REVIEW ARTICLE

Ticagrelor: A new antiplatelet drug for acute coronary syndromes

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ABSTRACT

Coronary heart disease and acute coronary syndrome (ACS) are a significant cause of morbidity and mortality all over the world. Antiplatelet agents play an essential role in the treatment of acute coronary syndrome (ACS), usually with aspirin and a thienopyridine. Currently, clopidogrel, a second generation thienopyridine, is the main drug of choice, and the combination of aspirin and clopidogrel is administered orally for the treatment of ACS. Clopidogrel, the most commonly used thienopyridine, is limited by a high degree of interpatient variability and inconsistent inhibition of platelets. Ticagrelor, a new, oral, direct-acting P2Y12 receptor antagonist, produces a more profound and consistent antiplatelet effect than clopidogrel. The U.S. Food and Drug Administration approved Ticagrelor on July 20, 2011. Furthermore, ticagrelor has at least one active metabolite, which has pharmacokinetics that are very similar to the parent compound. Therefore, ticagrelor has a more rapid onset and more pronounced platelet inhibition than other antiplatelet agents. The safety and efficacy of ticagrelor compared with clopidogrel, in an ACS patient, has been recently evaluated by the *PLATelet inhibition and patient Outcomes* (PLATO) trial. Clinical studies of patients with both ST-elevation and non-ST-elevation ACS have shown that ticagrelor, when compared with clopidogrel, reduces the rates of vascular death and myocardial infarction. The clinical data currently available indicate that ticagrelor is a promising option for the treatment of patients with ACS and may be of particular use in those at high risk for ischemic events or in those unresponsive to clopidogrel.

Key words: Acute coronary syndrome, adenosine diphosphate receptor, platelet aggregation, ticagrelor

Introduction

The term acute coronary syndrome (ACS) is used to refer to a group of clinical symptoms associated with acute myocardial ischemia.[1] It encompasses unstable angina, non-ST segment elevation myocardial infarction, and ST segment elevation myocardial infarction.[2]ACS is usually the result of an acute or subacute primary reduction of the myocardial oxygen supply, provoked by the disruption of an atherosclerotic plaque, associated with inflammation, thrombosis, vasoconstriction, and microembolization.[1,3]

Each year, in the United States, approximately 1.36 million hospitalizations are required for ACS (listed either as a primary or a secondary discharge diagnosis), of which 0.81 million are for myocardial infarction (MI) and the remainder are for unstable angina (UA). Roughly two-thirds of the patients with MI have Non-ST elevation myocardial infarction (NSTEMI); the rest have ST Elevation Myocardial Infarction (STEMI).[4,5]

A number of factors are directly responsible for the development and progression of endothelial dysfunction and atherosclerosis, including hypertension, age, male gender, tobacco use, diabetes, obesity, elevated plasma homocysteine concentrations, and dyslipidemias.[6,7]

A patient with ACS is generally treated with, (1) anti-ischemic and analgesic therapy (nitrates, morphine sulfate, beta-adrenergic blockers, calcium channel blockers, inhibitors of the renin-angiotensin-aldosterone system, intra-aortic balloon counter pulsation, analgesic therapy), (2) antiplatelet therapy (aspirin, ticlopidine, clopidogrel), (3) anticoagulants (unfractionated heparin, low molecular–weight heparin, long-term anticoagulant-like warfarin), and (4) platelet GP IIb / IIIa receptor antagonists.[8]

Ticagrelor

The combination of aspirin and thienopyridine

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