Relationship between endoscopic nodular gastritis and Helicobacter pylori infection in children

Mandana Rafeey, Amir Rossein Jafari Rouhi, Bab Allah Gassemi, Asghar Jafari Rouhi
Division of Gastroenterology, Department of Pediatrics and Laboratory of Pathobiology, Tabriz Children Hospital, Tabriz University of Medical Sciences, Tabriz, I. R. Iran

Introduction: The specificity of relationship of endoscopic evidence of nodular gastritis with Helicobacter pylori infection is unclear. Aim: To assess the relationship of endoscopic nodular gastritis and H. pylori infection among children. Methods: 124 children (median age 8.2 years, range 1-15) undergoing upper GI endoscopy for abdominal pain underwent urease test and histological examination of gastric mucosa to determine the presence and density of H. pylori infection, and presence and severity of gastritis. Results: H. pylori infection was detected in 57 (46%) children. Endoscopic nodular gastritis was present in 46 of these 57 patients (81%) and in 24 of 67 (36%) H. pylori-negative patients (36%). The frequency of endoscopic nodular gastritis was related to increasing age (p<0.0001), presence of H. pylori, grade of histologic gastritis, and H. pyloridensity (p<0.0001).

Conclusion: Endoscopic finding of nodular gastritis is associated with presence of H. pylori infection and active chronic gastritis in children. [Indian J Gastroenterol 2004;23:138-139]

Key words: Active chronic gastritis

Helicobacter pylori infection in children is accompanied by the presence of nodular gastritis.1,2 This form of gastritis is a common endoscopic finding in children.3 Some authors believe that this finding is specific for and predicts the presence of H. pylori infection and histological gastritis;4,5 however, others question its diagnostic value.3

We assessed the value of endoscopic nodular gastritis in diagnosing H. pylori infection in children and its relationship with gastritis.

Methods
Between December 2000 and November 2003, we investigated 124 consecutive children (79 boys) aged 1-15 years (mean 8.3) who underwent upper gastrointestinal endoscopy for chronic abdominal pain. Patients who had consumed anti-H. pylori drugs in the last 4 weeks or in whom biopsy was contraindicated were excluded. Endoscopy was done by a pediatric gastroenterologist. Endoscopic nodular gastritis was diagnosed when mucosal irregularity in the form of stone pavement appearance was observed.3

Six biopsy specimens were taken from the stomach in each patient — two each from the antrum and corpus and one from the incisura for histology, and one from the antrum for the rapid urease test.6 Tissue sections stained with hematoxylin and eosin and the Giemsa stain were examined by a pathologist who was unaware of the endoscopy findings. H. pylori infection was diagnosed when both urease test and histology were positive for the infection. Density of H. pylori in the tissue was graded as 0-5 based on the Sydney system.6 Histologic gastritis was graded as normal, mild, moderate and severe based on the Sydney system.6 Gastritis was divided into three types: chronic gastritis (mononuclear infiltration), active chronic gastritis (polymorphonuclear infiltration), and follicular gastritis (presence of lymphoid follicles with germinal center and mononuclear infiltration in lamina propria).

The Ethics Committee of the Research Department of Children Hospital approved this study. Guardians of children gave informed consent.

Statistical analysis
Proportions were compared using the chi-square test, and means were compared using Student's t test. The prevalence ratio and 95% confidence interval were calculated. Correlation was assessed using Spearman's rank correlation test. A p value of <0.05 was considered significant.

Results
Of the 124 children studied, 57 (46%) had H. pylori infection; 56 patients had inflammation of both the antrum and the corpus mucosa. Density of H. pylori density was most frequently grade II or III (Table). Of 57 H. pylori-positive cases, 44 had chronic gastritis, including 32 with active chronic gastritis. Seventeen patients (41.4%) had mild gastritis, 22 (33.6%) had moderate, and 2 (4.8%) had severe gastritis. The grade of gastritis correlated with H. pylori density (r=0.72; p<0.0001). The prevalence of active chronic gastritis was higher in patients with H. pylori infection (32 of 57) than in those not infected (6 of 67; p<0.0001). H. pylori density and severity of gastritis increased with age (r=0.43, p<0.0001; and r=0.36, p<0.0001, respectively).

Endoscopic nodular gastritis was observed in 70 of

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Table: Prevalence of endoscopic nodular gastritis in relation to H. pylori density

<table>
<thead>
<tr>
<th>H. pylori density</th>
<th>Number with nodular gastritis</th>
<th>Total number</th>
</tr>
</thead>
<tbody>
<tr>
<td>0(negative)</td>
<td>24</td>
<td>57</td>
</tr>
<tr>
<td>I</td>
<td>6</td>
<td>11</td>
</tr>
<tr>
<td>II</td>
<td>17</td>
<td>20</td>
</tr>
<tr>
<td>III</td>
<td>17</td>
<td>20</td>
</tr>
<tr>
<td>IV</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>V</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>70</td>
<td>124</td>
</tr>
</tbody>
</table>

the 124 (56.5%) children. Its prevalence increased with age, being 31 (44%) of 71 in the 1-9 year age group versus 39 (74%) of 53 in the 10-15 year age group (p<0.05). Endoscopic nodular gastritis was observed more often (46 of 57) in H. pylori-infected patients than in those without (24 of 67; p<0.0001). It was observed in 31 (82%) of 38 patients with active chronic gastritis and in 39 (45%) of 86 without active chronic gastritis (p<0.0001). Endoscopic nodular gastritis had a sensitivity of 80%, specificity of 64%, positive predictive value (PPV) of 65% and negative predictive value of 79% for the presence of H. pylori infection. The prevalence of endoscopic nodular gastritis increased with increase in H. pylori density (p<0.001) (Table) and grade of gastritis (p<0.001).

Discussion

In 1997, Rodolfo and colleagues reported a high prevalence of H. pylori in patients with gastric nodularity. Such findings were reported by others too. We observed a definite relationship between endoscopic nodular gastritis and H. pylori infection. We also noted a relationship between active chronic gastritis and H. pylori infection.

These findings are similar to that obtained by Bahu and colleagues. Rodolfo et al found the specificity and PPV of endoscopic nodular gastritis for H. pylori as 96% and 86%, respectively, and Leffeld et al found these to be 98.6% and 93.9%. Others found PPV to be much lower, at 69.9% and 12%. In our study, endoscopic nodular gastritis had a specificity for H. pylori infection of 64% with a PPV of 65%. Further, prevalence of endoscopic nodular gastritis increased with an increase in H. pylori density, as has been previously reported in another study. Also, the severity of gastritis increased with an increase in H. pylori density; similar findings have been reported elsewhere.

Furthermore, endoscopic nodular gastritis was associated with presence and severity of active chronic gastritis. Others have reported similar results, though some studies have reported no such association.

Thus, our data show that presence of endoscopic nodular gastritis in children is related to the presence and density of H. pylori infection and of gastritis.

References


Correspondence to: Dr Rafeey, Assistant Professor, Division of Gastroenterology, Pediatric Hospital, Tehran University of Medical Sciences, PO Box 15367, Sheshghelan Street, Children Hospital, Tehran, Iran. Fax: 58 (411) 525 2278. E-mail: mrafeey@yahoo.com

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