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Management of Various Bleeding Problems in Dental Patients: An Approach to Normal Hemostatic System and the Patient's Specific Coagulation Defect

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Abstract
Management of various bleeding problems in Dental patients is very critical issue in today clinical practice. Dentists must be conscious of the collision of bleeding disorders on the supervision of their patients. Proper dental and medical evaluation of patients is therefore necessary before handling, especially if an invasive dental method is planned. Patients with gentle bleeding disorders can be treated in a most important care setting after conference with the hematologist, while patients with a reasonable to severe level of bleeding disorder who require all-encompassing dental procedures are best treated in a hospital setting. Dental care providers must straight away report cases of prolonged bleeding, dysphagia, or involvedness speaking and breathing following dental events to the patient's hematologist.
Keywords: *Bleeding Problems, Dental Patients.*

Introduction

Dentists must be conscious of the collision of bleeding disorders on the supervision of their patients. Proper dental and medical evaluation of patients is therefore necessary before handling, especially if an invasive dental method is planned. Patient evaluation and history should commence with standard medical questionnaires. Patients should be queried about any previous extraordinary bleeding episode after surgery or injury, impulsive bleeding and easy or frequent bruising.

Most reported bleeding episodes are minor and do not have need of a visit to the dentist or the crisis department and do not affect dental action significantly (Lockhart *et al*., 2003; Meechan & Greenwood, 2003).

An inclusive drug history is important. If a patient is captivating anticoagulant drugs, it will be important to consult his or her physician before any major surgical practice. In addition, a number of medications may get in the way with homeostasis and prolong bleeding. Drugs of abuse, such as alcohol or heroin, may also cause excess bleeding2 by causing liver injure consequential in altered production of coagulation factors (Lockhart *et al*., 2003; Meechan & Greenwood, 2003).

A universal examination of the patient might point toward a tendency to bleed. Multiple purpurae of the skin, bleeding wounds, obvious hematomas or swollen joints may be evident in patients with severe bleeding defects. In adding together, patients may show signs of underlying systemic disease. Patients with liver disease may have jaundice, spider nevi, ascites and other signs of impaired hepatic function (Lockhart *et al*., 2003; Meechan & Greenwood, 2003).

A cardiac patient can show tachycardia or hypertension, which may make hemostasis more difficult to achieve. Evidence of petechiae, ecchymoses, hematomas or excessive gingival bleeding should direct the practitioner's concentration in the direction of a possible underlying bleeding disorder. When a bleeding disorder is suspected, laboratory investigations, including blood counts and clotting studies, should be carried out (Lockhart *et al*., 2003; Meechan & Greenwood, 2003).

Basic Mechanisms of Homeostasis and Their Interactions

Interaction of several basic mechanisms produces normal homeostasis. For clarity and understand-ding, these are presented separately. Homeostasis can be separated into four general phases:

The vascular phase; the platelet phase; the coagulation cascade phase, consisting of intrinsic, extrinsic, and common pathways; and the fibrinolytic phase. The first three phases are the principal mechanisms that stop the loss of blood subsequent vascular injury. Briefly, when vessel truthfulness is disrupted, platelets are activated, adhere to the site of injury, and form a platelet plug that reduces or temporarily arrests blood loss (Davie *et al*., 1991). The exposure of collagen and commencement of platelets also initiates the coagulation cascade, which leads to fibrin configuration and the production of an insoluble fibrin clot that strengthens the platelet plug (Davie *et al*., 1991). Fibrinolysis is the major means of disposing of fibrin after its haemostatic function has been satisfied, and it can be considered the

rate-limiting step in clotting. It leads to fibrin degradation by the proteolytic enzyme plasmin. The coagulation cascade is happening within 10 to 20 seconds of injury, an initial haemostatic plug is formed in 1 to 3 minutes, and fibrin has been generated and added to steady the clot by 5 to 10 minutes.

Vascular Phase

After tissue injury, there is an immediate reflex vasoconstriction that may alone be haemostatic in small vessels. Various mediators such as serotonin, histamine, prostaglandins, and other materials are vasoactive and produce vasoconstriction of the microvascular bed in the area of the injury.

