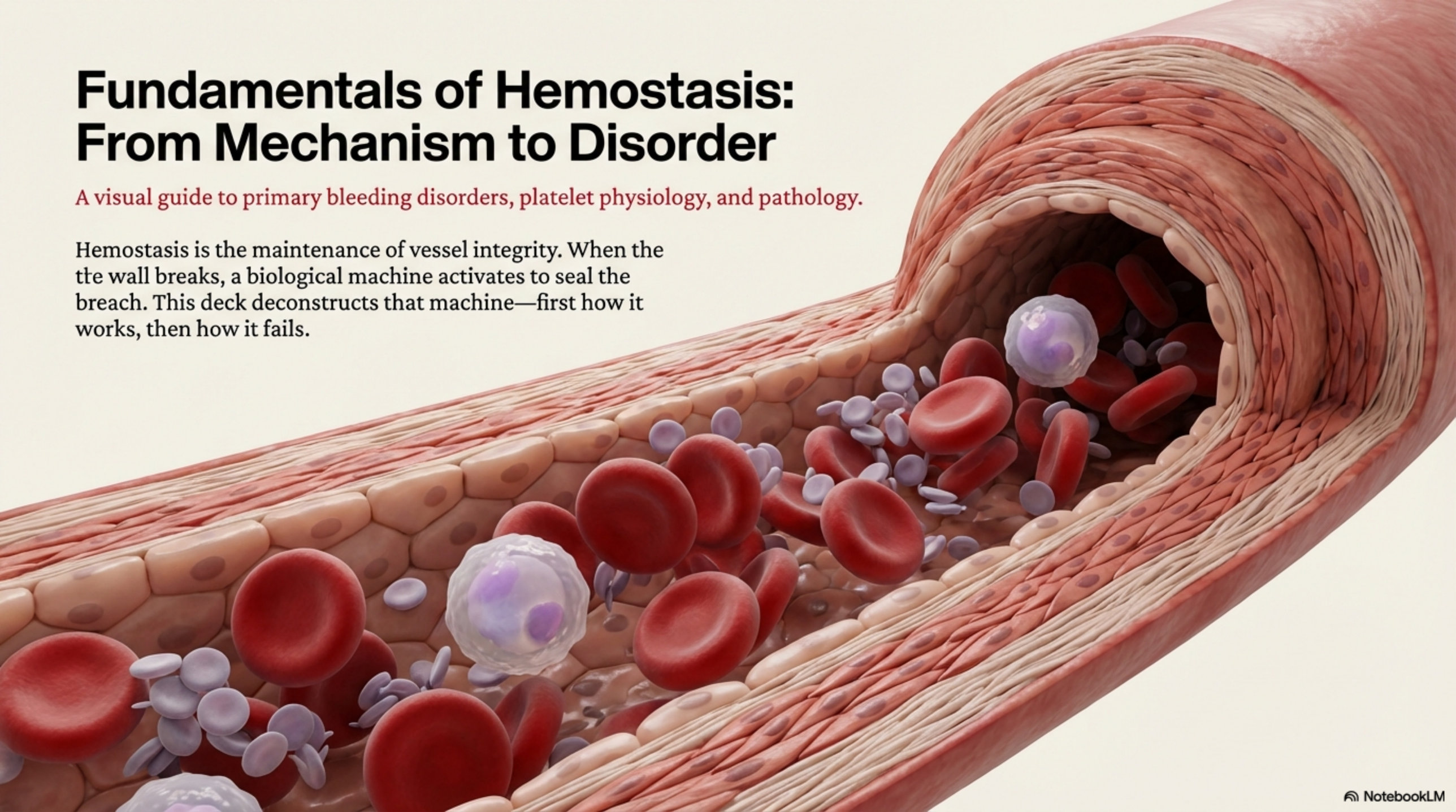
Fundamentals of Hemostasis: From Mechanism to Disorder

A visual guide to primary bleeding disorders, platelet physiology, and pathology.

Hemostasis is the maintenance of vessel integrity. When the tte wall breaks, a biological machine activates to seal the breach. This deck deconstructs that machine—first how it works, then how it fails.



