# Optimal Anticoagulant Therapy in ST Elevation Myocardial Infarction Interventions

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**KEYWORDS**

• STEMI • Anticoagulant • Bivalirudin • PCI

**KEY POINTS**

- Optimal anticoagulant therapy combined with an adequate reperfusion therapy in patients with ST elevation myocardial infarction (STEMI) is associated with lower mortality, reduced bleeding episodes, and lower hospitalization costs.
- Bivalirudin is a direct thrombin inhibitor that has proved to be useful in the management of STEMI in patients undergoing percutaneous coronary intervention.
- Bivalirudin is an anticoagulant with favorable pharmacokinetic and numerous pharmacodynamic benefits compared with unfractionated heparin.

## INTRODUCTION

According to the World Health Report, 17.3 million deaths per year were caused by cardiovascular diseases (CVDs) in 2008, representing 30% of global deaths. CVDs represent the largest cause of death and disability in the world. Of these, an estimated 7.3 million (42%) were caused by coronary artery disease and 6.2 million (35.8%) were caused by stroke. By 2030, 23.6 million people are predicted to die from CVDs, leaving coronary artery disease as the leading cause of death.[1]

The pathophysiology of acute coronary syndromes is characterized by disruption of atherosclerotic plaques, activation and aggregation of platelets, and formation of an arterial thrombus (**Fig. 1**A–C).[2] Thrombus formation can produce transient or persistent occlusion. Inhibition of the different pathways of platelet activation might offer the best management conduct. Antithrombotic therapy and mechanical reperfusion have changed the management of acute coronary syndromes by improving the outcome and survival rates of the patients. Anticoagulants prevent thrombus formation at the site of arterial lesion, the guidewire, and in the catheters used for percutaneous coronary intervention (PCI).[3] Anticoagulation combined with antiplatelet therapy is considered the gold

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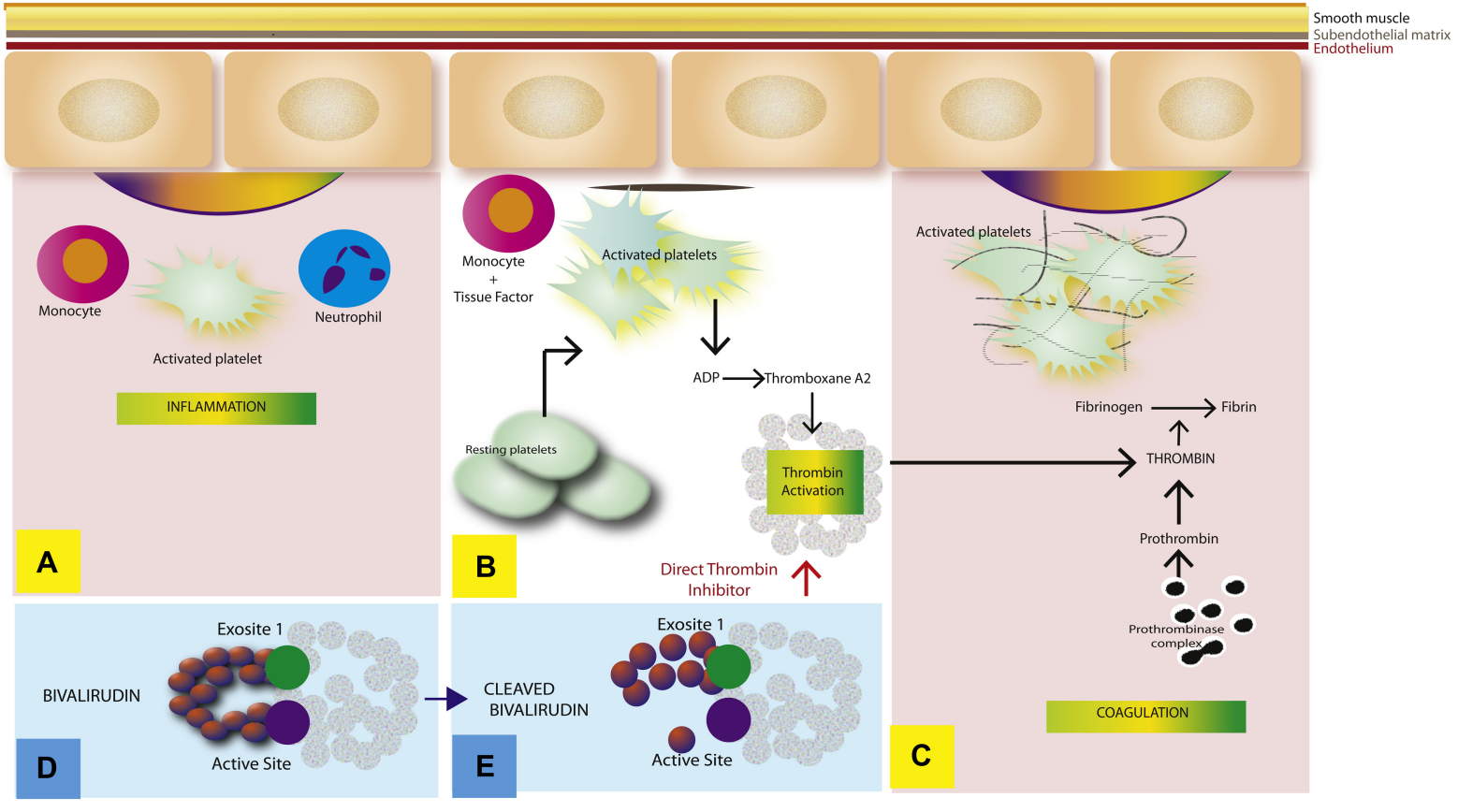


**Fig. 1.** Role of thrombin and bivalirudin. (*A*) Thrombin is involved in inflammation. It activates neutrophils and platelets, releasing cytokines, chemotactic factors, and growth factors. (*B*) Monocytes express tissue factor, triggering the extrinsic pathway of the coagulation cascade, thereby activating platelets. It leads to the activation of thrombin. (*C*) Thrombin is a trypsinlike serine protease produced by the enzymatic cleavage of 2 sites on prothrombin by factor Xa, which is enhanced by binding of the prothrombinase complex (factor Va). Prothrombin is proteolytically cleaved to form thrombin. Thrombin converts soluble fibrinogen into insoluble strands of fibrin (cross-linked framework). (*D*) Bivalirudin directly inhibits thrombin by binding to the active catalytic site and to the anion-binding exosite 1 of circulating and clot-bound thrombin. (*E*) The binding is reversible because thrombin cleaves the bivalirudin-thrombin bond, resulting in the recovery of the thrombin active binding site. ADP, adenosine diphosphate.