

GINGIVAL BLEEDING – AN INSIGHT

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Abstract: Bleeding gingiva can be a sign that the patient is at risk for gingival disease. It may be due to serious medical conditions such as leukaemia and bleeding and platelet disorders. The main cause of the gingival bleeding is the build-up of plaque at the gum line. This article explains deeply about the etiological agents, risk factors, microscopic features associated with gingival bleeding and care for oral health.

Keywords: Gingiva, gingival bleeding, gingival sulcus, leukaemia, haemostasis.

1. Introduction:

Oral health is considered as an integral part of general health which is achieved by good oral hygiene¹. Oral diseases qualify as one of the foremost public health dilemma owing to their higher prevalence in general population². Poor oral hygiene is the main leading cause for the development of oral diseases which heavily affect general health³. The plaque microorganisms which shows its effect on periodontium by releasing certain products which may cause damage to the epithelial and connective tissue. The intercellular spaces between the junctional epithelial cells are destroyed and may permit the bacterial products or bacteria themselves to gain access into the connective tissue causing gingival diseases and it further leads to Periodontitis and tooth loss⁴. Bleeding on probing and bleeding on brushing now have become a gold standard measure of gingivitis⁵. Bleeding gums and other oral diseases

are more common in people with low socio-economic status having poor oral hygiene and not aware about oral health⁶.

What is gingival bleeding?

It is a sign of gingival inflammation which indicates destruction and erosion to the line of sulcus or ulceration of sulcular epithelium. Blood comes from lamina propria which is a thin layer of connective tissue after the ulceration of the lining epithelium which is referred as gingival bleeding or bleeding gums or bleeding on probing⁷.

Significance of gingival bleeding:

Bleeding from the sulcus is the earliest symptoms of gingivitis and causes discolouration and swelling and therefore, gingival colour changes are less often during the early period of gingivitis. Bleeding from the sulcus is the **clear-cut sign of pathology**⁸.

- Bleeding on probing helps to determine whether the lesion is in an active lesion or inactive lesion. In inactive lesion, little or no bleeding while probing, whereas active lesions bleed more on probing.
- The severity of bleeding which indicates the intensity of the inflammation.

Etiological factors responsible for gingival bleeding:

Gingival bleeding can be caused due to series of factors, which can be grouped broadly in to two categories such as:

- Local factors:
 - a. Acute bleeding
 - b. Chronic or recurrent bleeding.
- Systemic factors

local factors:

1. Infectious
 - Acute:
 - a. ANUG (Acute Necrotizing Ulcerative Gingivitis)
 - b. AHGS (Acute Herpetic Gingivostomatitis)
 - Chronic:
 - a. Gingivitis
 - b. Periodontitis
2. Trauma:
 - a. Tooth brush trauma
 - b. Impaction of sharp pieces of hard food
 - c. Gingival burns from hot foods or chemicals
 - d. Biting into solid foods such as apples
 - e. Tooth pick injury
3. Post-surgical treatment

2. SYSTEMIC FACTORS:

1. Deficiencies:
 - a. Hereditary: haemophilia A & B, von Willebrand disease
 - b. Liver disease: Vitamin-k deficiency
 - c. Deficiency of factor II, VII, IX, X

- d. Idiopathic: Thrombocytopenic purpura
 - e. Leukaemia
 - f. Nutritional: Vitamin A&C, and protein deficiency
2. Dysfunction:
- a. Multiple myeloma
 - b. Systemic Lupus Erythematosus
3. Drugs & chemical allergies:
- a. Salicylates
 - b. Anticoagulants such as Dicumarol and Heparin.
4. Thrombocytosis
5. Defective platelet aggregation
6. Hereditary Haemorrhagic telangiectasia
7. Hormones: pregnancy, menstruation

GINGIVAL BLEEDING ASSOCIATED WITH SYSTEMIC CHANGES:

With some systemic disorders, gingival haemorrhage occurs spontaneously or after irritation and it is excessive and **difficult to control which is high risk.**

Disease involving specific blood factors:

Haemophilia: (Bleeder's disease, disease of the Hapsburg, the disease of Kings)

Haemophilia A is a congenital deficiency in clotting factor VIII.