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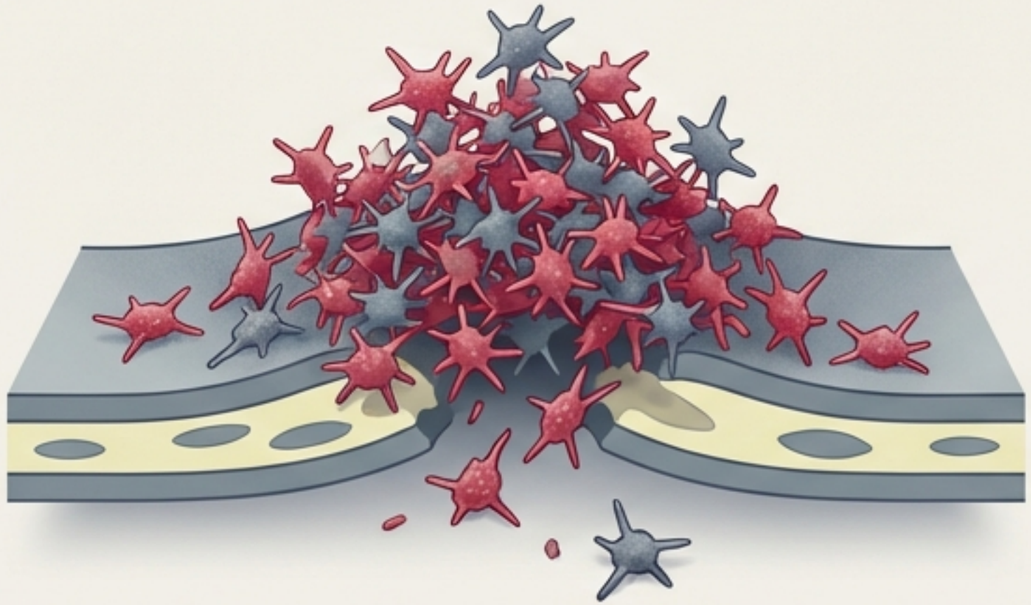
The Two Pillars of Hemostasis

Focus of this Presentation

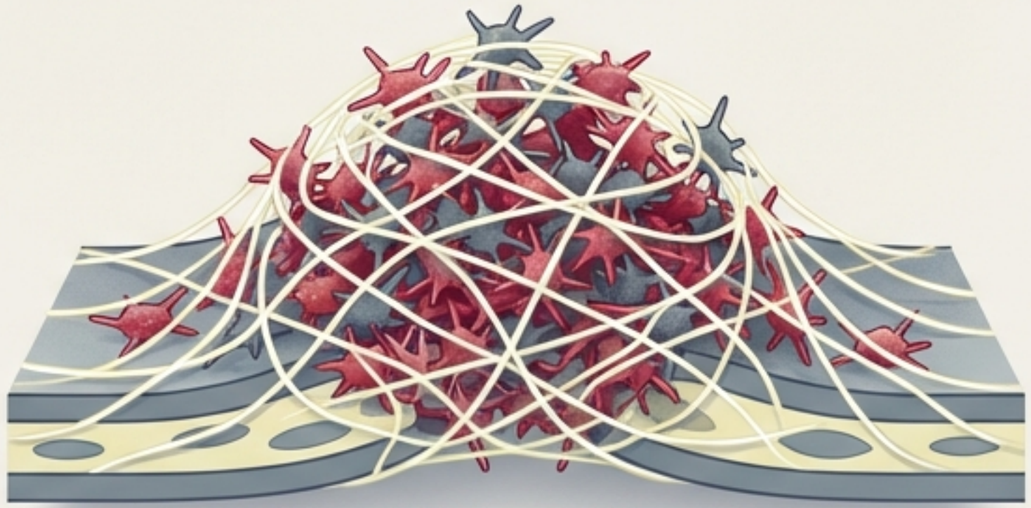
Primary Hemostasis (Platelet Plug)

Weak. Immediate. Interaction between platelets and vessel wall.



Secondary Hemostasis (Coagulation Cascade)

Stable. Stabilizes the plug via the coagulation cascade.



