Hemostasis and Bleeding Disorders (Part 2)

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Learning Objectives:

By the end of this session, you will be able to:

1. Describe Disorders of Primary Hemostasis.
2. Describe Congenital Bleeding Disorders.
3. Describe Acquired Bleeding Disorders.
4. Outline Clinical Assessment of Hemostasis.
5. Outline Investigations for Bleeding Disorders.

Bleeding Disorders:

If blood does not clot sufficiently

=

due to clotting problems.

This Reviewing Session Aim To :

1. **Provide Dentists with guided information on how to address the bleeding problems**
2. **Providing simple and updated treatment of these Conditions.**

❑Must:

- ❑ **Not forget that hematologist or internist who controls the patient's medical condition, is a cornerstone for planning and implementation of treatment.**

- ❑**Remember that in certain circumstances: treatment should be performed in a hospital setting.**

Hemostasis means the whole body's physiological processes whose ultimate goal is to prevent blood loss on altering the integrity of the vascular system structures.

If this delicate balance is disturbed it appears = both clinical bleeding (Hemorrhagic Diathesis) + Hypercoagulable (thromboembolic) syndromes.

1.. Disorders of Primary Hemostasis

=Failure of Platelet Plug Formation :

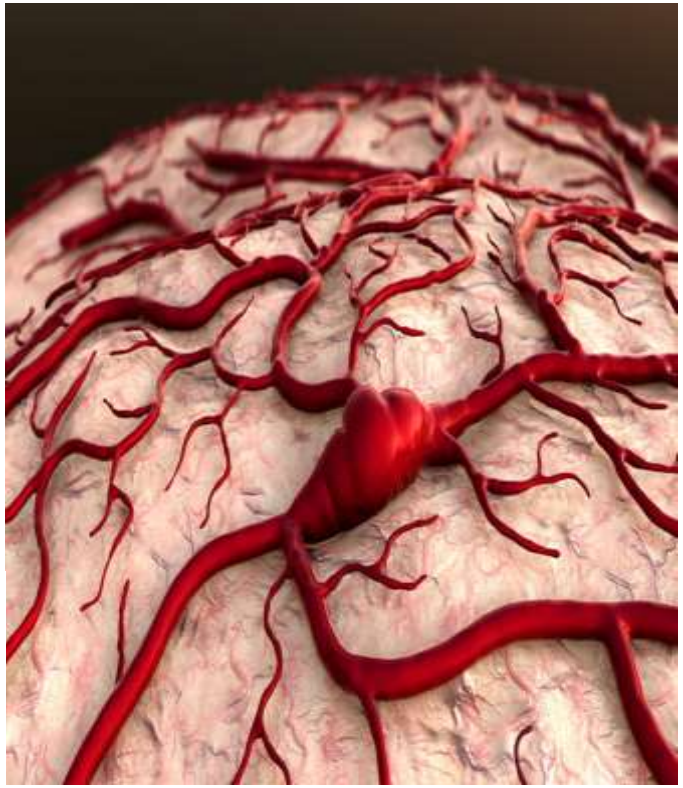
1. Diseases Affecting Vessel Wall
2. Platelet Disorders
3. Von- Willebrand Disease

1. Vessel Wall Abnormalities:

Hereditary Hemorrhagic Telangiectasia:

- ❑ **Autosomal Dominant Inheritance**
- ❑ **Abnormalities of vascular modeling + Larger arterio-venous malformations**
- ❑ **Patients present with:**

1. **Recurrent bleedings (particularly Epistaxis)**
2. **Iron deficiency (due to occult GI Bleeding).**



Treatment:

1. **Iron therapy**
2. **Local cautery**
3. **Laser therapy to prevent bleeding.**

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:

1) **Platelet Deficiency**

2) **Abnormal Platelet Function**

3) **Abnormal Platelet Distribution.**

1)Thrombocytopenia (Low Platelet Counts):

Main Causes:

- **Decreased Platelet Production:**
  - **Diseases / Conditions Affecting Bone Marrow :**

1) **Aplastic anemia**
2) **Chemotherapy**
3) **Radiotherapy**
4) **Metastatic diseases**

- **Viral infections:** (HIV, CMV, Rubella)
- **Drugs.**
- **Increased platelet destruction:**

