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Mini-Review

Management of Bleeding in Dental Surgery: A Mini Review

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Abstract

Bleeding is a common and potentially serious complication in dental surgery. Proper management is crucial to prevent morbidity and ensure patient safety. Dental surgery, encompassing a wide range of procedures from simple tooth extractions to complex bone augmentation procedures, inherently carries the risk of bleeding complications. These can be exacerbated in patients with underlying conditions such as anticoagulation therapy or hereditary bleeding disorders. Managing bleeding effectively in these scenarios is a significant challenge for dental practitioners. Hemostasis is a normal process that occurs immediately after such procedures, involving a balance between fibrinolytic factors and the hemostatic system through three steps: vascular spasm of blood vessels and smooth muscles, aggregation of platelets to form a plug, and the coagulation sequence by extrinsic and intrinsic factors. Interruption of this process may be caused by certain medical conditions and medications. Knowledge of the normal hemostatic cascade can help clinicians detect individuals with a high risk of bleeding risk and prevent surgical complications.

***Keywords:** Dental Surgery, Bleeding Management, Hemostatic Cascade.*

Introduction

Hemostasis

To understand how to manage bleeding in dental surgery, it is crucial to comprehend hemostasis and the clotting cascade. Hemostasis is the physiological process that keeps blood fluid within the blood vessels and, if these vessels are damaged, induces the formation of a hemostatic plug to stop bleeding. The process of hemostasis can be divided into primary hemostasis and secondary hemostasis.

Primary hemostasis occurs immediately after an injury and begins with vasoconstriction, regulated by endothelin, an endothelium-derived vasoconstrictor. Simultaneously, the extracellular matrix (ECM) is exposed, facilitating platelet adhesion to the endothelium and forming a hemostatic plug through platelet aggregation. Within about 30 minutes of damage to the blood vessels, vascular spasm leads to vasoconstriction. At the site of the disrupted endothelial lining, the ECM/collagen becomes exposed to the blood components.[1]

The ECM releases cytokines and inflammatory markers that lead to platelet adhesion and aggregation at the site, forming a platelet plug and sealing the defect.[2]

Secondary hemostasis, or coagulation, involves the activation of the coagulation cascade by the interaction of fibrinogen and the products secreted by platelets. This process activates thrombin, which converts soluble fibrinogen to insoluble fibrin, forming a secondary stable coagulum. In the extrinsic pathway, the tissue factor binds to factor VII and activates it. The activated factor VII (factor VIIa) further activates factor X and factor IX via proteolysis. Activated factor IX (factor IXa) binds with its cofactor, activated factor VIII (factor VIIIa), which leads to the activation of factor X (factor Xa). Factor Xa binds to activated factor V (factor Va) and calcium, generating a prothrombinase complex that cleaves prothrombin into thrombin.[3]

The intrinsic pathway involves thrombin production converting factor XI to activated factor XI (factor XIa). Factor XIa, with activated factor VII and tissue factor, converts factor IX to activated factor IX (factor IXa). Activated factor IX combines with activated factor VIII (factor VIIIa) and activates factor X. Activated factor X (factor Xa) binds with activated factor V (factor Va) and converts prothrombin to thrombin.[4]

The final steps in the coagulation cascade involve the conversion of fibrinogen to fibrin monomers, which polymerize to form a fibrin polymer mesh, resulting in a cross-linked fibrin clot. This reaction is catalyzed by activated factor XIII (factor XIIIa), which stimulates the lysine and glutamic acid side chains, causing cross-linking of the fibrin molecules and forming a stabilized clot.[5]

Clot Resolution or tertiary hemostasis occurs when activated platelets contract their internal actin and myosin fibrils in their cytoskeleton, leading to shrinkage of the clot volume. Plasminogen then activates plasmin, promoting lysis of the fibrin clot and restoring blood flow in the damaged or obstructed blood vessels.[6]

Conditions and medications affecting hemostasis

There are several conditions and medications that can disrupt the normal hemostatic process and increase the risk of bleeding in dental surgery.

Hemophilia is an inherited bleeding disorder that affects secondary hemostasis, presenting in two main types: Hemophilia A, caused by a deficiency in factor VIII, and Hemophilia B, resulting from a deficiency in factor IX. Consequently, deficiencies in either protein result in similar bleeding symptoms, including excessive bruising and spontaneous bleeding into joints, muscles, internal organs, and the brain. Hemophilia primarily affects males due to the X-linked nature of the genes involved, with Various mutations in factor VIII or IX can cause different severities of hemophilia, ranging from severe (<1% function) to moderate (1-5%) and mild (5-20%). Treatment for Hemophilia A and B primarily involves infusions of recombinant factor VIII or IX.[7]

Von Willebrand disease (VWD) is a bleeding disorder caused by a deficiency or defect in von Willebrand factor (VWF), which is essential for platelet aggregation and as a carrier for factor VIII. Type 1 VWD involves a partial quantitative defect, while type 3 results from a complete absence of VWF. Type 2 VWD features normal amounts of VWF but with functional defects and is further subdivided into several categories. VWD is autosomal and affects men and women equally. For type 1 (mild) and most type 2 VWD, the primary treatment is desmopressin (DDAVP), a vasopressin analog that stimulates the release of VWF from endothelial cells, temporarily increasing VWF levels in the blood.[8]

