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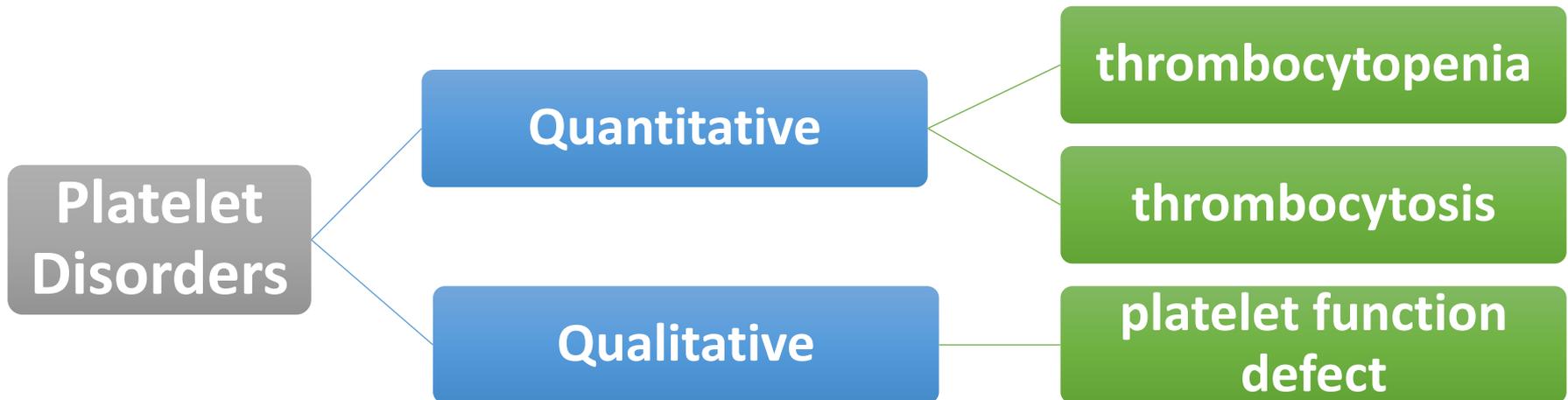
# Platelet Disorder

