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Review Article

Safe tooth extraction on aspirin: no pause for the cause

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ABSTRACT:
Tooth extraction in patients with cardiovascular disease presents a significant clinical challenge due to the widespread use of antiplatelet agents such as aspirin and clopidogrel, which may increase the risk of perioperative and postoperative bleeding. Traditionally, dental practitioners have recommended discontinuation of aspirin therapy 7 to 10 days prior to surgical procedures. However, the potential for rebound thromboembolic events, including cerebrovascular accidents and myocardial infarction, poses a serious risk, making the cessation of antiplatelet therapy a clinical dilemma.
This review aims to evaluate the safety and clinical outcomes of dental extractions performed without interruption of low-dose aspirin therapy. Cardiovascular patients undergoing simple tooth extractions were assessed with particular emphasis on intraoperative and postoperative bleeding, wound healing, and the necessity for additional haemostatic interventions. Local haemostasis can be effectively achieved using standard techniques such as pressure application, suturing, and the use of haemostatic agents. Furthermore, adjunctive measures including topical collagen sponges and the local application of tranexamic acid have demonstrated efficacy in controlling bleeding and preventing secondary haemorrhage in patients receiving antiplatelet therapy.
In conclusion, the continuation of antiplatelet therapy during dental extraction is strongly supported, as the potential for life-threatening thromboembolic complications associated with therapy interruption far outweighs the minimal and manageable bleeding risk inherent to routine dental procedures.

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INTRODUCTION

Aspirin, or acetylsalicylic acid, is one of the most widely used and longest-standing drugs in clinical medicine. First introduced by Bayer in 1899, aspirin has maintained its central role in modern therapeutics for over a century. Derived from salicylic acid, originally extracted from willow bark, aspirin was synthesized to reduce gastric irritation while retaining its therapeutic properties. Since then, it has progressed from a simple analgesic and antipyretic to a cornerstone of cardiovascular and cerebrovascular disease prevention, owing to its unique antiplatelet action.

Pharmacologically, aspirin belongs to the group of non-steroidal anti-inflammatory drugs (NSAIDs). Its mechanism of action is distinct from other agents because it irreversibly acetylates cyclooxygenase (COX) enzymes, primarily COX-1 in platelets, thereby inhibiting the synthesis of thromboxane A_2. This results in a significant reduction in platelet aggregation and thrombus formation. Importantly, the inhibition is irreversible and persists for the lifespan of the platelet (7–10 days). This unique property explains aspirin's efficacy as a long-term antithrombotic agent and distinguishes it from the remaining NSAIDs. While aspirin also inhibits prostaglandin synthesis, conferring analgesic, antipyretic, and anti-inflammatory properties at higher doses, its most important clinical role today lies in its low-dose antiplatelet effect.

Currently, aspirin is predominantly prescribed in low doses (75–150 mg/day) for cardiovascular and cerebrovascular protection. It is well established in the secondary prevention of myocardial infarction, ischemic stroke, transient ischemic attacks, and in patients with coronary stents or post-coronary artery bypass grafting. Aspirin may also be used in specific conditions such as preeclampsia prevention in pregnancy and, less commonly today, for analgesic or anti-inflammatory purposes at higher doses. Consequently, many dental patients, especially older individuals and those with cardiovascular disease, are prescribed long-term aspirin therapy.

This widespread use presents a frequent clinical dilemma in dentistry especially inoral surgery. The primary concern is whether aspirin should be continued or discontinued prior to invasive dental procedures, such as tooth extraction. On one hand, aspirin's antiplatelet effect increases the risk of perioperative and postoperative bleeding, which may be challenging to control in some cases. On the other hand, discontinuing aspirin, even temporarily, carries a substantial risk of thrombotic complications, including myocardial infarction, ischemic stroke, or stent thrombosis. These risks can be catastrophic and often outweigh the inconvenience of controlled local bleeding.

From a dental perspective, several studies[1,2,4]have shown that bleeding associated with aspirin use during tooth extraction is usually mild to moderate and can be adequately controlled using local measures such as suturing, pressure packs, local haemostatic agents (oxidized cellulose, gelatine sponge, fibrin glue), and tranexamic acid mouthwash. The evidence strongly supports the position that the benefits of continuing aspirin therapy far outweigh the risks of discontinuation, particularly given the serious complications of aspirin withdrawal.[2]

Ardekian et al. conducted a prospective study to evaluate the risk of bleeding after tooth extraction with the use of aspirin 100 mg/day. Suturing of the extraction socket and pressure pack had been used to achieve haemostasis in all the patients

Given these considerations, it is crucial for oral health professionals to have a clear understanding of aspirin's pharmacology, its clinical indications, and the implications of withdrawal. A structured approach to patient evaluation, risk assessment, and intraoperative and postoperative bleeding management can ensure safe dental care without compromising the patient's systemic health.

