Aspirin, Plavix, and Other Antiplatelet Medications
What the Oral and Maxillofacial Surgeon Needs to Know

Andre E. Ghantous, MD[a],
Elie M. Ferneini, DMD, MD, MHS, MBA[b,c,d,*]

KEYWORDS

• Antiplatelet therapy • Aspirin • Bleeding • Myocardial infarction • Clopidogrel
• Coronary artery disease • Cardiac stenting • Angioplasty

KEY POINTS

- Most patients with coronary artery disease and peripheral vascular disease are on long-term antiplatelet therapy and dual therapy.
- Achieving a balance between ischemic and bleeding risk remains a challenge in patients undergoing surgery who are treated with dual antiplatelet therapy.
- For most outpatient oral and maxillofacial surgical procedures, maintenance and continuation of the antiplatelet therapy are recommended.

INTRODUCTION

Long-term antiplatelet therapy is an important component of secondary prevention after a cerebrovascular accident (CVA), transient ischemic attack, myocardial infarction (MI), or myocardial revascularization, or for patients with a diagnosis of peripheral arterial disease (PAD) or acute coronary syndrome (ACS). In fact, dual antiplatelet therapy (eg, aspirin and clopidogrel) is commonly used in surgical patients. This therapy prevents stent thrombosis following percutaneous coronary intervention with placement of bare-metal or drug-eluting stents. In the perioperative period, the indication for antiplatelet agents is reinforced by the increased platelet activity following surgery; however, they also increase the risk of perioperative as well as postoperative surgical bleeding. The oral and maxillofacial surgeon must decide whether the risk of hemorrhage with antiplatelet therapy is lower than the risk of thrombosis when antiplatelet agents are stopped or disrupted.

Antiplatelet agents are categorized according to their mechanism of action: thromboxane/cyclooxygenase (COX) inhibitors, adenosine diphosphate receptor inhibitors, phosphodiesterase inhibitors, adenosine reuptake inhibitors, and glycoprotein IIb/IIIa inhibitors (Fig. 1).

THROMBOXANE/CYCLOOXYGENASE INHIBITOR
Aspirin (Acetylsalicylic Acid)

In 1763, Hippocrates prescribed willow tree bark extract for headaches.[1] Salicylic acid, the active ingredient of aspirin, was isolated from the bark

[a] Division of Cardiology, Department of Medicine, Yale University School of Medicine, 333 Cedar St., New Haven, CT 06510, USA; [b] Private Practice, Greater Waterbury OMS, 435 Highland Avenue, Suite 100, Cheshire, CT 06410, USA; [c] Beau Visage Med Spa, 435 Highland Avenue, Suite 100, Cheshire, CT 06410, USA; [d] Division of Oral and Maxillofacial Surgery, Department of Craniofacial Sciences, University of Connecticut, 263 Farmington Avenue, Farmington, CT 06030, USA
* Corresponding author. Beau Visage Med Spa, 435 Highland Avenue, Suite 100, Cheshire, CT 06410, USA.
E-mail address: eferneini@yahoo.com

Oral Maxillofacial Surg Clin N Am ■ (2016) ■–■
http://dx.doi.org/10.1016/j.coms.2016.06.003
1042-3699/16/$ – see front matter © 2016 Elsevier Inc. All rights reserved.

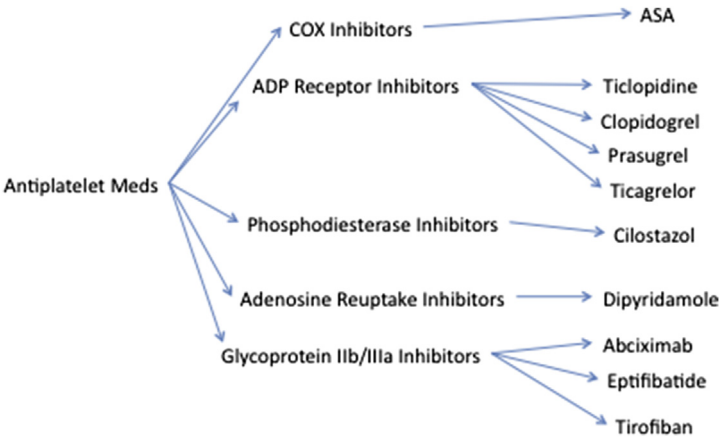

Fig. 1. Antiplatelet agents.