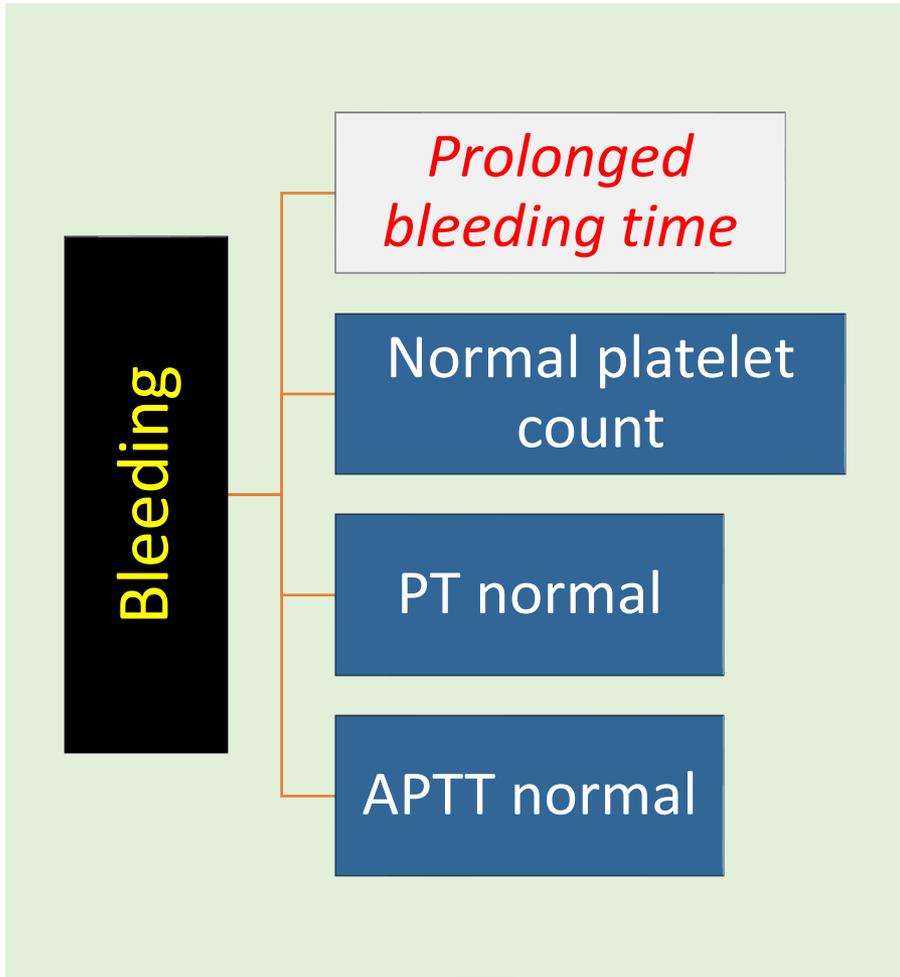


Departemen Patologi Klinik dan Kedokteran  
Laboratorium FKKMK UGM/RSUP Dr. Sardjito  
Yogyakarta

