Management of Antiplatelet Agents and Anticoagulants in Patients with Gastrointestinal Bleeding

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KEYWORDS

- Gastrointestinal hemorrhage • Upper gastrointestinal bleeding
- Lower gastrointestinal bleeding • Adverse effects • Thienopyridine
- Novel oral anticoagulant

KEY POINTS

- Antithrombotic drugs are associated with a clinically significant risk of gastrointestinal bleeding.
- An important consideration is if endoscopic hemostasis (in itself) constitutes a high vs. low-risk procedure.
- A better understanding of the pharmacology, mechanism of action and clinical indications for common antiplatelet drugs is imperative for sound decision-making regarding drug cessation or continuation in the peri-endoscopic period.
- Management of anticoagulant associated bleeding in the emergent and urgent setting is still grounded in the principles of A (airway), B (breathing), and C (circulation).
- There is remarkably little data to inform the endoscopist's decision of resumption of antithrombotic therapy.

INTRODUCTION

Current estimates of antithrombotic use in the United States are limited. The Reduction of Atherothrombosis for Continued Health (REACH) registry suggests that 70% of Americans (n = 25,686) are on acetylsalicylic acid (ASA) monotherapy; 13% are on ASA with a thienopyridine antiplatelet agent (ie, dual antiplatelet therapy [DAPT]), 8% are on anticoagulant or thienopyridine antiplatelet agent monotherapy, 4% are on ASA plus anticoagulant, and 1% are on thienopyridine agent plus anticoagulant or on all 3 antithrombotic agents

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concurrently.[1] Data from the Department of Veterans' Affairs (n = 78,133) show that 50.5% are on DAPT, 29.3% are on ASA plus anticoagulant, 13.8% are on anticoagulant plus thienopyridine antiplatelet agent, and 6.3% are on triple therapy with ASA plus anticoagulant plus thienopyridine agent.[2] It is projected that, by 2030, greater than 40% of US adults (>25 million individuals) will have at least 1 form of cardiovascular disease, accompanied by an expected aggressive increase in antithrombotic drug use for prevention of myocardial infarction (MI), stroke (cardiovascular accident [CVA]), and thromboembolic disorders (deep venous thromboembolism or pulmonary embolism) in patients who have already had a prior event (ie, for secondary cardioprophylaxis).[3]

These drugs are associated with an important and clinically relevant gastrointestinal bleeding (GIB) risk. Abraham and colleagues[2] showed the magnitude of risk associated with the use of antithrombotic drugs used in dual and triple combinations. The 1-year number needed to harm for common dual therapy strategies (ASA plus thienopyridine agent, ASA plus anticoagulant, or anticoagulant plus thienopyridine agent) as well as triple therapy (ASA plus thienopyridine agent plus anticoagulant) is less than 93 patients to incur 1 additional upper gastrointestinal (GI) bleed, less than 23 to incur 1 additional lower GI bleed, less than 51 to incur 1 additional blood transfusion, and less than 67 patients to incur 1 additional GI bleed–related hospitalization.

These estimates may represent just the "tip of the iceberg" because they fail to include the impact of GIB associated with the new oral anticoagulants, which are known to increase the risk of GIB 3-fold when combined with ASA and a thienopyridine agent.[4] Furthermore, with the aging US population, GIB is likely to increase because of the presence of multiple concomitant risk factors in this population: (1) advancing age, (2) multiple medical comorbidities, and (3) increased use of antiplatelet and anticoagulant agents in combination.[5] The synergism of these risk factors is likely to change the epidemiology of GIB in North America.[3]

This article focuses on the management of antithrombotic agents in the periendoscopic period surrounding an acute, clinically significant GIB, requiring endoscopic intervention. These patients include those with hemodynamic compromise, greater than or equal to a 2-g reduction in hemoglobin, or overt signs of GIB (melena, hematemesis, coffee-ground emesis, and hematochezia).

This article addresses the following clinical questions:

1. Is endoscopic hemostasis considered a high-risk or low-risk procedure?
2. How should antiplatelets be managed when the patient is bleeding?
3. How should anticoagulants be managed when the patient is bleeding?
   a. How should the novel oral anticoagulants (NOACs) be managed in the urgent setting?
   b. What are the new target-specific NOAC reversal agents?
4. Should the patient be bridged if stopping anticoagulation?
5. When should antithrombotics be restarted?

Is Endoscopic Hemostasis Considered a High-risk or Low-risk Procedure?

An important consideration is whether endoscopic hemostasis (in itself) constitutes a high-risk versus low-risk procedure. The American Society of Gastrointestinal Endoscopy considers a low-risk procedure to be a procedure that is associated with a clinical rate of bleeding of 1.5% or less, in the absence of antithrombotic therapy.[6] If a procedure with a risk greater than 1.5% is considered high risk, many of the commonly performed hemostatic procedures would be in this category.[7] Some procedures (such as hemostatic clip placement, injection) remain ill-defined in terms of postprocedural bleeding risk (**Table 1**). However, few endoscopic procedures are