Optimal Antiplatelet Therapy in ST-Segment Elevation Myocardial Infarction

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KEYWORDS

• Antiplatelet agents • ST-segment elevation myocardial infarction • STEMI • $P2Y_{12}$ inhibitors • GP IIb/IIIa inhibitors

KEY POINTS

- Antiplatelet therapy remains the cornerstone of treatment of myocardial infarction (MI).
- Advances in antiplatelet agents have resulted in improved outcomes for patients with ST-segment elevation MI (STEMI).
- Guideline recommendations for the use of antiplatelet agents in STEMI are discussed.
- Landmark clinical trials are reviewed.

INTRODUCTION

Cardiovascular disease is the leading cause of death worldwide, with an estimated 1 in every 3 deaths in 2012 attributed to this condition.[1] In the United States, heart disease has remained the leading cause of death since 1935, resulting in approximately 611,105 deaths and 735,000 MIs in 2013.[2] Coronary heart disease death rates in the United States have fallen steadily since 1968, with approximately 21% of this decrease attributed to initial treatments of MI and secondary preventive therapies.[3] Similarly, the case fatality of STEMI continues to decrease.[4–6]

Coronary artery disease is a chronic inflammatory vasculopathy characterized by endothelial dysfunction, smooth muscle cell proliferation, and the development of an atheromatous plaque within the intimal layer of coronary arteries.[7] STEMI most commonly occurs as a result of plaque rupture or endothelial erosion, leading to thrombus formation within the arterial lumen that impedes blood flow.[8] Since the 1960s, platelets have been recognized as playing a central role in the pathogenesis of MI by forming a thrombus and releasing chemical mediators (serotonin, ADP, AMP, collagen, and thrombin) that perpetuate the hemostatic cascade.[9] For this reason, antiplatelet therapy remains the cornerstone in the medical management of acute coronary syndromes (ACSs), particularly in patients presenting with STEMI and undergoing percutaneous coronary intervention (PCI).

Current American College of Cardiology Foundation (ACCF)/American Heart Association (AHA) guidelines present 3 different classes of antiplatelet agents that are approved for use in patients with STEMI (Table 1): cyclooxygenase (COX) inhibitors (aspirin), $P2Y_{12}$ receptor antagonists (clopidogrel, prasugrel, and ticagrelor), and glycoprotein (GP) IIb/IIIa inhibitors (abciximab, eptifibatide, and tirofiban). This article provides an overview of these agents as well as the evidence supporting their use in STEMI.

CYCLOOXYGENASE INHIBITOR: ASPIRIN

Arachidonic acid is converted to thromboxane in a chemical reaction catalyzed by the COX enzyme (Fig. 1). Thromboxane is a potent vasoconstrictor and promotes platelet activation and

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Table 1
2013 American College of Chest Physicians/American Heart Association guideline for the management of ST-segment elevation myocardial infarction: antiplatelet therapy to support percutaneous coronary intervention in ST-segment elevation myocardial infarction

	Level of Evidence
Class I	
Aspirin, 162–325 mg, before primary PCI	B
Aspirin, 81–325 mg, daily should be continued indefinitely after PCI	A
A loading dose of a $P1Y_{12}$ inhibitor should be given as early as possible or at time of primary PCI	
Clopidogrel, 600 mg	B
Prasugrel, 60 mg	B
Ticagrelor, 180 mg	B
$P2Y_{12}$ inhibitor therapy, in addition to aspirin, should be given for 1 y to patients who receive a stent (BMS or DES) during primary PCI	
Clopidogrel, 75 mg daily	B
Prasugrel, 10 mg daily	B
Ticagrelor, 90 mg twice a day	B
Class IIa	
It is reasonable to use aspirin, 81 mg daily, over higher doses after primary PCI	B
It is reasonable to use intravenous GP IIb/IIIa receptor antagonist at time of primary PCI in selected patients who are receiving unfractionated heparin	
Abciximab	A
High-bolus-dose tirofiban	B
Double-bolus eptifibatide	B
Class IIb	
It may be reasonable to administer intravenous GP IIb/IIIa receptor antagonist in the precatheterization laboratory setting (ambulance or emergency department) to patients with STEMI for whom primary PCI is intended	B
It may be reasonable to administer intracoronary abciximab to patients with STEMI undergoing primary PCI	B
Continuation of $P2Y_{12}$ inhibitor beyond 1 y may be considered in patients undergoing DES placement	C
Class III	
Prasugrel should not be administered to patients with a history of stroke or TIA	B

Abbreviations: BMS, bare metal stents; DES, drug eluting stents.

Data from O'Gara PT, Kushner FG, Ascheim DD, et al. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation 2013;127(4):e362–425.

aggregation.[10–13] Aspirin irreversibly inhibits the COX enzyme, thereby impairing the platelet hemostatic response to plaque rupture and thrombus formation in a coronary artery.

The efficacy of aspirin in STEMI was initially described in the Second International Study of Infarct Survival (ISIS-2). In this study, 17,187 patients were randomized to streptokinase infusion, aspirin (162 mg for 1 month), both treatments, or neither after the onset of suspected acute MI. Aspirin resulted in 23% reduction in vascular mortality at 5 weeks compared

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