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

Clinical Features of Platelet Deficiency:

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

= When Platelet Count = *< 20 000 cells/mm^3.*

Management:

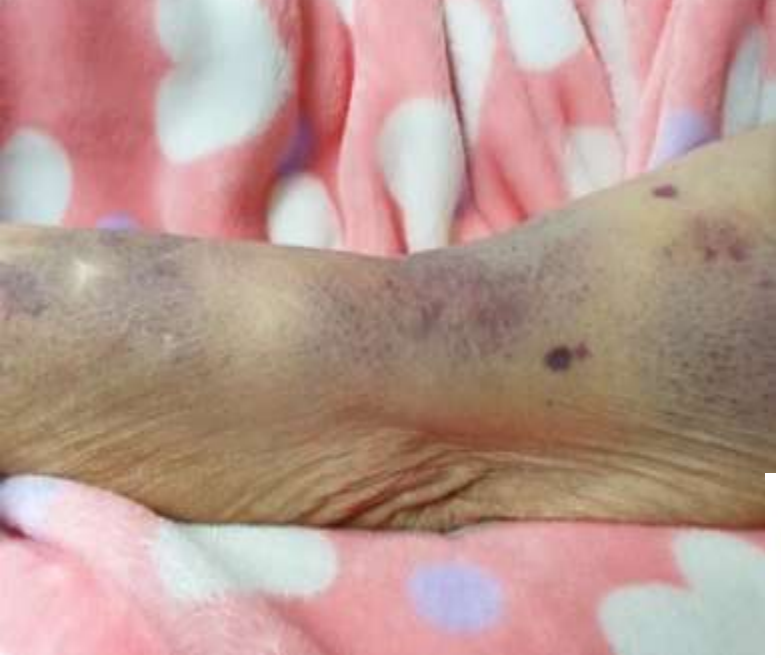
Platelet Infusion:

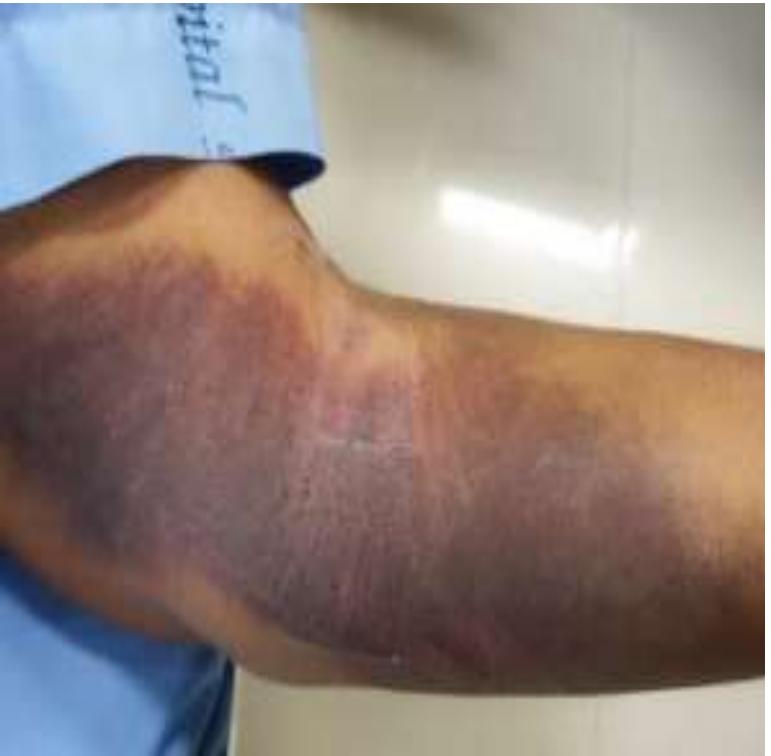
- **Risk of infection = blood- borne agents**