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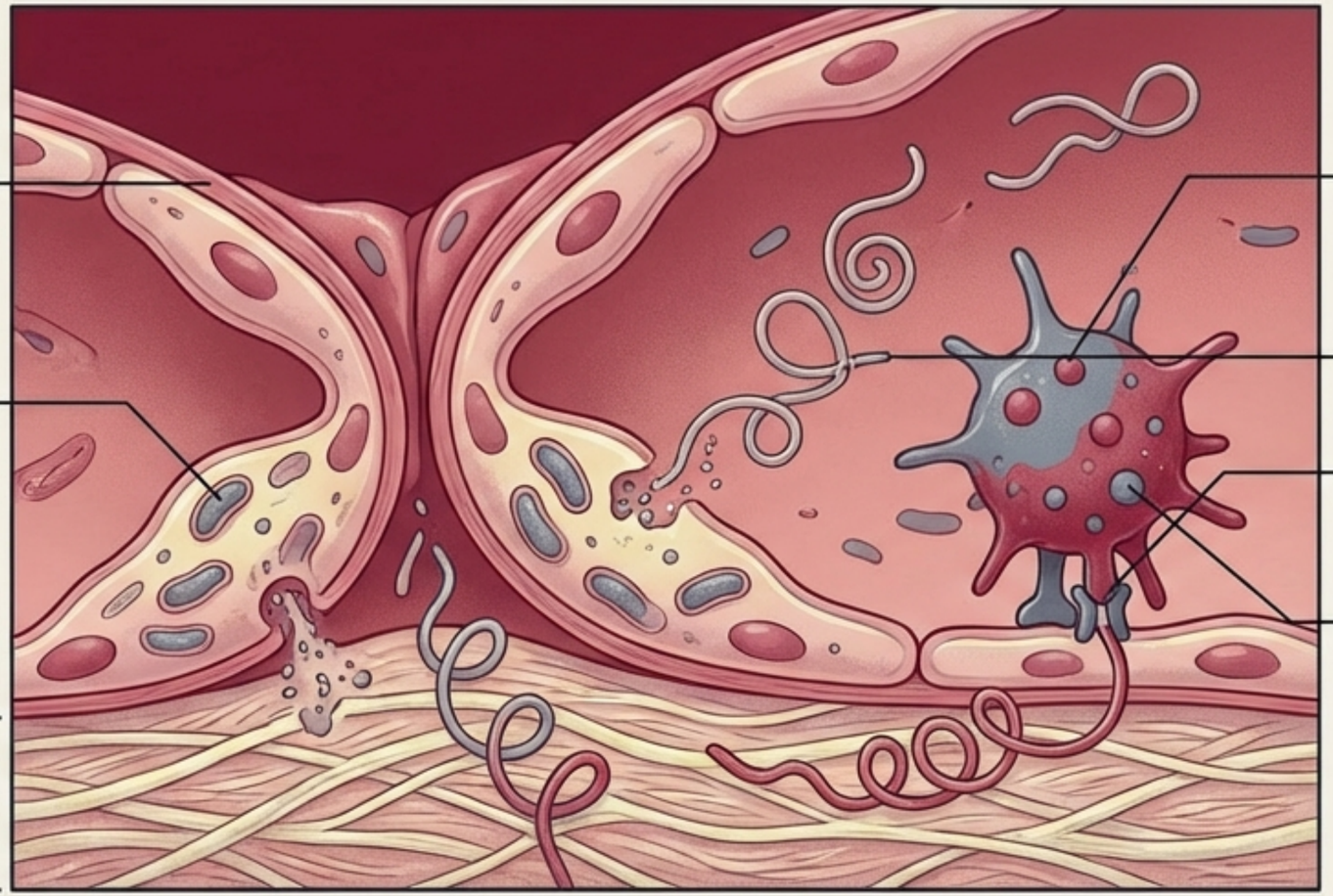
The Setup: Vasoconstriction and Adhesion



Key Concept: vWF is the bridge. GPib is the hand. Collagen is the anchor.

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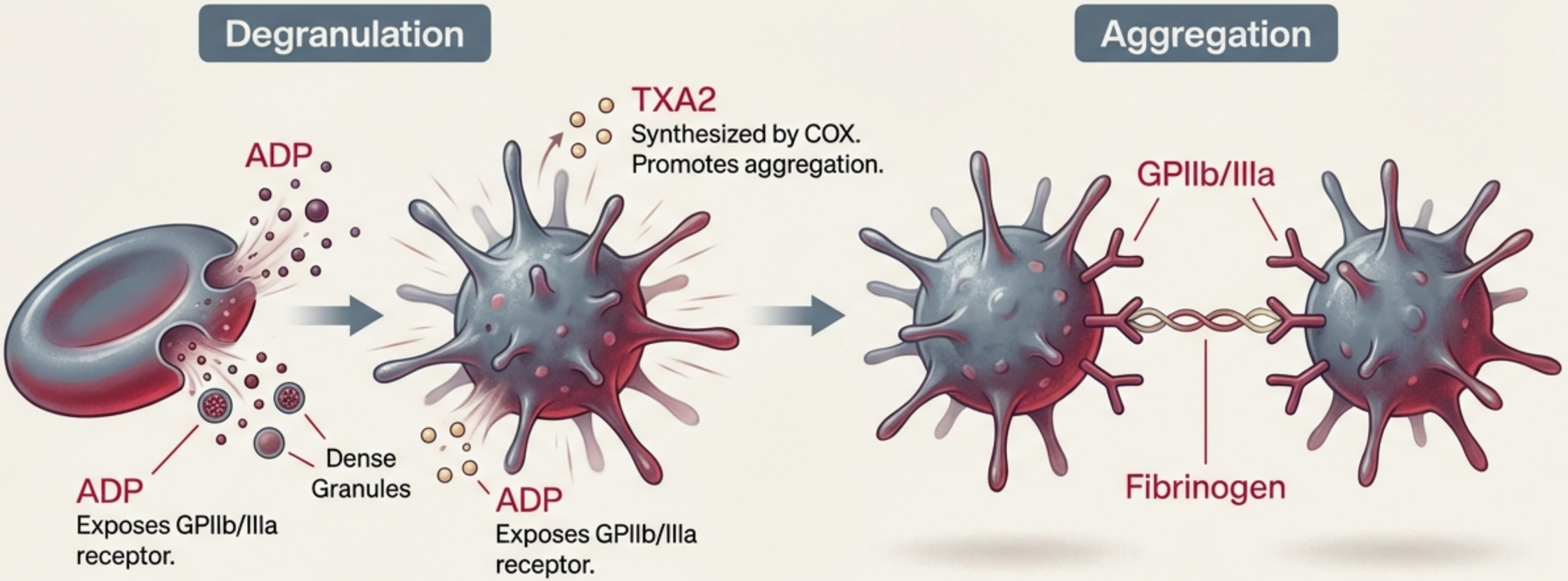
The Climax: Degranulation and Aggregation