The aim of this review is to provide a comprehensive overview of safe tooth extraction in patients receiving aspirin therapy, with emphasis on the clinical relevance of continuing versus discontinuing aspirin, the phenomenon of aspirin withdrawal syndrome, and practical strategies for dental practitioners to balance bleeding risk with systemic thrombotic risk.

Aspirin Withdrawal

When aspirin therapy is discontinued, newly formed platelets gradually restore full cyclooxygenase-1 (COX-1) activity, resulting in increased thromboxane Aproduction, enhanced platelet aggregation, and the establishment of a transient prothrombotic state [1,2]. This rebound effect generally begins within 48–72 hours of withdrawal, with the risk peaking between 7 and 14 days [3]. Clinically, aspirin discontinuation has been strongly associated with serious thrombotic complications, including acute myocardial infarction, ischemic stroke, and stent thrombosis. Evidence indicates that the risk of major vascular events rises approximately three- to five-fold following withdrawal, underscoring the importance of maintaining therapy in at-risk patients.

Aspirin withdrawal syndrome

Aspirin withdrawal syndrome" (AWS)—a paradoxical increase in thrombotic events after discontinuation of chronic aspirin therapy. This phenomenon has significant clinical implications, particularly in patients with a high baseline cardiovascular risk.

Pathophysiology

The pathogenesis of aspirin withdrawal syndrome is multifactorial and involves a rebound prothrombotic state:

1. Rebound Platelet Hyperactivity:

Aspirin irreversibly inhibits thromboxane A_2 (TXA_2) synthesis. After discontinuation, newly formed platelets—unexposed to aspirin—rapidly restores TXA_2 production.

This rebound effect may overshoot baseline levels, leading to heightened platelet aggregation.

2. Endothelial Dysfunction:

Withdrawal of aspirin removes its anti-inflammatory and Vaso protective effects.

This results in a prothrombotic vascular environment.

Neurohormonal andHemodynamic Effects: Discontinuation of aspirin triggers sympathetic nervous system activation, which leads to increased catecholamine release. This results in enhanced vasoconstriction, elevated heart rate, and higher blood pressure, all of which increase shear stress on vascular endothelium. The resulting hemodynamic instability creates a prothrombotic milieu by promoting platelet activation, reducing blood flow at vulnerable sites, and encouraging thrombus formation on pre-existing atherosclerotic plaques. Together, these neurohormonal and hemodynamic changes amplify the rebound prothrombotic effect observed after aspirin withdrawal.

Patients experiencing aspirin withdrawal syndrome (AWS) may develop serious thrombotic complications. The most common clinical manifestations include acute coronary syndromes such as unstable angina and myocardial infarction, ischemic stroke or transient ischemic attack, stent thrombosis in post-percutaneous coronary intervention (PCI) patients, and in severe cases, sudden cardiac death.

The risk of such events is significantly higher in individuals with underlying cardiovascular and cerebrovascular disease. Those with a recent myocardial infarction, coronary stent implantation, cerebrovascular disease, or peripheral arterial disease are particularly susceptible. Additional comorbidities such as diabetes mellitus, hypertension, and advanced age further compound the vulnerability to life-threatening events after aspirin discontinuation.

Clinical Implications

The recognition of aspirin withdrawal syndrome has direct consequences for perioperative and dental practice:

- Continuation vs Discontinuation:

Most guidelines recommend continuing low-dose aspirin during minor surgical and dental procedures, as the bleeding risk is minimal and manageable, while the thrombotic risk from discontinuation is substantial.[7]

- If Discontinuation is Inevitable:
  - The interruption should be as short as possible (ideally < 5 days).
  - Aspirin should be resumed promptly after surgery.
  - Bridging strategies are generally not recommended for aspirin.
- Patient Counselling:

Patients on long-term aspirin must be educated never to stop therapy without medical supervision.

