tionship between OP-induced impairment in performance of a spatial memory task and the protracted decrease in the expression of cholinergic receptors in specific brain regions caused by asymptomatic exposure to OP compounds (18).

Although these findings are difficult to extrapolate directly to human low-level exposures to OPs, they indicate that short cognitive impairments without clinically manifested disturbance of central cholinergic nervous system could occur in humans following the inhalation exposure to asymptomatic concentrations of sarin.

Acknowledgements

The authors thank to Mrs. H. Antlová and Mrs. E. Vodiaková for their skilful technical assistance and to Mr. V. Bliha for the statistical evaluation.

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References


sive gastritis and acute erosive gastritis. The eradication was described as histologically negative examination (grade 0).

Results

The patient characteristics and distribution of risk factors for gastritis were shown in Table 1. There were no statistically significant differences between groups in age, alcohol intake, smoking, non steroidal antiinflammatory drug use and HP positivity.

At the end of the treatment, eradication was achieved in 20 patients (66.6 %) in group I, 15 patients (50 %) in group IV, 0 patients (0 %) in group V, 27 patients (90 %) in group VI and 7 patients (23.3 %) in group VII. The eradication rates were compared with standard treatment group.

No statistically significant difference between group I and II, IV and VI, V and VII were higher and the differences were statistically significant (p < 0.001, p < 0.001, p < 0.01 respectively). In group III eradication rate was significantly low. No eradication was achieved in group V.

To assess the efficiencies of drug therapies beyond the cases in which complete eradication has achieved we compared average points of HP colonisation grade before and after treatment in each group. The findings were given in Table 3. Rates of reduction in HP colonisation were similar to eradication rates in all groups. There were no additional adverse effects with the use of AA, BC and Allicin in combination with standard regimen. Monotherapy of these agents tolerated well also.

Discussion

Despite numerous studies on HP eradication there is not an accepted golden standard treatment regimen yet. In most studies two or three antimicrobial agents in combination with proton pump inhibitors were shown to be most effective. In previous studies clarymein, amoxicillin and lansoprazol combination were shown to eradicate HP in 60 % - 96 % of cases and accepted as one of the most successful regiments (11,16). On the other hand emerging antibiotic resistance, high treatment costs and drugs side effects necessitates development of new treatment modalities.HP induces infiltration of the gastric mucosa by polymorphonuclear cells and macrophages, as well as T and B lymphocytes (2,6). Paradoxically, this strong immune/inflammatory response cannot clear the infection, and thus leaves the host prone to complications resulting from chronic inflammation. The attracted immune cells produce inflammatory mediators that include reactive oxygen species (ROS). These mediators impart oxidative stress on the cells in the immediate vicinity in the gastric epithelium (15). Normally, oxidative stress is neutralized by natural antioxidants as vitamin C, beta carotene (4). However, levels of this antioxidant in the gastric juice are decreased during HP infection (2,7,13,19,25). On the other hand vitamin C is shown to be bactericidal for HP in vitro. In a clinical study high dose vitamin C (5 g/day for four weeks) successfully eradicated eight of 27 patients (30%) (10). In our study, we used 1200 mg/day Allicin for two weeks in two patient groups, each consisting of ten patients (1). They found that grade of gastritis and HP density were unchanged in all cases. In our study, we used 1200 mg/day Allicin for two weeks in two groups, each consisting 30 patients. It is the first study that Allicin was used in combination with standard eradication regimen. Also Allicin dose was higher than the previous studies. Addition of Allicin in standard regement group from 66.7% to 90%. In patients taking only Allicin eradication was achieved in 24.4%. Our study supports the hypothesis derived from in vitro studies that Allicin can be used in HP eradication. It is particularly advisable to combine Allicin with standard therapies. The effect may be dose related hence use of higher doses can be warranted. Allicin may help to overcome drug failure which was very high in our standard treatment group.

Conclusions

Ascorbic acid and beta carotene was found to be ineffective in HP eradication in vivo. On the other hand Allicin seemed to be potentially effective against HP in vivo.

References

5. F: Female
6. Male
7. M: Male(+/-): used or not used
8. NSAID: Non Steroidal Anti Inflammatory Drugs. There was no statistically difference between the groups, P>0.05.
9. NSAID Non Steroidal Anti Inflammatory Drugs. There was no statistically difference between the groups, P>0.05.
10. Student’s t test was used for comparison of the age between the groups. Chi square test were used for comparison of results of treatment groups. Spierman test was used for correlation of HP positivity and symptoms. P < 0.05 was considered to indicate statistical significance.