Key Insight

ADP opens the door (GPIIb/IIIa). Fibrinogen is the glue. This forms the primary plug.

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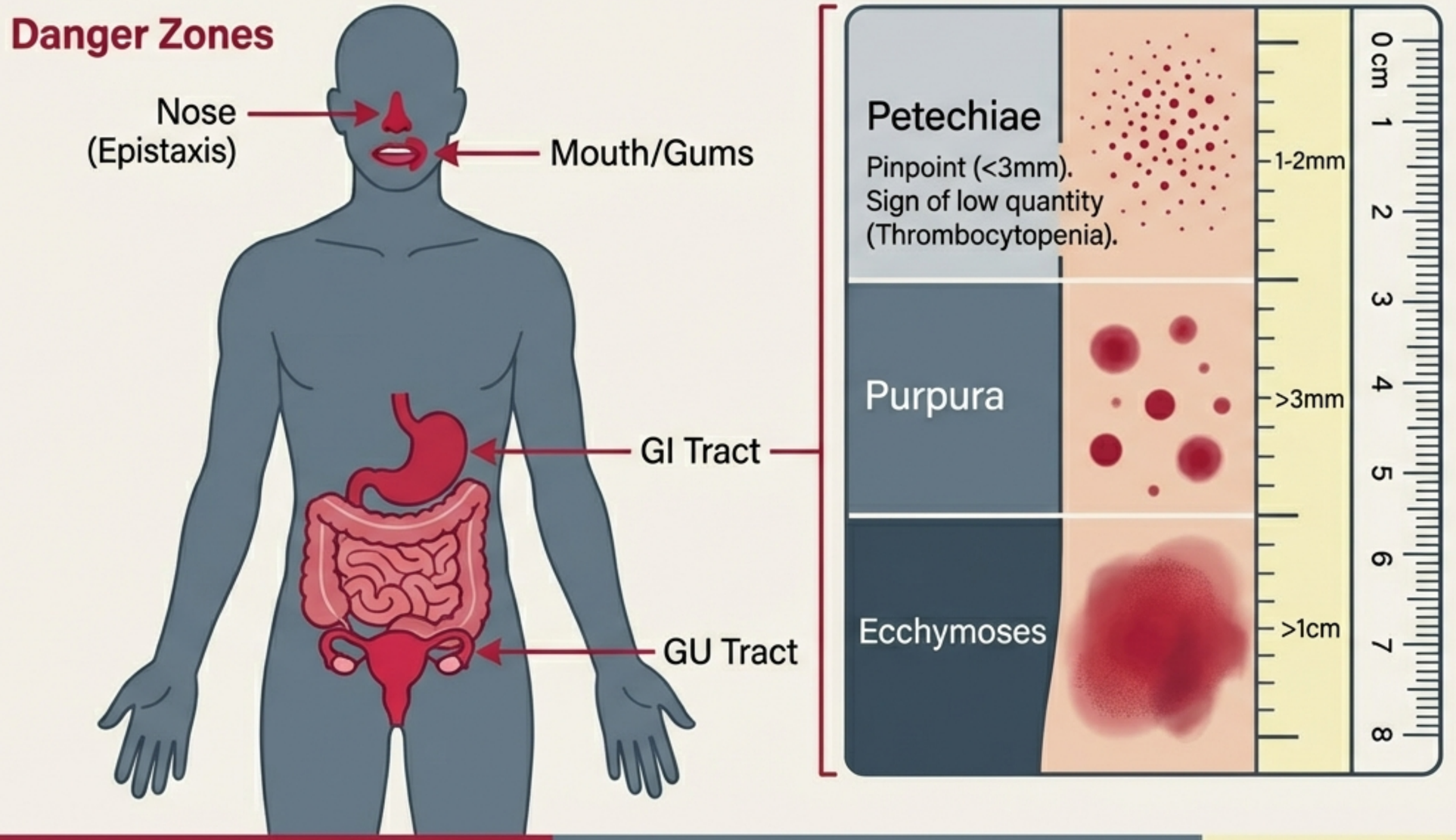
Clinical Manifestations: Mucosal and Skin Bleeding



Disorders of primary hemostasis are superficial.

