Platelet Inhibitors

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KEYWORDS

• Nursing • Acute coronary syndrome • Platelets • Antiplatelet • Thrombosis
• Bleeding

KEY POINTS

- Antithrombotic medications have become standard of care for management of acute coronary syndromes (ACSs).
- Platelet adhesion, activation, and aggregation are essential components of platelet function; platelet-inhibiting medications interfere with these components and reduce incidence of thrombosis.
- Active bleeding is a contraindication for administration of platelet inhibitors.
- There is currently no reversal agent for platelet inhibitors, although platelet transfusion may be used to correct active bleeding after administration of platelet inhibitors.

INTRODUCTION

Platelets are an essential part of a complex protective pathway used by the body to maintain hemostasis. After adhering to the site of vascular injury, platelets activate and secrete substances that attract other platelets, resulting in an aggregation of platelets around the site of injury in an attempt to stop acute blood loss. Under some pathologic conditions, such as atherosclerosis and inflammatory disorders, this process leads to thrombosis and arterial occlusion. Subsequently, a variety of potential complications may result, including life-threatening conditions, such as ACSs.[1] According to recent statistics provided by the American Heart Association, an estimated 15.5 million Americans 20 years of age or older has coronary heart disease.[2] The American Heart Association also projects that, based on available research, 635,000 Americans will have a new coronary event, and another 300,000 will have a recurrent event.[2]

Strategies used to reduce risk of thrombosis in ACSs include antiplatelet agents, such as antithrombin drugs (eg, unfractionated and low-molecular-weight heparin

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and direct thrombin inhibitors), aspirin, thienopyridines, and glycoprotein (Gp)IIb/IIIa inhibitors.[3] Previous trials evaluating the use of oral antiplatelet agents in ACSs have supported the combined administration of thienopyridines and aspirin; current standards include a course of treatment of up to 1 year after an ACS event.[3] This article discusses platelet formation and life cycle, the antiplatelet medications most commonly used in the treatment of ACSs, and guidelines for drug administration and implications for nursing care.

PLATELET FORMATION AND LIFE CYCLE

Functional platelets are developed through the intricate process of thrombopoesis. Thrombopoietin, a Gp hormone produced by sinusoidal and parenchymal cells in the liver and proximal convoluted tubule cells kidneys, is responsible for regulating the growth, number, and differentiation of megakaryocytes in the bone marrow.[4–6] These progenitor cells extend small budlike pseudopodia into the bone marrow sinusoids, releasing anucleate cell fragments that migrate into systemic circulation.[7] Platelets undergo their final maturation processes while circulating in the blood stream, although there are some data to support that the pulmonary system may be involved in this process as well.[8] Once fully mature, the average lifetime of a disk-shaped platelet is approximately 8 to 10 days.[9]

PLATELET FUNCTION

Platelets are commonly associated with their involvement in thrombosis and hemostasis; however, they also plan significant roles in inflammation and immunity.[10] The primary insult triggering a platelet response is vascular injury involving the intimal layer of the blood vessel. After vascular compromise, the stepwise process of platelet adhesion, activation, and aggregation occurs to protect the host from further physiologic consequences. For the purpose of this article, the discussion of pathways involved in these processes is largely limited to those directly associated with pharmacologic platelet inhibitor management.

ADHESION

Vascular injury can stem from multiple sources; atherosclerosis, sheer force, and direct vessel compromise, however, remain the primary mechanisms through which platelets are activated. Damaged vascular endothelium exposes platelets to both activated von Willebrand factor (vWF) and collagen in subendothelium, triggering 2 simultaneous mechanisms that are foundational for platelet adhesion to the site of injury (**Fig. 1**). The first process takes place when activated vWF binds with GpIb-V-IX complex, a receptor on the platelet cell membrane. The second process occurs when exposed collagen from damaged endothelial cells binds with platelet GpVI, a separate receptor on the platelet cell membrane. Collaboratively, these processes work to tether the platelet to the site of vascular injury.[1,11]

ACTIVATION

Once the platelet has been bound to the site of injury, activation of the platelet occurs (**Fig. 2**). The activated platelet releases several mediators, including ADP, thromboxane A_2 (TxA_2), and thrombin. ADP amplifies platelet activation and recruits additional platelets to the site of compromise to achieve hemostasis.[12] Additionally, ADP is instrumental in changing the morphology of the platelet from its disklike shape