Evidence on Bleeding Risk in Dental Extractions

Clinical trials [4]have shown that patients on low-dose aspirin experience only a slight increase in bleeding time, which can be effectively managed with local haemostatic measures. Post-extraction bleeding is typically mild, self-limiting, and controllable without systemic complications. No evidence supports aspirin discontinuation for routine dental procedures. [5]

"Aspirin Continuation Recommended for Dental Surgery"

The American Dental Association (ADA) notes that most dental treatments do not require altering antiplatelet therapy, including aspirin, and any bleeding can be effectively controlled with local haemostatic measures. Similarly, the Scottish Dental Clinical Effectiveness Programme (SDCEP, 2nd edition, 2022) [5]advises continuing aspirin for outpatient dental care, with treatment planned to incorporate appropriate local haemostatic techniques. These recommendations emphasize that the risk of thrombotic events from discontinuing aspirin far outweighs the manageable risk of minor procedural bleeding.

NEWER STUDIES ON BLEEDING RISK WITH ASPIRIN

2023 systematic review & meta-analysis (Journal of Stomatology, Oral and Maxillofacial Surgery):

No significant increase in immediate or delayed bleeding with aspirin monotherapy vs healthy controls after extractions (RR 1.26, p=0.5; RR 2.17, p=0.09). Higher bleeding seen with dual antiplatelet therapy (DAPT). [6]

2023 narrative/clinical reviews:

Dental procedures can generally proceed without altering antiplatelets; local measures (suturing, cellulose/gelatine sponges, tranexamic mouthwash) are effective. [8]

Oral Surgery Considerations

Preoperative**:** Assess medical history, consult physician if dual therapy.

Intraoperative: Use atraumatic extraction techniques, minimize soft tissue trauma, achieve meticulous haemostasis. Local Measures: Oxidized cellulose, collagen sponge, fibrin sealants, tranexamic acid rinse, gauze pressure.

Postoperative:

Provide written instructions, avoid spitting/rinsing first 24h, cold packs, elevate head, report persistent bleeding 2–3 hours despite applying firm pressure

Comparison: Continuing vs Discontinuing Aspirin

Aspects	Continuing aspirin	Discontinuing aspirin
Bleeding risk	Slightly increased	Reduced bleeding risk but not eliminated
Systemic risk	Minimal systemic risk, patient remains protected against thrombotic events.	High systemic risk, associated with aspirin withdrawal syndrome, rebound platelet hyperactivity, increased chance of myocardial infarction, stroke, or stent thrombosis
Mortality/morbidity	Very low, safe when managed with proper local measures	Potentially high Serious complication, including fatal cardiovascular events.
Guideline recommendations	Recommend continuing aspirin for routine extraction and minor oral surgery.	Generally, not recommend except in very risk bleedingprocedures.

Clinical practicality	when aspirin therapy is continued, bleeding is usually mild, easily controlled with local measures, and patient safety is prioritized.	When aspirin therapy is discontinued, it may avoid minor bleeding inconveniences but exposes the patient to life-threatening systemic thromboembolic events.

Haemorrhage can usually be controlled with the aid of local haemostatic methods.

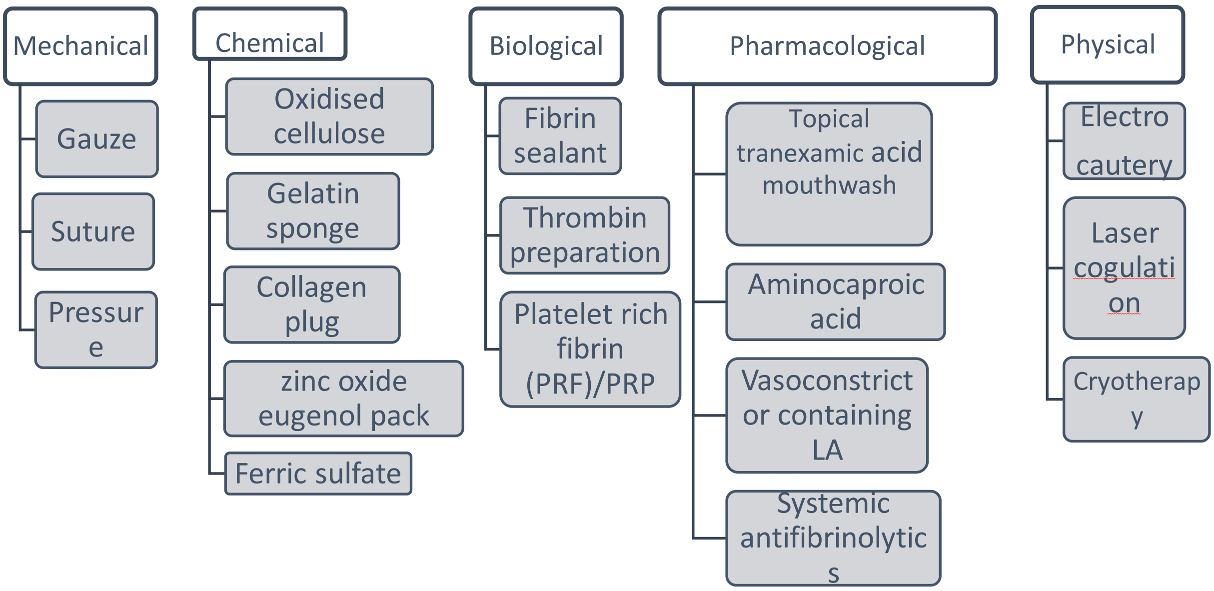
- **Haemostatic Agents in Oral Surgery.**

