

Bleeding Disorders

(Part 2)

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Learning Objectives:

This Reviewing Session Aim To :

1. Provide Dentists with guided information to:

Describe the bleeding problems like:

Disorders of Primary Hemostasis

Congenital Bleeding Disorders

Acquired Bleeding Disorders

2. Provide Dentists with simple Outline of Clinical Assessment & Lab. Investigations for Bleeding Disorders and updated treatment of these Conditions.

❑ Dentists Must Know that hematologist or internist = Corner Stone for planning and implementation of treatment.

❑ Dentists Must Remember that treatment in certain circumstances should be done in a hospital setting.

Hemostasis

Means = *delicate balance* *to prevent*
both:

- ☐ *Clinical bleeding (= Hemorrhage)*
- ☐ *Hypercoagulable*
(thromboembolic) clotting syndromes.

Clotting Disorders:

***Due to problems when blood
does not clot Properly:***

- 1. Hemorrhagic syndromes***
- 2. Thromboembolic/ clotting
syndromes***

1.. Disorders of Primary Hemostasis

=Failure of Platelet Plug Formation :

1. Diseases Affecting Vessel Wall

2. Platelet Disorders

3. Von- Willebrand Disease

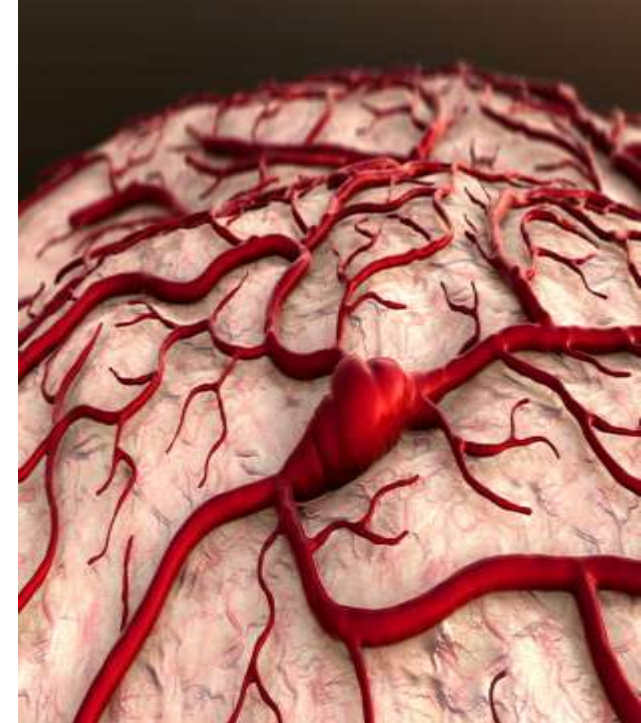
1. Diseases Affecting Vessel Wall:

Hereditary Hemorrhagic Telangiectasia:

- ☐ Autosomal Dominant Inheritance
- ☐ Arterial-Venous Malformations
- ☐ Patients present with:
 1. Recurrent bleedings (particularly Epistaxis)
 2. Iron deficiency (due to GI Bleeding).

Treatment:

1. Iron therapy = For bleedings
2. Local cautery + Laser therapy.



Tortuous veins:

- ☐ 4 mm - 5 mm in diameter = Varicose
- ☐ 1 mm - 4 mm in diameter = Reticular
- ☐ <1 mm in diameter = Telangiectasia

2. Platelet Disorders

Caused by:

A. Deficiency

B. Abnormal Function

A) Main Causes of Platelet Count Deficiency (=Thrombocytopenia):

□ Decreased Production:

□ Diseases / Conditions Affecting Bone Marrow :

- 1. Aplastic an emia**
- 2. Chemotherapy**
- 3. Radiotherapy**
- 4. Metastatic diseases**

□ Viral infections: (HIV, CMV, Rubella)

□ Drugs.

