ADA CE Program ARTICLE 3

ORAL AND MAXILLOFACIAL SURGERY

Antiplatelet therapy and exodontia

Willem H. Schreuder, DMD, MD; Zachary S. Peacock, DMD, MD, FACS

ABSTRACT

Background. This is a review of the management considerations regarding exodontia for patients taking antithrombotic medications that affect platelet function or aggregation.

Methods. The authors reviewed the literature, focusing on the indications and mechanisms of antiplatelet therapy and the perioperative management of patients taking these agents who require exodontia or other dentoalveolar surgery.

Results. Dentists making management decisions regarding patients taking antiplatelet therapy should consider the patient's risk of experiencing perioperative hemorrhage against the risk of experiencing complications associated with thromboembolic events. The risk of perioperative bleeding complications is low for patients taking single or dual antiplatelet therapy. Intraoperative and postoperative bleeding can be controlled with local hemostatic measures.

Conclusion. For patients taking antiplatelet medication, bleeding risk for exodontia is generally lower than the risk of experiencing thromboembolic events owing to cessation of therapy.

Practical Implications. Dentists can safely complete exodontia in patients who continue taking antiplatelet therapy. The dentist should consult the patient's prescribing physician before considering altering the patient's antiplatelet therapy regimen.

Key Words. Tooth extraction; exodontia; antiplatelet; aspirin; NSAID; thienopyridine; clopidogrel; dipyridamole; postoperative bleeding.

JADA 2015:146(11):851-856

http://dx.doi.org/10.1016/j.adaj.2015.04.024

In general, dentists can complete exodontia for patients taking antiplatelet therapy without the occurrence of bleeding sequelae.[1] Local hemostatic measures are effective in limiting intra- and postoperative bleeding with continuation of antiplatelet medications. For each case that involves deciding whether to alter antiplatelet regimens, the dentist must compare the patient's risk of experiencing perioperative hemorrhage with that of a thromboembolic event. Alteration of therapy always requires the dentist to consult with the patient's prescribing physician.

MANAGEMENT CONSIDERATIONS

In this review, we will describe the indications for and the mechanisms of antiplatelet therapy in the context of perioperative management of patients requiring exodontia or other dentoalveolar surgery. When assessing a patient on antiplatelet therapy, the dental practitioner must weigh the patient's risk of experiencing early and late postoperative bleeding against the risk of experiencing thromboembolic events with discontinuation of therapy. Dental practitioners should take note of the following management considerations:

- The dental practitioner must consider the patient's complete medical history and all comorbidities, including the indications for antiplatelet therapy and the time

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since the event or intervention that prompted the patient to start antiplatelet therapy.

- Whenever possible, the dental practitioner should maintain the patient's intake of aspirin and other antiplatelet therapies, including dual antiplatelet therapy.
- The dental practitioner should not interrupt the patient's dual antiplatelet therapy until at least 12 months after placement of a coronary stent. The practitioner should not perform elective procedures requiring alteration of therapy during this time.
- The dental practitioner must consider the extent of the procedure and local factors involved when determining a patient's risk of bleeding. Extraction of multiple teeth with existing inflammation (that is, periodontal disease) may increase the risk of experiencing perioperative bleeding.
- The practitioner can aid local hemostasis at the time of extraction and in the event of postoperative bleeding with the use of sutures, gelatin (for example, Gelfoam [Pfizer]), cellulose (for example, Surgicel [Ethicon]), and application of continuous pressure with gauze. The use of local anesthetic with vasoconstrictor decreases peri- and postoperative blood loss.[2] Caution must be used in patients with a history of cardiac ischemia.[3]
- Special considerations for procedures that are associated with an elevated risk of bleeding (for example, full-mouth extractions in patients taking dual antiplatelet therapy) include completing the extractions in stages, scheduling the procedure early in the day and week to allow time to manage bleeding, or considering the alteration of the patient's antiplatelet therapy.
- The practitioner should address questions regarding antiplatelet therapy with the patient's prescribing physician.

BACKGROUND AND DISCUSSION

An increasing number of anticoagulant medications are being prescribed for patients with cardiovascular comorbidities. Performing invasive dental procedures in patients who are taking anticoagulants requires knowledge of normal hemostasis and the various ways it can be inhibited.

Antithrombotic medications include inhibitors of platelet function and aggregation (for example, aspirin) as well as those that inhibit the coagulation cascade (for example, warfarin). Generally, antiplatelet agents are prescribed for the purpose of primary and secondary prevention of acute ischemic cerebrovascular and cardiovascular events.[4,5] Guidelines also recommend the use of antiplatelet agents for patients with occlusive peripheral vascular disease and to prevent thrombosis after the implantation of bare metal or drug-eluting stents (DES).[6,7]

The risk of intra- and postoperative bleeding is increased in the presence of platelet inhibition.[8] Dentists are confronted routinely with the dilemma of whether to request that patients modify or discontinue the use of these agents before having invasive dental treatment. Dentists always must consider the indication for antiplatelet medications and the patient's underlying medical conditions.

Normal hemostasis. The physiologic mechanism of hemostasis occurs to prevent extravasation of blood in the event of blood vessel injury. The coagulation process is divided into 3 distinct, interrelated phases[9]: primary hemostasis (the formation of a platelet aggregate), secondary hemostasis (the formation of a fibrin clot by activating coagulation factors), and fibrinolysis (the breakdown of the clot after tissue and vascular repair).

Disruption of the endothelial lining of a blood vessel leads to platelet binding (via the von Willebrand factor) to proteins of the subendothelial matrix (that is, platelet adhesion).[10,11] Platelets change in shape as the coagulation process becomes activated further and bind to other platelets via another membrane integrin, glycoprotein (GP) IIb-IIIa during platelet aggregation.[10] Platelet activation results in exocytosis of dense and alpha granules containing adenosine diphosphate (ADP), thromboxane A_2, epinephrine, and thrombin, which promotes further activation and aggregation.[9]

The primary clot consists of a fibrin mesh containing the platelet aggregate, together with entrapped red and white blood cells. The formation of fibrin starts early during platelet activation.[12] The coagulation cascade results from a complex interaction of clotting factors as well as cellular surfaces producing a fibrin clot.[12]

Antiplatelet medications. Increased platelet activity also is implicated in pathologic thrombosis, atherosclerosis, and coronary artery disease. This has led to the development of various classes of antiplatelet medications (Figure).

Acetylsalicylic acid (aspirin) irreversibly inactivates the enzyme cyclo-oxygenase (COX) type 1, through selective acetylation.[13] The enzyme is responsible for the formation of prostaglandins and thromboxane A_2, which are critical to platelet activation and aggregation.[13] Nonsteroidal anti-inflammatory drugs (NSAIDs) reversibly inactivate the COX pathway, with their effect on platelet function dependent on plasma half-life.[14] COX type 2 selective inhibitors (for example, celecoxib) have less effect on primary hemostasis.[15]

Thienopyridines (for example, clopidogrel, ticlopidine, prasugrel) selectively and irreversibly inhibit the platelet ADP receptor ($P2Y_{12}$ receptor).[16] The most common, clopidogrel (Plavix [Bristol-Myers Squibb/Sanofi Pharmaceuticals Partnership]), affects the ADP-dependent activation of the GP IIb-IIIa complex in platelet

ABBREVIATION KEY. **ADP:** Adenosine diphosphate. **COX:** Cyclo-oxygenase. **DES:** Drug-eluting stent. **GP:** Glycoprotein. **NSAID:** Nonsteroidal anti-inflammatory drugs. **TxA_2:** Thromboxane A_2.