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Pharmacology and Metabolism of Clopidogrel

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Description

Clopidogrel,which was the second largest commerce embedded medicine in the US in 2011 with$8.9 billion in deals with an unrecoverable P2Y12 receptor antagonist designated for reduction of arteriosclerotic actions in cases with recent stroke or MI, and established supplemental arterial complaint. Clopidogrel is a alternate- group for thienopyridine, that has largely replaced ticlopidine, the first- generation thienopyridine with analogous efficacity, due to more tolerability, reduced prevalence of haematological side effects, more rapid-fire onset of action and an accessible (formerly-diurnal) dosing authority In recent times, binary antiplatelet medication with aspirin and P2Y12 receptor antagonists clopidogrel, prasugrel or ticagrelor has come the clinical trophy standard for cases with ACS and/ or witnessing percutaneous coronary interventions (PCI) due to the significant enhancement of long- term clinical outgrowth. Although clopidogrel is safe and effective in numerous cases, there's substantial variability in response between individualities. Some of these cases continue to have cardiovascular events although they undergo clopidogrel treatment. This lack of efficacity has, in part, been attributed to the reduced response to clopidogrel in cases, performing in high on-treatment platelet reactivity (HPR) and the development of atherothrombotic complications.

Metabolism of clopidogrel

This relative non-responsiveness to clopidogrel medicine has been detected “clopidogrel resistance” and is allowed to affect 5 – 44 of cases entering standard- cure clopidogrel treatment. On the other hand, some cases also witness medicine- influenced bleeding due to inordinate platelet inhibition

It's virtually dissolvable in water at neutral pH but freely liable at pH 1. It also dissolves freely in methanol, dissolves sparingly in methylene chloride, and is virtually not dissolvable in ethyl ether. It has a specific optic rotation of about 56°c.

Clopidogrel is a pro drug, one of which metabolites an asset of platelet aggregation. A variety of medicines that inhibit platelet function have been shown to drop morbid actions in people with cardiovascular atherosclerotic problem and with substantiated by stroke or flash ischemic attacks, myocardial infarction, unstable angina or the need for vascular bypass or angioplasty. This indicates that platelets share in the inauguration and/ or elaboration of these events and that inhibiting platelet function can reduce the event rate.

Clopidogrel must be metabolized by CYP450 enzymes to produce the active metabolite that inhibits platelet aggregation. The active metabolite of clopidogrel widely inhibits the list of adenosine diphosphate (ADP) to its platelet P2Y12 receptor and the posterior ADP mediated activation of the glycoprotein GPIIb/ IIIa complex, thereby inhibiting platelet aggregation.

The active metabolite of clopidogrel selectively inhibits the binding of adenosine diphosphate (ADP) to its platelet P2Y12 receptor and the subsequent ADP mediated activation of the glycoprotein GPIIb/ IIIa complex, thereby inhibiting platelet aggregation. Amplification is a process of platelet activation by released ADP. Because the active metabolite is formed by CYP450 enzymes, some of which are polymorphic or lead to inhibition by other drugs, not all patients will have adequate platelet inhibition. Inhibition of platelet aggregation can be seen 2 hours after single oral doses of Plavix based on dose. Repeated doses of 75 mg Plavix per day inhibit ADP-induced platelet aggregation on the first day, and inhibition reaches steady state between Day 3 and Day 7. At steady state, the average inhibition level observed with a dose of 75 mg Plavix per day was between 40% and 60%.

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