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Review of *Helicobacter pylori* Infection and Chronic Renal Failure

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Abstract

Chronic renal failure patients receiving hemodialysis and continuous ambulatory peritoneal dialysis often encounter gastrointestinal troubles over their long treatment period. *Helicobacter pylori* infection has close association with development of peptic ulcer, gastric cancer and gastric lymphoma, and is thought to be one of the major risk factors for gastrointestinal troubles in dialysis patients. However, it is unclear whether *H. pylori* infection is directly associated with progression of renal dysfunction and prognosis of chronic renal failure patients. Recent consensus shows that the prevalence of *H. pylori* infection in chronic renal failure patients is significantly lower than in subjects with normal renal function. In the natural history of *H. pylori* infection in hemodialysis patients, the prevalence of infection decreases as dialysis periods progressed, in particular within the first four years after the start of treatment. However, the chance of natural eradication becomes rare for patients receiving dialysis treatment for a long time. Moreover, chronic renal failure patients with *H. pylori* infection have a higher incidence of gastroduodenal diseases, and therefore, are recommended to receive eradication therapies, especially for those receiving treatment for a long time and with higher risks of complication. Intensive endoscopic check-ups for the prevention of gastrointestinal events and the discovery of peptic ulcer and neoplastic diseases at an early phase may be required.

Keywords

Chronic renal failure; Continuous ambulatory peritoneal dialysis; Eradication therapy; *Helicobacter pylori*; Hemodialysis; Peptic ulcer

Chronic renal failure (CRF) patients receiving hemodialysis treatment consist of more than 1.1 million people in the world, and the size of this population is expanding at a rate of 7% per year according to the progress in medical and dialysis machine technique (1). Although heart failure, angina pectoris, hypertension, parathyroid-related disease, amyloidosis, renal anemia and infection are well known to occur by receiving hemodialysis and continuous ambulatory peritoneal dialysis (CAPD) for long periods (2), those patients also often suffer from gastrointestinal troubles including peptic ulcer, hemorrhage, abdominal symptoms, constipation, diarrhea, ileus, and perforation (3–12).

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Helicobacter pylori is a spiral-shaped, microaerophilic Gram-negative flagellate bacterium that was isolated in 1983 from gastric biopsy specimens of patients with chronic gastritis (13). The gastric mucosa of approximately 50% of the world's population is infected with *H. pylori*, and the infection levels exceed 70% in some developing areas (14,15). *Helicobacter pylori* infection plays a crucial role in the development of gastrointestinal diseases, such as peptic ulcer, gastric hyperplastic polyps, gastric adenoma, gastric cancer, and gastric mucosa associated-lymphoid tissue lymphoma, both in individuals with normal renal function and in chronic renal failure patients receiving hemodialysis and CAPD (Tables 1 and 2) (4–9,16,18–32,34,36–45,47–60).

Patients with chronic renal failure often have gastrointestinal symptoms caused not only by *H. pylori* infection, but also by high urea levels, decline of gastrointestinal motility, amyloid protein deposition (59,60), and decreased sensory disturbance. Therefore, the quality of life in patients with chronic real failure is usually poor, which affects their nutrition status leading to the development of malnutrition, which is a potent predictor of morbidity and mortality. Moreover, patients with chronic renal failure may have higher risks of gastric mucosal damages compared with individuals with normal renal function because of systemic and/or local chronic circulatory failure (61,62), hypergastrinemia (32), high ammonia levels (55), and enhanced inflammation. Especially, 25–75% of those patients suffer from a number of gastrointestinal lesions and complications including gastric erosions, peptic ulcers, angiodysplasia and gastrointestinal bleeding (3–12,17).

It is unclear about the actual condition of gastroduodenal diseases in hemodialysis and CAPD patients related with *H. pylori* infection, because previous reports have controversial results due to small sample size and short observation periods. In this review article, we therefore initially reviewed the association between *H. pylori* infection and chronic renal failure, the natural history of *H. pylori* infection according to the treatment periods, and then summarize the *H. pylori* eradication therapy for chronic renal failure patients.

Helicobacter pylori infection in patients receiving hemodialysis and CAPD

Helicobacter pylori infection has an influence on intake of proton pump inhibitors (PPI) and antibiotics and eradication therapy of *H. pylori*. The prevalence of *H. pylori* infection in chronic renal failure patients receiving CAPD or hemodialysis was shown to be equal or lower compared to subjects with normal renal function in various different geographic populations irrespective to presence/absence of gastric symptoms (4–9,16,18–32,34,36–45,47–60) (Table 1). Recently, in the investigation of 539 Japanese hemodialysis patients with treatment periods of mean 8.4 ± 0.3 years, the prevalence of *H. pylori* infection was reported to be 48.6% ([95% CI: 44.3–52.9%]), which was significantly lower than in dyspepsia patients with normal renal function (78.5% [74.1–82.4%], 314/400, $P < 0.001$) and individuals with normal renal function receiving annual health exams (69.4% [60.3–77.5%], 84/121, $P < 0.001$) (48). Moreover, the prevalence of *H. pylori* infection in hemodialysis patients is significantly lower (27.5%) compared with non-hemodialysis chronic renal failure patients (56.0%) (36), and the prevalence in individuals with normal renal function is similar with patients receiving hemodialysis treatment for less than 1-year period (48). These data suggest that hemodialysis treatment, but not uremia by chronic renal failure itself, plays a role in the lower prevalence of *H. pylori* infection. The start of hemodialysis treatment is thought trigger the decrease in the prevalence of *H. pylori* infection.

Previously, eight studies showed significantly lower prevalence of *H. pylori* infection in patients receiving hemodialysis (8/36 studies, 22.2%) (20,21,25,36,38,48,53,57) compared with controls and one study has found a significantly lower prevalence of *H. pylori* infection

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in patients receiving CAPD (1/5 studies, 20.0%) (21); whereas only one study reported that Iranian hemodialysis patients (63.0%) and chronic renal failure patients (66.2%) had significantly higher prevalence of *H. pylori* infection compared with normal individuals (27.5%) (Table 1) (9). However, since the prevalence of *H. pylori* infection in the Iranian population is reported to be more than 60% in other reports (63,64), further studies are required to clarify this discrepancy in the Iranian population. In a combined analysis using previous reports (4–9,16,18–32,34,36–45,48–60), the prevalence of *H. pylori* infection in patients receiving chronic hemodialysis and CAPD are 43.9% ([95% CI: 42.2–45.6%], 1435/3 272) and 34.8% ([29.6–40.2%], 113/325), respectively, which is significantly lower than in individuals with normal renal function both with and without gastrointestinal symptoms (49.8% [48.0–51.7%], 1476/2961, $P < 0.001$).

