The Relationship Between Ghrelin Levels and the Presence of Helicobacter Pylori in the Gastric Mucosa in Uremic Patients

The Relationship Between Ghrelin Levels and the Presence of Helicobacter Pylori in the Gastric Mucosa in Uremic Patients

Üremik Hastalarda Grelin Düzeyleri ile Mide Mukozasında Helicobacter Pylori Varlığı Arasındaki İlişki

ABSTRACT

OBJECTIVE: Helicobacter pylori (H. pylori) infection is an important risk factor for chronic gastritis and peptic ulcer. Ghrelin is mostly secreted by gastric mucosa. The aim of the study is to examine the relationship between H. Pylori and ghrelin levels in uremic patients.

MATERIAL and METHODS: A total of 91 patients [control group (CG, n=29), hemodialysis group (HD, no=21), peritoneal dialysis group(PD, no=12), predialysis group(PreG, n=29)] were involved. Patients using drugs active on H. pylori and/or gastric acidity were excluded. Besides demographic and biochemical parameters, patients were examined endoscopically; and H. Pylori was searched histopathologically.

RESULTS: H. pylori was found to be positive in 62 patients (68.1%). Those patients were not different from H. pylori negative group regarding demographic and biochemical parameters. H. pylori positivity was statistically similar in patient groups. Ghrelin levels of uremic groups (HD, PD and PreG) were significantly higher than that of CG (4.22±1.00ng/ml vs. 2.81±0.37ng/ml, p<0.001). Ghrelin level was higher in the predialysis group compared with the HD group (4.49±1.18ng/ml vs. 3.81±0.98ng/ml, p=0.040). Ghrelin levels of H. pylori positive and negative patients were not different (3.83±1.2ng/ml vs. 3.65±1.0ng/ml, p=0.50).

CONCLUSION: Ghrelin levels are elevated in uremia, but there is no relationship between ghrelin level and H. pylori positivity in uremic patients.

KEY WORDS: Helicobacter pylori, Ghrelin, Uremia

ÖZ


BULGULAR: H. pylori 62 hastada (%68.1) pozitif saptandı. Bu hastalar demografik ve biyokimyasal parametreler açısından H. pylori negatif gruptan farklı değerlendi. H. pylori pozitifliği hasta gruplarında istatistiksel olarak benzerdi. Grelin düzeyleri üremik gruplarda (HD, PD ve PreG) kontrol grubundan anlamlı derecede yüksekti (4.22±1.00ng/ml ve 2.80±0.37ng/ml, p<0.001). Grelin düzeyleri prediyaliz grupta HD grubuna kyasla daha yüksek bulundu (4.49±1.18 ng/ml ve 3.81±0.98 ng/ml, p=0.040). Grelin düzeyleri H. pylori pozitif ve negatif hastalarda farklı değildi (3.83±1.2 ng/ml ve 3.65±1.0 ng/ml, p=0.50).

SONUC: Grelin düzeyleri üremide yüksektir, fakat grelin düzeyleri ile H. pylori pozitifliği arasında ilişki yoktur.

ANAHTAR SÖZCÜKLER: Helicobacter pylori, Grelin, Üremi

Meltem GÜRSU1
Sami UZUN1
Ali Hakan KAYA2
Şule POTUROĞLU3
Savaş ÖZTÜRK1
Burcu KAYA2
Serhat KARADAĞ1
Zeki AYDIN1
Oktay ÖZKAN1
Birol BAYSAL3
Hikmet FEYİZOĞLU2
Rümeyza KAZANCIOĞLU4

1 Haseki Training and Research Hospital, Clinic of Nephrology, Istanbul, Turkey
2 Haseki Training and Research Hospital, Clinic of Internal Medicine, Istanbul, Turkey
3 Haseki Training and Research Hospital, Clinic of Gastroenterology, Istanbul, Turkey
4 Bezmialem Vakif University, School of Medicine, Department of Nephrology, Istanbul, Turkey

Received : 11.02.2013
Accepted : 04.03.2013

Correspondence Address:
Meltem GÜRSU
Haseki Eğitim ve Araştırma Hastanesi,
Nefroloji Kliniği, İstanbul, Turkey
Phone : +90 212 529 44 00
E-mail : meltem1401@yahoo.com
INTRODUCTION