Unlike coagulation factor defects (which bleed into joints), platelet defects bleed from the skin and mucosa.

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The Diagnostic Toolkit

Platelet Count

Quantity check.
Normal: **150-400 K/µL**.
Symptoms **< 50 K**.

Bleeding Time

Function check.
Normal: **2-7 min**.
Prolonged in **quantitative** AND qualitative disorders.

Blood Smear

Visual check.
Assesses number and size.

Bone Marrow Biopsy

Factory check.
Assesses megakaryocytes.

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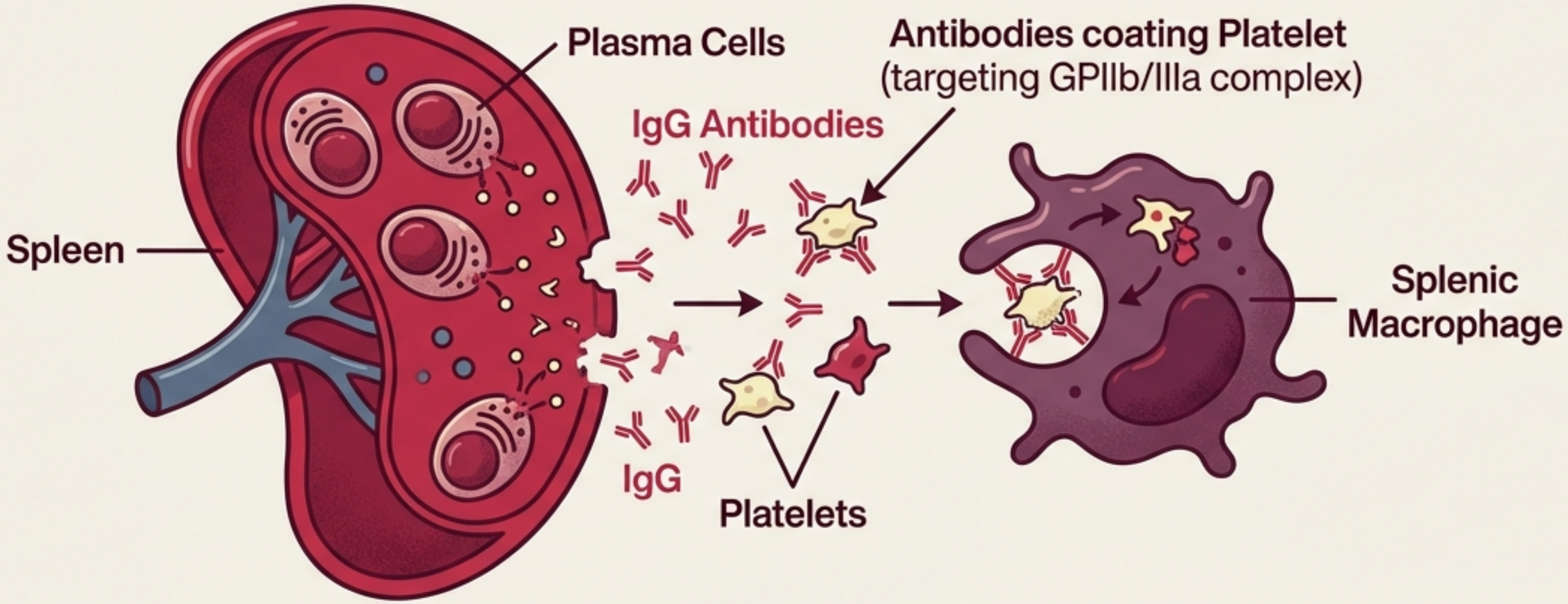
Immune Thrombocytopenic Purpura (ITP)



Acute ITP

Children.
Post-viral/immunization.
Self-limited (weeks).

Chronic ITP

Adults (Women of childbearing age).
Associated with SLE.
IgG can cross placenta.

Labs

- Low Platelets (<50K) | Normal PT/PTT
- Increased Megakaryocytes

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Managing ITP: Suppress, Distract, or Remove

Corticosteroids (First Line)

Suppress immune system/antibody production.

IVIG

The Distraction. Splenic macrophages eat IVIG instead of platelets. Short-lived effect.

Splenectomy

Refractory cases only. Eliminates the source of antibody AND the site of destruction.