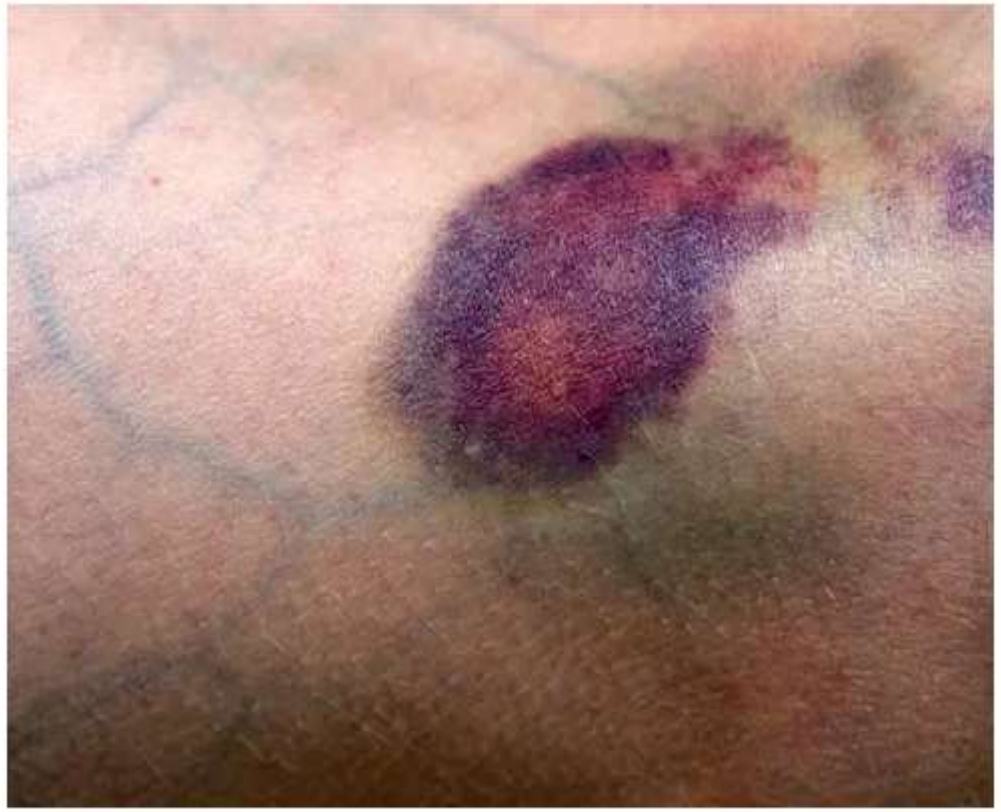
- **Transfusions provide only temporary relief because survival of platelets is a few days**

Indications for platelet transfusion include:

1. **A platelet count < 10 000 cells/mm^3.**
2. **Troublesome bleeding = Persistent Epistaxis.**
3. **Life-threatening bleeding = GI Hemorrhage.**