Platelet Phase

When circulating platelets are uncovered to injured vascular surfaces (in the presence of functionally normal vWF, endothelial cells, collagen or collagen-like materials, basement membrane, elastin, microfibrils, and other cellular debris), platelets are activated to experience physical and chemical changes (Mustard & Packman, 1970). These changes fabricate an environment that causes the platelets to undergo the aggregation-and release phenomenon and form the primary vascular plug that reduces blood loss from small blood vessels and capillaries. These platelet plugs stick on to exposed crypt membranes (Mustard & Packman, 1970).

Coagulation Phase

The generation of thrombin and fibrin the end product of the third phase of hemostasis, the coagulation phase. This process involves multiple proteins, many of which are synthesized by the liver (fibrinogen, prothrombin, Fs V, VII, IX, X, XI, XII, and XIII) and are vitamin K dependent (Fs II, VII, IX, and X). The progression of coagulation essentially involves three separate pathways. It initially proceeds by two separate pathways (intrinsic and extrinsic) that congregate by activating a third (common) pathway. The

blood clotting mechanism is the most studied unit; it was outlined originally in 1903 by Markowitz as the prothrombin- to-thrombin and fibrinogen-to-fibrin conversion system. In 1964, the "cascade" or "waterfall" theory was proposed (Davie & Ratnoff, 1964; MacFarlane, 1964). It offered a useful machine for understanding this complex system and its control, as well as the clinically significant associated laboratory tests. The design of reaction is a bio amplification, in which a precursor is altered to an active form, which, in turn, activates the next pioneer in the sequence. Beginning with an undetectable biochemical reaction, the coagulation mechanism results in a final explosive change of a liquid to a gel. The major steps involve the adaptation of a precursor protein to an "activated" form, which activates another precursor protein, and so on down the cascade.

The coagulation of blood also requires the presence of both calcium ions and phospholipid (or a phospholipid-containing membrane fragment derived from blood platelets). The intrinsic pathway is initiated when F XII is activated by surface get in touch with (eg, with collagen or subendothelium), and it involves the communication of F XII and F XI. The next step of intrinsic coagulation, the activation of F IX to F IXa, requires a divalent cation. Once activated, F IXa forms a complex with F VIII, in a reaction that requires the company of both calcium ions and phospholipid, which, in turn, converts F X to an activated form— F Xa.

The extrinsic pathway is initiated by the release of tissue thromboplastin, also called tissue factor, and does not require contact activation. Tissue thromboplastin binds to F VII in the presence of calcium, and this complex is capable of activating Fs IX and X, linking the intrinsic and extrinsic pathways. It is the activation of X that begins the frequent trail. Once activated, F Xa converts prothrombin to thrombin in a reaction similar to the activation of F X by F IXa. Thrombin converts fibrinogen to fibrin, the building block of hemostatic plug.

Fibrinolytic Phase

The fourth phase of homeostasis is Fibrinolysis; this is well thought-out the major means of disposing of fibrin after its haemostatic function has been fulfilled. Once the micro vascular bed is sealed and primary homeostasis is complete, the secondary homeostasis pathway has previously commenced in equivalent.
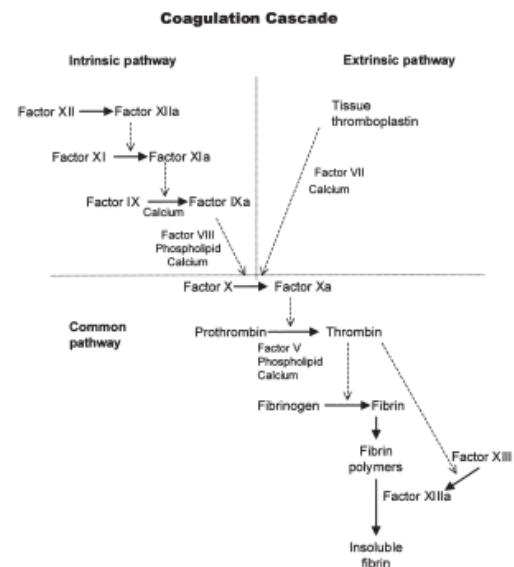

Figure No.1 The coagulation cascade

Types of Bleeding Disorders

Bleeding disorders can be classified as coagulation feature deficiencies, platelet disorders, vascular disorders or fibrinolytic defects (Patton, 2003; Blinder).

