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Bleeding and its Management

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ABSTRACT

Blood is a connective tissue in fluid form. It carries oxygen from lungs to all parts of the body and CO2 from all parts of body to lungs. Blood contains blood cells (RBC, WBC, PLATELETS) and liquid portion known as plasma.

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Introduction

Bleeding is also called as hemorrhage. It is the loss of blood or escape of blood from blood vessels. Bleeding occurs internally or externally either through opening in the body or through a break in the skin. Bleeding of oral cavity is caused by trauma during dental procedure, platelet disorder, coagulation disorder, vessel wall disorder, drugs like aspirin, coumarin, anticoagulants, chemotherapeutic agents [1].

Laboratory investigations for screening major defects of Hemostasis include platelet count (normal range:1,50,000-4,50,000 per cu mm. <10,000- spontaneous clinical hemorrhage, <50,000- surgical hemorrhage), bleeding time (Normal range 1-6minutes, Extrinsic pathway of coagulation phase by prothrombin time (normal 11-13 seconds),common coagulation pathway evaluated by activated normal thromboplastin time (normal range is 15-35 sec)Other tests are Thrombin time, Fibrin degradation products, Fibrinogen assay, Coagulation factor assays, and Coagulation factor inhibitor assay [2,3].

Causes of Bleeding

Local Factors

- Gingivitis
- Periodontitis
- Improper use of floss
- Vigorous brushing
- Poor oral hygiene
- Decayed tooth

Systemic Factors

- Drug induced bleeding
- Diabetic Mellitus
- Pregnancy
- Vitamin-k deficiency vitamin -c deficiency
- Leukemia
- Von Willi brands disease
- Platelet disorder
- Aplastic anemia

Local Factors

The most common cause of abnormal gingival bleeding is chronic inflammation The bleeding is chronic or recurrent and it is caused by mechanical trauma (tooth brushing, food impaction, biting solid foods etc.)

Acute episodes of bleeding occur spontancously caused by acute gingival disease.

Systemic Factors

Drug induced Gingival Bleeding

Drugs such as antiplatelet medications (e.g. Aspirin) or anticoagulants (e.g. Warfarin) that are prescribed for certain medical conditions also increase the bleeding tendencies of gingival tissues. Women taking oral contraceptives are significantly more prone to gingivitis and therefore to gingival bleeding.

Diabetic Mellitus

Diabetic mellitus is a metabolic disorder characterized by deficiency of Insulin. Consists of 2 types Type 1 – Insulin dependent diabetes, Type 2- Non insulin dependent diabetes Gingival bleeding is caused by increased blood sugar level. Author witzum suggested free oxygen radicals lead to tissue destruction and increase the gingival inflammation. The high blood glucose level in GCF (Gingival crevicular fluid alters the plaque microbial flora and increases the gingival bleeding)

Pregnancy

Gingival bleeding at the time of pregnancy due to fluctuating estrogen and progesterone levels on the periodontium start as early as puberty Gingival bleeding characterized by mild to severe levels along with inflammation, pain and hyperplasia. Most times it will resolve automatically after delivery, when the hormonal levels return to normal.

Vitamin -k Deficiency

Vitamin-k is required for the production of blood clotting factors, and essential for coagulation. Deficiency of vitamin-k leads to lack of active prothrombin in the circulation. The result is that blood coagulation is adversely affected. Vit-k deficiency can lead to gingival bleeding, nose bleeding and increased clotting time [4].

Vitamin -C Deficiency (Scurvy)

Vitamin c is required for collagen production, maintenance of normal connective tissue and wound healing process. Severe deficiency of Vit -c causes bleeding gums, spongy or sore gums, loose teeth, delayed wound healing process. Due to the impairment in the synthesis of collagen, and antioxidant property of Vit -c leads to delayed wound healing [4].

Leukemia

The Leukemia is a malignant neoplasm of the hematopoietic stem cells characterized by diffuse replacement of the bone marrow by neoplastic cells. The abnormal leukemic cells disseminate into peripheral blood, so the blood cell count increases. Leukemia is the primary disorder of the bone marrow. The depression of normal bone marrow function leads to anemia, fever, fatigue and repeated infection (due to absence of matured leukocytes.) can easily occur. Leukemic cells can directly affect the lymph nodes, central nervous system and gingiva [5].