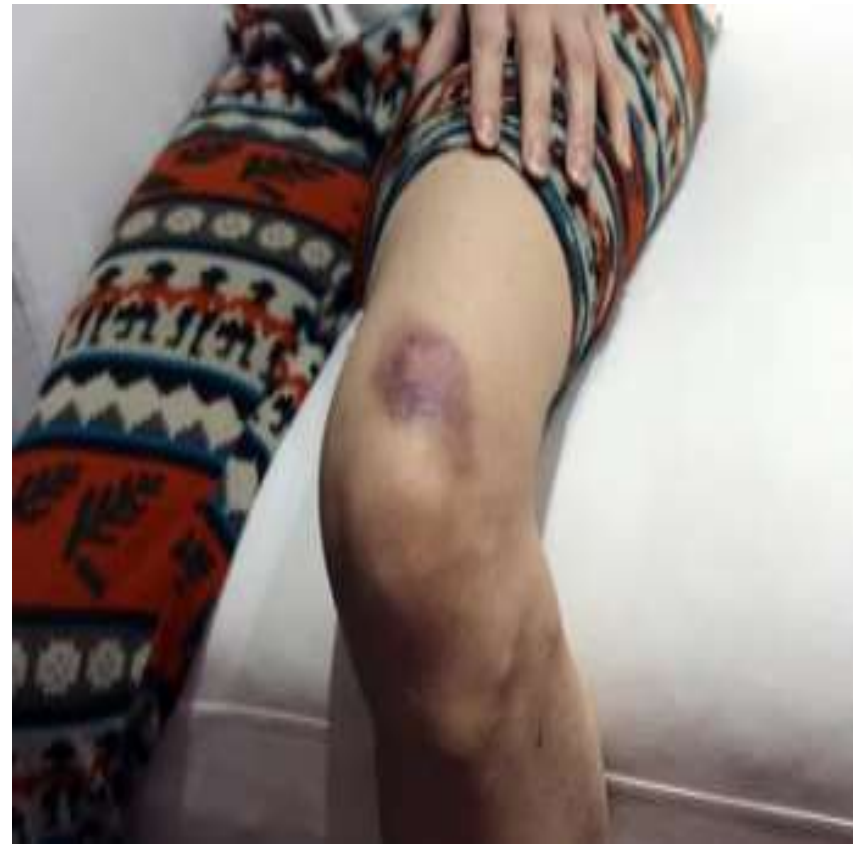




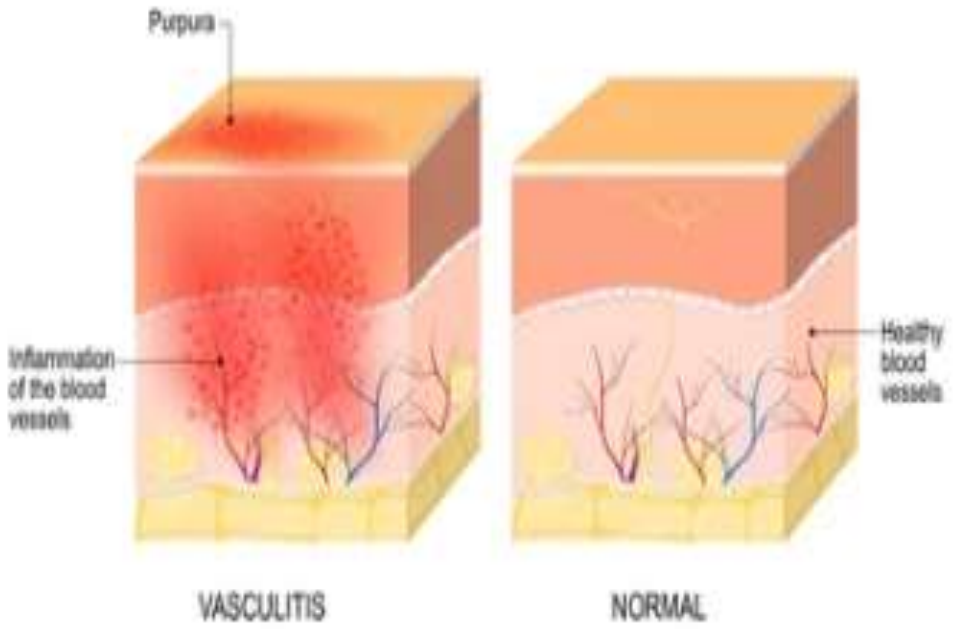


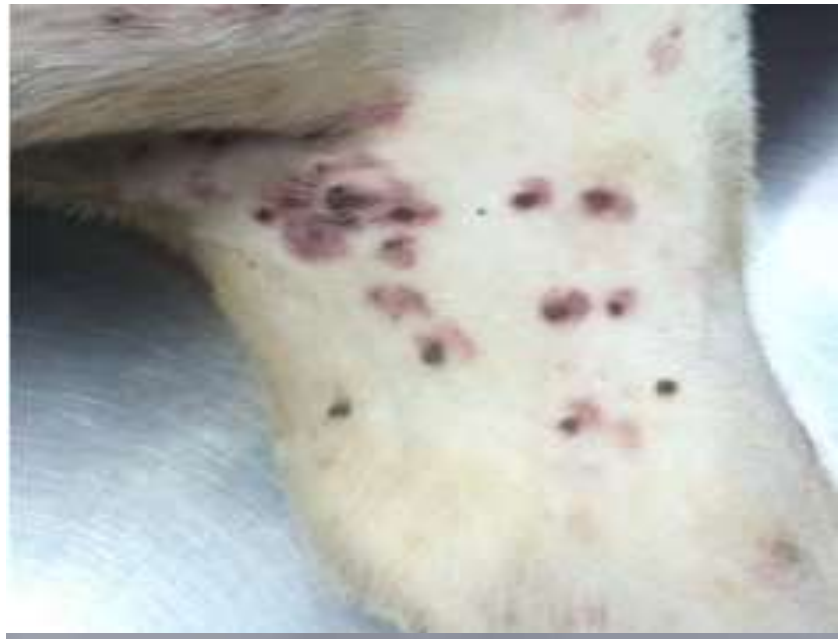
Picture of a ecchymosis.


