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Pharmacogenetics of P2Y12 Receptor Inhibitors

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Abstract : For cardiovascular illness, oral P2Y12 inhibitors including clopidogrel, prasugrel, and ticagrelor are frequently recommended. Each of these medications has advantages and disadvantages. In the absence of genotyping, it has been demonstrated that the stronger platelet aggregation inhibitors prasugrel and ticagrelor are superior than clopidogrel at preventing significant adverse cardiovascular events following an acute coronary syndrome and percutaneous coronary intervention (PCI). Both, nevertheless, come with a higher risk of bleeding unrelated to a coronary artery bypass. As a prodrug, clopidogrel needs to be bioactivated, principally by the CYP2C19 enzyme. A CYP2C19 no function allele and diminished or absent CYP2C19 enzyme activity are present in about 30% of people. The reduced exposure to the active metabolite of clopidogrel and reduced inhibition of platelet aggregation among clopidogrel-treated carriers of a CYP2C19 no function allele likely contributed to the reduced efficacy of clopidogrel in clinical trials. Clopidogrel's pharmacogenetic results are strongest when used in conjunction with PCI, but evidence for other indications is growing. One of the most typical examples of clinical pharmacogenetic application is CYP2C19 genotype-guided antiplatelet medication following PCI. Guidance is available from expert consensus groups and regulatory bodies to assist with incorporating genetic information into P2Y12 inhibitor prescribing decisions. Here, we examine the data supporting genotype-guided P2Y12 inhibitor selection's effects on clopidogrel response and outcomes and discuss tips for pharmacogenetic implementation. We also discuss procedures for using genotype data to choose P2Y12 inhibitor therapies as well as any unmet research needs. Finally, choosing a P2Y12 inhibitor medication that optimally balances the atherothrombotic and bleeding risks may be influenced by both clinical and genetic factors.

Keywords : inhibitors, cardiovascular events, coronary intervention, pharmacogenetic implementation
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