Patients who are obese and with high cholesterol or other cardiovascular risks may also have a higher risk of bleeding due to the effect of certain anticoagulants or antiplatelet medications they may be taking to reduce the risk of thrombosis. Some of these medications, such as aspirin, clopidogrel, or warfarin, may interfere with normal hemostasis and increase the risk of bleeding after dental procedures.[9, 10]

Thrombosis is commonly managed with drugs that inhibit platelet aggregation. Acetylsalicylic acid (aspirin) inhibits cyclo-oxygenase (COX)-1 in the thromboxane A2 synthesis pathway, preventing platelet activation.[11] Clopidogrel (Plavix) and prasugrel (Effient) inhibit the ADP receptor P2Y12, thereby blocking platelet activation.[12]

Long-term prophylaxis has often employed vitamin K antagonists like warfarin (Coumadin) which inhibit the post-translational processing of vitamin K-dependent coagulation factors II, VII, IX, and X.[13] Patients on warfarin need closer monitoring and dose adjustment to maintain a therapeutic INR to ensure adequate anticoagulation without increasing the risk of bleeding.[14] Warfarin also has a number of food and drug interactions that need to be considered.[15]

A new class of anticoagulants directly inhibit factor Xa or thrombin, many of which are being developed as oral medications to prevent and treat deep vein thrombosis and pulmonary embolism as well as reduce the risk of strokes. Rivaroxaban, Apixaban, and Edoxaban are direct factor Xa inhibitors whilst Dabigatran is a direct thrombin inhibitor. These drugs are used at a fixed dose, and have a short half-life and predictable pharmacokinetics, eliminating the need for routine coagulation monitoring.[16]

Tests to assess bleeding

Several tests are available to evaluate bleeding risk. One such test is the bleeding Time test, where a small incision is made in the skin to activate the hemostatic mechanisms necessary for coagulation. Normally, bleeding stops within 7 to 9 minutes without external pressure. This test measures the time taken to form a platelet plug. Abnormal results may indicate thrombocytopenia, which can be identified through a platelet count, or abnormal platelet function, which can be diagnosed with platelet function studies and a complete blood count.[17]

Two widely used screening assays for detecting coagulopathy are the activated partial thromboplastin time (aPTT) and prothrombin time (PT). These functional assays evaluate the coagulation cascade and are not specific to any single defect. The aPTT measures the activity of the intrinsic pathway coagulation proteins, assessing factors such as VIII, IX, and XI. Normal aPTT values range from 25 to 35 seconds. Prolonged aPTT may indicate deficiencies in these clotting factors, hemophilia, or the presence of inhibitors. Conversely, a shortened aPTT may suggest an increased risk of thrombosis and could be associated with elevated factor VIII levels.[18] The PT measures the activity of the extrinsic pathway coagulation proteins, it is also used to monitor patients on warfarin. Normal PT values range from 9 to 13 seconds. Elevated PT values indicate a prolonged clotting time, suggesting potential issues with clotting factors such as fibrinogen, factor V, VII, X, and prothrombin. Abnormal PT values may point to liver disease, vitamin K deficiency, or the presence of anticoagulants.[19] The international normalized ratio (INR) standardizes PT measurements, ensuring consistency across different laboratories and is used to monitor warfarinzed individuals. A standard INR value is 1, while the therapeutic range typically runs from 2.0 to 3.5.[20] Both aPTT and PT can be influenced by factors in the common pathway (factors X, V, and II). These values can become abnormal at different thresholds for each clotting factor and may vary by the performing laboratory.[21]

Patient management

Before treatment, dental providers should take a comprehensive history to assess the bleeding risk associated with the intended surgical procedure. This assessment should include a review of medications, blood pressure, and liver function. Patients undergoing soft tissue surgery or those with severe periodontal disease may have an increased risk of bleeding during treatment. Based on the findings, it may be necessary to conduct clotting tests such as INR and APTT before treatment, as well as obtain medical clearance from a hematologist or the physician managing the patient's condition or medication. For dental procedures, an INR of up to 4 may be acceptable in some European countries,[22] while the American Academy of Oral Medicine has determined that an INR of up to 3.5 can be acceptable.[23] The INR should be checked at least 72 hours before minor oral surgery.

The consensus is that patients should not interrupt their anticoagulant or antiplatelet therapy[22] with the European Heart Rhythm Association and the American College of Chest.