BRUISE



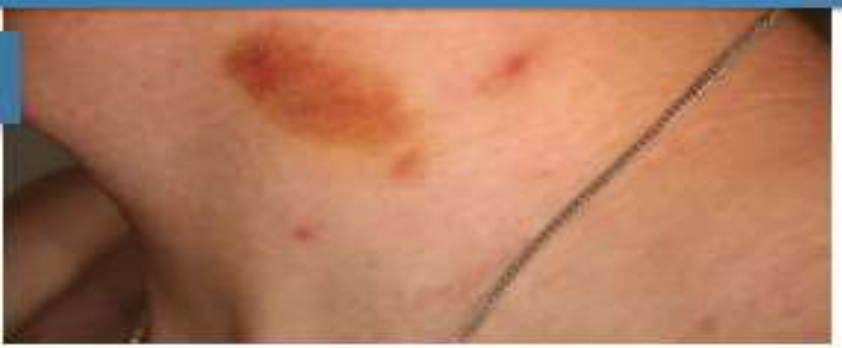
Petechiae= (tiny purple, red or brown spots in the skin due to tiny blood vessel **breakage**



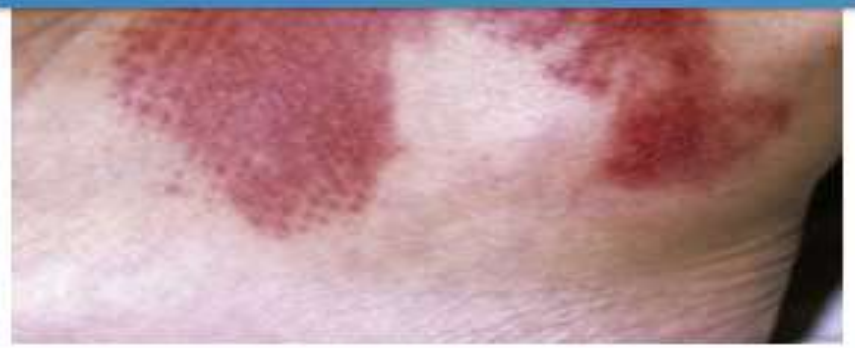


Petechial Skin problem

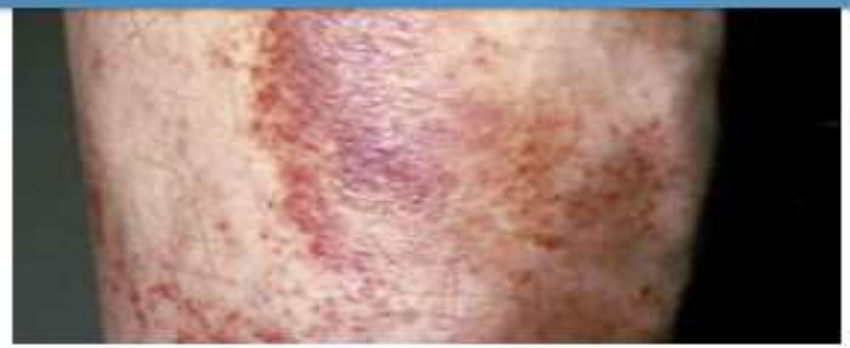
Different Causes of Purpura



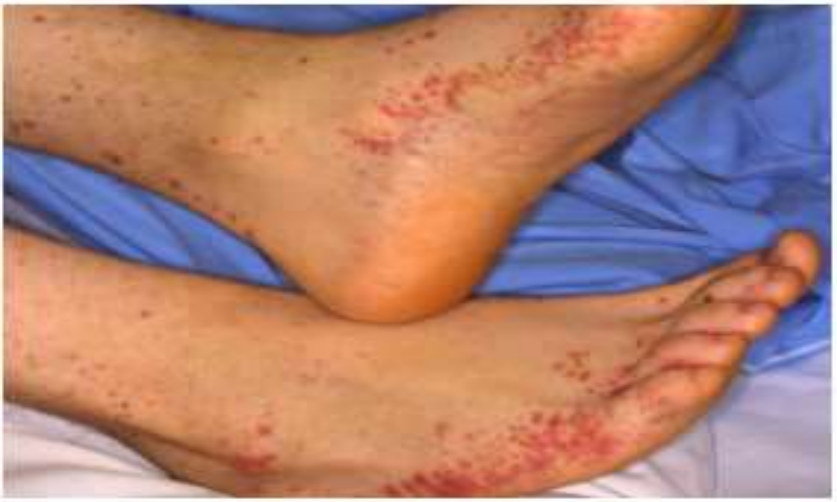
Suction bruise



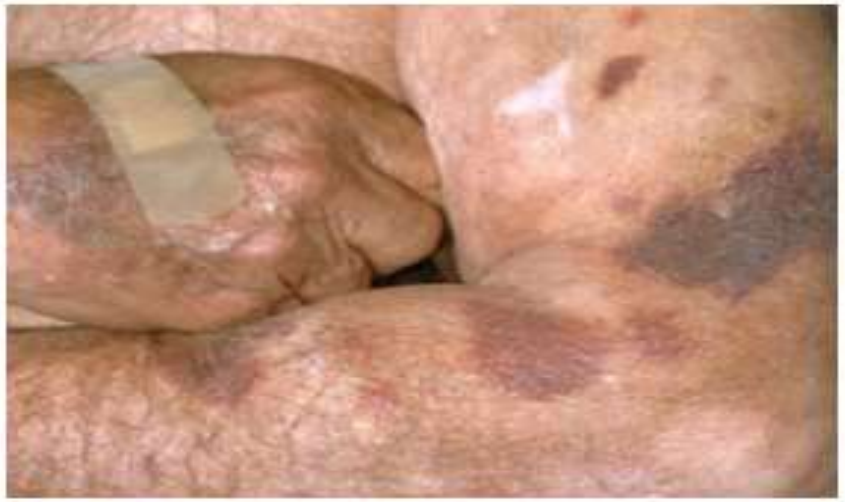
Injury



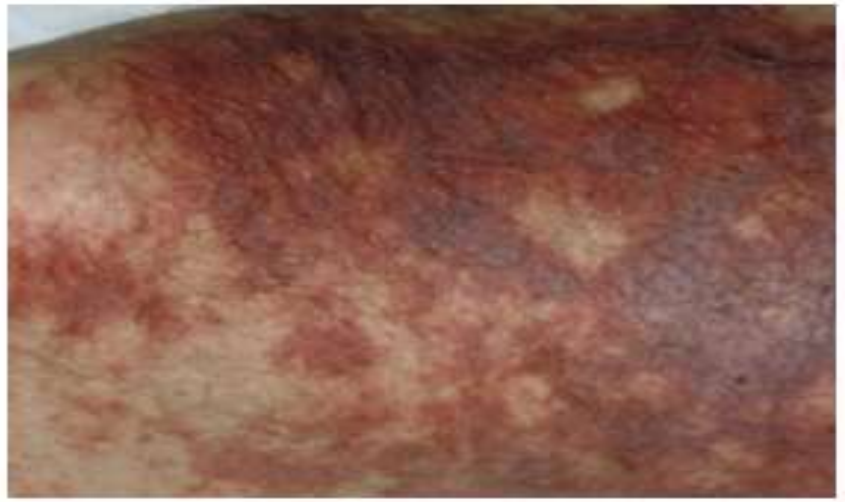
Venous stasis



Vasculitis



Steroid purpura



Disseminated intravascular coagulation

Purpura:= (purple spots in the skin due to small vessel bleeding).

2) Abnormal Platelet Function=

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

Management:

1..local mechanical measures

2.. Anti-fibrinolytic (Tranexamic acid)

3.. Platelet transfusion [severe bleeding] .

2. Drugs= *NSAIDs, aspirin and clopidogrel* = The platelet count is not affected by any one of these drugs….These drugs (except NSAIDs)= permanently affect the platelets for 7–10 days while The NSAIDs is temporary lasting 1–2 days .

3. Idiopathic Thrombocytopenic Purpura (ITP) =

Autoantibodies against platelets = platelet destruction / more commonly affects females / insidious onset.

Management:

1. **Oral prednisolone.**
2. **Platelet transfusion>>> Life-threatening bleeding.**
3. **Splenectomy >>>. patients with relapsing disease.**

3) Von Willebrand disease (vWD):

=Called PseudoHemophilia.

