Gastrointestinal Bleeding Caused by Complications in a Patient Receiving Warfarin Therapy

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ABSTRACT
A 76-year-old woman receiving warfarin for the prevention of stroke in the context of atrial fibrillation, a prior transient ischemic attack, a known enlarged left atrium, and depressed left ventricular function secondary to an old myocardial infarction presented with hypotension and melena. She was previously quite well, except as noted above. She had started passing bloody stools approximately 1 week earlier. On initial examination, the hemoglobin level was found to be 7.6 g/L and the prothrombin time international normalized ratio (INR) was 8.04. She was diagnosed with gastric ulcer bleeding. Here, we report a case of gastrointestinal bleeding caused by complications in a patient undergoing warfarin therapy.

Key words: • atrial fibrillation • anticoagulation • gastrointestinal bleeding

Introduction
Anticoagulation therapy is effective for the prevention and treatment of both arterial and venous thromboembolism in patients with atrial fibrillation (AF). This therapy produces bleeding complications in an approximately dose-dependent fashion: the risk of bleeding intensifies on increasing doses or using multiple antithrombotic agents. The risk of bleeding varies both with the anticoagulant type and patient characteristics. We report a case of gastrointestinal bleeding caused by complications in a patient undergoing warfarin therapy.

Case
A 76-year-old woman presented to the emergency department after 3~4 instances of passing melena stools. She had been experiencing epigastric discomfort that she found difficult to describe; it was episodic in nature and mild in intensity and there were no provocative or palliative factors. She felt dizzy while standing and fell to the ground, but did not lose consciousness.
She was transported to the hospital by ambulance.

In 2006, she had developed atrial fibrillation and cerebral infarction for which she was being treated with warfarin. Her current medications included low-dose aspirin (100 mg orally once daily), extended-release diltiazem (180 mg orally once daily), valsartan (80 mg orally once daily), and warfarin (3.5 mg orally once daily). In the emergency department, she was found to be diaphoretic with an irregular pulse of 141 bpm and a blood pressure of 80/50 mmHg. Abdominal examination showed no abnormalities, but rectal examination showed melena. Her electrocardiogram showed atrial fibrillation with a heart rate of 141 bpm (Figure 1).

Two intravenous accesses were established for the patient and she received crystalloids and was observed in a monitored setting. Her laboratory tests showed a hemoglobin level of 7.6 g/dL (hemoglobin level at 1 month prior to presentation, 13.7 g/dL), a white blood cell count of 9,000/μL, and a platelet count of 151×10^3/μL. Her prothrombin time international normalized ratio (INR) was 8.04 and her urea level was 59 mg/dL; the electrolyte, creatinine, and liver enzyme levels were normal.

She was transfused with 2 units of packed red blood cells, given 5 mg of vitamin K subcutaneously and was transfused with 1,000 U of prothrombin complex concentrate (a mixture of clotting factors II, VII, IX, and X, and proteins C and S). After 12 hours, she was hemodynamically stable and underwent a gastroduodenoscopy under conscious sedation. Several variably sized gastric ulcers were noted at the gastric antrum and pylorus (Figure 2). Her hemoglobin level increased to 10.0 g/dL and her INR was 1.4.

She was hospitalized for 7 days, during which she received intravenous proton pump inhibitor (PPI) therapy for 72 hours; thereafter, oral PPI therapy was initiated. In addition, warfarin therapy without aspirin was resumed.

The patient began eating 24 hours after the endoscopy and was discharged after 7 days.

**Discussion**

This patient had several risk factors for stroke and the use of warfarin for long-term primary prophylaxis of stroke would be strongly recommended by consensus guidelines. However, the patient showed evidence of acute bleeding (in the form of melena). It is probable that a gastrointestinal ulcer or malignancy was the cause of her presentation.

The risk for thromboembolic complications and bleeding in AF varies in different medical conditions.¹ The CHADS₂ score is a simple validated

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**Figure 1.** Electrocardiogram showed atrial fibrillation with rapid ventricular rhythm
measure of risk. Not much data is available on survival and bleeding rates in relation to the CHADS2 risk score. Thus, the threshold at which the benefit of oral anticoagulation for the prevention of stroke in AF exceeds the risk for bleeding is unclear. There are several other stroke prediction measures, including the CHA2DS2–VASc score, but they all have only a modest discrimination ability for individual patients and are not very different from the CHADS2 risk score in this respect. International AF guidelines uniformly recommend the use of warfarin for patients with a CHADS2 score of 2 or higher. Warfarin is largely underused because of concerns over the need for systematic monitoring and the risk of bleeding associated with its use. There is consequently a need for new agents that can function as alternatives to warfarin for long-term use in AF. Dabigatran and rivaroxaban have been recently approved by the United States Food and Drug Administration for stroke prevention in patients with AF. Recent meta-analysis demonstrated that the new oral anticoagulants lower the risk for intracranial bleeding and may decrease the overall risk for major bleeding events in patients with AF. Increasing CHADS2 scores are associated with increased risk for stroke or systemic embolism, major and intracranial bleeding, and death in patients with AF who are treated with warfarin. With respect to major bleeding complications, new oral agents may be considered as alternatives in patients with AF.

References