There is variation in *H. pylori* infection rates among different countries. It may, therefore, be better to evaluate the infection rate in various countries. In East Asian countries, the prevalence of *H. pylori* infection in patients receiving chronic hemodialysis is 44.5% ([95% CI: 41.5–47.6%], 474/1065), which is significantly lower than in individuals with normal renal function (54.0% [50.9–57.1%], 560/1038, $P < 0.001$). On the other hand, because the prevalence of *H. pylori* in other areas, such as Europe, Middle East, and South Asia has a wide variation, it is difficult to evaluate the prevalence of *H. pylori* infection in those areas.

The prevalence of *H. pylori* infection in patients receiving a kidney transplant was 39.0% ([33.9–44.2%], 143/367) (Table 2). Kidney transplant patients with sufficient renal function at present also have a lower *H. pylori* infection rate, as well as hemodialysis and CAPD patients. This may be explained by a situation in which most patients received hemodialysis and/or CAPD anytime before kidney transplantation.

The evaluation of an association between *H. pylori* infection and causes of chronic renal failure is limited. The prevalence in patients whose chronic renal failure was caused by diabetic nephropathy (60.1%) is reported to be significantly higher than that in chronic glomerulonephritis patients (46.1%) irrespective of the decreased immune system of diabetes patients (48). This suggests that disorders of the immune system, such as severe diabetic nephropathy, does not lose the maintenance of *H. pylori* infection.

Treatment periods of hemodialysis and *H. pylori* infection

Only a few reports have evaluated the relationship between *H. pylori* infection and dialysis treatment periods, and the mean dialysis duration in *H. pylori*-positive patients was significantly shorter than in uninfected patients (28,29,36,41,48). Munoz de Bustillo et al. (28) reported that mean hemodialysis periods in *H. pylori* negative patients receiving dialysis was 66.5 months, which was significantly longer than in positive patients (24.7 months). Nakajima et al. (36) reported that the prevalence of *H. pylori* infection markedly decreases when the treatment duration is two years or more. Recently, when the treatment period of hemodialysis and the *H. pylori* infection rate were simply measured at one time in a hospital, the infection rate was reported to gradually decrease according to dialysis treatment periods until four years after starting hemodialysis followed by a plateau (Table 3) (48). The prevalence of *H. pylori* infection is inversely associated with treatment periods of hemodialysis and the decreasing pattern is characteristic (Table 3). However, there are no data on the relationship between *H. pylori* infection and the time course of CAPD treatment periods.

The follow-up survey of *H. pylori* infection

To investigate whether *H. pylori* infection was actually eradicated after beginning hemodialysis treatment, a follow-up survey using the same dialysis patients with *H. pylori*

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infection is required. When the natural history of *H. pylori* infection was investigated using more than 300 patients for a 4 year-follow up survey, although nobody received eradication therapy, the prevalence of *H. pylori* infection was reported to be 51.6% at first year, 42.9% two years later, and 38.3% four years later, indicating that the infection rate gradually decreased during dialysis treatment (Table 3). In other words, 26.7% of dialysis patients naturally cured *H. pylori* infection over four years. Patients who received hemodialysis and CAPD are expected to eradicate *H. pylori* not only naturally but also by environmental improvement and *H. pylori* eradication.

The mechanisms of lower prevalence of *H. pylori* infection in hemodialysis and CAPD patients

There are at least three explanations as to why dialysis patients have low prevalence of *H. pylori* infection: (i) Patients receiving dialysis have higher levels of pro-inflammatory cytokines, including interleukin (IL)-1β, IL-6, IL-8 and tumor necrosis factor (TNF)-α, from activated inflammatory cells infiltrating the gastric mucosa (65). As a result, gastric atrophy progresses, accompanied by increased pH, and finally *H. pylori* are not able to live in gastric mucosa (66). (ii) Blood urea levels as well as urea nitrogen levels in gastric secretions are higher in dialysis patients than in patients with normal renal function, and high urea levels may inhibit *H. pylori* growth in the stomach (67). (iii) *H. pylori* infection might be cured upon antibiotic treatment, both because antibiotics are commonly used during the initial treatment periods and most dialysis patients had many chances of bacterial infection during the treatment, and because antibiotic concentrations are higher and more prolonged in patients with renal failure than in individuals with normal renal function. Importantly, there was no significant difference in the prevalence of *H. pylori* infection between patients with normal renal function and patients receiving less than one year of dialysis treatment (48). Therefore, urea concentration and antibiotic usage are unlikely to be contributors in decreasing the prevalence of infection. One possible explanation for decreased *H. pylori* prevalence is therefore up-regulation of pro-inflammatory cytokine resulting from dialysis enhanced severe inflammation of gastric mucosa, and progression to gastric atrophy. However, further studies to investigate the histopathology of gastric inflammation and atrophy are necessary.

Necessity of *H. pylori* eradication therapy for chronic renal failure patients

The prevalence of peptic ulcer in hemodialysis and CAPD patients with *H. pylori* infection is higher than in individuals with normal renal function (9). Moreover, the incidence of tumor death is 2–4 times higher in dialysis patients than in the general population, and it is known that the most frequently detected malignant tumor is gastric cancer (68).

For hemodialysis and CAPD patients, one of the severe gastroduodenal events is a hemorrhage from gastroduodenal lesions. Because most chronic renal failure patients receive anti-coagulant drugs and/or anti-platelet drugs for prevention of hypertension and brain infarction, and thus anti-coagulant drugs, such as heparin sodium, are usually used during hemodialysis treatment, hemorrhage from gastroduodenal lesions easily occurs in such patients and often causes fatal blood loss. Moreover, gastric mucosa is known to be fragile in hemodialysis and CAPD patients. Therefore, to prevent hemorrhage from gastrointestinal diseases, intragastric pH in patients receiving hemodialysis and CAPD should be kept at a higher level by doses of acid inhibitory drugs (e.g. proton pump inhibitors) and by limiting doses of anticoagulant drugs.