Physicians recommending maintaining anticoagulant therapy during dental procedures such as the extraction of 1-3 teeth, periodontal, and implant surgery to minimize the risk of thromboembolism.[24]

In most cases, placing a hemostatic agent and suturing is sufficient to control bleeding.[25] If the risk of bleeding is very high, such as in patients with liver impairment or alcoholism; kidney failure, and chemotherapy, managing any interruption of anticoagulant or antiplatelet therapy should involve a multidisciplinary approach.[26]

The choice of surgical technique can significantly impact the likelihood of bleeding. Minimally invasive techniques that preserve soft tissue and bone structures tend to result in less bleeding compared to more extensive procedures. However, certain complex surgeries, such as orthognathic surgery or extensive bone grafting, inherently carry a higher risk of bleeding. The skill and experience of the surgeon also play a crucial role in managing bleeding, as meticulous handling of tissues and precise execution of techniques can minimize trauma to blood vessels.[27]

In the event that excessive bleeding occurs, first identify the source of the bleed and apply pressure. Applying direct pressure to the bleeding site is one of the simplest and most effective methods for controlling intraoperative bleeding. This can be achieved using gauze, which helps compress the vessels and promote clot formation. In cases of minor bleeding, continuous pressure for several minutes is often sufficient to achieve hemostasis. Several agents can be placed into the socket or surgical site and secured with resorbable sutures, including gelatin, derived from acid partial hydrolysis of purified animal collagen, available as a sponge or film which can be used dry or moistened with saline.

These products adapt easily to wounds, making them suitable for irregular surfaces.[27] Gelatin products are low-cost, efficient, and can be completely absorbed within 4–6 weeks.[28] Collagen hemostats provide a matrix for clot formation and consolidation, improving clotting factor release and platelet aggregation.[29] Complete reabsorption occurs within 14 –56 days.[30]

Oxidized regenerated cellulose is also a common choice for managing bleeding, acting as an absorbable mechanical barrier that promotes clotting. These products are ready-to-use, absorb 7–10 times their weight, and assist in clot formation by creating a gelatinous mass upon absorption. They are absorbed within 4–8 weeks.[31]

Active agents can directly affect the clotting cascade, including, Thrombin, derived from bovine or human plasma or produced via recombinant DNA techniques, which can be applied topically or combined with gelatin sponges. Fibrin sealant or glue, made from bovine and/or human blood components, simulates the final phases of the coagulation cascade, forming a fibrin clot to control bleeding locally and diffusely.[32]

Another agent is cyanoacrylate glue, An adhesive is maintained in a liquid state by an acidic stabilizer, is partially ionized molecules of water, and has the action of neutralizing the inhibitor. Once you apply the adhesive to tissue it starts to gradually polymerize to a solid state in 10 seconds after contact with blood or saliva.

Chemical hemostatic agents can effectively control bleeding. Ferric sulfate achieves hemostasis by creating a sealing membrane over injured blood vessels. When applied to tissue, ferric sulfate combines blood proteins with ferric and sulfate ions, resulting in a ferric ion-protein complex that physically obstructs the cut vessels. Tranexamic acid, an antifibrinolytic agent, can be used off-label as a 4.8% oral rinse to stabilize clots. It works by inhibiting the conversion of plasminogen to plasmin, thereby preventing fibrinolysis and promoting clot stability. Silver nitrate is useful in situations where sutures are impractical, such as on the hard palate. It acts as a cauterizing agent by releasing free silver ions that bind to the tissue, forming an eschar that blocks the blood vessels.

Bone wax, a non-absorbable mixture of beeswax, paraffin, isopropyl palmitate, and a wax- softening agent, helps control bleeding by occluding bleeding channels in bone. Bone wax is rarely used today due to potential foreign body reactions and its negative impact on bone healing.[33]

For severe vascular hemorrhage, methods such as ligation, and electrocautery may be necessary. While not common in primary care, these techniques are used postoperatively to control bleeding from larger or smaller blood vessels. Electrocautery seals damaged vessels with heat, while ligation ties off vessels with sutures. In cases where appropriate equipment or expertise is lacking, patients should be referred to the emergency department for immediate care.

To minimize the risk of bleeding complications, it's recommended to treat patients in the morning and at the start of the week. This scheduling allows sufficient time to manage any immediate or early bleeding issues, which typically arise within the first 48-72 hours.[34] For post-operative care, patients should be instructed to avoid rinsing or spitting for at least 24 hours to prevent disruption of the socket. Additionally, they should steer clear of hot foods, hot liquids, and chewing on the treated area. Patients should be provided with gauze and instructed to apply pressure to the site for at least 20 minutes if bleeding occurs. If bleeding continues, they should seek medical assistance promptly.

Conclusion

Effective management of bleeding in dental surgery is essential for patient safety and successful outcomes. Understanding the mechanisms of hemostasis and the factors that can disrupt this process allows dental practitioners to identify high-risk patients and implement appropriate measures. Various hemostatic agents and techniques, from chemical applications like ferric sulfate and tranexamic acid to mechanical methods such as pressure and suturing, play a crucial role in controlling bleeding during and after dental procedures. In complex cases or when severe bleeding occurs, advanced methods like ligation and electrocautery may be necessary. By combining thorough preoperative assessment with the judicious use of hemostatic strategies, dental professionals can minimize the risks associated with bleeding, ensuring optimal care for their patients.

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Conflict of Interest

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