Von -Willibrands Disease

It is the hereditary blood clotting disorder characterized by spontaneous bleeding from mucous membranes, excessive bleeding from wounds, menorrhagia and prolonged bleeding time in the presence of normal platelet count' In case of severe deficiency continuous bleeding and post-operative bleeding after tooth extraction can occur [5].

Platelet Disorders: Thrombocytopenic Purpura

Thrombocytopenic purpura characterized by a low platelet count, a prolonged clot retraction and bleeding time, normal or slightly prolonged clotting time. Small tiny blood clots and hemorrhagic vesicles occur in the oral cavity. Gingiva is swollen, soft, friable and gingival bleeding occurs spontaneously. Removal of local irritants can reduce the severity of gingival bleeding [6].

Aplastic anemia

It is a hematological disorder, rare type, applied to pancytopenia characterized by 1) anemia 2) Neutropenia 3) Thrombocytopenia. If the neutrophil counts less than 0.2x 10%, then it is considered as severe form of Aplastic anemia [7].

Orally spontaneous gingival bleeding due to decreased platelet level, severe periodontal diseases, pallor, and oral ulceration are found [8]. Before treating the patient in dental clinics one must consult with hematologist and be advised to do the treatment on the day of platelet infusion [9]. In the uncontrolled bleeding patient the patient should take antifibrinolytics before the procedure is begun. It will reduce the bleeding especially mucosal bleeding. Aplastic anemia leads to many infections so one must postpone the dental treatment until the patient reaches normal blood cell count and must prescribe proper antibiotics prior to the treatment [10].

Classification

Hemorrhage can be classified in several ways for ease of identification and treatment.

Based on The Source of Blood Loss

- Arterial hemorrhage
- Venous hemorrhage
- Capillary hemorrhage

The vessel from which the bleed is occurring can be identified by the colour, pulsation, vigor of flow and the presence of a spurt.

Based on the Time of Occurrence

- Primary hemorrhage

It is the bleeding that occurs at the time of injury or surgery.

Secondary Hemorrhage

It is also recurrence of bleeding, it occurs weeks after injury or even later than that.

- Infection is the cause for reopening of the bleeder vessel in most cases.
- It is typically seen in patients with retained root tips or foreign materials in extraction socket.

Reactionary (intermediate) Hemorrhage

- It is recurrence of the bleeding within 24 hours of the injury or surgery.
- It is caused by dislodgement of the clot following rise of blood pressure after the injury.
- Restlessness in the post injury or postoperative period can also lead to either dislodging of the clot or slipping of the ligature.
- Coughing and vomiting increases the venous pressure, especially in the neck veins and this is often the cause for bleeding from extraction site or thyroidectomy wounds in the immediate postoperative period.
- This may also occur after the vasoconstrictive effect of the local anesthetic wears off. Adrenaline or epinephrine causes vasoconstriction by its action on the alpha receptors of the smooth muscles in the peripheral arterioles.

Based on Visualisation of the Haemorrhage

- External hemorrhage: Bleeding onto the exterior as in skin laceration
- Internal hemorrhage: It occurs in injuries to abdominal viscera. It is typically seen in rupture of the spleen. The diagnosis has to be made from history and clinical signs of blood loss like pallor, rising pulse rate and falling BP [11].

Based on Clinical Signs of Haemorrhage

- Petechial hemorrhage
- Ecchymosis
- Hematoma

Factors of Coagulation Cascade
Clotting factors

FACTOR	COMMON NAMES	FUNCTION
FI	Fibrinogen	Helps control bleeding by assisting with fibrin clot formation.
FII	Prothrombin	Assist FIXa,FVIIIa, FVa in formation of thrombin.
FIII	Tissue factor (TF)	Assists FVII and FIV in activation of FIX and FX
FIV	Calcium ions (Ca2+)	Necessary for the activation of multiple clotting factors.
FV	Proaccelerin (labile factor)	Assists FVilla, Fixa, FXa and FIl in formation of thrombin
FVII	Proconvertin (stable factor)	Assists TF and FIV in activation of FIX and FX
FVIII	Antihemophilic factor	Activated by thrombin tov amplify additional formation of thrombin
FIX	Plasma thromboplastin	Assist FVa, FVIIIa,FXa, with formation of thrombin
FX	Stuart power factor	Assist FVa,FVIIIa,Fixa with formation of thrombin
FXI	Plasma thromboplastin Antecedent (PTA)	Activated by thrombin in extrinsic pathway to increase the production of thrombin inside fibrin clot through the intrinsic pathway helps slow down fibrinolysis
FXII	Hageman factor	Contact activator of the kinin system
FXIII	Fibrin stabilizing factor	Activated by thrombin and helps with the formation of bonds between fibrin strands during secondary hemostasis
FXIV	Prekallikerin	Serine protease zymogen
FXV	HMWK	Co- factor
FXVI	VWF	Binds to VIII mediates platelet adhesion
FXVII	Antithrombin III	Inhibits IIa, Xa, and other proteases
FXVIII	Heparin cofactor II	Inhibits IIa
FIX	Protein C	Inactivates Va and VIIIa
FXX	Protein S	Co factor of activated protein C