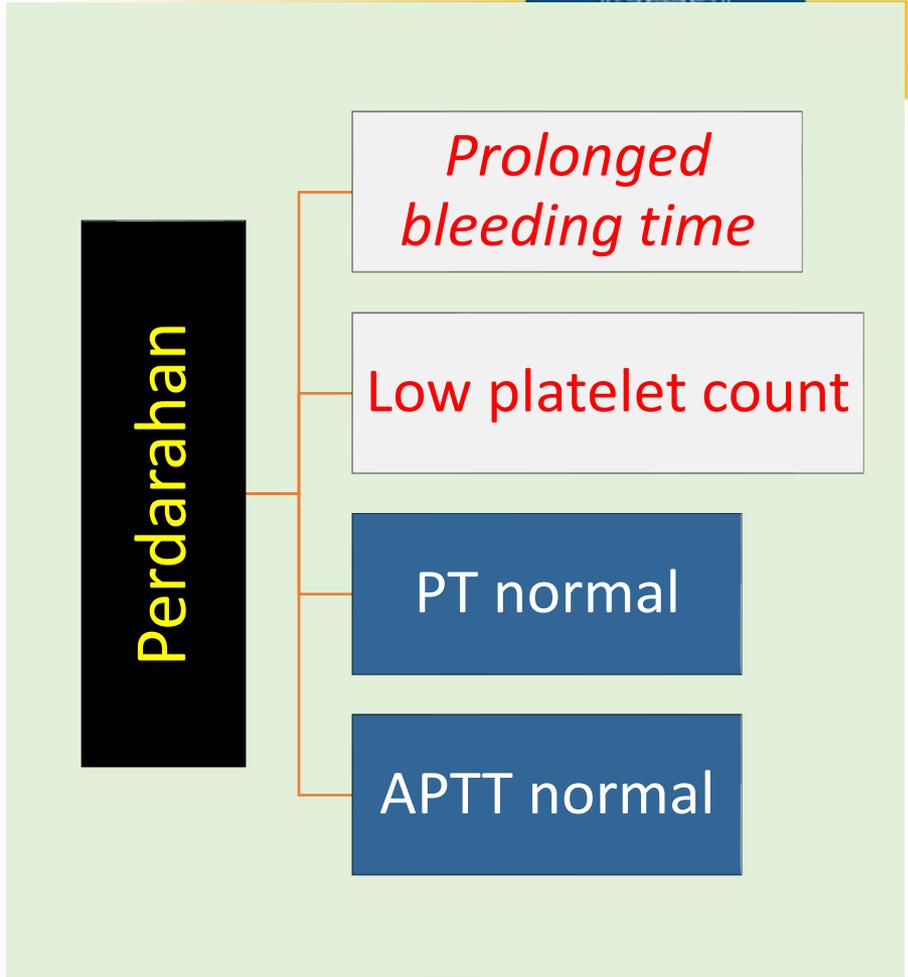
2020

# PLATELET DISORDERS





Platelet dysfunction



Thrombocytopenia

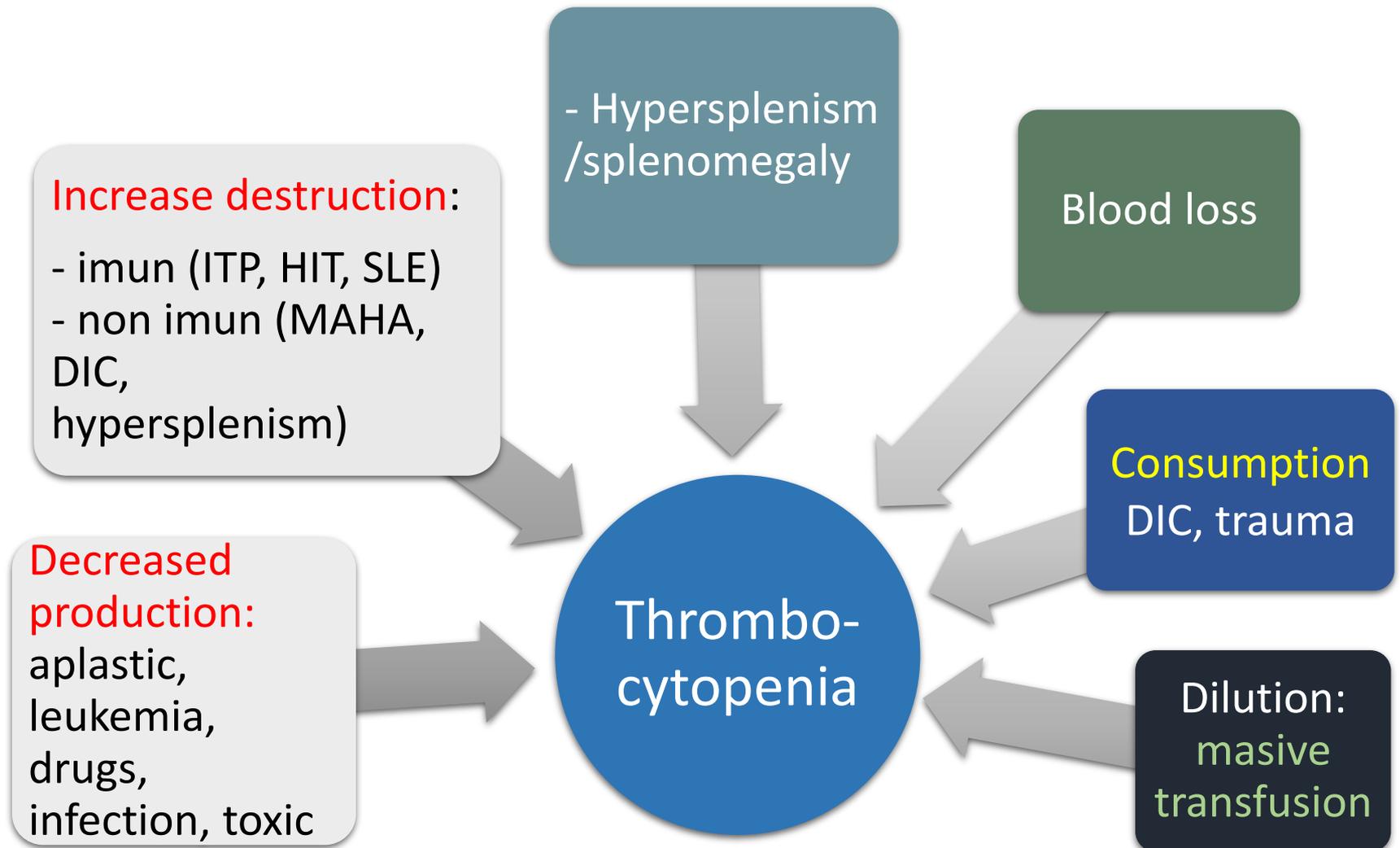
# Thrombocytopenia

- Reduction in platelet number **< 100.000/uL**
- However, **spontaneous bleeding does not become evident until the count falls below 20.000/uL**
- Spontaneous bleeding associated with thrombocytopenia most often involves **small vessels**
- **The common sites:**
  - skin and the mucous membranes of the gastrointestinal and genitourinary tracts
  - Intracranial bleeding is a threat to any patient with a markedly depressed platelet count





