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Compared with Clopidogrel, Ticagrelor Treatment for St-Segment Elevation Myocardial Infarction Patients

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Introduction

Platelet activation complicates the pathogenesis of Acute Coronary Syndrome (ACS). Functional receptors on the platelet surface, such as P2Y12, aid platelet adhesion and aggregation by altering the rheological properties of the blood. Platelet receptors can also activate a number of pro- and anti-inflammatory chemicals, allowing them to actively modulate immune responses. Subclinical inflammation has long been recognized to be a critical pathophysiological reaction that plays a role in the initiation and progression of serious cardiovascular diseases like ACS.

New-generation antiplatelet medicines differ in their methods of action, specific interference in platelet activation, and potential impact on biological, clinical, and adverse effects. All antiplatelet medicines have the potential to modify platelet receptor function. Following ACS, the combination of two antiplatelet medications with separate mechanisms of action lowers the likelihood of poor outcomes when compared to aspirin immunotherapy. Dual antiplatelet therapy results in a faster and more powerful antiplatelet action, but it also results in more bleeding. However, in most cases, the benefit outweighs the risk. As a result, current clinical guidelines regard prolonged dual antiplatelet therapy as the gold standard of care for patients with ACS, emphasizing the dangers of stopping it too soon.

After an ACS, dual antiplatelet medication has been demonstrated to be effective. Using clopidogrel with aspirin reduces the risk of cardiovascular death, Myocardial Infarction (MI), and stroke, according to the Clopidogrel in Unstable Angina to Prevent Recurrent Events (CURE) trial. The CURE researchers found that taking aspirin plus clopidogrel lowered the chance of a heart attack or stroke. In the multicenter PLATO trial, the combination of aspirin and ticagrelor was found to be helpful in those with ACS. The therapeutic superiority of ticagrelor versus clopidogrel increases after a year. Individuals who used aspirin with ticagrelor for three years had a decreased incidence of cardiovascular mortality, MI, and stroke, according to the PEGASUS study. However, a decreased risk of unfavorable cardiovascular events is linked to a higher risk of severe bleeding. The superiority of ticagrelor over aspirin in reducing recurrent cardiovascular events in patients with MI has been proven in several multicenter trials.

Specific concerns, such as the impact of switching antithrombotic medications on the risk of adverse events, have been identified and will require further research. To quantify the rate of adverse events and assess the switch's safety in terms of bleeding risk, it's critical to examine the effects of switching antiplatelet drugs on platelet aggregation inhibition and pro-inflammatory effects of platelets. As a result, the purpose of this study was to determine how switching from clopidogrel to ticagrelor altered clinical outcomes in ST-segment elevation myocardial infarction patients during their in-hospital stay and one year after discharge.

Myocardial Infarction Patients

Ticagrelor (AZD6140) is the first reversible oral P2Y(12) receptor antagonist to inhibit ADP-induced platelet aggregation. The pharmacological and therapeutic effects of ticagrelor are not dependent on metabolic activity in humans. Ticagrelor has a faster onset and offset of effects than clopidogrel, as well as bigger and more consistent effects. Ticagrelor was found to be superior to clopidogrel in the PLATO research because it reduced platelet aggregation more efficiently (%age of platelet aggregation in the clopidogrel and ticagrelor groups was 44% 15% and 28% 10%, respectively, p0.0001).

The new investigation's findings are consistent with the PLATO findings. Because circulating platelets have a seven-day lifespan, the clopidogrel-blocking platelets will also regenerate in seven days. Because of this, we chose this time period to assess cytokine levels. Patients who remained to take ticagrelor on day 7 after switching antiplatelet medicines had more severe platelet aggregation inhibition than those who continued to take clopidogrel.

In the PLATO research, ticagrelor was found to be superior to clopidogrel in reducing cardiovascular mortality (21%) and MI (16%) in ACS patients. There was a significant reduction in overall mortality in subgroups of MI patients who received both conservative and invasive therapy. On the other hand, the exceptional effects of ticagrelor could scarcely be explained just by its antiplatelet effect.

Since the eighteenth century, there has been an inflammatory theory of atherosclerosis, and the role of inflammation in the development of ACS is a hot issue. This notion is supported by increased levels of inflammatory markers (CRP, IL-6, fibrinogen, etc.) in blood samples of people with cardiovascular diseases. Oh et al. described the deposition of CRP in the ischemia myocardium in a rat acute MI model. In a pig MI model, Guo et al. discovered similar results after thrombolysis. A number of studies have discovered a relationship between CRP and IL-6 levels and prognosis in people who have heart disease.

Increased CRP levels at baseline in healthy guys, according to Ridker et al., suggest the risk of future MI and thromboembolic stroke. According to a new study including 60 patients, the levels of pro-inflammatory markers are considerably higher following CABG in patients with prior MI.

On the other side, pro-inflammatory substances can decrease platelet function, resulting in hyper coagulation. As a result, Bester and Pretorius determined that IL-1?, IL-6, and IL-8 are important factors in platelet hyper activation. According to Thomas et al., P2Y12 inhibitors have a significant impact on inflammatory factors and the prothrombotic pathway. Platelets were discovered to have a crucial role in bacterial endotoxin-induced systemic inflammation. P2Y12

inhibitors were connected to lower mortality in sepsis patients in clinical trials.

Platelet aggregation was significantly reduced in the ticagrelor group. Furthermore, the ticagrelor group exhibited lower CRP and IL-6 levels on day 7 after switching antiplatelet medications than the clopidogrel group. The ticagrelor group exhibited a trend toward fewer endpoints than the clopidogrel group one year after STEMI was identified. The fact that ticagrelor has a higher pro-inflammatory effect than clopidogrel could explain this pattern. As a result, the PLATO trial's lower total mortality in the ticagrelor group could be explained by ticagrelor's ability to reduce platelets involved in the production of pro-inflammatory factors.

Switching from clopidogrel to ticagrelor has no risk of hemorrhagic complications. In the in-hospital period and 1 year after STEMI, there was no significant increase in the rate of bleeding in the ticagrelor group compared to the clopidogrel group. Ticagrelor and clopidogrel both had the same risk of major bleeding (11.6% vs. 11.2%, respectively) and fatal and/or life-threatening hemorrhages, according to PLATO criteria (5.8% in both groups). However, in the PLATO research, the ticagrelor group had a greater rate of major and mild bleeding (16.1%) than the clopidogrel group (14.6%), p=0.0084.