Definition

Blood coagulation or blood clotting is defined as the process in which blood loses its fluidity and becomes a jelly-like mass few minutes after it is shed out or collected in a container.

Factors Involved in Blood Clotting

Coagulation of blood occurs through a series of reactions due to the activation of a group of substances. The substances necessary for clotting are called clotting factors. Thirteen clotting factors are identified.

Stages of Blood Clotting

Blood Clotting Occurs in three Stages

1. Formation of prothrombin activator.
2. Conversion of prothrombin into thrombin.
3. Conversion of fibrinogen into fibrin.

Formation of Prothrombin Octivator

Blood clotting commences with the formation of a substance called prothrombin activator. This process is initiated by substances produced either within the blood itself or outside the blood. Thus, formation of prothrombin activator occurs through two pathways:

A. Intrinsic pathway.
B. Extrinsic pathway.

Intrinsic Pathway for the Formation of Prothrombin Activator. In this, the formation of prothrombin activator is initiated by platelets, which are within the blood itself. Sequence of events in intrinsic pathway [12].

- During injury, the blood vessel is ruptured. Endothelium is damaged and collagen beneath the endothelium is exposed.
- When factor XII (Hageman factor) comes in contact with collagen, it is converted into activated factor XII in the presence of kallikrein and HMW kininogen (high molecular weight kininogen).
- Activated factor XII converts factor Xl into activated factor XI in the presence of HMW kininogen. Activated factor XI activates factor IX in the presence of factor IV (calcium).
- Activated factor IX activates factor X in the presence of factor VIII and calcium [5-12].
- When platelet comes in contact with collagen of damaged blood vessel, it gets activated and releases phospholipids.
- Now, the activated factorX reacts with platelet phospholipid and factor V to form prothrombin activator. This needs presence of calcium ions.
- Factor V is also activated by positive feedback effect of thrombin.

Extrinsic Pathway for The Formation of Prothrombin Activator

In this, the formation of prothrombin activator is initiated by the tissue thromboplastin which is formed from the injured tissues [13].

Sequence of Events in Extrinsic Pathway

- Tissues that are damaged during injury release factor
- Ill, i.e. tissue thromboplastin. The thromboplastin contains proteins, phospholipid and glycoprotein, which act as proteolytic enzymes.
- Glycoprotein and phospholipid components of thromboplastin convert factor X into activated factor X, in the presence of factor VII.
- Activated factor X reacts with factor V and phospholipid component of tissue thromboplastin to form prothrombin activator. This reaction requires the presence of calcium ions.