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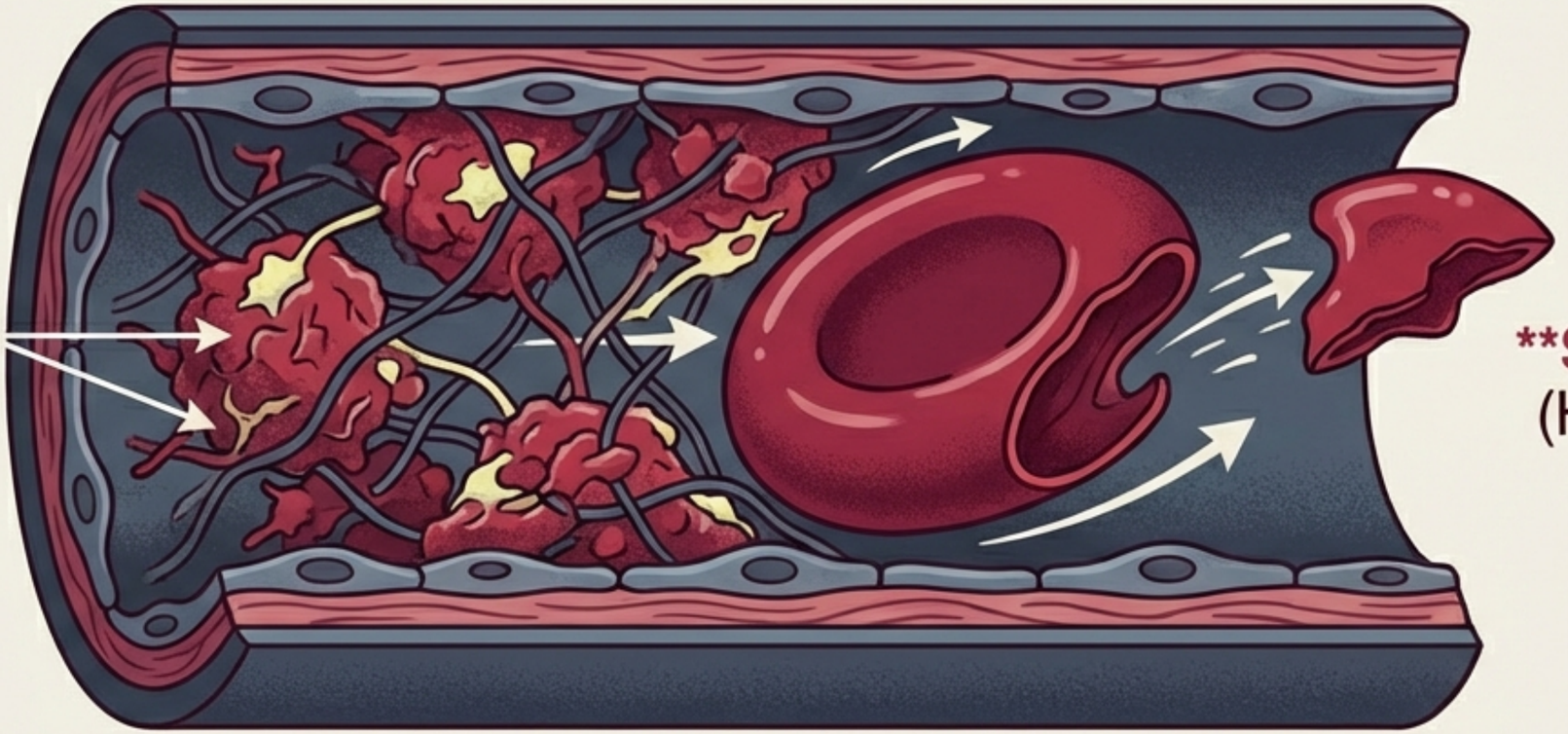
Microangiopathic Hemolytic Anemia (MAHA)



Pathologic formation of platelet microthrombi consumes platelets (Thrombocytopenia) and mechanically shears RBCs (Hemolytic Anemia).