Uremia affects the upper gastrointestinal system (GIS) as well as the other systems. Nausea, epigastric burning and pain are frequent symptoms in patients with chronic renal failure (CRF). Although, CRF has been suggested to increase the risk for peptic ulcer disease since many years, some studies have found no significant difference in peptic ulcer disease incidence among CRF patients when compared to the normal population (1, 2). Helicobacter pylori (H. pylori) infection, as one of the most common worldwide infections, is a main factor for chronic gastritis and peptic ulcer disease and it is also a risk factor for gastric cancer. The increased urea in CRF patients may predispose to colonization of H. pylori in the stomach (3). Conflicting results have been obtained related to prevalence of H. pylori in dialysis patients. Increased prevalence of H. pylori was detected in all CRF patients (4, 5). On the other hand, peptic ulcer recurrence after H. pylori eradication was shown to be higher in end-stage renal disease than non-uremic patients (6).

Ghrelin is a recently defined hormone that is mainly secreted from gastric mucosa and it stimulates appetite and the release of growth hormone (7, 8). Recently, serum ghrelin level was found to be 2.8 times more in those with CRF than non-uremic individuals (9). This increased ghrelin level was attributed to impaired renal function in breaking down and eliminating the ghrelin hormone. Increased ghrelin levels were also detected in hemodialysis (HD) and peritoneal dialysis (PD) patients (10).

There are several studies reporting the relationship between plasma ghrelin levess and H. pylori infection in non-uremic population, but it is still controversial. Some of them reported that plasma ghrelin levels increased or decreased after the eradication of H. pylori (11-14). There are also studies reporting that H. pylori infection has no effect on plasma ghrelin levels (15). There is no study investigating the relationship of plasma ghrelin levels and H. pylori in uremic population. Hence, our study aimed to investigate the prevalence of H. pylori in uremic patients and the correlation between the presence of H. pylori and ghrelin levels in different patient groups with uremia and compared with the normal population.

SUBJECTS and METHOD

All the participants were sequentially selected among the patients who were referred to our endoscopy unit between March 2009 and May 2010. Uremic patients were selected and divided into three groups:

Hemodialysis group (HD group): Patients on chronic hemodialysis program thrice a week for more than three months.
Peritoneal dialysis group (PD group): Patients on chronic peritoneal dialysis program for more than three months.
Predialysis group (PreG): Patients with stage 3-4 CRF.

These patients were compared with the control group (CG) involving patients with gastric symptoms and normal renal function (estimated glomerular filtration rate more than 90 ml/min and proteinuria less than 300 mg/day).

The study was started after gaining approval from local ethical committee; and informed consent was taken from all participants. Patients who did not give informed consent; patients aging less than 18 years, those who had been using drugs effective on gastric acidity (H2 receptor blockers, proton pump inhibitors, anti-acids, steroids, non-steroidal anti-inflammatory drugs) within the last months; patients with active gastrointestinal bleeding, history of GIS operation, previously diagnosed chronic GIS disease like inflammatory bowel diseases; those who received H. pylori eradication treatment within the last two years; patients with advanced cardiac, hepatic, pulmonary or malignant disease, any systemic infection within the last two weeks and pregnant patients were excluded from the study.

Demographic and clinical data of patients including age, gender, primary kidney disease, comorbid conditions, medications received within the last three months, past medical history and symptoms of upper GIS disease were recorded for each patient. In each patient the endoscopic and histopathological diagnoses (esophagitis, gastritis, duodenitis, ulcer, histopathological positivity for H. pylori) were obtained. Besides, blood samples were obtained after 12 hours of fasting in the morning of the endoscopic examination for determination of blood glucose, urea, creatinine, phosphorus, calcium, parathyroid hormone, albumin, white blood cell (WBC), hemoglobin, hematocrit, iron, total iron-binding capacity (TIBC), ferritin, C-reactive protein (CRP) and ghrelin levels. Ghrelin level was measured using Enzyme-Linked Immunosorbent Assay (ELISA).

