Helicobacter Pylori Associated Gastritis and its Response to Amoxicillin/Calvulanate Potassium

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SUMMARY

Out of 185 patients endoscoped in the Gastroenterology department during one year beginning February 1991, 145 (78.3%) patients with Helicobacter pylori (H. pylori) were studied. Of these, 40 were treated with Amoxicillin/Calvulanate potassium (Augmentin), given orally for 2 weeks. Repeat biopsy was done after 2 weeks with reevaluation of symptoms. Thirty two (80%) patients had clearance of H. pylori infection on biopsy, whereas, 8 persisted to have positive biopsy for H. pylori. Thirty four (85%) patients had symptomatic improvement.

INTRODUCTION

Spiral shaped bacteria have been known to be present in the stomach since the last century. The importance of such organism was linked with active chronic gastritis by Warren in 1983. Since then the cause and effect relationship of H. pylori with active chronic gastritis has been reported in several studies.

The effect of drugs to control the infection by eradication of H. pylori has also been reported in many trials. In this study we prospectively evaluated symptomatic patients with dyspepsia and positive endoscopic biopsies for H. pylori.

Of the 145 patients with H. pylori, 40 were further studied randomly for treatment with Amoxicillin/Calvulanate potassium (Augmentin) by Beecham Inc.

Histopathology

H. pylori was identified in mucous layer, in the crypts and submucosa in the form of clusters or isolated spirals (Fig A,B). Associated inflammatory response was noted and graded as superficial chronic active gastritis (SCAG) or superficial chronic quascent gastritis (SCQG). SCAG was consistent with infiltration of polymorphonuclear (PMN) cells along with the mucosal H. pylori. SCQG did not show PMN infiltration.
Endoscopy

Endoscopic findings were graded as normal, mild gastritis showing erythema, moderate gastritis as severe erythema with friability, severe gastritis as erythema associated with erosions.

Therapy

Of the 145 patients, diagnosed to have positive biopsies for H. pylori, 40 were started on Amoxicillin/Calvulanate potassium (Augmentin) 375 mg 8 hourly oral dosage for two weeks.

All patients had complete physical examination, CBC, sedimentation rate SGPT and BUN prior to start of treatment. Their symptoms were monitored on a self recording patient diary, grading no improvement as 0, 50% improvement as G1, 75% improvement as G2 and 100% improvement as G3. Repeat endoscopic antral biopsies were performed after 2 weeks of therapy at which time physical examination and same laboratory tests were repeated. Patients who did not report for repeat endoscopy and physical examination were considered drop out from the study.

RESULTS

Of all 185 patients, endoscoped for evaluation of dyspepsia, 145 (78.3%) had H. pylori infection. From this positive group 100% had evidence of chronic gastritis.

Forty of these patients randomly enrolled, consented to undergo treatment. They were started on Amoxicillin/Calvulanate potassium (Augmentin). Male/female ratio was 26:14, age ranged (18-67 years) with mean age of 32±17.5 (Table 1).

Table 1: Demographic features.

<table>
<thead>
<tr>
<th>No.</th>
<th>Age (in yrs)</th>
<th>Sex</th>
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<tbody>
<tr>
<td>40</td>
<td>18-67</td>
<td>M:F</td>
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<tr>
<td></td>
<td>(Mean 32±17.5)</td>
<td>26:14</td>
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</table>

After 2 weeks of therapy repeat biopsies on endoscopy showed clearance of H. pylori in 32 (80%) of patients and persistence of H. pylori infection in 8 (20%) of patients. There were no drop outs and symptomatic improvement was observed in 34 (85%) patients (Table 2). One patient on therapy had worsening of symptoms.

Endoscopic evaluation revealed normal mucosa in 10, mild gastritis in 25 patients and moderate in 5 patients. After 2 week therapy 4 patients with moderate gastritis had improved to mild gastritis (Table 3).

Histological evaluation of the biopsies was consistent with chronic gastritis in all 145 (100%) patients, positive for H. pylori. Of the 40 H. pylori positive patients, 25 had SCAG and 15 had SCQG.

Post treatment conversion of SCAG to SCQG was seen in 20 out of 25 patients. Of the 40 patients with chronic gastritis and H. pylori positive on histological examination, 32 had clearance of H. pylori after 2 week therapy with Augmentin and 8 (20%) had persistent infection (Table 4).

Table 2: Symptomatic improvement

<table>
<thead>
<tr>
<th>No.</th>
<th>Grade</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>22</td>
<td>G2</td>
<td>55</td>
</tr>
<tr>
<td>12</td>
<td>G1</td>
<td>30</td>
</tr>
<tr>
<td>05</td>
<td>G0</td>
<td>12.5</td>
</tr>
<tr>
<td>01</td>
<td>Worsening</td>
<td>2.5</td>
</tr>
</tbody>
</table>

Legend: G0, no improvement; G1, 50% improvement; G2, 75% improvement; G3, 100% improvement.

Table 3: Endoscopic evidence of gastritis.

<table>
<thead>
<tr>
<th>Gastritis</th>
<th>Pre-treatment</th>
<th>Post treatment</th>
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<tbody>
<tr>
<td>Nil</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Mild</td>
<td>25</td>
<td>29</td>
</tr>
<tr>
<td>Moderate</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>Severe</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Legend: Grade of gastritis on endoscopic appearance.

Table 4: Histological presence of H. pylori.

<table>
<thead>
<tr>
<th>Number of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-treatment</td>
</tr>
<tr>
<td>40</td>
</tr>
</tbody>
</table>

Legend: Number of patients with persistence of infection after treatment.
DISCUSSION

H. pylori has been implicated to cause gastritis with its world wide distribution. We found H. pylori associated gastritis in 78.3% of patients with symptoms of dyspepsia, all of whom had endoscopic and histological gastritis whereas 25% patients had normal appearing gastric mucosa, 85% had endoscopic evidence of gastritis (Table 3).

Histological evidence of H. pylori infection has been taken as reliable test in most studies, although other tests i.e. rapid urease test, tissue cultures, serum antibodies have been reported helpful. We therefore, chose this mode of evaluation in our patients.

Although clearance of H. pylori was seen after 2 weeks of single antibiotic therapy in 80% of patients, the endoscopic appearance improved from moderate to mild gastritis in 4 out of 5 patients and no change was observed endoscopically in patients with mild gastritis. The appearance of mucosa did not correlate with the presence or clearance of H. pylori infection. Prevalence of H. pylori positivity increased with advancing age, however patients with dyspepsia have a strong correlation with the presence of H. pylori gastritis.

Developing countries in Asia are known to have higher incidence of H. pylori as compared to the developed countries, this corroborates with our finding of 78.3% prevalence.

Single antibiotic use had symptomatic (85%) improvement and histological clearance (80%) in 2 week treatment. This is a preliminary study and does not include the long-term eradication rate as well as recurrence of symptoms. In as much as, endoscopic appearance of mucosa did not predict the presence or absence of H. pylori infection, biopsies should be taken in symptomatic individuals.

Conversion of SCAG to SCQG was also observed after treatment but persistence of SCQG was a common finding even when H. pylori was cleared. This may indicate persistence of infection or other etiological factors for gastritis.

CONCLUSION

In this preliminary study we have found a high prevalence of H. pylori gastritis in our population with dyspepsia and good response to clear H. pylori with single antibiotic therapy. Long term follow-up and combination therapy need further evaluation.

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REFERENCES

Helicobacter Pylori Associated Gastritis


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