MED-1 · MED-411 · HEMATOLOGY

Bleeding Disorders

Hemostasis, the bleeding history, the first panel and the common disorders

Husain Alkhaldy · King Khalid University College of Medicine



These slides are an outline. Read them with the handbook, the casebook and the question bank.

Blood must keep flowing to sustain the body. When a vessel breaks, it must clot at that site, and only there.

Bleeding disorders are failures of that second step.

CASES

Four patients

A

A boy, aged 2

Painful, swollen knees, again and again. A large bruise after a small fall.

B

A woman, aged 24

Three weeks of bruises and bleeding gums. A viral illness a month ago.

C

A man, aged 70

Sudden, large bruises. Has had teeth out and surgery without trouble.

D

A woman, aged 19

Heavy periods since her teens. Bled for days after her wisdom teeth.

> We return to each one during the lecture. Two more patients (E and F) come later.

OBJECTIVES

By the end, you can

1. Tell a **local bleed** from a **bleeding disorder**
2. Say whether **primary or secondary hemostasis** failed
3. Decide **inherited or acquired** from the history
4. Read the **first panel**: count, film, PT, aPTT, fibrinogen
5. Recognise the **common disorders** and their first-line care
6. Know **when to call hematology** the same day

01

How bleeding stops

START, MAKE, STOP, CLEAN, and the test for each step.

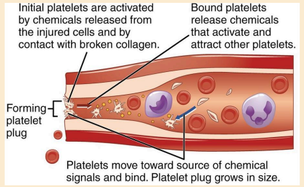
THE FRAMEWORK

Two arms of hemostasis

PRIMARY HEMOSTASIS

The platelet plug

- Vessel wall (endothelium)
- Platelets
- Von Willebrand factor (VWF)