All the endoscopic procedures were performed by the same endoscopist. Oral intake of patients was stopped at least 8-10 hours before the procedure. Local oropharyngeal anaesthesia was performed using Xylocaine pump spray 10% before the procedure. Midazolam 1-5 mg IV was administered as a sedative when needed. The endoscopic examination procedure of upper GIS was performed by video endoscopy. At least four gastric biopsies, two from the antrum and two from the corpus were obtained. The obtained biopsy samples were fixed and embedded in paraffin; and were evaluated histopathologically in the pathology department of the same hospital.

Statistical analysis: Statistical analysis was performed using SPSS (Statistical Package for Social Sciences) for Windows 16.0 package program. Numerical data were expressed as mean±standard deviation (SD). In the comparison of two groups, Student t-test or Mann Whitney U test was used when appropriate. Chi-square test and Fisher’s exact test were used for categorical variables. In the comparison of more than two groups, ANOVA or Kruskal Wallis-H variance analysis was used. The correlation analysis between quantitative parameters.
was performed using Pearson and Spearman’s (rho) correlation test for parameters with normal and abnormal distribution, respectively. p<0.05 was accepted as statistically significant. Multivariate linear regression analysis model was applied with “backward” method.

RESULTS

The study involved 91 (48 male, 43 female, mean age: 54±15 years) patients. There were 29 patients in the CG and predialysis group, 21 patients in the HD and 12 patients in the PD group. The baseline demographic characteristics of the groups and laboratory results are presented in Table I and Table II, respectively. The groups were well matched for primary kidney disease (p=0.553). The number of diabetic patients in HD, PD and predialysis groups were eight, two and ten, respectively (p=0.390). Consistent with these findings, blood glucose levels were higher in the HD and predialysis groups compared with the CG.

The symptoms necessitating endoscopic examination were epigastric pain in 36 (39.6%), epigastric burning in 30 (33%), retrosternal pain in 2 (2.2%), dyspepsia in 9 (9.9%), nausea in 2 (2.2%), chronic diarrhea in 2 (2.2%) and other symptoms in 10 (11%) patients. There was no difference between the groups in terms of symptomatology or existence of *H. pylori*.

The gastroscopic findings of the groups and the pathological evaluation of the biopsies obtained during gastroscopy are presented in Table III. 62 (68.1%) of the patients had *H. pylori* positivity histopathologically, while the remaining 29 (31.9%) were negative. *H. pylori* positivity rate was 65% in the control group, 58% in the PD group and 76% in the predialysis group. There was no statistically significant difference between the groups regarding this subject. *H. pylori* positive group was not different from negative group in terms of age, gender, serum urea, creatinin, albumin, calcium, phosphorus, parathyroid hormone, iron, TIBC, ferritin, hemoglobin, hematocrit and ghrelin levels. The entire *H. pylori* positive group had histopathologically proven gastritis whereas gastritis was prominent in 24 (83%) patients in the *H. pylori* negative group (p=0.001). Only presence of loose cardia was significantly higher in the *H. pylori* positive group than the negative one [11 (18%) patients vs. 1 (3%) patient, respectively]. Other histopathological and gastroscopic findings were similar between *H. pylori* positive and negative patients. *H. pylori* existence rate also did not differ statistically between the uremic groups (HD, PD and predialysis groups) and CG (Table III).

Mean serum ghrelin level was lower in CG than all other groups (Table II) (p<0.001). In addition, ghrelin level in HD group was lower than predialysis group (p=0.040). Serum ghrelin levels was not different in patients with or without *H. pylori* positivity (3.83±1.2 vs. 3.65±1.0 ng/ml, p=0.50). This was true for all groups when analyzed separately. Serum ghrelin level of the uremic groups was significantly higher than control group (2.81±0.37ng/ml vs. 4.22±1.00ng/ml, p<0.001).

The parameters found to be related with serum ghrelin levels in univariate analyses were as follows: CRP (r=0.20, p=0.05), phosphorus (r=0.341, p=0.001), glucose (r=-0.241, p=0.21), creatinine (r=0.403, p<0.0001), urea (r=0.403, p<0.0001), age (r=0.189, p=0.073), and parathyroid hormone (r=0.380, p<0.0001). When these parameters together with age, gender, group, *H. pylori* positivity and being uremic were analyzed by linear regression analysis; only the patients group (B=0.335, Beta=0.372, p=0.009) and being uremic (B=0.703, Beta=0.295, p=0.036) were found to be related to the serum ghrelin level.

