Management of Anticoagulation in the Critically Ill Patient

Katy M. Toale, PharmD, BCPS

KEYWORDS

• Anticoagulation • Anticoagulant protocols • Heparin
• Low-molecular-weight heparin • Direct thrombin inhibitors • Bleeding

KEY POINTS

- Anticoagulation is often used in the intensive care unit for indications such as venous thromboembolism treatment and prophylaxis, acute coronary syndrome, atrial fibrillation, and heparin-induced thrombocytopenia.
- The most commonly used anticoagulation agents are heparin and low-molecular-weight heparins.
- Low-molecular-weight heparins have a favorable pharmacokinetic and adverse effect profile and the anticoagulation effect is more predictable; however, heparin is often preferred because of its shorter half-life and ability to be completely reversed in the setting of bleeding or the need for an urgent procedure.
- Direct thrombin inhibitors can be used for patients with heparin-induced thrombocytopenia or potentially as an alternative to traditional therapies in percutaneous coronary interventions.

INTRODUCTION

Anticoagulation is often needed in the critically ill patient for many reasons including prevention and treatment of venous thromboembolism (VTE), acute coronary syndrome (ACS), atrial fibrillation, and heparin-induced thrombocytopenia (HIT). The agent most often used is heparin because of its short half-life and the availability of a reversal agent. Protocols are needed because of the difficulty in dosing and monitoring of these agents, and nurse-driven protocols play an essential role in management.

HEPARIN

For many decades, heparin has been available as an anticoagulant for use in the intensive care unit (ICU). Heparin works by binding to antithrombin, which then inactivates

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Department of Pharmacy, MD Anderson Cancer Center, 1515 Holcombe Boulevard, Unit 377, Houston, TX 77030, USA
E-mail address: khanzelka@mdanderson.org

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primarily thrombin (factor IIa) and factor Xa (Fig. 1). As a result, heparin prevents fibrin formation and inhibits activation of platelets.[1]

Because heparin is a mixture of molecules that vary in length, the anticoagulant effect can vary from patient to patient. Another disadvantage of heparin is that it is highly protein bound, which can make the anticoagulation effects unpredictable. Heparin is a large molecule; therefore, it is unable to bind to clot-bound thrombin (Fig. 2). Clot-bound thrombin maintains the ability to activate the coagulation cascade, which could be detrimental to the patient. Heparin can only inactivate the clotting process; it does not lyse blood clots.[2]

Dosing

Heparin infusions are dosed based on the indication for treatment of VTE, ACS, or VTE prophylaxis, and the recommended dosages are presented in Box 1. Controversies exist on whether heparin should be used in the acute management of ischemic stroke. With the current evidence available, urgent anticoagulation is not recommended for management of acute ischemic stroke.[3] Originally, heparin infusions used standard dosing scales; however, research has shown that weight-based dosing is superior in achieving therapeutic anticoagulation.[4,5] Nurse-driven protocols allow safe and effective titration of heparin to achieve therapeutic anticoagulation in a timelier manner. Examples of dosing protocols used at our institution are presented in Figs. 3 and 4. The half-life of heparin can vary based on the dosage administered. Half-life variability occurs because initial doses of heparin are rapidly cleared through a saturable process. Once these mechanisms are saturated, the elimination of heparin is much slower. As a result, larger bolus doses result in a longer half-life of the medication and could potentially result in supratherapeutic anticoagulation.[2]

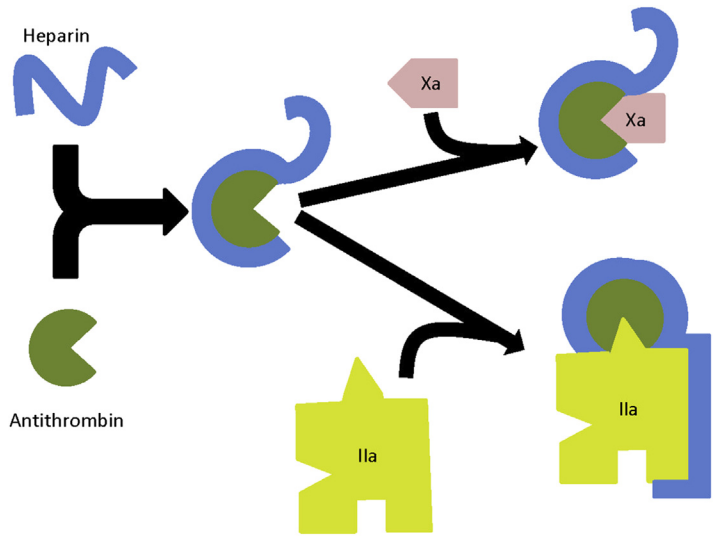

Fig. 1. Heparin mechanism of action. Heparin binds to antithrombin, which subsequently binds to and inactivates thrombin (factor IIa) and Xa. Inhibition of thrombin requires heparin chain lengths of at least 18 saccharide units. LMWHs are generally not large enough to bind thrombin; therefore they have greater activity against factor Xa.