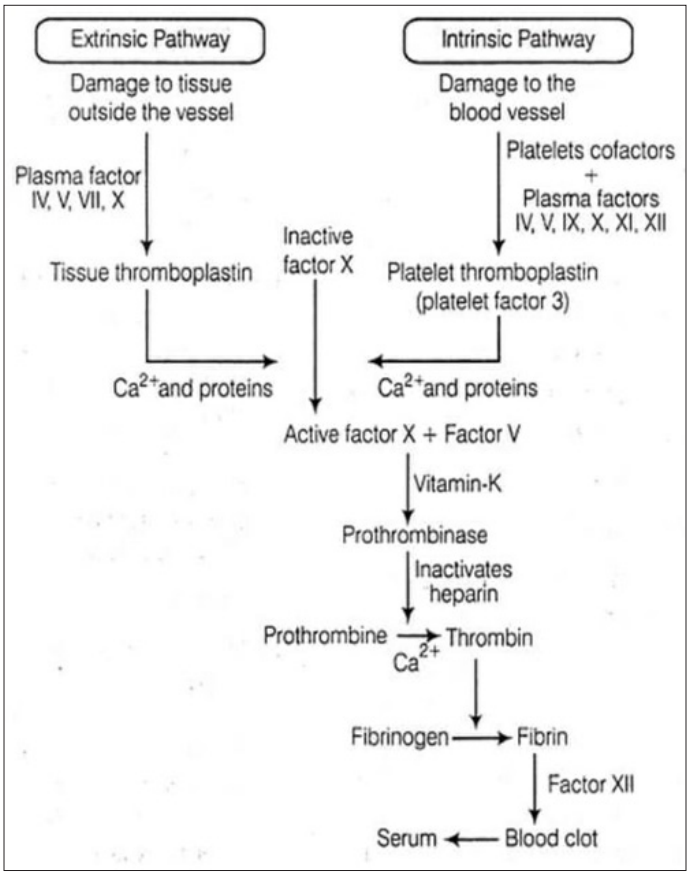
Conversion of Prothrombin into Thrombin

Blood clotting is all about thrombin formation. Once thrombin is formed, it definitely leads to clot formation [2].

Sequence of Events in Stage

- Prothrombin activator that is formed in intrinsic and extrinsic pathways converts prothrombin into thrombin in the presence of calcium ions (factor IV).
- Once formed thrombin initiates the formation of more thrombin molecules. The initially formed thrombin activates factor V in turn accelerates formation of both extrinsic and intrinsic prothrombin activator which converts prothrombin into thrombin. This effect of thrombin is called positive feedback effect.
- STAGE 3: CONVERSION OF FIBRINOGEN INTO FIBRIN
- Final stage of blood clotting involves the conversion of

fibrinogen into fibrin by thrombin.
- Sequence of Events in Stage 3
- Thrombin converts fibrinogen into activated fibrinogen which is called fibrin monomer.
- Fibrin monomer polymerizes with other monomer, molecules and form loosely arranged strands of fibrin Later these loose strands are modified into dense and llight fibrin threads.



Management
- Pressure
- Hemostat
- Sutures and ligation

Chemical methods
- Adrenaline
- Thrombin
- Surgicel
- Surgicel fibrillar
- Oxycel
- Gelatine sponge: Gelfoam / surgifoam
- Microfibrillar collagen (Avitene)
- Fibrous glue
- Styptics and astringents
- Alginic acid
- Natural collagen sponge
- Fibrin sponge
- Bone wax
- Ostene: a new water-soluble bone haemostatic agent

Thermal agents
- Eletrocautery / surgical diathermy
- Monopolar diathermy
- Bipolar diathermy
- Cryosurgery
- Lasers

Mechanical Methods

Pressure Firm pressure should be applied over the bleeding site using either fingers or gauze for at least 5 minutes. This would control most hemorrhages by counteracting the hydrostatic pressure of the bleeding vessel [10]. Hemostat Application of hemostat at the bleeding points helps in direct occlusion of the bleeding vessel. Sutures and ligation Severed blood vessels may be sutured with ligatures. A ligature replaces the hemostat as a permanent method of effective hemostasis. For large pulsatile artery, a trans-fixation suture to prevent slipping is indicated. Non-resorbable sutures such as silk and polyethylene are used as they evoke less tissue reaction.

Chemical Methods

Adrenaline Topical application of adrenaline brings about vasoconstriction of bleeding capillaries. Adrenaline is available in ampoule, which is applied with the help of gauze. The concentration of 1 in 10000 is used for hemostasis over the oozing site.ThrombinThrombin helps in converting fibrinogen into fibrous clot and acts as hemostat. Surgicel It is as an oxidized cellulose polymer obtained by dissolving pure alpha-cellulose in an alkaline solution. It acts by forming acid products from partial dissolution that coagulates the plasma proteins to form a black or brown sticky gelatinous clot. The applied surgicel resorbs from the site in 4 to 8 weeks. However, the disadvantage is that the surgicel clot is not formed by normal physiological mechanism. There is modified surgicel or oxidized regenerated cellulose in lavers that can be adapted to irregular surfaces and inaccessible areas. Complete resorption occurs in 2 weeks.

Oxycel is an oxidized cellulose polymer product. This absorbable hemostatic material is manufactured by controlled oxidation of cellulose using nitrous dioxide. The cellulosic acid (cytotoxic acid) present in oxycel has affinity for hemoglobin which leads to the formation of artificial clot. It should be applied on the dry surface as the acid formed during the wetting process inactivates the thrombin. It has bacteriostatic property because of its relatively low pH. It is available in gauze form or pellet form. Oxycel is composed of hollow 'twisted tubule' fibers in comparison to the irregular solid fibers of surgicel. Gelatine sponge or Gelfoam or Surgifoam Gelfoam is manufactured from purified pork skin gelatine. This is a non-antigenic and completely absorbable material. It has the capacity to absorb 45 times its weight in blood. It resorbs completely in 4 to 6 weeks.

