 years, through fecal-oral, oral-oral or gastro-oral transmission [12]. The infection may be life-long in the absence of treatment. Children with symptomatic H. pylori infection associated with duodenal and gastric ulcers, lymphoma or atrophic gastritis with intestinal metaplasia require treatment to eradicate the bacterium [13].

The relationship between H. pylori and gastroesophageal reflux disease (GERD) has been controversial in published literature in the past decade [14-26]. H. pylori infection has been associated with a significantly reduced risk of developing GERD, Barrett’s esophagus and esophageal adenocarcinoma in contrast to its increased risk for peptic ulcer disease and gastric cancer [14-16]. Labenz et al. [9, 10] who first reported in 1997 that there was an increased risk of development of GERD after H. pylori eradication, postulated a possible protective role of H. pylori infection. Subsequently, McColl and others [19-23] suggested that H. pylori infection might protect against GERD in infected patients who had atrophic gastritis and reduced gastric acid secretion. The regional distribution and severity of gastritis appeared to be a more important risk factor than the mere presence of H. pylori in the gastric mucosa [20]. However, several papers contradicted Labenz’s findings showing that H. pylori eradication may not induce GERD symptoms in adults and in fact the infection may have no association [24-16].

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There are limited data regarding the relationship between \textit{H. pylori} infection and GERD in the pediatric population [27-30]. Levine \textit{et al.} [27] reported that eradication of \textit{H. pylori} was not associated with increased symptoms of GERD in children. Pollet \textit{et al.} [28] found that \textit{H. pylori} eradication did not provoke or worsen GERD in neurologically impaired children. Özçay \textit{et al.} [29] found no significant difference in reflux esophagitis between children with and without \textit{H. pylori} infection while Daugule \textit{et al.} [30] showed a significantly higher prevalence of \textit{H. pylori} in children with RE compared to children with hyperemic gastropathy.

The aim of this study was to evaluate potential relationships between \textit{H. pylori} infection and GERD in pediatric patients based on a retrospective examination of biopsy specimens in conjunction with clinical and demographic characteristics.

\section*{MATERIALS AND METHODS}

Data of all patients less than 18 years old that were undergone endoscopy in Children’s Medical Center of Tehran for ruling out GERD disease between 1\textsuperscript{st} Dec 2010 and 1\textsuperscript{st} Dec 2012 entered our study. These data consisted of Sex, age, result of Giemsa staining for diagnosing \textit{H. pylori}, signs and symptoms and the duration of these symptoms. All data entered SPSS version 11 and analyzed with descriptive tests and T-test.

\section*{RESULTS}

In the two years of study data of 114 patients entered our study. The results of Giemsa staining on biopsy specimens revealed that only 5 patients (4.4\%) had \textit{H. pylori} infection and other 109 (95.6\%) had no infection. Distribution of age and sex among \textit{H. pylori} and no- \textit{H. pylori} groups are shown in Table 1.

\section*{DISCUSSION}

In the present study the prevalence of \textit{H.pylori} among patients under 18 years was 4.4\%. In a study by Alborzi \textit{et al.} [31] the prevalence of this bacteria was 82\%, 98\%, 88\%, 89\% and 57\% in under 9 months old, 9 months to 2 years, 2-6 years, 6-10 years and 10-15 years respectively. This high prevalence of \textit{H.pylori} among non-GERD patients and not in GERD patients suggests that there should be a supportive mechanisms of \textit{H.pylori}

Graph 1: Distributions of signs and symptoms among pediatric patients

FTT: Failure to Thrive, AP: Abdominal Pain
(Some patients have more than one sign or symptom)

Table 1: Distribution of age and sex among H. pylori and no- H. pylori groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Age</th>
<th>Sex</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;5</td>
<td>&gt;5</td>
</tr>
<tr>
<td>\textit{H. pylori}</td>
<td>4(80%)</td>
<td>1(20%)</td>
</tr>
<tr>
<td>Non-\textit{H. pylori}</td>
<td>69(63.3%)</td>
<td>40(36.6%)</td>
</tr>
<tr>
<td>Total</td>
<td>73(64%)</td>
<td>41(36%)</td>
</tr>
</tbody>
</table>

Distributions of signs and symptoms among the patients that entered the study can be seen in graph 1.
and chance of getting GERD. This idea can be more visible when we review other studies in this area. For example Brazowski et al. [31] found the prevalence of *H. pylori* 18.1% in GERD patients and found this rate low in comparison with non-GERD patients and suggested that there should be a role in this difference. Epidemiologic studies does not support a role of *H. pylori* in pathophysiology of GERD disease but studies showed that there are decreased rate of *H. pylori* in GERD patients [33]. Zerbib et al. suggested that *H. pylori* eradication does not exacerbate GERD symptoms and may, in some cases, improve them [34]. In a review article Sharma and Vakil suggested that the prevalence of *H. pylori* infection in patients with reflux disease is probably no greater than that in those without reflux and there are conflicting data indicating that reflux symptoms or erosive esophagitis develop after *H. pylori* eradication. It is also unclear whether *H. pylori* augments the antisecretory effects of proton pump inhibitors or accelerates the development of atrophic gastritis [35]. At the other hand there are some studies that indicate the high prevalence of *H. pylori* among GERD patients. For instance Masjedizadeh et al. announced high prevalence of *H. pylori* (88.2%) among GERD patients [36].

The prevalence of GERD in childhood varies by age. In the current study 64% of the patients with GERD were under 5 years old. The study conducted by Gold Benjamin et al. [37] revealed the highest prevalence of GERD in the patients 1-2 years old. These results are in conductance with the results of our study. In both studies, male patients consisted the majority of the patients.

**CONCLUSION**

The association between GERD and *H. pylori* is not very clear in the literature, but our results showed a low prevalence of this bacteria among GERD patients and support the idea that suggests it has a protective role for developing GERD symptoms. As suggested in previous studies, GERD was more prevalent in the younger and male patients.

**REFERENCES**