Haemophilia B is a congenital deficiency in clotting factor IX.

These congenital deficiencies cannot produce sufficient thrombin in the intrinsic pathway of coagulation cascade which causes haemorrhage.

Aetiology:

Haemophilia is an X-linked recessive disorder.

Type

Clotting factor deficiency

Haemophilia A

Plasma Thromboplastinogen (antihemophilic globulin, AHG, factor VIII) Haemophilia B

Plasma Thromboplastin component (PTC, factor IX)

Haemophilia C

Plasma Thromboplastin Antecedent (PTA, factor XI)

Oral Manifestations:

Haemorrhage from many sites in the oral cavity is a common finding in haemophilia, and gingival haemorrhage may be massive and prolonged. Even the physiologic processes of

tooth eruption and exfoliation may be attended with severe prolonged haemorrhage. The problem of dental extractions is a difficult one in haemophiliacs.

Treatment and Prognosis.

There is no known cure for Haemophilia. Haemophiliac patients should be aware of traumatic injuries. Initially in early 1960's concentrates of Anti Hemophilic Globulin ⁹ and **glycine precipitated factor VIII** were used in the treatment of haemophilia ¹⁰. Then in 1970's and 80's plasma derived products and **cryoprecipitate** were used to treat haemophilia ¹¹. But this increased the incidence of HIV and Hepatitis A and B in haemophiliacs and increased the development of antibodies to factors VIII and IX ^{12,13,14}. By the beginning of 1990, **Recombinant- DNA** derived Antihemophilic factors were used to treat haemophilia ^{15,16}. Following that **Desmopressin and Immunosuppressive Therapy** were used ^{17,18}. Activated Prothrombin complex concentrate was used to treat patients with inhibitors ¹⁹. In patients with liver cirrhosis or hepatitis due to **transfusion**, liver transplantation was done which provided good results and cured factor VIII and factor IX deficiencies^{21,21,22}.

Coagulation disorders-congenital:

von Willebrand disease:(Pseudo haemophilia, vascular haemophilia, vascular purpura)

when a traumatic injury occurs, the blood vessels get injured and results in bleeding, so the platelets together with clotting factors form a plug at the site of injury and it stops bleeding. The plasma protein in the blood which helps the platelets and forms a clump is the von Willebrand factor (VWF). It also carries factor VIII. When plasma level reduced or any defects in the von Willebrand factor, blood clotting capacity reduced which leading to a continuous bleeding after an injury and it is called as Von Willebrand disease (VWD).

The name was given originally by Erik von Willebrand in 1926.

Normal vWF level is **10mg/L**, with a half-life of **6-15 hours**.90% of individuals are with vWD are unaware of their condition.

Oral manifestation:

Gingival bleeding after brushing is a common manifestation among vWD

(Von Willebrand Disease) patients. Rarely, the bleeding may be spontaneous. The disease may be discovered after dental extraction because of the prolonged and excessive bleeding. The bleeding continues indefinitely and result in an almost unmanageable flow ²³.

Treatment:

Patients scheduled for any surgical treatments can be treated with Desmopressin or infusion of combined factor VIII and vWF.

Liver disease-Vitamin K deficiency

Causes include obstructive jaundice or, less commonly, Malabsorption. In emergency, vitamin K can be given orally and its effectiveness can be checked by the Prothrombin time ²⁴.

Idiopathic Thrombocytopenic Purpura:

Acute ITP mainly affects children of any age but peaks between 1 and 6 years. It develops suddenly and resolves within 6 months

Chronic ITP often develops in adults of age 30 and 40 years and it has most insidious course than the acute form and it affects females two to three times more than males.

Oral manifestation:

The major sign is **gingival bleeding**. This haemorrhage may be spontaneous and the tendency for excessive bleeding contraindicates any oral surgical procedure, particularly tooth extraction, until the deficiency has been compensated.

Treatment:

Corticosteroids have been used and it shows good results. The prognosis for patients with this disease is fairly good and remissions are more common. As with other platelet disorders, Aspirin and other Anti-inflammatory Analgesics should be avoided ²⁵.