### Table I: The demographic characteristics of the groups.

<table>
<thead>
<tr>
<th></th>
<th>CG (n=29)</th>
<th>HD group (n=21)</th>
<th>PD group (n=12)</th>
<th>Predialysis group (n=29)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>48±13</td>
<td>57±11</td>
<td>51±13</td>
<td>59±17</td>
<td>0.036&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Gender (male/female)</td>
<td>16/13</td>
<td>15/6</td>
<td>3/9</td>
<td>14/15</td>
<td>NS</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td></td>
<td>8</td>
<td>2</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Hypertensive nephrosclerosis</td>
<td>6</td>
<td>3</td>
<td>9</td>
<td></td>
<td>NS</td>
</tr>
<tr>
<td>ADPKD</td>
<td></td>
<td>2</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Chronic glomerulonephritis</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic pyelonephritis</td>
<td></td>
<td>-</td>
<td>2</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Others</td>
<td></td>
<td>3</td>
<td>3</td>
<td>5</td>
<td></td>
</tr>
</tbody>
</table>

**ADPKD:** Autosomal dominant polycystic kidney disease.

<sup>a</sup>: CG-Predialysis group (p=0.039)
Table II: Laboratory findings.

<table>
<thead>
<tr>
<th></th>
<th>CG (n=29)</th>
<th>HD group (n=21)</th>
<th>PD group (n=12)</th>
<th>Predialysis group (n=29)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Urea (mg/dl)</strong></td>
<td>27.2±13.6</td>
<td>110.6±40.2</td>
<td>91.7±36.1</td>
<td>83.7±47.3</td>
<td>&lt;0.001&lt;sup&gt;a,b,c&lt;/sup&gt;</td>
</tr>
<tr>
<td><strong>Creatinine (mg/dl)</strong></td>
<td>0.83±0.15</td>
<td>6.21±1.67</td>
<td>7.06±2.40</td>
<td>2.60±1.47</td>
<td>&lt;0.001&lt;sup&gt;a,b,c&lt;/sup&gt;</td>
</tr>
<tr>
<td><strong>Glucose (mg/dl)</strong></td>
<td>95±17</td>
<td>138±72</td>
<td>120±50</td>
<td>135±73</td>
<td>0.006&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td><strong>Albumin (g/dl)</strong></td>
<td>4.4±0.7</td>
<td>3.8±0.6</td>
<td>3.6±0.4</td>
<td>3.7±0.7</td>
<td>0.01&lt;sup&gt;a,b,c&lt;/sup&gt;</td>
</tr>
<tr>
<td><strong>Calcium (mg/dl)</strong></td>
<td>9.2±0.5</td>
<td>8.6±0.9</td>
<td>9.3±1.1</td>
<td>9.2±0.6</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Phosphorus (mg/dl)</strong></td>
<td>3.4±0.8</td>
<td>4.9±1.6</td>
<td>4.2±1.0</td>
<td>4.4±1.1</td>
<td>NS</td>
</tr>
<tr>
<td><strong>PTH (pg/ml)</strong></td>
<td>48±20</td>
<td>374±283</td>
<td>384±330</td>
<td>161±107</td>
<td>&lt;0.001&lt;sup&gt;b,c&lt;/sup&gt;, 0.04&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td><strong>WBC (/mm3)</strong></td>
<td>8648±1943</td>
<td>7365±2614</td>
<td>7408±1751</td>
<td>8058±2916</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Hemoglobin (g/dl)</strong></td>
<td>11.9±2.3</td>
<td>11.1±2.7</td>
<td>10.7±1.8</td>
<td>10.6±1.9</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Hematocrit (%)</strong></td>
<td>36±7</td>
<td>33±8</td>
<td>33±6</td>
<td>32±5</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Iron (mc/dl)</strong></td>
<td>66±57</td>
<td>77±47</td>
<td>71±45</td>
<td>48±30</td>
<td>NS</td>
</tr>
<tr>
<td><strong>TIBC (mcg/dl)</strong></td>
<td>311±79</td>
<td>229±65</td>
<td>214±40</td>
<td>254±83</td>
<td>0.02&lt;sup&gt;a&lt;/sup&gt;, 0.01&lt;sup&gt;b,c&lt;/sup&gt;</td>
</tr>
<tr>
<td><strong>Ferritin (ng/ml)</strong></td>
<td>92±183</td>
<td>632±549</td>
<td>369±230</td>
<td>219±422</td>
<td>NS</td>
</tr>
<tr>
<td><strong>CRP (mg/L)</strong></td>
<td>13.7±23.1</td>
<td>21.2±31.3</td>
<td>13.5±17.9</td>
<td>24.2±30.6</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Ghrelin (ng/ml)</strong></td>
<td>2.81±0.37</td>
<td>3.81±0.98</td>
<td>4.31±0.62</td>
<td>4.49±1.18</td>
<td>&lt;0.001&lt;sup&gt;a,b,c&lt;/sup&gt;, 0.04&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<sup>a</sup>: Control group versus predialysis group; <sup>b</sup>: Control group versus hemodialysis group; <sup>c</sup>: Control group vs. peritoneal dialysis group; <sup>d</sup>: Predialysis group vs. hemodialysis group; <sup>e</sup>: Predialysis group vs. peritoneal dialysis group

