Published in final edited form as:

J Clin Gastroenterol. 2002 January ; 34(1): 62–63. doi:10.1097/00004836-200201000-00012.

The Novel Use of an Intravenous Proton Pump Inhibitor in a Patient With Short Bowel Syndrome

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Abstract

Recent evidence suggests that parenteral proton pump inhibitors (PPIs) can effectively control gastric acid hypersecretion. Intravenous PPI (omeprazole) can substantially reduce the risk of recurrent bleeding in patients with peptic ulcer disease. We describe a patient with short bowel syndrome who had recurrent life-threatening upper gastrointestinal bleeding from severe gastric and esophageal ulcerations. The patient had failed long-term, maximal-dose intravenous ranitidine therapy but was successfully treated and maintained on long-term therapy with an intravenous PPI (pantoprazole). To our knowledge, this is the first case report in the literature describing the use of an intravenous PPI to treat upper gastrointestinal bleeding in a patient with complete intestinal resection. Intravenous PPIs should be considered as the first line of treatment of erosive esophagitis and peptic ulcer disease in patients with short bowel syndrome and in patients who are nil per os and who fail intravenous H₂-receptor antagonist treatment. Parenteral PPI may also be the drug of choice in intensive care patients who have erosive esophagitis. Furthermore, this is the first case report describing the novel use of intravenous pantoprazole to treat erosive esophagitis in a patient with short bowel syndrome, suggesting that intravenous PPI may also be useful for the treatment of ulcer prophylaxis in patients undergoing intestinal transplantation.

Keywords

Intravenous proton pump inhibitor; Pantoprazole; Short bowel syndrome

Recent evidence suggests that parenteral proton pump inhibitors (PPIs) can effectively control gastric acid hypersecretion in pentagastrin-induced healthy subjects¹ and in patients with Zollinger–Ellison syndrome.² Intravenous PPI (omeprazole) can substantially reduce the risk of recurrent bleeding in patients with peptic ulcer disease.³ This case report describes a 54-year-old man with short bowel syndrome with recurrent life-threatening upper gastrointestinal bleeding from severe gastric and esophageal ulcerations who was successfully treated and maintained on long-term therapy with an intravenous PPI (pantoprazole). To our knowledge, this is the first case report in the literature describing the

use of an intravenous PPI to treat upper gastrointestinal bleeding occurring in the setting of a patient with complete intestinal resection.

CASE REPORT

In 1996, the patient presented with aortic dissection complicated by ischemic bowel and multiorgan failure. Management included subtotal resection of the small and large intestine with placement of a duodenostomy tube for upper gastrointestinal drainage. The patient required hemodialysis and total parenteral nutrition for long-term management. After the bowel resection, the patient received intravenous ranitidine 50 mg/d for his recurrent nausea, vomiting, and occasional heartburn, with minimal relief. In September 1998, the patient developed line-related sepsis and massive upper gastrointestinal bleeding with hemodynamic compromise.

Management at that time included transfusion of 5 units packed red blood cells, intensive care unit admission, and tracheal intubation for airway protection. Emergent upper endoscopy (esophagogastroduodenoscopy) revealed a Dieulafoy lesion in the fundus of the stomach, which was successfully treated with epinephrine injection and gold probe cauterization. Additional findings included severe erosive esophagitis (Grade 4, LA classification) with multiple large linear esophageal ulcerations and a small gastric ulcer. The patient was not taking nonsteroidal antiinflammatory drugs, and the dose of ranitidine was limited by his reduced renal clearance and hemodialysis. Failure of H₂-receptor antagonist medications to control mucosal injury was an indication for PPI therapy; however, the patient's subtotal small bowel resection precluded use of oral PPIs as an option for therapy because PPIs in oral formulation (e.g., lansoprazole) require absorption in the small bowel. The intravenous ranitidine dosage was increased to 50 mg every 8 hours, which was the recommended maximal dosage in patient with renal failure.