Tab. 1: Patients characteristic

<table>
<thead>
<tr>
<th>Group/Characteristic</th>
<th>Group I N=30 (%)</th>
<th>Group II N=30 (%)</th>
<th>Group III N=30 (%)</th>
<th>Group IV N=30 (%)</th>
<th>Group V N=30 (%)</th>
<th>Group VI N=30 (%)</th>
<th>Group VII N=30 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (year)</td>
<td>38±9±10</td>
<td>40±9±10</td>
<td>39±10</td>
<td>41±10</td>
<td>38±10</td>
<td>37±10</td>
<td>40±13</td>
</tr>
<tr>
<td>Sex (F/M)</td>
<td>18/12</td>
<td>16/14</td>
<td>18/12</td>
<td>19/16</td>
<td>16/14</td>
<td>15/15</td>
<td>16/14</td>
</tr>
<tr>
<td>Smoking (+/-)</td>
<td>7/23</td>
<td>7/23</td>
<td>9/21</td>
<td>8/22</td>
<td>8/22</td>
<td>8/22</td>
<td>7/25</td>
</tr>
</tbody>
</table>

P: value for eradication rates: Group I and II, IV >0.05, Group I and III, <0.001, Group I and VI, VII <0.01.

Tab. 2: At the end of the treatment eradication rates

<table>
<thead>
<tr>
<th>Group/Characteristic</th>
<th>Group I N=30 (%)</th>
<th>Group II N=30 (%)</th>
<th>Group III N=30 (%)</th>
<th>Group IV N=30 (%)</th>
<th>Group V N=30 (%)</th>
<th>Group VI N=30 (%)</th>
<th>Group VII N=30 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eradication (+)</td>
<td>20 (66.7)</td>
<td>15 (50)</td>
<td>3 (10)</td>
<td>15 (50)</td>
<td>- (0)</td>
<td>27 (90)</td>
<td>7 (24.4)</td>
</tr>
<tr>
<td>Eradication (-)</td>
<td>10 (33.3)</td>
<td>15 (50)</td>
<td>27 (90)</td>
<td>15 (50)</td>
<td>30 (100)</td>
<td>3 (10)</td>
<td>23 (76.6)</td>
</tr>
</tbody>
</table>

P: value for eradication rates: Group I and II, IV >0.05, Group I and III, <0.001, Group I and VI, VII <0.01.

Tab. 3: Density of HP colonisation before and after treatment

<table>
<thead>
<tr>
<th>Group/Characteristic</th>
<th>Group I N=30 (%)</th>
<th>Group II N=30 (%)</th>
<th>Group III N=30 (%)</th>
<th>Group IV N=30 (%)</th>
<th>Group V N=30 (%)</th>
<th>Group VI N=30 (%)</th>
<th>Group VII N=30 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HP colonisation before treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 point</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>2 points</td>
<td>9</td>
<td>10</td>
<td>12</td>
<td>12</td>
<td>14</td>
<td>14</td>
<td>15</td>
</tr>
<tr>
<td>3 points</td>
<td>18</td>
<td>15</td>
<td>10</td>
<td>13</td>
<td>15</td>
<td>13</td>
<td>18</td>
</tr>
<tr>
<td>4 points</td>
<td>6</td>
<td>5</td>
<td>4</td>
<td>4</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Aver. points</td>
<td>2.8</td>
<td>2.8</td>
<td>2.8</td>
<td>2.8</td>
<td>2.8</td>
<td>2.8</td>
<td>2.8</td>
</tr>
</tbody>
</table>

P values for average points before treatment: There was no statistically difference between the groups. P value for average points after treatment: Group I and II, IV >0.05, Group I and III, <0.001, Group I and VI, VII <0.01.
sive gastritis and acute erosive gastritis. The eradication was described as histologically negative examination (grade 0).

**Results**

The patient characteristics and distribution of risk factors for gastritis were shown in Table 1. There were no statistically significant differences between groups in age, alcohol intake, smoking, non-steroidal anti-inflammatory drug use and HP positivity.