- **Autosomal Dominant Inheritance**
- **Deficiency of von Willebrand factor (vWF) which:**

1. ***Mediates platelet aggregation***
2. ***Mediates platelet adhesion to damaged endothelium***
3. ***Acts as a carrier for factor VIII.***

Management:

1) Mild hemorrhage: Tranexamic acid or Desmopressin :

Desmopressin stimulates release of von Willebrand factor (vWF) from the endothelial cells (with subsequent increase in factor VIII= 3 to 5-fold)

2) Severe bleeding: factor VIII concentrates.

Congenital Bleeding Disorders

1.. Hemophilia A= (Factor VIII deficiency):

10 times more common than hemophilia B

X-linked recessive Inheritance:

=Affects males only //// females are carriers.

Normal plasma= contains 1 unit of factor VIII/ml

= This factor VIII level is defined as 100%.

❑ *Classification of Hemophilia A is according to the level of factor VIII :*

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 =

1) Babies after the age of 6 months = become more mobile and experience bruising.
2) Bleeding at any site BUT joints and muscles are the most common sites / Intracranial hemorrhage is often fatal.
3) Abnormal prolonged bleeding after Dental extractions

All Laboratory Investigations Findings:

=Normal…. Except:

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

Management of Hemophilia A:

- **In mild hemophilia=**

Anti-Fibrinolytic Agents = Tranexamic Acid.

1. **Treat a mild bleed**
2. **Cover minor surgery such as dental extraction.**

- **In Severe Bleeding Episodes =**

Intravenous factor VIII concentrate = prepared by recombinant technology=

more expensive= but much safer than those derived from plasma

(= Because many patients infected with hepatitis B, hepatitis C and HIV)

❑ Hemophilia B = (Christmas disease)≡ (Deficiency of Factor IX)

❑X-linked recessive Inheritance:

Clinically Not distinguished from Hemophilia A

less common than it.

❑Management of Hemophilia B:

❑ **Replacement therapy with synthetic factor IX.**

- ***Hemophilia C = (Deficiency of Factor XI)=***
- **Autosomal dominant Inheritance:**
- **This deficiency results in rapid fibrinolysis.**
- **Management of Hemophilia C:**

1) **Fresh-frozen plasma**
2) **Factor XI is required.**

Acquired Hemophilia

- Rare disorder
- **CAUSE:** Circulating Antibodies to Factor VIII
- Unknown origin
- **But:** Rarely found in autoimmune disorders: such as [Rheumatoid Arthritis].
- **In contrast to congenital hemophilia=**
- **affected females = affected males.**

Acquired Bleeding Disorders

Main Cause = Anticoagulant Therapy

= Prophylaxis or Treatment of thromboembolic events:

1) **Atrial fibrillation**
2) **IHD**
3) **MI**
4) **DVT**
5) **CVA**
6) **Pulmonary embolism.**

IHD: Ischemic heart disease

MI: Myocardial infarction

DVT: Deep vein thrombosis

CVA: Cerebrovascular accident

The common anticoagulant drugs are:

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

❑Heparin:

- ❑ Soon = block conversion of fibrinogen to fibrin.
- ❑ Anticoagulant Effect lost after 6 hours
- ❑ TEST: Prolonged PT + PTT

Warfarin:

- ❑ Effect begins after 8 hours and persists for 72 hours
- ❑ Vitamin K antagonist
- ❑ Inhibits the 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 Anti-Platelet agent.
- Inhibits platelet aggregation
- = TEST: Increased Bleeding Time

Clopidogrel (Plavix)

- ❑ Common Antiplatelet Agents.
- ❑ Mechanism of action: prevent platelet aggregation.

Scurvy:

- Vitamin C deficiency
- Affects normal synthesis of collagen
- **Bleeding Disorders =**

1. Petechia
2. Bruising
3. Sub-periosteal bleeding.

- **Diagnosis = Dietary history.**

Severe Liver Diseases:

Bleeding For Many Causes:

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

>>>>>>>>>treated with parenteral vitamin K<<<<<<<<<<<<<<<<<<<

Advanced Renal Failure :

Platelet dysfunction [GI bleeding… especially].

Thank You For Your Attention