Despite this dosage increase, the patient continued to have nausea, vomiting, and occasional coffee ground emesis. He required multiple intensive care unit admissions and regular blood transfusions (2–4 units of packed red blood cells each month) to maintain a hemoglobin level of more than 9 to 10 g/dL. A repeated emergent esophagogastroduodenoscopy found persistent severe erosive esophagitis with multiple clean-based ulcers in the distal esophagus. No other possible bleeding source was localized, and hemostasis was not performed, as no stigmata for early recurrent bleeding were identified. In November and December 1999, the patient had two major episodes of massive upper gastrointestinal bleeding that required 4 to 5 units of packed red blood cells transfusions during each intensive care unit admission. The presentation in both instances included bright red blood draining from the duodenostomy and concomitant hypotension. An emergent esophagogastroduodenoscopy demonstrated severe erosive esophagitis with multiple clean based ulcerations, which was unchanged from the prior examinations. Subsequently, the intravenous ranitidine dosage was increased to 150 mg every 8 hours, without resolution of the bleeding.

Further increases in the ranitidine dosage were not considered given the history of end-stage renal disease and the poor overall response to therapy (the patient had already failed long-

term, maximal-dose intravenous ranitidine therapy at dosages of 150–350 mg/d for 4 months). Consequently, compassionate-use approval was obtained from the Food and Drug Administration for the use of intravenous pantoprazole in the treatment of this patient (40 mg/d).

Pantoprazole therapy was initiated in January 2000, and the patient's nausea, vomiting, and heartburn symptoms subsided completely within 3 days. Follow up over the subsequent 9 months (to date) has demonstrated no evidence of recurrent gastrointestinal bleeding. Analysis of gastric basal acid output (0.237 mEq/h) confirmed satisfactory suppression of acid production. Endoscopy performed 3 months after the start of pantoprazole treatment demonstrated complete resolution of the esophageal and gastric ulcerations, as well as no evidence of erosive esophagitis (Grade 0, LA classification). The patient's hemoglobin level has been stable, without the need for further transfusion. The patient tolerated intravenous pantoprazole very well, with no reported side effects. There were no major changes in serial liver function tests and complete blood count. Although no formal ophthalmologic examinations were performed, the patient reported no changes in either visual acuity or visual fields. We anticipate maintaining the patient on life-long therapy with intravenous pantoprazole to prevent the recurrence of gastrointestinal hemorrhage.

DISCUSSION

To our knowledge, this is the first case report in the literature to describe the chronic use (>1 month) of an intravenous PPI and to document complete resolution of erosive esophagitis and peptic ulcer disease in a patient with short bowel syndrome. On reviewing the literature, there has been no reported incidence or prevalence data and no treatment strategy of erosive esophagitis and peptic ulceration in patients with short bowel syndrome.

Current data suggests that prolonged gastric acid suppression with oral PPIs therapy is safe and rarely produces adverse events.^{4–6} One recent prospective longitudinal study found long-term oral omeprazole therapy (11 years) is highly effective and safe for control of reflux esophagitis.⁶ On annual endoscopic and histologic examination of the gastric corpus, there was no increased incidence of dysplasia or neoplasm. In a recent large double-blind trial, after initial endoscopic hemostasis, patients with bleeding peptic ulcer were randomized to either high-dose intravenous omeprazole infusion (8 mg/h for 3 days) or placebo. The risk of recurrent bleeding at 30 days after endoscopy was significantly reduced the omeprazole-treated group (6.7%, $p < 0.001$) compared with that of the placebo group (22.5%).³

This case is particularly relevant with the recent release of the first intravenous PPI (pantoprazole) in the United States. Pantoprazole for intravenous administration is distributed in vials containing the equivalent of 40 mg of pantoprazole as a lyophilized power. For intravenous administration, the powder is reconstituted with 10 mL of normal saline and administered over a 15-minute interval. Our patient received only the standard dose of intravenous pantoprazole and had immediate relief of symptoms, with subsequent complete healing of his erosive esophagitis and peptic ulcer disease. Our experience with intravenous PPI in this patient with short bowel syndrome and end-stage renal disease

provided additional evidence that the intravenous PPI is highly effective and safe in patients who cannot tolerate H₂-receptor antagonists or oral PPI treatment. Intravenous PPIs should be considered as the first line of treatment of erosive esophagitis and peptic ulcer disease in patients with short bowel syndrome and in patients who are nil per os and who failed intravenous H₂-receptor antagonists treatment. Parenteral PPI may also be the drug of choice in intensive care unit patients who have erosive esophagitis. Furthermore, to our knowledge, this is the first case report describing the novel use of intravenous pantoprazole to treat erosive esophagitis in a patient with short bowel syndrome, suggesting that it may also be useful for ulcer prophylaxis in patients undergoing intestinal transplantation.

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