***Helicobacter pylori* Antibodies in Patients on Chronic Hemodialysis**

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Table 1. Characteristics of hemodialysis patients

Hp(-)

Dear Sir,

A variety of gastrointestinal tract disorders are seen in uremic patients and gastritis, gastroduodenal ulceration, and bleeding are frequent complications of uremia [1]. Reduced gastric motility, changes in gastric morphology and histology, and increased gastrin levels may induce gastroduodenal lesions [2]. *Helicobacter pylori* (HP), a gram-negative spiral bacterium, has been shown to be strongly associated with gastritis, peptic ulcer disease and nonulcer dyspepsia, to be increased with gastritis, peptic ulcer disease and nonulcer dyspepsia [3]. Increased urea content of the gastric mucus could be a risk factor for HP in patients with chronic renal failure [4]. We measured the prevalence of HP in 91 hemodialysis (HD) patients and the results were correlated with dialysis duration, blood urea levels, receiving antacids or not. Ninety-one HD patients, 55 males and 36 females, aged between 16 and 70 years (mean age 41.4 ± 1.4 years) were studied. The mean duration of HD was 22.9 ± 2.2 months (range: 6-120 months). Sixty-eight HD patients (74.7%) were receiving aluminium antacids and calcium carbonate as phosphate binders. Serum samples for blood urea and Hp were taken before dialysis. Thirty-five age-matched healthy subjects with normal renal function were used as controls. Patients who had received antibiotics and colloidal bismuth preparations prior to blood sampling were excluded. IgG anti-

bodies against HP were measured by using the IgG ELISA test where the sensitivity and specificity of this technique was near 95%. The χ^2 test and the t test were used for statistical analysis.

NS = Not significant.

HP was detected in 13 (14.3%) of 91 patients undergoing regular HD. The mean predialysis serum urea in HP positive (+) and HP negative (-) patients was 127.3 ± 16.1 and 108.6 ± 19.8 mg/dl, respectively, the difference being significant at $p < 0.001$. There were no difference between HP (+) and HP (-) patients with respect to sex, age, and HD duration. The receiving

antacids in HP (+) and HP (-) were 38.4% (5/13) and 80.7% (63/78), respectively, the difference being statistically significant at $p < 0.001$. Sixty-five percent (9/13) of HP (+) patients had symptomatic dyspepsia compared to 50% (38/78) in the HP (-) patients, and smoking status was similar in seropositive and seronegative patients (table 1). In 8 of 35 (22.8%) healthy controls HP was positive.

We found antibodies against HP in 14.3% of HD patients and in 22.8% of the controls with normal renal functions. In the literature, the prevalence of HP ranges from 24 to 43% in patients with chronic renal failure [5, 6]. It has been accepted that the prevalence of HP may be related to blood levels of patients with renal insufficiency. But, it has also been reported that high blood urea levels have not been a risk factor for HP infection.

Hp(+)

p value

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In our study, predialysis blood urea levels of seropositive patients were significantly higher than those of seronegative HD patients. In the meantime, there was a significant difference between seropositive and seronegative patients who were receiving antacids. This may indicate that HP antibodies are particularly determined in patients who are not receiving antacids.

In conclusion, the prevalence of HP was found to be decreased in patients with chronic renal failure compared to controls. We suggest that this may be related to those prescribed antacids more frequently and that the high predialysis urea level may be a risk factor for HP infection.

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Nephron 1996;72:725-726

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