Haemostatic agents prevent bleeding by inhibiting the conversion of plasminogen to plasmin, thereby producing an antifibrinolytic effect. Since the oral mucosa contains abundant plasminogen activators, these agents are particularly effective in controlling oral bleeding. Of the many available options, only tranexamic acid (TXA) and epsilon-aminocaproic acid (EACA) have been evaluated and widely used as mouth rinses in dental practice[11-13]. TXA is commercially available as an intravenous injection (100 mg/mL), while EACA is available as a 500 mg tablet, an injectable solution (250 mg/mL), and a flavoured oral syrup (250 mg/mL)[13]. Importantly, TXA is approximately 6–10 times more potent than EACA, as demonstrated in both in vitro and in vivo studies. Neither drug is marketed directly as a mouthwash, but EACA syrup can be readily adapted for this purpose. Alternatively, EACA tablets may be compounded, or the injectable forms of TXA or EACA may be diluted with sterile water and prepared fresh on the day of surgery to ensure stability and sterility.

The adverse effects of antifibrinolytics are generally dose-dependent, with the most common being nausea, vomiting, abdominal pain, and diarrhoea. Rare cases of thromboembolism have been reported in post-marketing surveillance, though current evidence is insufficient to quantify the risk or establish causality. Because of the potential thromboembolic risk, systemic administration is not recommended in anticoagulated patients. Local use as a mouth rinse is considered safer and equally effective, with fewer systemic side effects compared to systemic therapy.

Local and systemic haemostaticagents

Local haemostatic agent



Local haemostatic agents

1. Mechanical

Gauze pressure pack**:** Provides direct pressure, facilitating platelet plug formation.

- Suturing (simple, figure-of-eight, mattress)**:** Achieves tissue approximation and socket compression to stabilize the clot or haemostatic material.
- Periodontal/pressure dressing**:** Acts as a protective barrier and exerts localized pressure over gingival wounds.

2. Chemical / Topical Agents

- Oxidized cellulose (Surgical): Forms an artificial scaffold for clotting and produces local vasoconstriction through low ph.

- Gelatine sponge (Gel foam): Porous matrix that traps platelets and promotes coagulation.
- Collagen plug/sponge: Induces platelet aggregation and supports wound healing.
- Zinc oxide–eugenol pack**:** Provides physical protection with mild astringent action.
- Ferric sulphate / Aluminium chloride**:** Induce haemostasis via protein precipitation and vasoconstriction.

3. Biological

- Fibrin sealant/glue: Mimics the final steps of the coagulation cascade to form a stable fibrin clot.
- Topical thrombin: Converts fibrinogen to fibrin, enhancing local clot formation.
- Platelet-rich fibrin (PRF)/PRP**:** Autologous platelet concentrates providing growth factors and a fibrin matrix that promotes haemostasis and healing.

4. Pharmacological

- Aminocaproic acid**:** Alternative antifibrinolytic agent with similar mechanism to TXA.
- Vasoconstrictor-containing local anaesthetics**:** Reduce intraoperative bleeding through α-adrenergic vasoconstriction.

5. Physical

- Electrocautery**:** Achieves haemostasis by thermal coagulation of bleeding vessels.
- Laser coagulation**:** Provides precise photothermal coagulation with added antimicrobial effect.
- Cryotherapy**:** Produces vasoconstriction and reduces fibrinolysis, thereby limiting bleeding and edema.

ANTIFIBRINOLYSIS

- Antifibrinolysis refers to the physiological process that prevents the premature breakdown of blood clots, thereby promoting haemostasis and stabilizing clot formation. This is particularly crucial in managing conditions associated with excessive bleeding, such as trauma, postpartum haemorrhage, and certain surgical procedures.