# THROMBOCYTOPENIA & ETIOLOGY



# THROMBOCYTOPENIA (Decreased production)

- **Decreased megakaryocytes**
  - Normal platelet life span
  - Good response to platelet transfusion
- **Neoplastic Causes**
  - Leukemias
  - Aplastic Anemia
  - Metastatic Carcinoma
  - Drugs
  - Radiotherapy
- **Primary Marrow Disorders**
  - Megaloblastic Anemias
  - Myelodysplastic syndromes
  - Myeloproliferative diseases
  - Some congenital syndromes

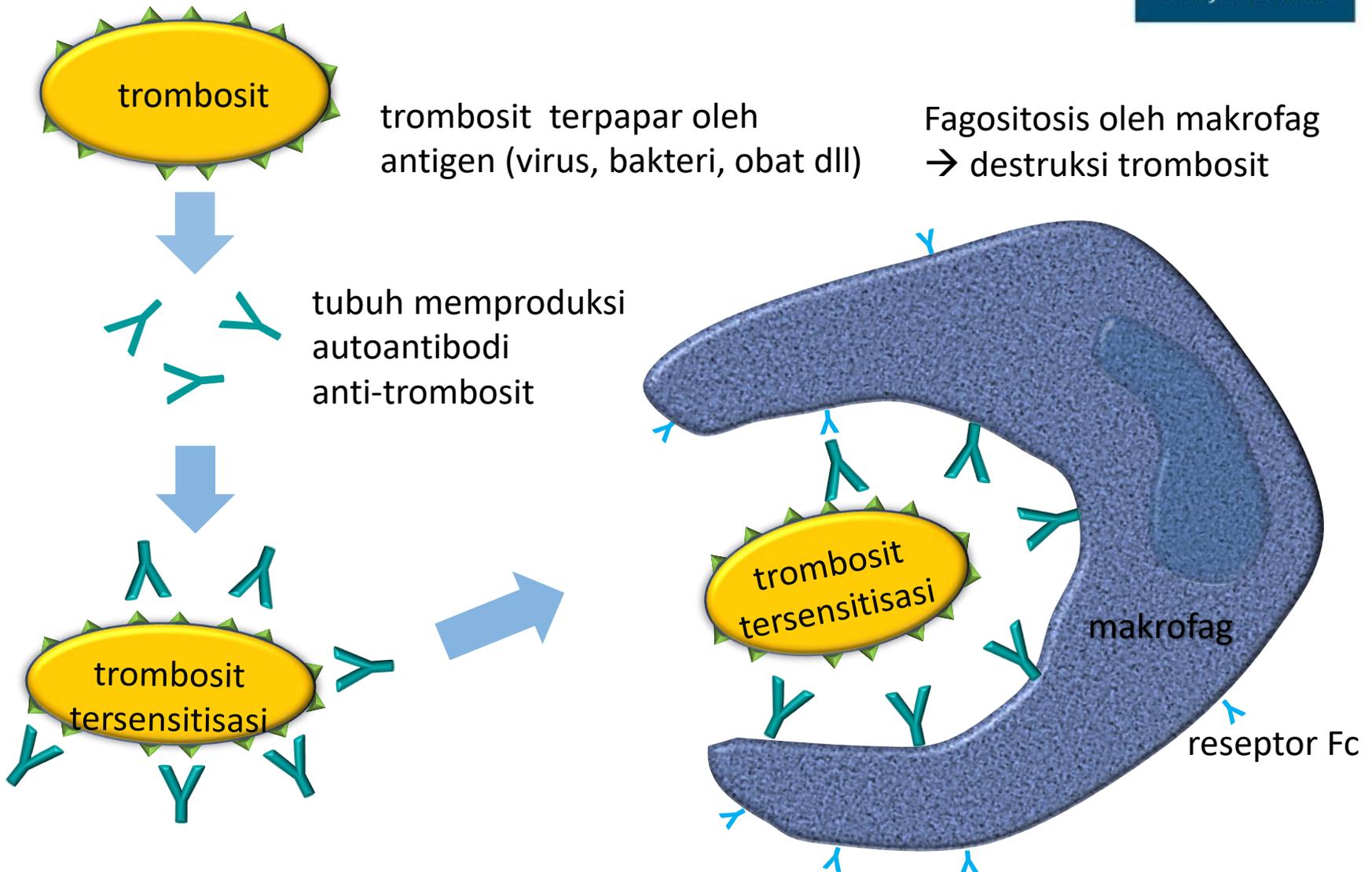
# THROMBOCYTOPENIA (Increased Destruction)