One important question is whether *H. pylori* eradication therapies are necessary for *H. pylori*-infected hemodialysis and CAPD patients to prevent gastroduodenal disease

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development. As described above, more than one-third of the patients receiving approximately four years of dialysis treatment have naturally cured *H. pylori* infections, especially in hemodialysis patients (48). However, the frequency of peptic ulcer in patients receiving dialysis treatment is known to be higher in *H. pylori* infected patients than in uninfected patients, as observed in patients with normal renal function (48). We therefore recommend eradication therapies for *H. pylori*-infected hemodialysis patients and for patients receiving hemodialysis treatment. Moreover, it is better to eradicate at an early time for patients with a higher risk of disease development, such as past history of peptic ulcer and/or gastroduodenal hemorrhage, usage of several kinds of anticoagulants and non-steroidal anti-inflammatory drugs (NSAIDs, Fig. 1).

Present condition *otH. pylori* eradication therapy for patients with chronic renal failure

One of the merits for *H. pylori* eradication therapy in hemodialysis and CAPD patients is that they keep a high plasma concentration of PPI and antibiotics even with low doses of drugs, although overdose of drugs must be carefully checked. On the other hand, one of the demerits for *H. pylori* eradication therapy in hemodialysis and CAPD patients is that there is a possibility that those patients are often infected with antibiotic-resistant strains since they have many occurrences of antibiotic intake due to an impaired immune system. In fact, 36.4% of patients with end-stage renal disease are reported to have clarithromycin-resistant strains, which is significantly higher than in non-uremic controls (15.2%) (69).

As summarized in Table 4 (4,28,31,32,35,38,46,70–75), each eradication regimen previously reported use of different PPIs (omeprazole, lansopprazole, esomeprazole) and/or antibiotics (clarithromycin, amoxicillin and metronidazole), and variation of the duration of treatment from 7 days to more than 3 weeks. However, there is no significant difference in the overall cure rates among the different studies listed. In multivariate analysis for eradication failure (age, gender, endoscopic findings, dialysis therapy, and previous eradication treatment), only history of previous treatment remained (38). This suggests that antibiotic resistance influences eradication success/failure, as observed in healthy non-dialysis patients. However, there are currently no data on antibiotic-resistant strains in eradication studies with dialysis patients (Table 4).

Antibiotics are known to have the possibility to progress renal dysfunction. Sheu et al. (74) reported that among chronic renal failure non-dialysis patients, those in the lansoprazole–clarithromycin–metronidazole regimen had a lower risk of acute renal failure than those in the lansoprazole–clarithromycin–amoxicillin regimen (2% vs. 18%, $P < 0.05$; relative risk, 0.128, 95%CI, 0.016–0.979). Because amoxicillin is mainly excreted from the kidney, toxic effects of amoxicillin on renal function in patients with chronic renal failure have been reported in various studies (76,77). However, most previous reports showed no severe adverse effects of amoxicillin in hemodialysis patients who received eradication therapy containing amoxicillin (4,28,31,32,35,38,70,71). Moreover, since PPI and antibiotics are removed by hemodialysis, plasma concentration of PPI and antibiotics is expected to decrease to a concentration that is less than effective. Therefore, it is necessary to set an optimal dosing plan in hemodialysis patients to increase cure rate and to use antibiotics more safely by using established pharmacological methods, such as monitored concentration.

Most patients who received eradication therapy were prevented from recurrence of peptic ulcer and had decreased risk of gastric cancer development (78–80). With eradication therapy in patients treated for early gastric cancer by endoscopic mucosal resection, the odds ratio for metachronous gastric cancer decreased significantly [0.353 (95% CI 0.161–0.775; $P = 0.009$)] (79). Patients who received hemodialysis often encountered gastric mucosal

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injuries, such as gastric erosion and gastric ulcer, irrespective to *H. pylori* infection due to NSAID intake and arteriosclerosis. Therefore, for patients receiving hemodialysis and CAPD, the most important benefit is the prevention of gastric cancer development.

CONCLUSION

Chronic renal failure patients receiving hemodialysis and CAPD have a low prevalence of *H. pylori* infection. More than one-third (36.2%) of patients receiving less than four years' dialysis had naturally cured *H. pylori* infection within the four-year observation period. However, because chronic renal failure patients have a higher risk of gastroduodenal disorders, it is recommended that all hemodialysis and CAPD patients receive endoscopic check-ups to reduce the chance of developing peptic ulcers. Moreover, patients with *H. pylori* infection should receive eradication therapy to prevent peptic ulcer and hemorrhage. The clinical protocol for a detailed endoscopic check-up and eradication therapy for dialysis patients should be evaluated in future studies.