Amongst the congenital coagulation defects, hemophilia A, hemophilia B (Christmas disease) and von Willebrand's disease are the most common. Hemophilia A is due to a shortage of clotting factor VIII or anti-hemophilic factor. It is an inherited X-linked recessive attribute establish in males. Symptoms may comprise prolonged bleeding, ecchymosis, deep hematomas, epistaxis, impulsive gingival bleeding and hemarthrosis. A factor VIII level of 6% to 50% of normal factor activity (mild hemophilia) is associated with bleeding during surgery or trauma; 1% to 5% (moderate hemophilia) with bleeding after mild injury; and < 1% (severe hemophilia) with spontaneous bleeding (Patton, 2003).

Management of hemophilia A among patients undergoing dental surgery consists of (Meechan &

Greenwood, 2003) mounting factor VIII levels, replacing factor VIII and inhibiting fibrinolysis. Desmopressin (DDAVP) is used to attain a transient increase in factor VIII level through the discharge of endogenous factor VIII in patients with hemophilia A and von Willebrand's disease. It may be adequate to achieve hemostasis in mild forms of these diseases. DDAVP may be mutual with antifibrinolytic agents to increase its effectiveness (Meechan & Greenwood, 2003).

Options for factor VIII substitute are factor VIII concentrates, fresh frozen plasma and cryoprecipitate. Highly purified forms of factor VIII concentrates, manufactured using recombinant or monoclonal antibody purification techniques, are favored because of their greater viral safety (Lusher & Roth, 2005; Schlesinger & Ragni, 2002). New generations of recombinant factor VIII are being developed that are free from human and animal proteins, in an attempt to additional improve their safety (Manno, 2003). In patients who manufacture antibodies to factor VIII, a higher dose of concentrated factors can be considered, but a focus on local measures is critical.

Anti-fibrinolytic therapy can be used postoperatively to defend the formed blood clot. Epsilon-aminocaproic acid and tranexamic acid are the ordinary agents used. Tranexamic acid in an oral soak helps stop postoperative bleeding from surgical wounds. Postoperative use of epsilon-aminocaproic acid can considerably diminish the level of factor compulsory to control bleeding when used in conjunction with pre surgical infusion of factor VIII concentrate (Webster *et al*., 1973; Walsh *et al*., 1971; Walsh *et al*., 1975). Hemophilia B is the consequence of factor IX deficiency. It is managed by replacement therapy with highly purified, virally inactivated factor IX concentrates. Prothrombin complex concentrates can also be used for factor IX replacement.

Von Willebrand's disease is the most common hereditary coagulation confusion with an incidence of 1 in 10,000. It is not sex linked. It is classified as Type I to Type IV and may vary in severity. For mild circumstances, use of DDAVP may be sufficient, but severe disease warrants factor VIII replacement.

Other than congenital diseases, coagulation defects may be acquired and from a variety of sources. In liver diseases, the synthesis of clotting factors may be condensed due to parenchymal damage or hindrance (Golla *et al*., 2004). These patients may have a variety of bleeding disorders depending on the extent of their liver disease. Management options for hemostatic defects in liver disease (Lusher & Roth, 2005) comprise vitamin K and fresh frozen plasma infusion (immediate but temporary effect) for long-drawn-out prothrombin time and partial thromboplastin time; cryoprecipitate for replacement of factor VIII deficiency; and replacement rehabilitation for disseminated intravascular coagulation. Patients suffering from viral hepatitis are a potential foundation of cross infection, and necessary precautions should be taken throughout procedures. Drug doses frequently need to be modified in these patients due to impaired liver function. The patient's physician should be consulted before making any changes in the drug schedule.

Dental Management

The management of patients with bleeding disorders depends on the severity of the circumstance and the invasiveness of the planned dental procedure. If the method has limited invasiveness and the patient has a mild bleeding disorder, only slight or no modification will be obligatory.

