Rebound hypersecretion after omeprazole and its relation to on-treatment acid suppression and *Helicobacter pylori* status

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**Abstract**

**Background & Aims:** There have been conflicting reports regarding acid secretion after treatment with omeprazole. This study examined acid secretion after treatment with omeprazole and its relation to *Helicobacter pylori* status and on-treatment gastric function.  

**Methods:** Twelve *H. pylori*-negative and 9 *H. pylori*-positive subjects were examined before, on, and at day 15 after an 8-week course of 40 mg/day omeprazole. On each occasion, plasma gastrin, intragastric pH, and acid output were measured basally and in response to increasing doses of gastrin 17.  

**Results:** In the *H. pylori*-negative subjects at day 15 after omeprazole treatment, basal acid output was 82% higher (*P < 0.007*) and maximal acid output 28% higher (*P < 0.003*) than before omeprazole. The degree of increase in maximal acid output was related to both on-treatment pH and on-treatment fasting gastrin levels, being 48.0% in subjects with an on-treatment pH of <4 vs. 21.0% in those with a pH of <4 (*P < 0.02*) and 49.2% in subjects with an on-treatment gastrin of >25 ng · L⁻¹ vs. 19.8% in those with a fasting gastrin of <25 ng · L⁻¹ (*P < 0.006*). At day 15 after omeprazole treatment, the *H. pylori*-positive subjects showed a heterogeneous response with some having increased acid output and others persisting suppression.  

**Conclusions:** Rebound acid hypersecretion occurs in *H. pylori*-negative subjects after omeprazole treatment. Its severity is related to the degree of elevation of pH on treatment. Persisting suppression of acid secretion masks the phenomenon in *H. pylori*-positive subjects.
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