□ Increased t destruction:

- 1) Immunological Attacks (Idiopathic Thrombocytopenic Purpura (ITP)**
- 2) Splenomegaly**
- 3) Disseminated Intravascular Coagulation (DIC).**

Clinical Features of Platelet

Deficiency:

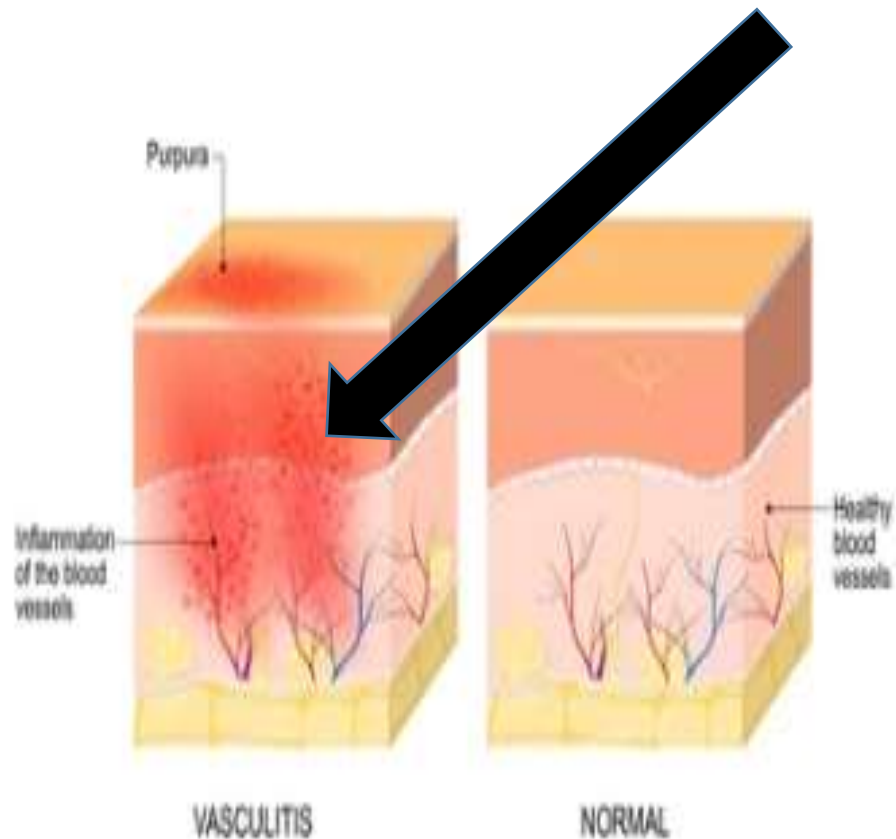
Petechia, Bruising, Ecchymosis and Bleeding (oral mucosa and skin) & Post-

Management = Platelet Infusion:

Indications:

- 1. A platelet count $< 10\,000$ cells/mm³.**
- 2. Persistent bleeding = Epistaxis.**
- 3. Life-threatening bleeding = GI Hemorrhage.**

Purpura:= (purple spots in the skin due to small vessel bleeding → Blood collect under the tissue in larger flat areas → When purpura spots are less than 4 mm in diameter, they are called Petechiae= (tiny purple, red or brown spots in the skin due to tiny blood vessel breakage



Petechial Skin problem





**Purpura spots
larger than 1 cm
(centimeter) are
called ecchymosis.**



Picture of a ecchymosis.



B) Abnormal Platelet Function=

1. Thrombasthenia = Defective Platelet Aggregation = Autosomal Recessive Inheritance.

Management:

1..local mechanical measures

2.. Anti-fibrinolytic Drugs.

3.. Platelet transfusion.

2. Drugs=

☐ **Aspirin + Clopidogrel = These drugs permanently affect the platelets for 7–10 days**

☐ **NSAIDs = These drugs permanently affect the platelets for 1–2 days**

3. Idiopathic Thrombocytopenic Purpura (ITP) =

Autoantibodies against platelets → platelet destruction

Management:

1. Prednisolone.

2. Platelet transfusion>>> Life-threatening bleeding.

3. Splenectomy >>> patients with relapsing disease.

3) Von Willebrand disease (vWD):

Autosomal Dominant Inheritance

❑ Deficiency of von Willebrand factor (vWF)= Important for:

1. Platelet aggregation
2. Platelet adhesion to damaged endothelium
3. As a carrier for factor VIII.

Management:

- 1) Mild hemorrhage: Tranexamic acid or Desmopressin :
stimulate release of von Willebrand factor (vWF) from the endothelial cells (+ subsequent increase in factor VIII)
- 2) Severe bleeding: factor VIII concentrates.