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REFERENCES

1. Lysaght MJ. Maintenance dialysis population dynamics: current trends and long-term implications. J Am Soc Nephrol. 2002; 13(Suppl l):S37–40. [PubMed: 11792760]
2. Go AS, Chertow GM, Fan D, McCulloch CE, Hsu CY. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. N Engl J Med. 2004; 351:1296–305. [PubMed: 15385656]
3. Ala-Kaila K. Upper gastrointestinal findings in chronic renal failure. Scand J Gastroenterol. 1987; 22:372–6. [PubMed: 3296137]
4. Sezer S, Ibis A, Ozdemir BH, et al. Association of *Helicobacter pylori* infection with nutritional status in hemodialysis patients. Transplant Proc. 2004; 36:47–9. [PubMed: 15013297]
5. Sotoudehmanesh R, Ali Asgari A, Ansari R, Nouraie M. Endoscopic findings in end-stage renal disease. Endoscopy. 2003; 35:502–5. [PubMed: 12783348]
6. Prakash J, Agrawal BK. Upper gastrointestinal mucosal lesions in chronic renal failure. Indian J Gastroenterol. 1991; 10:131–2. [PubMed: 1748493]
7. Gheissari A, Rajyaguru V, Kumashiro R, Matsumoto T. Gastrointestinal hemorrhage in end stage renal disease patients. Int Surg. 1990; 75:93–5. [PubMed: 2379997]
8. Tsai CJ, Hwang JC. Investigation of upper gastrointestinal hemorrhage in chronic renal failure. J Clin Gastroenterol. 1996; 22:2–5. [PubMed: 8776085]
9. Khedmat H, Ahmadzad-Asl M, Amini M, et al. Gastroduodenal lesions and *Helicobacter pylori* infection in uremic patients and renal transplant recipients. Transplant Proc. 2007; 39:1003–7. [PubMed: 17524875]
10. Milito G, Taccone-Gallucci M, Brancaleone C, et al. Assessment of the upper gastrointestinal tract in hemodialysis patients awaiting renal transplantation. Am J Gastroenterol. 1983; 78:328–31. [PubMed: 6344617]
11. Milito G, Taccone-Gallucci M, Brancaleone C, et al. The gastrointestinal tract in uremic patients on long-term hemodialysis. Kidney Int Suppl. 1985; 17:S157–60. [PubMed: 3867787]
12. Musola R, Franzin G, Mora R, Manfrini C. Prevalence of gastroduodenal lesions in uremic patients undergoing dialysis and after renal transplantation. Gastrointest Endosc. 1984; 30:343–6. [PubMed: 6392003]
13. Marshall BJ, Warren JR. Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. Lancet. 1984; 1:1311–15. [PubMed: 6145023]

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14. Rocha GA, Queiroz DM, Mendes EN, et al. Indirect immunofluorescence determination of the frequency of anti-*H. pylori* antibodies in Brazilian blood donors. Braz J Med Biol Res. 1992; 25:683–9. [PubMed: 1342599]
15. Perez-Perez GI, Taylor DN, Bodhidatta L, et al. Seroprevalence of *Helicobacter pylori* infections in Thailand. J Infect Dis. 1990; 161:1237–1. [PubMed: 2345304]
16. Offerhaus GJ, Kreuning J, Valentijn RM, et al. *Campylobacter pylori*: prevalence and significance in patients with chronic renal failure. Clin Nephrol. 1989; 32:239–41. [PubMed: 2582650]
17. Wee A, Kang JY, Ho MS, Choong HL, Wu AY, Sutherland IH. Gastroduodenal mucosa in uraemia: endoscopic and histological correlation and prevalence of *Helicobacter*-like organisms. Gut. 1990; 31:1093–6. [PubMed: 2083853]
18. Davenport A, Shallcross TM, Crabtree JE, Davison AM, Will EJ, Heatley RV. Prevalence of *Helicobacter pylori* in patients with end-stage renal failure and renal transplant recipients. Nephron. 1991; 59:597–601. [PubMed: 1766499]
19. Loffeld RJ, Peltenburg HG, vd Oever H, Stobberingh E. Prevalence of *Helicobacter pylori* antibodies in patients on chronic intermittent haemodialysis. Nephron. 1991; 59:250–3. [PubMed: 1956486]
20. Jaspersen D, Fassbinder W, Heinkele P, et al. Significantly lower prevalence of *Helicobacter pylori* in uremic patients than in patients with normal renal function. J Gastroenterol. 1995; 30:585–8. [PubMed: 8574329]
21. De Vecchi AF, Quatrini M, Boni F, et al. Epidemiology of *Helicobacter pylori* in dialysis patients. Pent Dial Int. 1995; 15:178–9.
22. Luzza F, Imeneo M, Maletta M, et al. *Helicobacter pylori*-specific IgG in chronic haemodialysis patients: relationship of hypergastrinaemia to positive serology. Nephrol Dial Transplant. 1996; 11:120–4. [PubMed: 8649619]
23. Seyrek N, Kocabas E, Hazar S, Paydas S, Aksaray N, Sagliker Y. *Helicobacter pylori* antibodies in patients on chronic hemodialysis. Nephron. 1996; 72:725–6. [PubMed: 8730459]
24. Krawczyk W, Gorna E, Suwala J, et al. Frequency of *Helicobacter pylori* infection in uremic hemodialyzed patients with antral gastritis. Nephron. 1996; 74:621–2. [PubMed: 8938694]
25. Abu, Farsakh NA.; Roweily, E.; Rababaa, M.; Butchoun, R. Brief report: evaluation of the upper gastrointestinal tract in uraemic patients undergoing haemodialysis. Nephrol Dial Transplant. 1996; 11:847–50. [PubMed: 8671907]
26. Hruby Z, Myszka-Bijak K, Gosciniak G, et al. *Helicobacter pylori* in kidney allograft recipients: high prevalence of colonization and low incidence of active inflammatory lesions. Nephron. 1997; 75:25–9. [PubMed: 9031266]
27. Ozgur O, Boyacioglu S, Ozdogan M, Gur G, Telatar H, Haberal M. *Helicobacter pylori* infection in haemodialysis patients and renal transplant recipients. Nephrol Dial Transplant. 1997; 12:289–91. [PubMed: 9132647]
28. Munoz de Bustillo E, Sanchez Tomero JA, Sanz JC, et al. Eradication and follow-up of *Helicobacter pylori* infection in hemodialysis patients. Nephron. 1998; 79:55–60. [PubMed: 9609463]
29. Yildiz A, Besisik F, Akkaya V, et al. *Helicobacter pylori* antibodies in hemodialysis patients and renal transplant recipients. Clin Transplant. 1999; 13:13–16. [PubMed: 10081629]
30. Fabrizi F, Martin P, Dixit V, et al. Epidemiology of *Helicobacter pylori* in chronic haemodialysis patients using the new RIBA H. pylori SIA. Nephrol Dial Transplant. 1999; 14:1929–33. [PubMed: 10462273]
31. Araki H, Miyazaki R, Matsuda T, Gejyo F, Koni I. Significance of serum pepsinogens and their relationship to *Helicobacter pylori* infection and histological gastritis in dialysis patients. Nephrol Dial Transplant. 1999; 14:2669–75. [PubMed: 10534510]
32. Gur G, Boyacioglu S, Gul C, et al. Impact of *Helicobacter pylori* infection on serum gastrin in haemodialysis patients. Nephrol Dial Transplant. 1999; 14:2688–91. [PubMed: 10534513]
33. Boran M, Cetin S. *Helicobacter pylori* and *Giardia lamblia* infection in hemodialysis patients. Nephrol Dial Transplant. 1999; 14:1803–4. [PubMed: 10435905]