Seen in TTP and HUS

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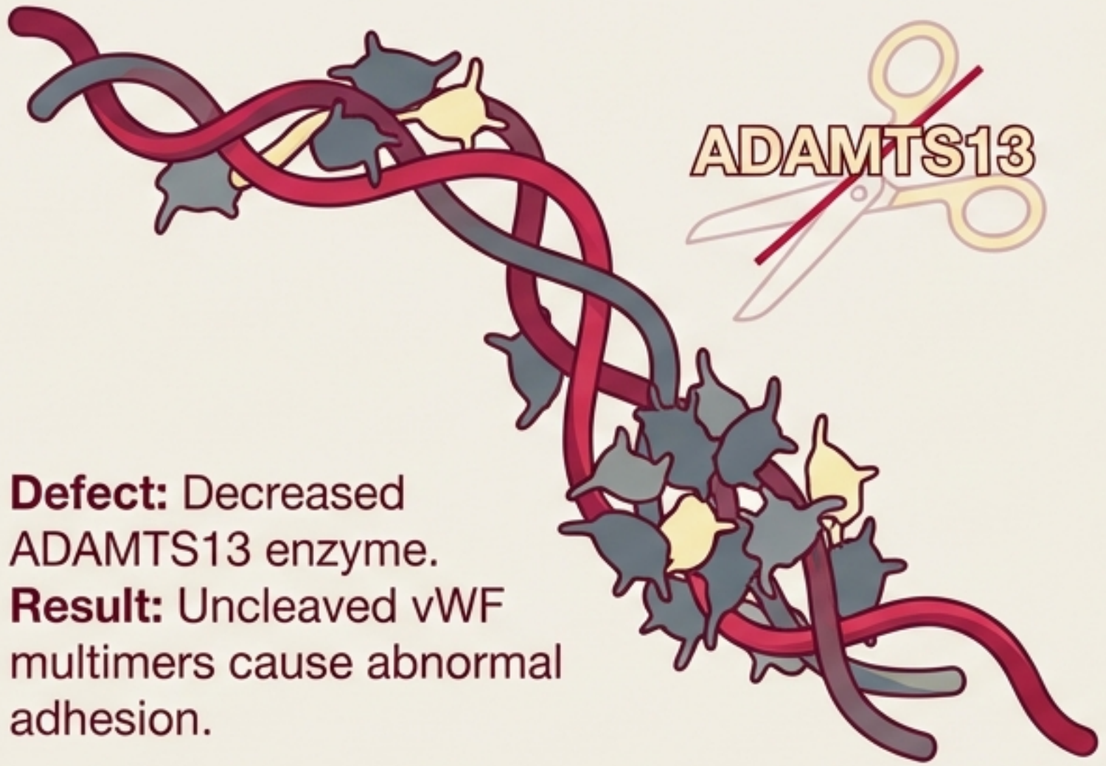
MAHA Etiologies: TTP vs. HUS

TTP (Thrombotic Thrombocytopenic Purpura)



Defect: Decreased ADAMTS13 enzyme.
Result: Uncleaved vWF multimers cause abnormal adhesion.

Adult Females

HUS (Hemolytic Uremic Syndrome)

Defect: Endothelial damage by *E. coli* O157:H7 Verotoxin (Undercooked beef).

Children

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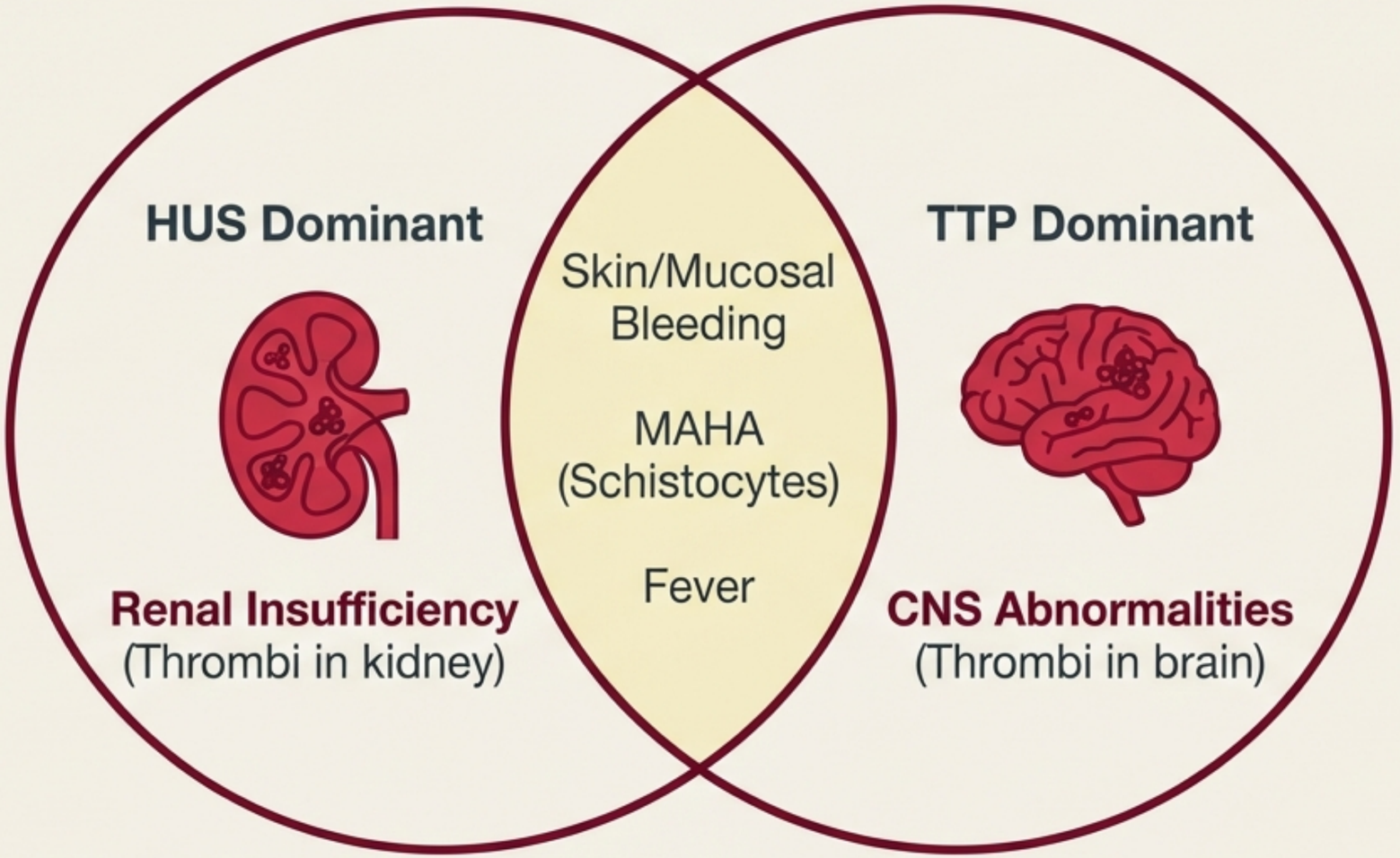
Distinguishing TTP from HUS



