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Selection of Antiplatelet Drugs in Dental Procedures in Myocardial Patients who Underwent Percutaneous Transluminal Coronary Angioplasty

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Editorial

Antiplatelet drugs in dental procedures are controversial as it's associated with bleeding. The practice of antiplatelet drugs in cardiac patients varies from patient to patient and clinician to clinician. The lack of guidelines has left clinicians with fewer choices but to manage these cases based on past experience. Cardiovascular and cerebrovascular problems have been most common in the elderly, and over a third of patients are seeking emergency dental treatment for co-morbidity. Furthermore, as per the recent estimation, CVDs are responsible for 17.8 million deaths in 2017 globally, and CAD and stroke were the most common cause among all cardiovascular disease. This mortality rate is 21.1% higher from data presented in 2007 on mortality due to cardiovascular disease. The most common cardiovascular diseases are frequently managed with anticoagulants or antiplatelet medication. Management of these patients provides a challenge for the dentist, which is set to continue with improvement in oral health, access to dentistry, and the ability to provide advanced treatments leading to retention of teeth for

longer [1].

Antiplatelet drugs are the most famed agents to reduce platelet adhesion, activation, and aggregation, which are integral steps in the formation of a thrombus after plaque disruption. The agents are categorized in various groups according to their effects on individual platelet receptors or/and pathways [2].

Aspirin is often prescribed and regularly consumed medication worldwide. Aspirin is a weak acid that is completely and rapidly absorbed in the upper gastrointestinal tract by passive diffusion after oral administration of immediate-release formulations. Aspirin has a permanent or irreversible effect on platelets function as it blocks the activation process of cyclooxygenase (COX-1), a membrane-bound glycoprotein expressed on the endoplasmic reticulum of platelets, including many other cells. Regeneration of COX enzyme is not possible; subsequently, Aspirin intake for these platelets and only newly formed platelets can generate more COX enzyme. It (COX-1) catalyzes the conversion of arachidonic acid to prostaglandin (PG)H2, which is the first step in prostanoid synthesis. Numerous bioactive prostanoids, including thromboxane A2 (TXA2) and PGI2, formed via PGH2. Aspirin results in inhibiting the conversion of arachidonate to thromboxane A2 (TXA2), which is a potent vasoconstrictor and platelet agonist. The inhibition is through the blockage of COX-1 by diffusing into the COX channel within the membrane).The aim of preferential use of low-dose aspirin to prevent cardiovascular and cerebrovascular disease is to protect the vessel wall against thrombosis at the fewer risk of bleeding events and inhibit platelet COX-1, spare endothelial COX with spare the minimal risk of dose-related adverse effects. The use of aspirin in dental procedures is controversial as this is associated with a severe bleeding score. Either abruption of the drug for 24-72 hours is recommended, or a very low dose is recommended if bleeding risk is less and there is no major dental procedure [3].

Clopidogrel (Plavix) is a second-generation thienopyridine derivative. It is chemically very similar to ticlopidine (both are prodrugs) and belongs to the ADP receptor/ P2Y12 inhibitors class. Clopidogrel is used as a potent antiplatelet agent and shows efficacy in preventing thrombotic events (MI, stroke, and vascular death) in high-risk patients. Clopidogrel is not active *in vivo*, requires oxidation via the cytochrome P450-3A in the liver to generate the active metabolite. Newly formed active metabolite expresses anti-aggregatory activity. The Clopidogrel Aspirin Stent International Cooperative Study (CLASSICS), showed that clopidogrel had a superior safety and tolerability profile. The results were when compared to ticlopidine following coronary artery stenting. This study shows the benefit of ticlopidine when initiated with a 300mg loading dose and led to the widespread adoption of clopidogrel for patients undergoing coronary stent implantation. Ticagrelor (cyclopentyltriazole pyrimidine molecule) is a novel, faster, highly potent, and reversible P2Y12 receptor inhibitor. Now a day, it is prescribed as a first-line drug for the treatment of ST-segment elevation myocardial infarction (STEMI) and non-ST segment elevation. Also, for all-cause mortality in ACS patients as recommended by both European and American guidelines. Ticagrelor undergoes hepatic biotransformation. It converts to an active metabolite (AR-C124910XX) by de-hydroxymethylation to exert its antiplatelet effect. Two cytochrome P450 enzymes, CYP3A4 and CYP3A5, are responsible for the metabolism of ticagrelor. AR-C124910XX is the primary active metabolite of ticagrelor, and other identified metabolites are clinically insignificant [4]. Suppose the patient is on dual antiplatelet drugs for MI and underwent PTCA recently. In that case, dental

procedures can be postponed as cardiologists do not recommend stopping antiplatelet drugs at least for 3-6 initial months. Later, drug can be stopped for 24-72 hours under strict monitoring where the bleeding risks are less.

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