Microfibrillar Collagen
- Collagen derived from bovine skin causes contact activation in addition to direct platelet aggregation.
- Its absorption time is 3 months.
- Styptics and astringents
- Precipitates protein and arrests bleeding.
- Commonly used styptics and astringents are
- Monsel's solution containing ferric sub-sulphate and tannic acid.
- Thrombin and gelatin sponge are now widely used.
- Alginic acid
- This is available in powder form in special 5-mg packages.
- It is placed over the bleeding sites, a protective film is formed over the bleeding site, this film compresses the capillaries and stabilizes the blood clot in place.
- Natural collagen sponge
- This is a white sponge material, non-antigenic and fully absorbable. It stimulates the platelet aggregation thereby enhancing hemostasis.
- It activates coagulation factors XI and XIII.

- It is preferred in patients who are susceptible for hemorrhage after dental surgical procedures.
- Fibrin sponge
- The fibrin sponge is non-antigenic and is obtained from bovine material.
- It is chemically treated to avoid allergic reactions.
- It is applied on the bleeding site especially in post-extraction socket.
- It stimulates coagulation thereby forming a normal clot; it also acts as a temporary plug over the small injured blood vessels.
- The fibrin sponge is fully absorbed by the tissues within 4-6 weeks.
- Bone wax
- Bone wax is a sterilized, non-absorbable mix of waxes.
- It consists of seven parts by weight of wax (white bees wax, paraffin wax and an isopropyl ester of palmitic acid), two parts of olive oil and one part of phenol.
- It is white and available as a solid rectangular plate weighing 2.5 g.
- It is indicated in cases of bleeding from the bone or from chipped edges of bone.
- The bone wax is softened with the fingers to a desirable consistency and then applied over the bleeding site.
- Its hemostatic mechanism is through mechanical obstruction of the osseous cavity containing the bleeding vessels.
- Frequent use may lead to the formation of wax granuloma (foreign body)
- Ostene (a watersoluble hemostatic agent)
- Earlier, the formulations containing naturally obtained bees wax were used as bone wax which interfered with the normal healing process and caused inflammatory reactions.
- Ostene is a new bone hemostatic agent, made of water-soluble alkaline oxide copolymers.
- With the use of the conventional bone wax, foreign body response including fibrous tissue infiltration by macrophages, giant cells and lymphocytes at the sites of the bone defects are not uncommon.
- Further bone wax also displaced the bone marrow and interfered with bone ingrowth into the defects.
- Ostene, a water-soluble bone hemostatic agent has shown no adverse tissue response or the interference with bone healing as seen with the use of bone wax.

Thermal Agents

- Surgical cutting instruments have been modified using thermal agents in order to achieve hemostasis during surgery. Delivery of heat or cold during the cutting can be done by electric current (electro cautery), laser beam (like argon), liquid nitrogen (cryosurgery), radio frequency energy etc. These thermal agents coagulate and seal the blood vessels as they cut achieving hemostasis and a bloodless field during surgery.
- Electro cautery/surgical diathermy
- Electro cautery is a surgical technique that depends on thermal effect of electric current. In electro cautery / surgical diathermy, a high frequency current is applied to a specific area of the body for the purpose of removal of unwanted tissue, coagulation, or to create a surgical incision.
- Cryosurgery
- Cryosurgery is the process of rapidly freezing tissue by exposing it to intensely low temperatures. Usually a probe containing liquid nitrogen is used. While it is not an ideal coagulating method, cryosurgery does minimize the extent of blood loss in extensive ablative surgeries.
- Effects of rapid freezing

o Increased concentration of intracellular solutes

- Reduction in intracellular water

o Cell membrane damage

o Formation of intracellular and extracellular ice crystals.

Laser helps in coagulating small blood vessels.

Conclusion

Clinicians are facing an ever-increasing number of conditions – inherited, acquired and drug-related – associated with abnormal hemostatic function. These raise the possibility of excessive blood loss, poor wound healing and infection. They must maintain clear and open communication with the patient and his hematologist. This will ensure that the dentist obtains complete information on the severity and control of the patient's condition and advice on management of the patient before, during and after surgery.

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