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Proton Pump Inhibitor for Treatment of *Helicobacter pylori* in Patients with Chronic Renal Failure: Is It Necessary?

Gaosi Xu^a, Jian Li^b, Pingshan Cheng^b, Weiping Tu^a

Departments of ^aNephrology and ^bScientific Education, Second Affiliated Hospital, Nanchang University, Nanchang, PR China

It has been demonstrated that the incidence of gastroduodenal disease is high in patients with chronic renal failure (CRF), although many studies have shown that these patients have normal-to-low acid secretion. *Helicobacter pylori* was first cultivated from human gastric mucosa in 1983 [1] and has been shown to play an important role in the development of gastritis and gastric ulcer. Dyspeptic symptoms and chronic gastritis are commonly observed in patients with CRF [2] and infection with *H. pylori* has also been described in dialysis patients.

H. pylori has several pathogenetic determinants, one of which is urease activity, converting urea to ammonia, which provides protection against low gastric pH (hydrogen ion concentration). Generally, CRF patients show an elevated concentration of gastric urea [3]. Thus, the gastric microenvironment in CRF patients should facilitate *H. pylori* mucosal colonization, which in turn can induce mucosal damage. The hypoacidity in CRF patients with *H. pylori* infection appears to result from neutralization of acid by ammonia as well as from gastric atrophy. The pH 3 holding time tests suggest that *H. pylori* infection does not significantly affect gastric acidity in subjects without CRF.

Aydemir et al. [4] reported that the uremia accelerates apoptosis and proliferation in gastric epithelial cells in patients with CRF. Increased luminal pH in CRF with *H. pylori* infection appears to result from neutralization of acid by ammonia overproduction, and also from decreased acid secretion due to gastric atrophy.

Hypotheses

Amoxicillin is conventionally used together with another antibiotic called clarithromycin to treat stomach ulcers caused by

H. pylori infection. This combination is often used with a stomach acid reducer called proton pump inhibitor (PPI). The 1-week course of PPI-based triple therapy has achieved a high eradication rate of *H. pylori* infection in CRF and hemodialysis patients [5].

Rabeprazole, a PPI, is able to rapidly achieve and maintain a pH of >4, although all PPI are activated at low pH levels (pH: 0–5), and some PPI lose their activation as the pH increases [6]. Although metronidazole is unaffected by gastric pH, amoxicillin and clarithromycin require pH values of 3 and 4, respectively, for maximum effectiveness. Therefore, achieving and maintaining a pH below 4 is very important for successful eradication of *H. pylori*. Profound inhibition of gastric acid secretion seems not to be necessary to improve the effect of amoxicillin on the cure rate for *H. pylori* infection in patients with duodenal ulcers.

Taken together, this evidence has led us to hypothesize that it is not necessary to treat *H. pylori* infection in CRF patients with PPI.

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Weiping Tu
Department of Nephrology, Second Affiliated Hospital
1, Minde Road
Nanchang 330006 (PR China)
Tel./Fax +86 791 629 1976, E-Mail weipingu6102@tom.com