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34. Huang JJ, Huang CJ, Ruaan MK, Chen KW, Yen TS, Sheu BS. Diagnostic efficacy of (13)C-urea breath test for *Helicobacter pylori* infection in hemodialysis patients. Am J Kidney Dis. 2000; 36:124–9. [PubMed: 10873881]
35. Wang YL, Sheu BS, Huang JJ, Yang HB. Noninvasive stool antigen assay can effectively screen *Helicobacter pylori* Infection and assess success of eradication therapy in hemodialysis patients. Am J Kidney Dis. 2001; 38:98–103. [PubMed: 11431188]
36. Nakajima F, Sakaguchi M, Amemoto K, et al. *Helicobacter pylori* in patients receiving long-term dialysis. Am J Nephrol. 2002; 22:468–72. [PubMed: 12381945]
37. Fabbian F, Catalano C, Bordin V, Balbi T, Di Landro D. Esophagogastroduodenoscopy in chronic hemodialysis patients: 2-year clinical experience in a renal unit. Clin Nephrol. 2002; 58:54–9. [PubMed: 12141407]
38. Tsukada K, Miyazaki T, Katoh H, et al. Seven-day triple therapy with omeprazole, amoxycillin and clarithromycin for *Helicobacter pylori* infection in haemodialysis patients. Scand J Gastroenterol. 2002; 37:1265–8. [PubMed: 12465723]
39. Marsenic O, Peco-Antic A, Perisic V, Virijevic V, Kruscic D, Kostic M. Upper gastrointestinal lesions in children on chronic haemodialysis. Nephrol Dial Transplant. 2003; 18:2687–8. [PubMed: 14605311]
40. Lopez T, Quesada M, Almirall J, Sanfeliu I, Segura F, Calvet X. Usefulness of non-invasive tests for diagnosing *Helicobacter pylori* infection in patients undergoing dialysis for chronic renal failure. Helicobacter. 2004; 9:674–80. [PubMed: 15610083]
41. Nakajima F, Sakaguchi M, Oka H, et al. Prevalence of *Helicobacter pylori* antibodies in long-term dialysis patients. Nephrology. 2004; 9:73–6. [PubMed: 15056265]
42. Al-Mueilo SH. Gastroduodenal lesions and *Helicobacter pylori* infection in hemodialysis patients. Saudi Med J. 2004; 25:1010–14. [PubMed: 15322589]
43. Trimarchi H, Forrester M, Schropp J, Pereyra H, Freixas EA. Low initial vitamin B12 levels in *Helicobacter pylori*—positive patients on chronic hemodialysis. Nephron Clin Pract. 2004; 96:C28–32. [PubMed: 14752251]
44. Blusiewicz K, Rydzewska G, Rydzewski A. Gastric juice ammonia and urea concentrations and their relation to gastric mucosa injury in patients maintained on chronic hemodialysis. Rocz Akad Med Bialymst. 2005; 50:188–92. [PubMed: 16358963]
45. Lentine KL, Parsonnet J, Taylor I, Wrone EM, Lafayette RA. Associations of serologic markers of infection and inflammation with vascular disease events and mortality in American dialysis patients. Clin Exp Nephrol. 2006; 10:55–62. [PubMed: 16544178]
46. Itatsu T, Miwa H, Nagahara A, et al. Eradication of *Helicobacter pylori* in hemodialysis patients. Ren Fail. 2007; 29:97–102. [PubMed: 17365917]
47. Gioe FP, Cudia B, Romano G, et al. Role and clinical importance of *Helicobacter pylori* infection in hemodialysis patients. G Chir. 2008; 29:81–4. [PubMed: 18366885]
48. Sugimoto M, Sakai K, Kita M, Imanishi J, Yamaoka Y. Prevalence of *Helicobacter pylori* infection in long-term hemodialysis patients. Kidney Int. 2009; 75:96–103. [PubMed: 18843261]
49. Antoniou S, Dimitriadis A, Kliridou M, Pavlitou K, Batzili H, Malaka E. Prevalence of *Helicobacter pylori* antibodies in CAPD patients. Nephron. 1997; 75:358–9. [PubMed: 9069462]
50. Aguilera A, Codoceo R, Bajo MA, et al. *Helicobacter pylori* infection: a new cause of anorexia in peritoneal dialysis patients. Perit Dial Int. 2001; 21(Suppl 3):S152–6. [PubMed: 11887811]
51. Lui SL, Wong WM, Ng SY, Chan TM, Lai KN, Lo WK. Sero-prevalence of *Helicobacter pylori* in Chinese patients on continuous ambulatory peritoneal dialysis. Nephrology. 2005; 10:21–4. [PubMed: 15705177]
52. Altay M, Turgut F, Akay H, et al. Dyspepsia in Turkish patients on continuous ambulatory peritoneal dialysis. Int Urol Nephrol. 2008; 40:211–17. [PubMed: 18196468]
53. Shousha S, Arnaout AH, Abbas SH, Parkins RA. Antral *Helicobacter pylori* in patients with chronic renal failure. J Clin Pathol. 1990; 43:397–9. [PubMed: 2370308]
54. Ala-Kaila K, Vaajalahti P, Karvonen AL, Kokki M. Gastric *Helicobacter* and upper gastrointestinal symptoms in chronic renal failure. Ann Med. 1991; 23:403–6. [PubMed: 1930936]