2. Congenital

Bleeding

Disorders

1.. Hemophilia A=

(Factor VIII deficiency):

= 10 times > hemophilia B

☐ **X-linked recessive Inheritance:**

=Affects males only

= females are carriers.

Normal factor VIII plasma level =

1 unit /ml= 100%.

Classification of

Hemophilia A=

(Factor VIII deficiency)

- 1) **Mild**= 5-30% of the Normal plasma Units
- 2) **Moderate**= 1-5% of the Normal plasma Units
- 3) **Severe** = less than 1% of the Normal plasma Units

☐ **Clinical Diagnosis of Hemophilia** **A=**

- 1) Babies after the age of 6 months = become experience bruising= more mobile.**
- 2) Bleeding at any site = joints / muscles / Intracranial hemorrhage.**
- 3) Severe prolonged bleeding after Dental extractions**

☐ **Laboratory Findings of Hemophilia** **A=**

- 1) Prolonged PTT**
- 2) Reduced levels of factor VIII.**

Management of Hemophilia A=

☐ **In mild Bleeding Episodes Like=**

☐ **To treat a mild bleed**

☐ **To Cover minor surgery (dental extraction).**

Anti-Fibrinolytic Agents = Tranexamic Acid.

☐ **In Severe Bleeding Episodes =**

Intravenous factor VIII concentrate

□ **Hemophilia B=** (Christmas disease)=

(Deficiency of **Factor IX**)

□ **X-linked recessive Inheritance:**

□ **Management of Hemophilia B=**

□ **Replacement therapy = synthetic factor IX.**

□ Hemophilia C = (Deficiency of Factor XI) =

□ Autosomal dominant Inheritance:

□ Results in rapid fibrinolysis.

□ Management of Hemophilia C =

1) Fresh-frozen plasma

2) Factor XI is required.

3. Acquired Hemophilia=

Affected females = Affected males

☐ **CAUSE= Unknown origin**

☐ **By Circulating Antibodies to Factor VIII**

☐ **Rare disorder= found in autoimmune disorders: such as [Rheumatoid Arthritis].**

4. Acquired Bleeding Disorders

Main Cause =

Anticoagulant Therapy

= When Used As Prophylaxis or Treatment of the thromboembolic events:

- 1) Atrial fibrillation**
- 2) IHD= Ischemic heart disease**
- 3) MI= Myocardial infarction =**
- 4) DVT= Deep vein thrombosis**
- 5) CVA= Cerebrovascular accident**
- 6) Pulmonary embolism.**

The common anticoagulant drugs are:

- 1) Warfarin = long-term treatment**
- 2) Heparin = short-term treatment**

Heparin:

- ❑ Block conversion of fibrinogen to fibrin.
- ❑ Anticoagulant Effect last for 6 hours
- ❑ TEST= Prolonged PT + PTT

Warfarin:

- ❑ Anticoagulant Effect last for 72 hours
- ❑ Vitamin K antagonist= Inhibits vitamin K-dependent synthesis of clotting factors= II, VII, IX and X, protein C and protein S).
- ❑ TEST= Prolonged PT + INR

Acetyl Salicylic Acid (Aspirin):

- **Most Common Antiplatelet Agents**
- **Inhibits platelet aggregation**
- **= TEST= Increased Bleeding Time .**

Clopidogrel (Plavix)

- **Common Antiplatelet Agents.**
- **Inhibits platelet aggregation**
- **= TEST= Increased Bleeding Time .**

Scurvy:

❑ Vitamin C deficiency

❑ Affects normal synthesis of collagen

❑ Causes Bleeding Disorders like =

1. Petechia

2. Bruising

3. Sub-periosteal bleeding.

❑ Diagnosis = History of Dietary Vitamin C deficiency .

Severe Liver Diseases:

Bleeding Causes:

1. Reduced Synthesis of Coagulation Factors
2. Cholestatic Jaundice → Reduces vitamin K absorption → Deficiency of factors II, VII, IX and X

Treatment → parenteral vitamin K

Advanced Renal Failure :

Platelet dysfunction [GI bleeding].

**Thank You For
Your Attention**