Leukaemia:

The primary oral manifestations of the disease may consist of gingivitis, gingival enlargement, gingival haemorrhage, petechiae and ulceration of the oral mucosa. The gingivae are boggy, oedematous and deep red which tends to bleed easily. The gingival bleeding which commonly occurs is mainly due to ulceration of the sulcular epithelium and necrosis of the underlying tissue.

Treatment:

Ideally, patients with leukaemia or any others having cytotoxic treatment, should have a proper oral hygiene to control the bacterial population, before complications develop for the patient. Mouth rinses (0.2% chlorhexidine or tetracycline and amphotericin) will often control severe gingival changes and superficial infections ²⁶.

Drugs and chemical allergies:

There are some drugs which have adverse effects that may cause gingival hemorrhage.

Patients taking anticoagulants such as Warfarin or Heparin may develop Gingival Hemorrhage and patients taking a combination of anticoagulants and antiplatelet drugs i.e, Warfarin and Clopidogrel after cardiac surgery, have an increased risk of spontaneous and prolonged gingival bleeding ²⁷. Patients on warfarin should have their International Normalised Ratio checked ²⁸

Microscopic Changes Associated with Gingival Bleeding on Probing

The following histological changes are seen:

a. Changes associated in the epithelium:

Sulcular epithelium is thinned and the microulcerations can be seen. Ulcerations of the sulcular epithelium can be due to the toxic substances released by plaque microorganisms which destroys the intercellular junctions and forms microulcerations.

b. In the connective tissue:

Dilation and engorgement of the capillaries takes place mainly in the connective tissue. Since the capillaries are engorged and closer to the surface which is already thinned and less protective, and it can cause rupture of the capillaries which may result in gingival haemorrhage.

Clinical Evaluation of gingival bleeding by gingival indices:

Papillary-marginal index :

The PMA index, given by Schour&Massler (1947) and then later it was described by Massler(1967). The index scores are based on the inflammation occurs in the interdental papilla (P) and it spreads to the marginal (M) and the attached gingiva (A). Each gingival unit is scored on the basis of score 0-4. Only the labial surfaces are examined. The number of affected units are counted for each individual and recorded.

Gingival Index (GI)

The Gingival Index given by loe and Silness in 1963 which is used to determine the gingival inflammation. This index scores the marginal and interproximal tissues of the gingiva separately on the basis of 0 to 3.

The criteria are:

0= Normal gingiva;

1= Mild inflammation – small change in colour and slight oedema and no bleeding on probing;

2= Moderate inflammation – bleeding on probing;

3= Severe inflammation – ulceration with spontaneous bleeding.

The bleeding is assessed by probing gently along the walls of the gingival sulcus. The scores are added and divided by four to give the Gingival index for the tooth²⁹

Sulcus Bleeding Index (SBI)

This is an index for assessment of gingival bleeding, developed by Muhlemann and Son.S in 1971. This index system is a modification of papillary-marginal index of muhlemann&mazor. P described the Sulcus Bleeding Index (SBI). The presence of early inflammatory gingival disease can be assessed by gentle probing on the gingival sulcus.

Gingival Bleeding Index (GBI)

In 1974, Carter and Barnes introduced this Gingival Bleeding Index, a dental flossis inserted into the interproximal gingival sulci to check the presence of gingival inflammation. By six segments the oral cavity is divided in an order of maxillary right, maxillary anterior, maxillary left, mandibular left, mandibular anterior and mandibular right. Bleeding is recorded as present or absent for the teeth.

Papillary Bleeding Index (PBI)

Saxer and Muhlemann in (1975) introduced first, and later described by Muhlemann (1977). A periodontal probe is gently inserted at the base of the papilla on the mesial aspect and the probe is moved coronally to the papilla tip and it is repeated on the distal aspect. The intensity of any bleeding is recorded as:

Score 0 – no bleeding;

Score 1 – A single discreet bleeding point

Score 2 – bleeding points or a single line of blood occurs

Score 3 – interdental areas filled with blood after probing

Score 4 – blood flows into the marginal sulcus³⁰

Eastman Interdental Bleeding Index (EIBI)

Caton & Polson in 1985 described the Eastman Interdental Bleeding Index (EIB). A small wooden interdental cleaner is inserted between the teeth from the buccal aspect, depressing the interdental area **1 to 2 mm** and it is repeated four times and the presence or absence of bleeding within 15 seconds is recorded ³¹.