- **Increased megakaryocytes**
  - Shortened platelet life span
  - Macroplatelets
  - Poor response to platelet transfusion
- **Causes**
  - **Immune**
    - ITP
    - Lymphoma
    - Lupus/rheumatic diseases
    - Drugs
  - **Consumption**
    - Disseminated intravascular coagulation
    - Thrombotic thrombocytopenic purpura
    - Hemolytic/uremic syndrome
  - **Septicemia**

# Immune Thrombocytopenia Purpura (ITP)

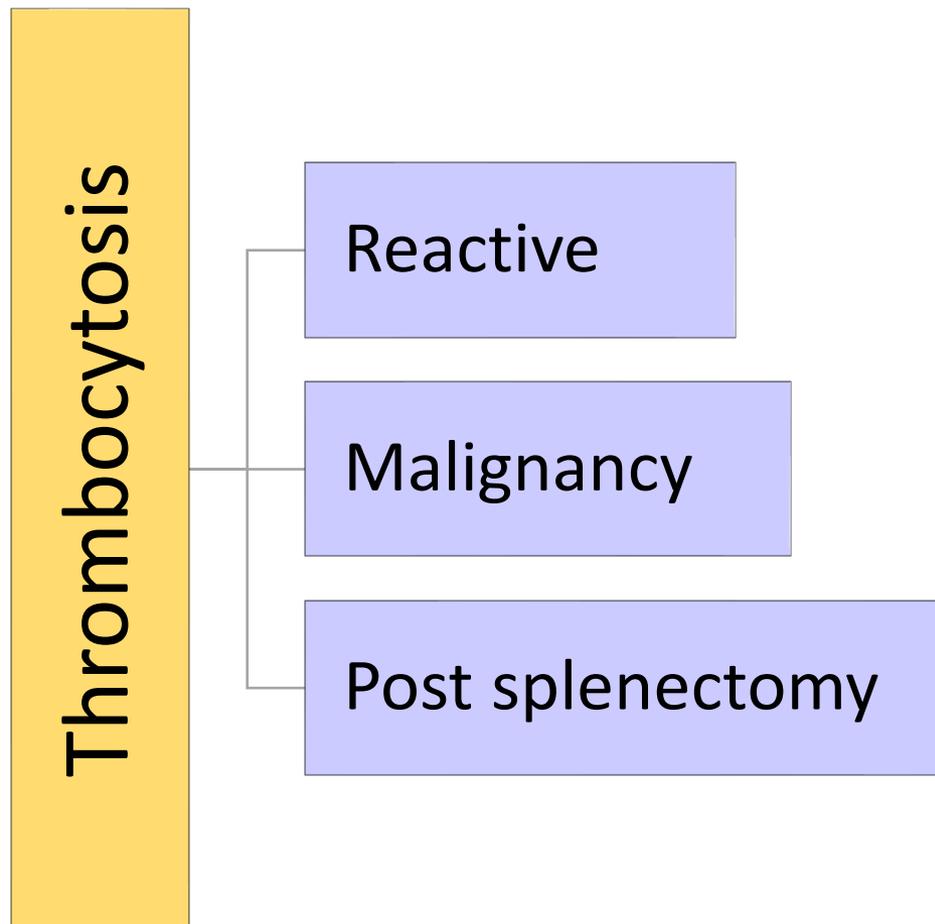
- The most common cause of thrombocytopenia due to increased **immune destruction of platelets**
- Acute and Chronic
- **Laboratory findings:**
  - **Platelet count: <<**
  - **Bleeding time: >>**
  - **PT and APTT: Normal**