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55. Neithercut WD, Rowe PA, el Nujumi AM, Dahill S, McColl KE. Effect of *Helicobacter pylori* infection on intragastric urea and ammonium concentrations in patients with chronic renal failure. J Clin Pathol. 1993; 46:544–7. [PubMed: 8331178]
56. Moustafa FE, Khalil A, Abdel Wahab M, Sobh MA. *Helicobacter pylori* and uremic gastritis: a histopathologic study and a correlation with endoscopic and bacteriologic findings. Am J Nephrol. 1997; 17:165–71. [PubMed: 9096448]
57. Misra V, Misra SP, Dwivedi M, et al. Decreased sensitivity of the ultrarapid urease test for diagnosing *Helicobacter pylori* in patients with chronic renal failure. Pathology. 1999; 31:44–6. [PubMed: 10212922]
58. Emir S, Bereket G, Boyacioglu S, Varan B, Tunali H, Haberal M. Gastroduodenal lesions and *Helicobacter pylori* in children with end-stage renal disease. Pediatr Nephrol. 2000; 14:837–40. [PubMed: 10955940]
59. Schoonjans R, Van VB, Vandamme W, et al. Dyspepsia and gastroparesis in chronic renal failure: the role of *Helicobacter pylori*. Clin Nephrol. 2002; 57:201–7. [PubMed: 11924751]
60. Strid H, Simren M, Stotzer PO, Abrahamsson H, Bjomsson ES. Delay in gastric emptying in patients with chronic renal failure. Scand J Gastroenterol. 2004; 39:516–20. [PubMed: 15223673]
61. Block GA, Raggi P, Bellasi A, Kooienga L, Spiegel DM. Mortality effect of coronary calcification and phosphate binder choice in incident hemodialysis patients. Kidney Int. 2007; 71:438–41. [PubMed: 17200680]
62. Nakamura S, Sasaki O, Nakahama H, Inenaga T, Kawano Y. Clinical characteristics and survival in end-stage renal disease patients with arteriosclerosis obliterans. Am J Nephrol. 2002; 22:422–8. [PubMed: 12381939]
63. Jafarzadeh A, Rezayati MT, Nemati M. Specific serum immunoglobulin G to *H pylori* and CagA in healthy children and adults (south-east of Iran). World J Gastroenterol. 2007; 13:3117–21. [PubMed: 17589930]
64. Hashemi MR, Rahnavardi M, Bikdeli B, Dehghani Zahedani M. *H pylori* infection among 1000 southern Iranian dyspeptic patients. World J Gastroenterol. 2006; 12:5479–82. [PubMed: 17006984]
65. Hwang IR, Kodama T, Kikuchi S, et al. Effect of interleukin 1 polymorphisms on gastric mucosal interleukin 1beta production in *Helicobacter pylori* infection. Gastroenterology. 2002; 123:1793–803. [PubMed: 12454835]
66. Wesdorp RI, Falcao HA, Banks PB, Martino J, Fischer JE. Gastrin and gastric acid secretion in renal failure. Am J Surg. 1981; 141:334–8. [PubMed: 7011077]
67. Gladziwa U, Haase G, Handt S, et al. Prevalence of *Helicobacter pylori* in patients with chronic renal failure. Nephrol Dial Transplant. 1993; 8:301–6. [PubMed: 8390002]
68. Ota K, Yamashita N, Suzuki T, Agishi T. Malignant tumours in dialysis patients: a nationwide survey. Proc Eur Dial Transplant Assoc. 1981; 18:724–30. [PubMed: 7329999]
69. Aydemir S, Boyacioglu S, Gur G, et al. *Helicobacter pylori* infection in hemodialysis patients: susceptibility to amoxicillin and clarithromycin. World J Gastroenterol. 2005; 11:842–5. [PubMed: 15682477]
70. Tamura H, Tokushima H, Murakawa M, et al. Eradication of *Helicobacter pylori* in patients with end-stage renal disease under dialysis treatment. Am J Kidney Dis. 1997; 29:86–90. [PubMed: 9002534]
71. Tokushima H, Tamura H, Murakawa M, et al. Eradication of *Helicobacter pylori* restores elevation of serum gastrin concentrations in patients with end-stage renal disease. Intern Med. 1998; 37:435–9. [PubMed: 9652896]
72. Mak SK, Loo CK, Wong AM, et al. Efficacy of a 1-week course of proton-pump inhibitor-based triple therapy for eradicating *Helicobacter pylori* in patients with and without chronic renal failure. Am J Kidney Dis. 2002; 40:576–81. [PubMed: 12200810]
73. Mak SK, Loo CK, Wong PN, et al. A retrospective study on efficacy of proton-pump inhibitor-based triple therapy for eradication of *Helicobacter pylori* in patients with chronic renal failure. Singapore Med J. 2003; 44:74–8. [PubMed: 14503780]

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74. Sheu BS, Huang JJ, Yang HB, Huang AH, Wu JJ. The selection of triple therapy for *Helicobacter pylori* eradication in chronic renal insufficiency. Aliment Pharmacol Ther. 2003; 17:1283–90. [PubMed: 12755841]

75. Tseng GY, Lin HJ, Fang CT, et al. Recurrence of peptic ulcer in uraemic and non-uraemic patients after *Helicobacter pylori* eradication: a 2-year study. Aliment Pharmacol Ther. 2007; 26:925–33. [PubMed: 17767477]

76. Arancibia A, Drouguett MT, Fuentes G, et al. Pharmacokinetics of amoxicillin in subjects with normal and impaired renal function. Int J Clin Pharmacol Ther Toxicol. 1982; 20:447–53. [PubMed: 7141752]

77. Jones DP, Gaber L, Nilsson GR, Brewer ED, Stapleton FB. Acute renal failure following amoxicillin overdose. Clin Pediatr. 1993; 32:735–9.