Bleeding on Interdental Brushing Index (BOIB- 2010)

Whereas measures of gingival inflammation through indices of bleeding on probing can be influenced by factors such as angulation of the probe, the probe insertion depth, direction, and motion of the probe and probing force and indices that use wooden interdental cleaner may cause trauma, Hofer et al in 2010 developed the Bleeding on Interdental Brushing Index (BOIB). It is performed by inserting a light interdental brush placed buccally, just under the contact point with a jiggling motion, And the presence or absence of bleeding within 30s is recored.

Laboratory Tests for Screening:

The four basic tests which is used to detect the defect of hemostasis:

- Bleeding time
- Platelet count
- Prothrombin time
- Partial Thromboplastin time

Management and treatment for haemorrhage:

Bleeding can be controlled by

- a. Agents acting locally
- b. Transfusional agents such as specific coagulation factors
- c. Nontransfusional agents

Agents acting locally:

The agents acting locally can control the oozing of a blood from a small vessel but it cannot control large vessel.

- **Thrombin:** Thrombin is obtained from bovine plasma. Topical thrombin is often used clinically. If given intra- venously, thrombin causes extensive thrombosis and death.
- **Thromboplastin:** Thromboplastin is a powder which is used for determination of prothrombin time is determined using thromboplastin powder and it is also used as a local haemostatic in surgery.
- **Fibrin:** It is used in the dehydrated form as sheets on the bleeding surfaces which can be cut at a desired size to arrest the bleeding. When it is combined with thrombin solution, mechanical barrier is formed and holds thrombin over the bleeding site.

Transfusional agents Fibrinogen:

Fibrinogen which is derived from human plasma, is used for acute afibrinogenemia by restoring normal fibrinogen levels in that condition.

- **Antihaemophilic Globulin (Ahg):** AHG is highly effective in the treatment of classical haemophilia-A which has a concentrate of factor VIII (AHG). It is produced by recombinant DNA technique.
- **Coagulation Factors:** Pure recombinant factor VII, VIII, IX are available at very expensive rates and it may be associated with a greater risk of inducing inhibitor formation of IgG antibodies for factor VIII and thus it reduces the efficacy of specific therapy.
- **Fresh Frozen Plasma:** Fresh frozen plasma is mainly used for the clotting disorder treatment, because it contains all the clotting factors. Factor VIII which is purified and partially purified preparation containing factors II, VII, IX and X are also available for specific deficiencies³².

Non-transfusal agents

- **Vitamin K:** Vitamin K is essential for the biosynthesis of 'active' prothrombin and factors VII, IX and X.
- **Aprotinin:** aprotinin is a serine protease inhibitor that inhibits **kallikrein trypsin, chymotrypsin** and **plasmin** at a concentration level. Fibrinolysis is inhibited and it reduces the bleeding site mainly in performing surgeries.
- **Epsilon Amino Caproic Acid:** It is a water-soluble lysine which binds to the lysine binding sites reversibly on plasminogen and plasmin and inhibits binding of plasmin to fibrin. It is absorbed rapidly after oral administration.

DRESSINGS & OTHER MEASURES: Bleeding on probing or any other dento alveolar surgery can most often be controlled by applying pressure with sterile cotton gauze.

Microfibrillar collagen, made from bovine skin collagen, is used topically to arrest certain haemorrhagic conditions that do not respond to conventional methods of haemostasis. If the bleeding cannot be controlled, assessment with a physician and systemic measures are necessary³³.

Treatment for gingival bleeding:

- Plaque control.
- Diet control.
- Removal of calculus and root planning.
- Correction of restorative and prosthetic irritational factors.
- Maintain good oral hygiene
- Anti-microbial therapy
- Avoid tobacco smoking and drinking alcohol

3. Conclusion:

Poor oral hygiene is the main factor for calculus deposits which is the leading cause for bleeding gums. Proper awareness should be given to the patients to consider gum bleeding is an early sign of gingival disease and if left untreated, it leads to periodontitis and tooth loss, so regular dental visit is much recommended.

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