Table III: Distribution of gastroscopic and histopathological findings.

<table>
<thead>
<tr>
<th></th>
<th>CG (n=29)</th>
<th>HD group (n=21)</th>
<th>PD group (n=12)</th>
<th>Predialysis group (n=29)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gastroscopic findings</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Esophagitis</td>
<td>3 (%10.3)</td>
<td>3 (%14.3)</td>
<td>2 (%16.7)</td>
<td>4 (%13.8)</td>
<td>0.947</td>
</tr>
<tr>
<td>Gastritis</td>
<td>29 (%100)</td>
<td>19 (%90.5)</td>
<td>12 (%100)</td>
<td>29 (%100)</td>
<td>0.111</td>
</tr>
<tr>
<td>Duodenitis</td>
<td>10 (%34.5)</td>
<td>12 (%57.1)</td>
<td>6 (%50)</td>
<td>7 (%24.1)</td>
<td>0.089</td>
</tr>
<tr>
<td>Ulcer</td>
<td>6 (%20.7)</td>
<td>3 (%14.3)</td>
<td>1 (%8.3)</td>
<td>2 (%6.9)</td>
<td>0.435</td>
</tr>
<tr>
<td>Loose Carida</td>
<td>1 (%3.5)</td>
<td>5 (%23.8)</td>
<td>1 (%8.3)</td>
<td>5 (%17.3)</td>
<td>0.129</td>
</tr>
<tr>
<td>GOR*</td>
<td>2 (%6.9)</td>
<td>3 (%14.3)</td>
<td>2 (%16.7)</td>
<td>3 (%10.3)</td>
<td>0.767</td>
</tr>
</tbody>
</table>

**Histopathological findings**

<table>
<thead>
<tr>
<th></th>
<th>CG (n=29)</th>
<th>HD group (n=21)</th>
<th>PD group (n=12)</th>
<th>Predialysis group (n=29)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastritis</td>
<td>27 (%93.1)</td>
<td>20 (%95.2)</td>
<td>11 (%91.7)</td>
<td>28 (%95.6)</td>
<td>0.907</td>
</tr>
<tr>
<td>Duodenitis</td>
<td>7 (%24.1)</td>
<td>2 (%9.5)</td>
<td>3 (%25.0)</td>
<td>2 (%6.9)</td>
<td>0.185</td>
</tr>
<tr>
<td>Malignancy</td>
<td>0 (%0.0)</td>
<td>1 (%4.8)</td>
<td>0 (%0.0)</td>
<td>0 (%0.0)</td>
<td>0.396</td>
</tr>
<tr>
<td>H. pylori</td>
<td>19 (%65.6)</td>
<td>14 (%66.7)</td>
<td>7 (%58.3)</td>
<td>22 (%75.9)</td>
<td>0.692</td>
</tr>
</tbody>
</table>