Lab Profile Section	• Thrombocytopenia • Anemia with Schistocytes • **Normal PT/PTT** (Crucial: Coagulation cascade is NOT involved)

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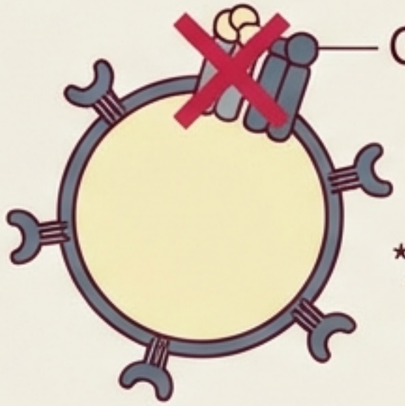
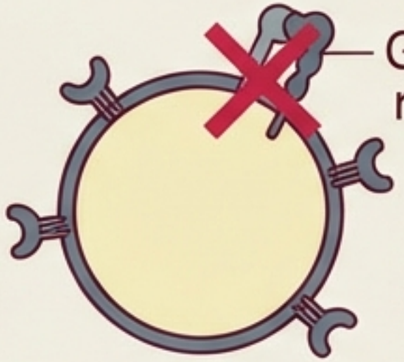
Qualitative Disorders: Broken Machinery

1. Bernard-Soulier Syndrome



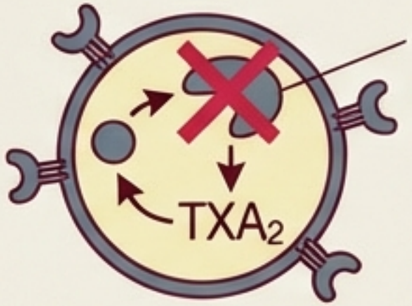
Adhesion Fails.
Genetic.
Note: Big Suckers
(Large Platelets).

2. Glanzmann Thrombasthenia



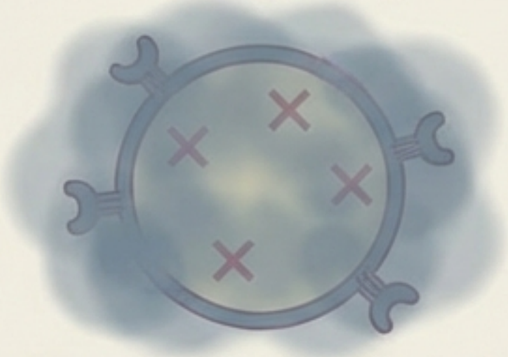
Aggregation Fails. Genetic.

3. Aspirin



No TXA2. Aggregation Impaired.

4. Uremia



Disrupts adhesion and aggregation.

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Summary of Primary Hemostasis Disorders

Disorder	Mechanism	Key Lab Finding	Clinical Association
ITP	Anti-GPIIb/IIIa IgG	Low Platelets	Post-viral (kids), SLE
TTP	Low ADAMTS13	Schistocytes, Normal PT/PTT	CNS symptoms, Adult females
HUS	*E. coli* Verotoxin	Schistocytes, Normal PT/PTT	Renal failure, Kids, Beef
Bernard-Soulier	No GPib	**Large Platelets**	Adhesion defect
Glanzmann	No GPIIb/IIIa	Normal count/morphology	Aggregation defect
Aspirin	No COX/TXA2	Normal count	Aggregation defect

Key Differentiation: Petechiae and mucosal bleeding point to platelets. Normal PT/PTT confirms the coagulation cascade is intact.

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