In patients with relentless bleeding disorders, the goal is to diminish the challenge to the patient by restoring the hemostatic system to satisfactory levels and maintaining hemostasis by local and adjunctive methods. The patient's physician should be consulted before invasive treatment is undertaken. In patients with drug-induced coagulopathies, drugs may be stopped up or the doses modified. For permanent coagulopathies,

replacement of absent factors may be necessary (Lockhart *et al.*, 2003; Flint *et al.*, 1988).

Pain Control

In patients with coagulopathies, nerve-block anesthetic injections are contraindicated unless there is no better substitute and prophylaxis is provided, as the anesthetic solution is deposited in a highly vascularized area, which carries a risk of hematoma formation (Nazif, 1970; Webster *et al.*, 1968). The frequently used blocks require minimum clotting factor levels of 20% to 30%. Extravasation of blood in the oropharyngeal area by an inferior alveolar block or in the pterygoid plexus can create gross swelling, pain, dysphasia, respiratory obstruction and risk of death from asphyxia (Archer & Zubrow, 1954; Leatherdale, 1960). Anesthetic infiltration and intra ligamentary anesthesia are potential alternatives to nerve block in many cases. An anesthetic with a vasoconstrictor should be used when promising. Alternative techniques, as well as sedation with diazepam or nitrous oxide–oxygen analgesia, can be in employment to reduce or eradicate the need for anesthesia. Patients undergoing widespread treatment requiring factor replacement may be treated under general anesthesia in a hospital operating room.

Oral Surgery

Surgical events carry the highest risk of bleeding, and safety precautions are needed. For coagulopathies, transfusion of appropriate factors to 50% to 100% of normal levels is not compulsory when a single bolus infusion is used in an outpatient setting. In patients with hemophilia, additional postoperative factor maintenance may be required after extensive surgeries. This can be done with factor infusion, DDAVP, cryoprecipitate or fresh frozen plasma depending on the patient's condition. The patient's hematologist should be consulted before planning, and patients with severe disease should be treated in specialty centers.

Local haemostatic agents and techniques such as pressure, surgical packs, sutures and surgical stents may be used individually or in grouping and may assist in the local delivery of hemostatic agents, such as topical thrombin and vasoconstrictors (Rackoz *et al.*, 1983).

Periodontal Procedures

Periodontal health is of critical significance in patients with bleeding disorders 3 as inflamed and hyperemic gingival tissues are at increased risk of bleeding. Periodontitis may cause tooth mobility and warrant extraction, which may be a convoluted process in these patients. Patients with coagulopathies may neglect their oral health due to fear of bleeding during tooth brushing and flossing, which leads to greater than before gingivitis, periodontitis and caries.

Periodontal probing, supragingival scaling and polishing can be done normally without the risk of considerable bleeding. Factor substitute is seldom needed for subgingival scaling and root planing if these procedures are done cautiously. Ultrasonic instrumentation may result in less tissue trauma (Rackoz *et al.*, 1983).

Restorative and Endodontic Procedures

General restorative events do not pose a significant risk of bleeding. Care should be taken to avoid injuring the gingiva while insertion rubber dam clamps, matrices, and wedges. A rubber dam should be used to put off laceration of soft tissues by the cutting instruments. Saliva ejectors and high-speed suction can injure the mucosa in the floor of the mouth and cause hematoma or ecchymosis; thus, they should be used carefully. Endodontic therapy is favored over extraction whenever possible in these patients. Endodontic therapy does not usually pose any significant risk of bleeding and can be performed routinely. Endodontic surgical procedures may necessitate factor replacement therapy (Rackoz *et al.*, 1983).

Prosthodontic Procedures

These procedures do not usually engage a considerable hazard of bleeding. Trauma should

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be minimized by careful post-insertion adjustments. Oral tissue should be handled precisely during the various clinical stages of prosthesis production to reduce the risk of ecchymosis. Careful modification of prostheses is needed to decrease trauma to soft tissue (Rackoz *et al*., 1983).

Orthodontic Procedures

Orthodontic therapy can be approved out without bleeding complications, although care should be taken that appliances do not interrupt on soft tissues and emphasis should be put on outstanding, traumatic oral hygiene.