<sup>*Gastroesophageal reflux</sup>
DISCUSSION

H. pylori infection is a main factor for chronic gastritis and peptic ulcer disease and also a risk factor for gastric cancer. The increased urea in CRF patients may predispose to colonization of H. pylori in the stomach (3). We aimed to investigate the previously unexamined relation between H. pylori and ghrelin in uremic patients. The groups were well matched for demographic parameters and primary kidney disease. The only difference among these parameters was between the control group and predialysis group regarding age. It is known that H. pylori positivity increases with age. However, the prevalence of H. pylori was similar in these mentioned groups, so this difference can be ignored. In our study, the gold standard of H. pylori was similar in these mentioned groups, so this difference positivity was detected histopathologically in 68% of the patients. Serum ghrelin level has been found higher in dialysis and predialysis groups than the control group (Table I), confirming previous similar studies comparing patients with HD and PD with non-uremic control group (10). Serum ghrelin level of H. pylori positive patients was nearly equal to the ones without H. pylori (3.83±1.2ng/ml vs. 3.65±1.0ng/ml, p=0.50). This similarity was present in each of the four groups.

Likewise, ghrelin level was similar in patients with or without histopathologically proven gastritis. These results are different from the studies demonstrating decreased ghrelin levels in H. pylori positive patients. No correlation was detected between H. pylori and serum ghrelin level in another study conducted in non-uremic population (15). Similarly, in the study of Osawa et al. studying the effect of H. pylori eradication on plasma ghrelin level in 134 non-uremic population, plasma ghrelin concentration increased in 50 patients whereas decreased in 84 patients after H. pylori eradication (18).

The studies conducted on the relationship between H. pylori and ghrelin levels yielded some confusing results. There are different studies in non-uremic population showing the potential relationship between H. pylori and gastric ghrelin (19). Some studies have found that ghrelin level decreased in the presence of H. pylori (13, 14). Additionally, in the aforementioned study of Osaka et al, it has been showed that expression of preproghrelin mRNA (which shows gastric ghrelin synthesis directly) increased following H. pylori eradication but serum ghrelin level decreased (18). However, there are also some studies indicating that this relation may be related with only atrophic gastritis. Furthermore, it has been found that H. pylori eradication decreased serum ghrelin levels in prepubertal children (20). Moreover, it has been considered that increased ghrelin may be associated with weight gain observed following H. pylori eradication (21).

It has been found that ghrelin plays a gastroprotective role and it shows this effect via prostaglandins (22). Ghrelin and H. pylori affect gastric mucosa contrarily in non-dialysis population. For example, ghrelin protects gastric mucosa from deterioration of ischemia/reperfusion by inhibiting the activation of nuclear factor-kappa B whereas H. pylori causes mucosal inflammation by the same factor (23). Additionally, ghrelin administered through central or peripheral circulation, increases the gastric movements, rate of gastric emptying and acidic secretion in rats and/or mice and ghrelin secretion is decreased by 70% following gastrectomy (24). Serum ghrelin level has been investigated in the studies conducted with respect to different GIS diseases and it has been found remarkably lower in the patients with chronic gastritis (25). The most common reason of the gastric inflammation in non-uremic population is H. pylori infection. H. pylori leads to gastric mucosal toxicity and consequently deteriorated gastric mucosa by converting ammonia to urea through the activity of urease (26). Since urea concentration in gastric mucosa is higher in CRF patients, effects of H. pylori may be expected to be changed in this patient group. As a matter of fact, it has been found in a study conducted by Aydemir et al that H. pylori increased the apoptosis of the gastric epithelial cell and this effect is more remarkable in uremic patients (27).

Also in uremic animal models, it has been found that gastric mucosal inflammation is different from the non-uremic ones and ischemic vascular deterioration is more remarkable (28). As known, frequency of peptic ulcer does not increase in uremic patients even in decreased gastric acid secretion (hypochlorhydia) present in the uremic patients (29). All of these findings suggest that gastric mucosal inflammation in uremic patients is different from the non-uremic patients and occurs through a different physiopathological mechanism. Toxins or factors different from H. pylori may have a role in gastric mucosal inflammation in the uremic patients.

Our study has some limitations. First of all the study is cross-sectional and the sample size is small in PD group and only patients with sufficient symptoms to warrant endoscopy were included.

CONCLUSION

Ghrelin concentration increases in uremic patients. However, high ghrelin levels in uremic population are independent of presence of H. pylori in gastric mucosa.

REFERENCES


Gürsu M et al: Ghrelin and Helicobacter Pylori in Uremia