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## Thrombocytopenia as a side effect of pantoprazole

*Pantoprozolün yan etkisi olarak trombositopeni*

To the Editor,

Proton-pump inhibitors (PPIs) are prescribed for treatment of peptic ulcer disease, gastroesophageal reflux disease, acute gastrointestinal bleeding, Zollinger–Ellison syndrome, and *Helicobacter pylori* eradication. These agents are generally well tolerated, with few commonly reported adverse effects (1). A rarely reported adverse effect consists of thrombocytopenia associated with the use of PPIs (2-5). We describe a case in which the patient developed thrombocytopenia while taking intravenous pantoprazole.

A 45-year-old man was admitted to the emergency room with upper gastrointestinal hemorrhage. The patient had a past medical history of a duodenal ulcer. The patient had taken nonsteroidal anti-inflammatory drugs. Upper gastrointestinal endoscopy had revealed a duodenal bulb deformity with a deep, penetrating ulcer over the anterior wall five months ago. Laboratory tests indicated hemoglobin 11 g/dl (13–15 g/dl), total leucocyte count 12,000 cells/cc (4,000–11,000 cells/cc) and platelet count 350x10<sup>3</sup>/cc. Prothrombin time was normal. He was resuscitated with intravenous fluids and started on intravenous pantoprazole, given as a bolus intravenous injection of 80 mg followed by an

infusion of 8 mg per hour for 72 hours. After resuscitation he underwent an upper gastrointestinal endoscopy, which showed a normal esophagus and stomach, and a non-bleeding, deep, duodenal ulcer approximately 1 cm in diameter on the anterior wall of the duodenal bulb. He was kept nil by mouth and given parenteral crystalloids and intravenous pantoprazole on hospital day 3. The hemoglobin level remained stable during the hospitalization. During the hospital course, the patient did not receive any blood products or fresh frozen plasma transfusions. He was then started on oral feeds on hospital day 4. The patient's platelet count decreased daily during the hospital course for 3 days, until it reached a nadir of 70 x 10<sup>3</sup>/cc. Peripheral smear showed a reduced platelet count with normal morphology. Pantoprazole was discontinued on hospital day 4, and by days 6 and 8, the platelet count had risen to 100x 10<sup>3</sup>/cc and 220 x 10<sup>3</sup>/cc, respectively. Pantoprazole was switched to oral rabeprazole on hospital day 8. The patient's platelet count remained stable during the hospitalization. The patient was given rabeprazole 40 mg/day, amoxicillin 2000 mg/day, and levofloxacin 500 mg/day for 2 weeks for treatment of *Helicobacter*

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**Table 1.** Reported cases of PPI induced thrombocytopenia

Cases (References)	Age	Sex	PPI
1. Zlabek JA, et al. 2002 (4)	85	M	Lansoprazole
2. Hayashibara T. 1998 (8)	80	F	Omeprazole
3. Rudelli A, et al. 1993 (9)	47	M	Omeprazole
4. Watson TD, et al. 2006 (2)	62	F	Pantoprazole
5. Watson TD, et al. 2006 (2)	42	M	Pantoprazole
6. Miller JL, et al. 2009 (3)	infant	F	Pantoprazole
7. Ranzino AM, et al. 2010 (5)	55	M	Esomeprazole
8. Present study	45	M	Pantoprazole

PPI: Proton-pump inhibitors, M: Male, F:Female

*pylori* infection. Forty days after eradication therapy, the urea breath test was negative. He was discharged after a total hospital stay of 23 days.

Drug-induced thrombocytopenia is defined as a drop in platelet count below  $100 \times 10^3/\text{cc}$  that resolves with discontinuation of the offending agent, and for which no other causes are identified, as in our case (6). Thrombocytopenia is not a commonly reported adverse effect of proton-pump inhibitors.

PPI-induced thrombocytopenia has been documented with lansoprazole, omeprazole, pantoprazole, and esomeprazole, but a causal relationship with rabeprazole has not been established (Table 1) (2-5,7-9). In these cases, discontinuation of PPIs was shown to improve thrombocytopenia, as in our case.

In conclusion, PPIs should be among the differential diagnosis of thrombocytopenia.

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