Choice of Medications

Many medications prescribed in dental practice, especially ASA, may interfere with hemostasis. In addition, many drugs interact with anticoagulants, increasing their potency and the risk of bleeding. When used for extended periods, ASA and nonsteroidal anti-inflammatory drugs (NSAIDS) can increase the effect of warfarin. Penicillins, erythromycin, metronidazole, tetracyclines and miconazole also have potentiating effects on warfarin (Rackoz *et al*., 1983).

Dental management of patients with inherited bleeding disorders

Bleeding disorders can result from inherited genetic defects or be acquired due to use of anticoagulant medications or medical circumstances such as liver dysfunction, chronic kidney disease, and autoimmune disease (Napeñas & Patton, 2015; Pototski & Amenábar, 2007; Lockhart *et al*., 2003). During blood vessel injury, hemostasis relies on interactions between the vascular vessel wall and activated platelets as well as clotting factors (De *et al*., 2013).Any marked defect at one of these stages results in bleeding disorders. Vascular wall defects, platelet defects, or deficiency of clotting factors can affect the severity level of bleeding episodes (Nizarali & Rafique, 2013). Thus; patients may have gentle, moderate, or severe episodes of bleeding.

Sources of inherited bleeding disorders
Vascular wall defects

A patient's bleeding disorder may be unrecognized, and bleeding episodes can come into view impulsively or after dental extraction. Patients with inherited bleeding disorders that are marked by a vascular defect, such as Marfan syndrome, hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome), and Ehlers-Danlos syndrome, rarely have a severe bleeding episode after dental extraction. Frequently, these patients can accept dental management in the primary care setting (Rafique *et al*., 2013). Management includes coordination with the hematologist, minimally invasive dentistry, local hemostatic measures, and avoiding analgesics such as aspirin and other nonsteroidal anti-inflammatory drugs (Scully *et al*., 2007).

Platelet defects

Inherited platelet defects result in qualitative dysfunction, and patients are best treated after conference with a hematologist, who might advocate for platelet transfusion prior to any invasive dental procedures (Valera *et al*., 2013; Seligsohn, 2012). Specific laboratory investigations are required to assess platelet function, which is deliberate by the platelet aggregation capacity. Different types of blood tests can be well thought-out to evaluate patients with inherited platelet defects during referral to the hematologist. These include the bleeding time, platelet aggregation test, and peripheral blood smear (Valera *et al*., 2013; Seligsohn, 2012).

Clotting factor defects

Management of patients with inherited clotting disorders, such as hemophilia A and von Willebrand disease (vWD), is not undemanding and requires consultation with a hematologist (Aoun *et al*., 2016).

Hemophilia A

Hemophilia A is an inherited coagulation disorder connecting a deficiency of factor VIII.1 The

prevalence of hemophilia A is dissimilar from country to country and depends on the national economies. For example, countries with a high income have the highest occurrence, which increases over time. Hemophilia A is inherited as an autosomal X linked recessive trait; therefore, the disease mainly affects males.16,17 However, females with a factor VIII activity level of less than 50% are considered to be carriers and are treated as patients with mild hemophilia (Anderson *et al.*, 2013).

Von Willebrand disease

Von Willebrand disease is an inherited disease noticeable by vWF deficiency. It is measured the most common congenital bleeding disorder, affecting 1% of the population of both sexes equally; symptomatic prevalence's are reported to range from 1 in 1000 to 1 in 10,000 of the population. In rare cases, acquired von Willebrand disease may develop in elderly patients and is linked with various underlying diseases (Franchini & Mannucci, 2013).

Conclusion

Patients with gentle bleeding disorders can be treated in a most important care setting after conference with the hematologist, while patients with a reasonable to severe level of bleeding disorder who require all-encompassing dental procedures are best treated in a hospital setting. Consultation with the hematologist previous to any dental procedure is suggested to assess the patient's needs for prophylactic substitution therapy. The use of aspirin and supplementary non steroidal anti-inflammatory drugs should be avoided in patients with bleeding disorders. Dental care providers must straight away report cases of prolonged bleeding, dysphagia, or involvedness speaking and breathing following dental events to the patient's hematologist.

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