78. Uemura N, Okamoto S, Yamamoto S, et al. *Helicobacter pylori* infection and the development of gastric cancer. N Engl J Med. 2001; 345:784–9. [PubMed: 11556297]

79. Fukase K, Kato M, Kikuchi S, et al. Effect of eradication of *Helicobacter pylori* on incidence of metachronous gastric carcinoma after endoscopic resection of early gastric cancer: an open-label, randomised controlled trial. Lancet. 2008; 372:392–7. [PubMed: 18675689]

80. Take S, Mizuno M, Ishiki K, et al. The effect of eradicating *Helicobacter pylori* on the development of gastric cancer in patients with peptic ulcer disease. Am J Gastroenterol. 2005; 100:1037–42. [PubMed: 15842576]

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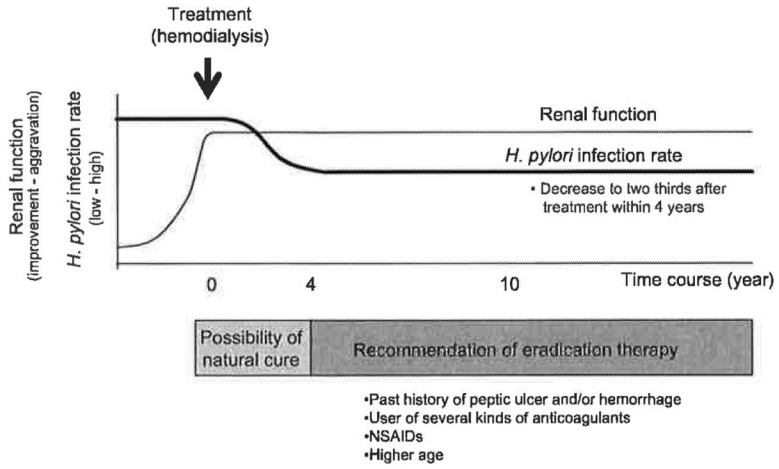

FIG. 1.

The schema of natural history of *Helicobacter pylori* infection in hemodialysis patients and treatment strategy.

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TABLE 1

List of previous reports: association of *Helicobacter pylori* infection prevalence and patients with hemodislysis

Year	Author	Period (months)	Patient (n)	Control (n)	Character of Control	PU	GIF
1989	Offerhaus GJA (16)	NA	44% (50)	45% (40)	Healthy	+	−
1990	Wee A (17)[†]	NA	31% (322)	NA		NA	+
1991	Davenport A (18)	38 (1–76)	34% (76)	30% (247)	Healthy	NA	−
1991	Loffeld RJ (19)	4 (2–16)	43% (30)	38% (344)	Healthy	NA	−
1995	Jasperson D (20)	NA	23% (34)[a]	37% (127)	Symptoms	+	+
1995	De Vecchi AF (21)	NA	59% (29)	72% (29)	Symptoms	+	−
1996	Luzza F (22)	74 ± 63	73% (103)	78% (103)	Symptoms	NA	−
1996	Seyrek N (23)	23 ± 2	14% (91)	23% (35)	Healthy	NA	−
1996	Krawczyk W (24)	28 ± 12	62% (21)	64% (22)	Dyspepsia	−	+
1996	Abu Farsakh NA (25)	18 (1–108)	57% (28)[a]	73% (100)		NA	+
1997	Hruby Z (26)	52 ± 54	35% (25)	44% (16)	Dyspepsia	NA	+
1997	Ozgur O (27)	29 ± 29	60% (47)	64% (100)	Symptoms	+	+
1998	Munoz de Bustillo E (28)	40 ± 42	64% (52)	NA	+	+	+
1999	Yildiz A (29)	33 ± 28	66% (47)	73% (55)	Healthy	NA	−
1999	Fabirizi F (30)	NA	56% (228)	53% (158)	Healthy	NA	−
1999	Araki H (31)[†]	91 ± 62	46% (63)	66% (64)	Healthy	+	+
1999	Gut G (32)	21 ~ 22	57% (44)	55% (45)	Dyspepsia	−	+
1999	Boran M (33)	52 ± 21	50% (50)	NA		NA	+
2000	Huang JJ (34)	28 (1–156)	47% (70)	60% (42)	Dyspepsia	+	+
2001	Wang YL (35)	53	50% (80)	60% (80)	Dyspepsia	+	+
2002	Nakajima F (36)	47 ± 58	28% (51)[a]	56% (25)	CRF (non-HD)	+	+
2002	Fabbian F (37)	51 ± 58	13% (38)	NA		−	4-
2002	Tsukada K (38)	76.5−57.4	30% (47)[a]	56% (55)	Dyspepsia	+	+
2003	Sotoudehmanesh R (5)	14 ± 29	59% (206)	NA		+	+
2003	Marsenic O (39)[‡]	NA	9% (22)	NA		−	+
2004	Lopez T (40)	56 ± 61	42% (86)	NA		NA	−

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Year	Author	Period (months)	Patient (n)	Control (n)	Character of Control	PU	GIF
2004	Sezer S (4)	67 ± 48	10% (163)	NA		−	+
2004	Nakajima F (41)	57 ± 62	37% (138)	62% (138)	Healthy	NA	−
2004	Al-Mueilo SH (42)	NA	63% (44)	63% (60)	Dyspepsia	+	+
2004	Trimarchi H (43)	19 ~ 20	28% (29)	NA		NA	−
2005	Blusiewicz K (44)	NA	63% (30)	71% (31)	Dyspepsia	+	+
2006	Lentine (45)	28	25% (97)	NA		NA	−
2007	Khedmat H (9)	47 ± 11	63% (73)[b]	35% (305)	Dyspepsia	+	+
2007	Itatsu T (46)	NA	40% (77)	NA	Symptoms	NA	+
2008	Gioe FP (47)	42–85	53% (142)	45% (132)	Symptoms	−	−
2009	Sugimoto M (48)	59 ± 0.4	49% (539)[a]	79% (400)	Symptoms	+	+

Treatment period was determined as median (range) or mean ± SE or SD according to reports. Gastrointestinal endoscopy (GIF)-positive (+) indicates that GIF was performed in the study, and peptic ulcer-positive (+) meant reports included peptic ulcer in both/either patient group and/or control group with normal renal function. CAPD, continuous ambulatory peritoneal dialysis; CRF, chronic renal failure; PU, peptic ulcer; NA, not available.

[a] $P < 0.05$ when significantly lower compared with control subjects;

[b] $P < 0.05$ when significantly higher compared with control subjects.

† Paper contains hemodialysis and CAPD patients

‡ Paper contains hcmodialysis and CRF patients.