of the willow tree by Edward Stone of Wadham College.[2] Felix Hoffmann, from Bayer, was credited with the synthesis of aspirin in 1897, which is a registered trademark by Bayer with the generic term of acetylsalicylic acid (ASA).[3] John Robert Vane was actually awarded the Noble Prize in 1971 for showing how ASA suppresses the production of prostaglandins and thromboxane.

Low-dose ASA (40–100 mg) irreversibly blocks thromboxane A2 in platelets, leading to an inhibitory effect on platelet aggregation during the life of the affected platelets. This antithrombotic property makes ASA useful for reducing cardiovascular as well as thrombotic events. In addition, ASA is nonselective and irreversibly inhibits both forms of cyclooxygenase isozymes (COX-1 and COX-2). Higher doses of ASA will also inhibit prostaglandin A2 synthesis. The most common side effect of ASA is bleeding, especially if associated with another antiplatelet or anticoagulant agent. Patients who use immediate-release ASA (not enteric coated) and take a single dose of ibuprofen 400 mg should dose the ibuprofen at least 30 minutes or longer after ASA ingestion, or more than 8 hours before ASA ingestion in order to avoid attenuation of ASA's effect.

ASA has been shown to reduce cardiovascular events (MI, CVA, and death) in patients with high cardiovascular risk factors as well as decrease the risk of colorectal and endometrial cancer.[4–6] ASA therapy has net benefits in the acute phase of an evolving MI and should be administered to all patients with an evolving acute MI. In fact, ASA therapy has statistically significant reductions in the risk of vascular mortality (23%), nonfatal reinfarction (49%), and nonfatal stroke (46%). To achieve an immediate clinical antithrombotic effect, an initial minimum loading dose of 162 mg of ASA should be used in an acute MI.[7] In addition, long-term ASA therapy has benefits on risk reduction of subsequent MI, stroke, and vascular death among patients with a wide range of prior manifestations of cardiovascular disease. The most widely tested regimen was a medium dose of ASA (75–325 mg/d). Most studies have shown no evidence that higher doses of ASA are more effective than daily ASA in this dose range.[8]

ADENOSINE DIPHOSPHATE RECEPTOR INHIBITORS

1. Thienopyridine class
   a. Ticlopidine (Ticlid)
   b. Clopidogrel (Plavix)
   c. Prasugrel (Effient)
2. Cyclopentyltriazolopyrimidine (CPTP)
   a. Ticagrelor (Brilinta)

Ticlopidine (Ticlid)

Ticlid, a thienopyridine, is an adenosine diphosphate (ADP) receptor inhibitor discovered in the 1970s and approved in 1978 in Europe for CVA therapy for patients who could not tolerate ASA. Thirteen years later, it was approved in the United States as a stronger antiplatelet agent than ASA to prevent coronary stent thrombosis when taken with ASA.[9] In addition to bleeding, ticlopidine has a serious adverse event, thrombotic thrombocytopenic purpura (TTP). After the approval of clopidogrel in 1997, which is less likely to cause TTP, the use of ticlopidine started declining, and as of April 2015, ticlopidine is no longer available on the US market.[10]

Clopidogrel (Plavix)

Clopidogrel is an oral thienopyridine antiplatelet that irreversibly inhibits the P2Y12 receptor on

Download English Version:

https://daneshyari.com/en/article/5642395

Download Persian Version:

https://daneshyari.com/article/5642395

Daneshyari.com