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**Discussion**

Despite numerous studies on HP eradication there is not an accepted golden standard treatment regimen yet. In most studies two or three antimicrobial agents in combination with proton pump inhibitors were shown to be most effective. In previous studies chlorylamphenicol, amoxicillin and tetracycline were used in combination with HP eradication regimen. It was found that chlorylamphenicol was more effective than amoxicillin or tetracycline. On the other hand Allicin was used in combination with standard eradication regimen. Also Allicin dose was higher than the previous studies. Addition of Allicin to eradication regimen from polypharmacological regimens and macrophages, as well as T and B lymphocytes (2,6). Paradoxically, this strong immune/inflammatory response cannot clear the infection, and thus leaves the host prone to complications resulting from chronic inflammation. The attracted immune cells produce inflammatory mediators that include reactive oxygen species (ROS). These mediators impart oxidative stress on the cells in the immediate vicinity in the gastric epithelium (15). Normally, oxidative stress is neutralized by natural antioxidants as vitamin C, vitamin E (4). However, levels of this antioxidant in the gastric juice are decreased during HP infection (2,7,19,25). On the other hand vitamin C is shown to be bactericidal for HP and HP eradication. In a clinical study high dose vitamin C (5 g/day for four weeks) successfully eradicated eight of 27 patients (30 %) (10). In our study, vitamin C treatment for shorter period (14 days) with lower dose (1 g/day) was effective in only three of (10 %) 27 cases. Vitamin C appears to not be an agent powerful enough to be used in eradication alone. On the other hand addition of vitamin C to standard regimen did not improve the eradication rates contrary to our expectations. Similar to vitamin C gastric juice beta-carotene concentration was shown to be markedly lower in patients infected with HP (10,20). Beta carotene was thought to act as a mucosal protector by scavenging the ROS (20). The authors did not encounter any previous clinical study that beta carotene had been used in HP treatment. In our study beta carotene treatment (120 mg/day for 14 days) – either alone or in combination with the standard regimen – was disappointing ineffective.

Garlic extracts have in vivo and in vitro anti HP activity (17). Many studies have been conducted on anti-HP activity of garlic extracts (21-24). Relative activities of garlic powder, garlic oil, diallyl sulphide and Allicin had been studied and Allicin was found to be most potent garlic ingredient (20). MIC of Allicin was 4.0 microgram/ml. The antibacterial activity of garlic was also studied after combination with a proton pump-inhibitor (omeprazol) and a synergistic effect was found (5,14). Chronic HP disease is reduced with Allium vegetable intake. Graham et al. studied effect of fresh garlic in eradication of HP (8). They used three test meals and measured urease activity by a breath test. The authors concluded that garlic was not effective in vivo. Aydin et al. used garlic oil 275 mg capsules containing 800 µg Allium t.l.d. and garlic oil capsules plus omeprazol 20 mg/day for two weeks in two patient groups, each consisting of ten patients (1). They found that grade of gastritis and HP density were unchanged in all cases. In our study, we used 1200 µg/g/day Allicin for two weeks in each group, each consisting 30 patients. It is the first study that Allicin and lansasoprol combination were shown to eradicate HP in 60 % -96 % of cases and accepted as one of the most successful regiments (11,16). On the other hand the emerging antibiotic resistance, high treatment costs and drugs side effects necessitates development of new treatment modalities.

**Conclusion**

Ascorbic acid and beta carotene was found to be ineffective in HP eradication (in vivo). On the other hand Allicin seemed to be potentially effective against HP (in vivo).

**References**

Background & Aim: Celiac disease is an autoimmune disease with the damage of the intestinal barrier. The aim of study was to measure gut permeability in patients with untreated celiac disease and during treatment with a gluten-free diet.

Summary: The aim of the study was to measure gut permeability in patients with untreated celiac disease and during treatment with a gluten-free diet. Small bowel permeability was measured using lactulose/mannitol and lactulose/D-xylose ratios. The results were higher in untreated celiac disease and decreased after treatment with a gluten-free diet.

Key words: Gut permeability, Lactulose, Mannitol, D-xylose, Celiac disease, Gluten-free diet

INTRODUCTION

Celiac disease is an autoimmune disease with presence of antibodies against gluten causing damage to enterocytes and villous atrophy in sensitive patients. Celiac disease is a suitable model of gut barrier damage with an increase of small bowel permeability. There are two theories explaining the increase of small bowel permeability in villous atrophy. One theory differentiates the various ways of absorption of saccharides according to molecular size: monosaccharides (e.g. mannitol, L-chamomose) are absorbed passively via enterocytes (passive transcellular absorption); other monosaccharides (D-xylose) are absorbed by facilitated diffusion (active transport after concentration); glucose, amino acids and dipeptides are absorbed by active, carrier mediated transport, which is Na+/K+-ATPase dependent. Disaccharides are absorbed paracellularly via tight junctions. If the small mucose is damaged, transcellular absorption decreases and paracellular absorption increases due to adaptation to worsening absorption.

According to the second theory both monosaccharides and disaccharides are absorbed paracellularly via tight junctions, monosaccharides on villous tips and disaccharides in crypts. In case of villous atrophy all saccharides are absorbed in crypts and absorption of disaccharides is relatively higher (2,9,12).

Patients

30 patients (7 males, 23 females) aged 39-12 years with celiac disease were included in the study. 30 patients (8 males, 22 females) aged 39-12 years without non-specific gastrointestinal problems served as controls (Table 1).

Diagnosis of celiac disease was made using histological and serological examinations (anti-gladine, anti-endomysium...