# Immune Thrombocytopenia Purpura (ITP)





# THROMBOCYTOSIS & ETIOLOGY



# Thrombocytosis



**Table 1**

## **Causes of Reactive Thrombocytosis**

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**Iron Deficiency**

**Inflammatory Diseases**

**Malignancy**

**Chronic Infections**

**Drugs (vinca alkaloids, epinephrine)**

**Post-splenectomy (or functionally asplenic states)**

**Rebound thrombocytosis (following acute blood loss, post-splenectomy)**

**Myelodysplastic syndromes (5q- syndrome, Sideroblastic anemia)**

**Table 2**

## **Myeloproliferative Disorders Associated with Thrombocytosis**

**Disorder**

**Distinguishing Feature**

**Chronic Myelogenous Leukemia**

**Bcr-abl gene rearrangement**

**Polycythemia Vera**

**Elevated red blood cell mass**

**Agnogenic Myeloid Metaplasia**

**Marrow fibrosis**

**Primary Thrombocythemia (Essential Thrombocytosis)**

**Elevated platelet count only**



# PLATELET FUNCTION DEFECTS

- Congenital
- Drugs
- Alcohol
- Uremia
- Hyperglobulinemias
- Fibrin/fibrinogen split products
- Thrombocythemia
- Cardiac Surgery

# PLATELET FUNCTION DEFECTS (Congenital)

- Bernard-Soulier disease (Decreased platelet adhesion)
- Glanzmann's thrombasthenia (Decreased platelet aggregation)
- $\gamma$  or  $\delta$ -storage pool disease (Defective platelet release)
- Gray platelet syndrome (Defective platelet release)
- Von Willebrand Disease

# PLATELET FUNCTION DEFECTS

## Inherited

Defect in Platelet Adhesion:

von Willebrand disease  
and Bernard-Soulier  
syndrome

Defect in Platelet Aggregation:

Glanzmann  
thrombasthenia

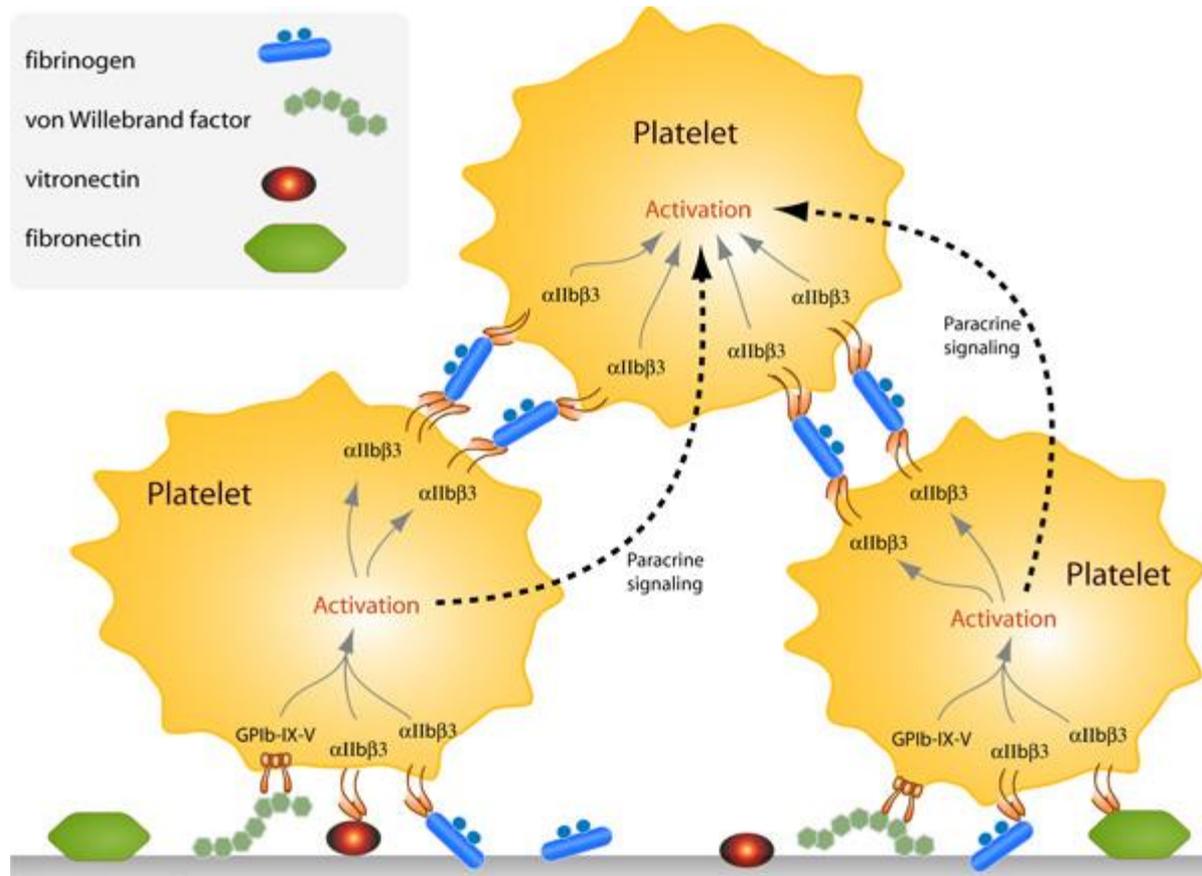
## Acquired

Drugs (**aspirin** and  
**NSAIDs**)

Toxins (**uremia**)