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TABLE 2

List of previous reports: association with *Helicobacter pylori* infection prevalence and patients with CAPD and Tx

Treatment	Year	Author	Period (month)	Patient (n)	Control (n)	Character of Control	PU	GIF
CAPD								
	1995	De Vecchi AF (21)	NA	53% (38)[a]	79% (38)	Symptoms	+	−
	1997	Antoniou S (49)	(1–108)	64% (39)	NA		NA	−
	2001	Aguilera A (50)	18 ± 13	33% (48)	NA		−	−
	2005	Lui SL (51)	54 ± 42	26% (136)	NA		NA	−
	2008	Altay M (52)	21 ± 18	27% (64)	NA		NA	−
TX								
	1991	Davenport A (18)	45 (6–219)	29% (202)	30% (247)	Healthy	NA	−
	1997	Hruby Z (26)	41 ± 35	62% (29)	44% (16)	Dyspepsia	NA	+
	1997	Ozgur O (27)	51 ± 40	70% (54)	64% (100)	Symptoms	+	+
	1999	Yildiz A (29)		38.6% (57)	73% (55)	Healthy	NA	−
	2007	Khedmat H (9)	52 ± 16	40% (25)	35% (305)	Dyspepsia	+	+
CRF								
	1990	Shousha S (53)		24% (50)[a]	42% (120)	Symptoms	−	+
	1991	Ala-Kaila K (54)		17% (89)	NA		NA	−
	1993	Neithercut WD (55)		48% (23)	NA		NA	+
	1995	Jaspcrson D (20)		24% (59)[a]	37% (127)	Symptoms	+	+
	1997	Moustafa FE (56)		40% (70)	NA		NA	+
	1999	Misra V (57)		56% (50)[a]	78% (50)	Healthy	−	+
	2000	Emir S (58)		63% (16)	NA		+	+
	2002	Schoonjans R (59)		46% (28)	NA		−	−
	2004	Strid H (60)		26% (31)	NA		NA	−
	2004	Nakajima F (41)		53.3 (30)	62% (138)	Healthy	NA	−
	2007	Khedmat H (9)		66% (71)[b]	35% (305)	Dyspepsia	+	+

Treatment period was determined as median (range) or mean ± SE or SD according to reports. Gastrointestinal endoscopy (GIF)-positive (+) indicates that GIF was performed in the study, and peptic ulcer-positive (+) meant reports included peptic ulcer in both/either patient group and/or control group with normal renal function. CAPD, continuous ambulatory peritoneal dialysis; CRF, chronic renal failure; PU, peptic ulcer; NA, not available; Tx, kidney transplant.

[a] $P < 0.05$ when significantly lower compared with control subjects;

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[b] $P < 0.05$ when significantly higher compared with control subjects.

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TABLE 3

Changes in *Helicobacter pylori* infection rate at the beginning, two years later, and 4 years later among total patients, chronic glomerulonephritis (CGN) patients, and patients with diabetes and others (48)

	Total	CGN	Diabetes	Others
Beginning	51.7%	48.8%	64.0 %[b]	48.8%
2 years later	42.9%[a]	41.3%	47.2%	46.5%
4 years later	38.3%[a]	38.2%	41.6%	32.6%

Others included lupus nephritis, polycystic kidney, chronic pyelonephritis and nephrosclerosis.

[a] $P < 0.05$ (vs. Beginning);

[b] $P < 0.05$ (vs. CGN and others groups).

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TABLE 4

Helicobacter pylori eradication therapy for chronic renal failure patients

Year	Author	Country	Number	Regimen	Eradication rate	Determine methods	Resistance
1997	Tamura H (70)	Japan	14	LPZ (30) oid/ 8 weeks, AMPC (500) oid/ 3 weeks, plaunotol (80) tid/ 24 weeks	78.6%	RUT, Culture, Histology	No data
1998	Munos de Bustillo E (28)	Spain	23	OPZ (20) bid, AMPC (500) tid/ 14 days	60.8%	UBT	No data
			23	Plus OPZ (20) bid, CAM (500) bid/ 14 days	82.6%		
1998	Tokushima H (71)	Japan	17	LPZ (30) oid/ 8 weeks, AMPC (500)/3 weeks	76.5%	RUT, Culture, Histology	No data
			10	LPZ (30) oid, AMPC (250), MNZ (250) bid/ 7 days	90.0%		
1999	Araki H (31)	Japan	17	OPZ (20) oid/ 8 weeks, AMPC (250) oid, CAM (200) oid/ 3 weeks, polaprizinc (0.5) bid/ 24 weeks	88.2%	IgG, Histology	No data
1999	Gur G (32)	Turkey	25	FAM (40) oid, CAM (500) bid, MNZ (250) bid/ 15 days	80.0%	Histology, RUT	No data
2001	Wang YL (35)	China	38	OPZ (20), AMPC (1000), CAM (500) bid/ 7 days	86.8%	Stool	No data
2002	Mak SK (72)	China	21 (CRF)	OPZ (20), AMPC (1000), CAM (500) bid/ 7 days	90.5%	RUT	No data
2002	Tsukada K (38)	Japan	39	OPZ (30) bid, AMPC (500) tid, CAM (400) bid/ 7 days	82.1%	UBT	No data
2003	Mak SK (73)	China	25 (CRF)	OPZ (20) or LPZ (30), AMPC (1000), CAM (5O0) bid/ 7 days	96.0%	Histology	No data
2003	Sheu BS (74)	China	38 (CRF)	LPZ (30), AMPC (750), CAM (500) bid/ 7 days	76.3%		
			40 (CRF)	LPZ (30), CAM (500), MNZ (500) bid/ 7 days	92.5%	Stool	No data
2004	Sezer S (4)	Turkey	17	OPZ (20), AMPC (1000), CAM (500) bid/ 14 days	94.1%	Endoscopy	No data
2007	Tseng GY (75)	China	34 (CRF)	ESO (40) or OPZ (20) bid, AMPC (1000) bid, CAM (500) bid/ 7 days	94.1%	UBT	No data
2007	Itatsu T (46)	Japan	11	LPZ (60), AMPC (750), CAM (400)/ 7 days	72.7%	RUT	No data
			9	LPZ (60), CAM (400)/ 7 days	33.3%		

AMPC, amoxicillin; CAM, clarithromycin; Eso, Esomeprazole; FAM, famotidine; LPZ, lansoprazole; MNZ, metronidazole; OPZ, omeprazole; RUT, rapid urease test; UBT, urea breath test.

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