Mechanism of Antifibrinolysis

- At the molecular level, antifibrinolysis involves the inhibition of fibrinolysis—the enzymatic breakdown of fibrin in blood clots. Plasminogen, a precursor to plasmin, binds to fibrin within a clot. Tissue plasminogen activator (tPA) and urokinase plasminogen activator (up A) convert plasminogen into plasmin, which then degrades fibrin, leading to clot resolution.
- Antifibrinolytic agents, such as tranexamic acid (TXA), function by inhibiting this process. TXA is a synthetic lysine analog that competitively binds to the lysine-binding sites on plasminogen, preventing its interaction with fibrin and subsequent activation into plasmin. By doing so, TXA stabilizes the fibrin clot and reduces bleeding.

Role of Tranexamic Acid (TXA)

- TXA has been extensively studied and utilized in clinical settings for its antifibrinolytic properties. It is particularly effective when administered early in the course of bleeding, as demonstrated by major clinical trials such as CRASH-2[2,10] andWOMAN[10]These studies found that early administration of TXA significantly reduced mortality in patients with traumatic haemorrhage and postpartum haemorrhage.
- In addition to its use in trauma and obstetrics, TXA is employed in various surgical disciplines to minimize blood loss. Its application extends to cardiac, orthopaedic, and dental surgeries, among others.

Considerations and Contraindications

- While TXA is generally well-tolerated, its use requires careful consideration of potential contraindications and risks. Patients with a history of thromboembolic events or those at high risk for such events should use TXA cautiously. Additionally, the timing of administration is critical; TXA is most effective when given within three hours of the onset of bleeding.
- In summary, antifibrinolysis plays a vital role in maintaining haemostasis during bleeding episodes. Agents like TXA enhance this process by inhibiting fibrinolysis, thereby stabilizing clots and reducing blood loss in various clinical scenarios.

Systemic haemostatic agent

Fresh Frozen Plasma (FFP)

Fresh Frozen Plasma (FFP) is a blood product that contains fibrinogen, protein C, antithrombin, protein S, albumin, and tissue factor pathway inhibitors. It acts as a haemostatic agent by replenishing coagulation factors lost during bleeding. FFP can be given alone or in combination with red blood cells (RBCs).

It is commonly used in patients with coagulation factor deficiencies and abnormal coagulation profiles who present with active bleeding. Other indications include reversal of warfarin-associated haemorrhage and use in trauma cases requiring massive transfusion.

Prothrombin Complex Concentrate (PCC)

Prothrombin Complex Concentrate (PCC) is a plasma-derived concentrate of vitamin K–dependent clotting factors, available in two formulations: four-factor PCC and three-factorPCC. The four-factor PCC contains factors II, VII, IX, X, along with anticoagulant proteins C and S, while the three-factor PCC contains the same factors but lacks factor VII.

Clinical studies, both retrospective and prospective, have shown that although PCC enables rapid correction of trauma-induced coagulopathy, its impact on overall mortality remains unclear. This highlights the need for larger, high-quality randomized clinical trials to establish its efficacy as a first-line therapy in haemorrhagic patients.

Advantages of PCC include:

- Rapid availability in both prehospital and emergency settings
- High concentration of clotting factors delivered in a small infusion volume

Tranexamic Acid (TXA)

Tranexamic Acid (TXA) is a synthetic antifibrinolytic drug that helps prevent the breakdown of blood clots. It acts by competitively inhibiting the conversion of plasminogen toplasmin, and at higher concentrations, it noncompetitively blocks plasmin activity, thereby stabilizing fibrin clots. In addition to its antifibrinolytic action, TXA may exert other beneficial effects in trauma patients independent of clot regulation.

Major clinical trials, including CRASH-2 [10] andWOMAN [11]demonstrated that TXA administration within three hours of injury significantly reduced mortality in haemorrhagic patients. More recently, TXA has gained increasing attention for use in prehospital and emergency settings.

CONCLUSION

From the above discussion, it can be concluded that tooth extraction in patients receiving long-term aspirin therapy is generally safe and does not require discontinuation of the drug. Although aspirin may cause a slight increase in postoperative bleeding, such bleeding is usually minor and can be effectively managed with local haemostatic measures such as pressure application, suturing, topical agents, and antifibrinolytic mouth rinses. On the other hand, stopping aspirin poses a significant risk of serious thromboembolic events like myocardial infarction and stroke. Therefore, it is advisable to continue aspirin therapy during dental extractions and manage any bleeding locally to ensure both effective haemostasis and overall patient safety.

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