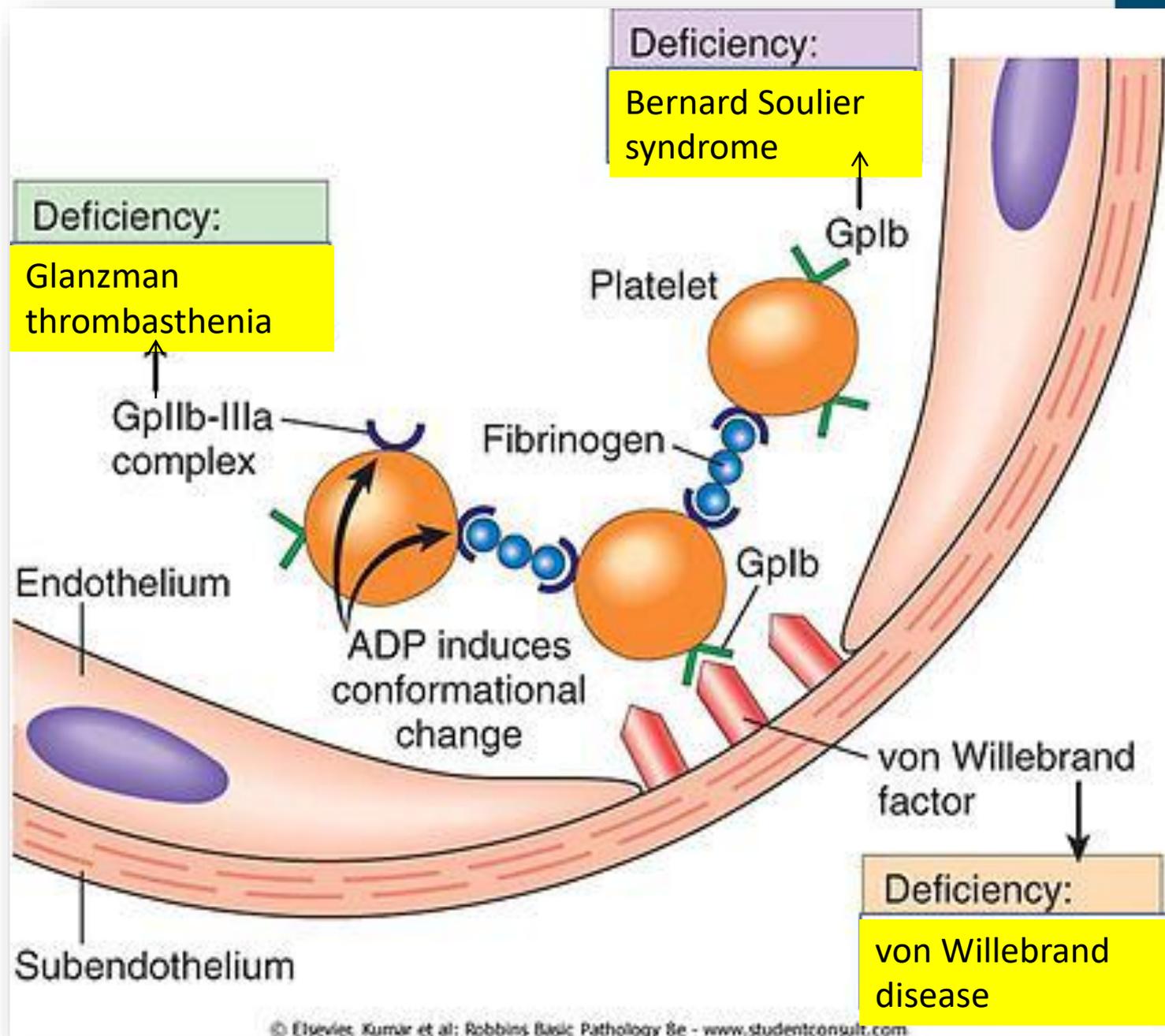


# Platelet receptors





# Inherited disorders of Platelet

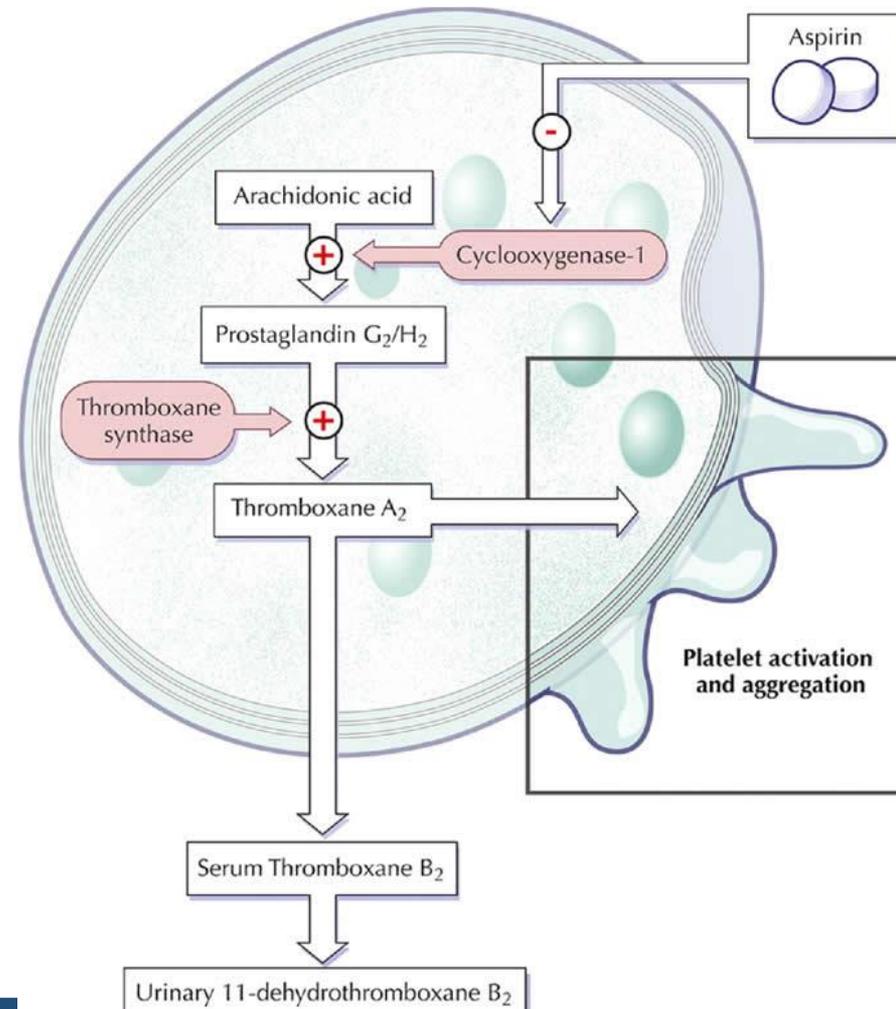


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# Acquired defect of platelet function

- **Aspirin** = irreversible inhibition of COX-1 → risk of hemorrhages that continue 3-7 days after stopping the administration
- **NSAID** = reversible inhibition of COX-1 (during the treatment period)







## Lab. Findings in Von Willebrand disease

- Platelet count: normal
- Bleeding time: >>
- APTT: Normal, (type 3b → APTT >>)
- Normal PT
- Platelet aggregation: <<
- VWF Ag: <<



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Thank You 😊