Bleeding: early, in skin and mucosa — petechiae, nose, gums, heavy periods

First tests: platelet count, blood film

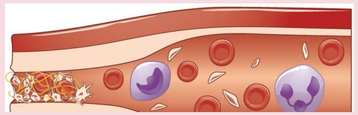
VWF

in both: holds platelets, carries factor VIII

SECONDARY HEMOSTASIS

The fibrin clot

- Coagulation factors
- Fibrinogen → fibrin

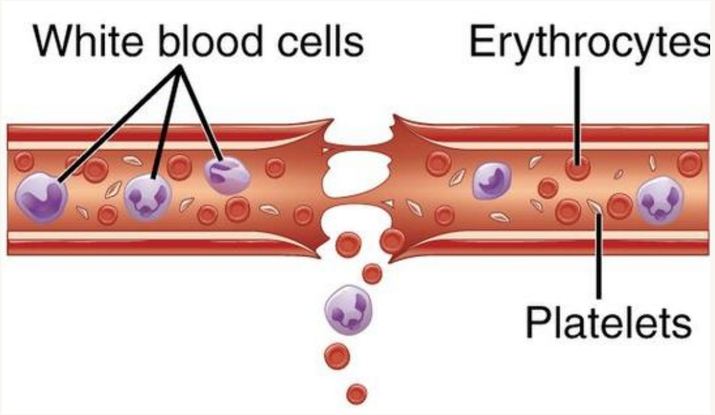


Bleeding: late, in deep tissue — joints, muscles

First tests: PT, aPTT, fibrinogen

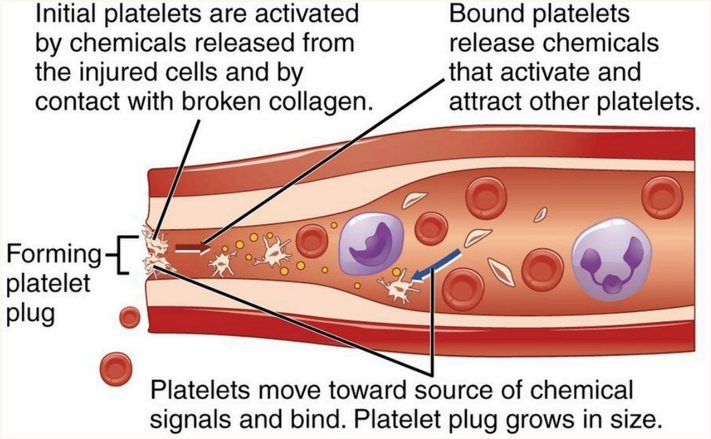
HEMOSTASIS

From injury to clot



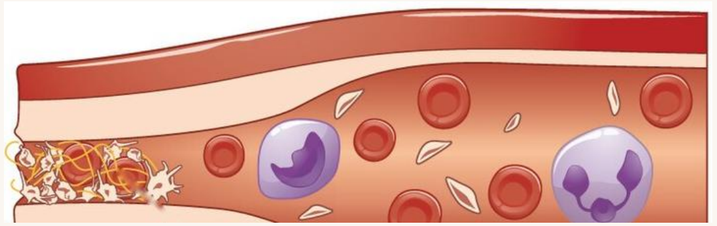
1 · INJURY

The vessel breaks and its wall constricts to slow the loss.



2 · PLATELET PLUG — PRIMARY

Platelets stick at the break and call in more. Seconds.



3 · FIBRIN CLOT — SECONDARY

Fibrin strands bind the plug. Minutes.

OpenStax, Anatomy & Physiology, fig. “1909 Blood Clotting”, CC BY 3.0, via Wikimedia Commons (cropped).

THE MAP FOR THIS LECTURE

Four verbs: START, MAKE, STOP, CLEAN

START

Primary hemostasis

Platelet plug

Test: Count, film

Fails in: Low platelets, VWD, aspirin

MAKE

Secondary hemostasis

Fibrin clot on the plug

Test: PT, aPTT, fibrinogen

Fails in: Hemophilia, warfarin, liver disease

STOP

Natural anticoagulants

Brakes keep the clot at the injury

Test: Protein C, protein S, antithrombin levels

Fails in: Higher risk of thrombosis (hereditary thrombophilia)

CLEAN

Fibrinolysis

Plasmin dissolves the clot

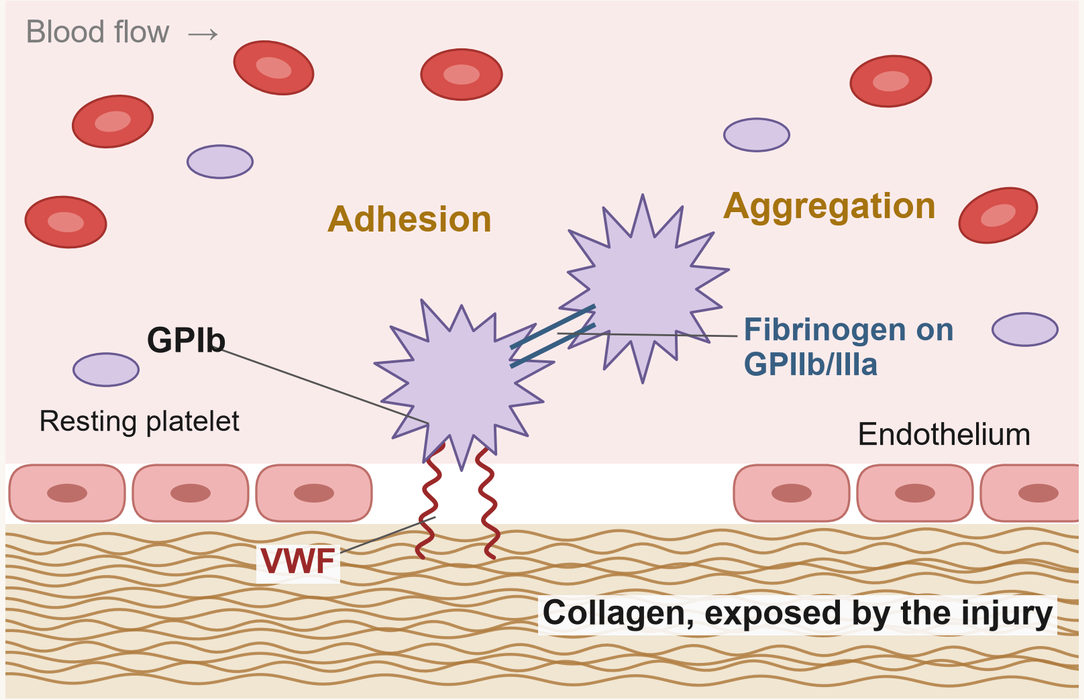
Test: D-dimer

Fails in: Too much: bleeding

PT: prothrombin time · aPTT: activated partial thromboplastin time · VWD: von Willebrand disease. These colours mark each step for the rest of the lecture.

PRIMARY HEMOSTASIS · START

The platelet plug



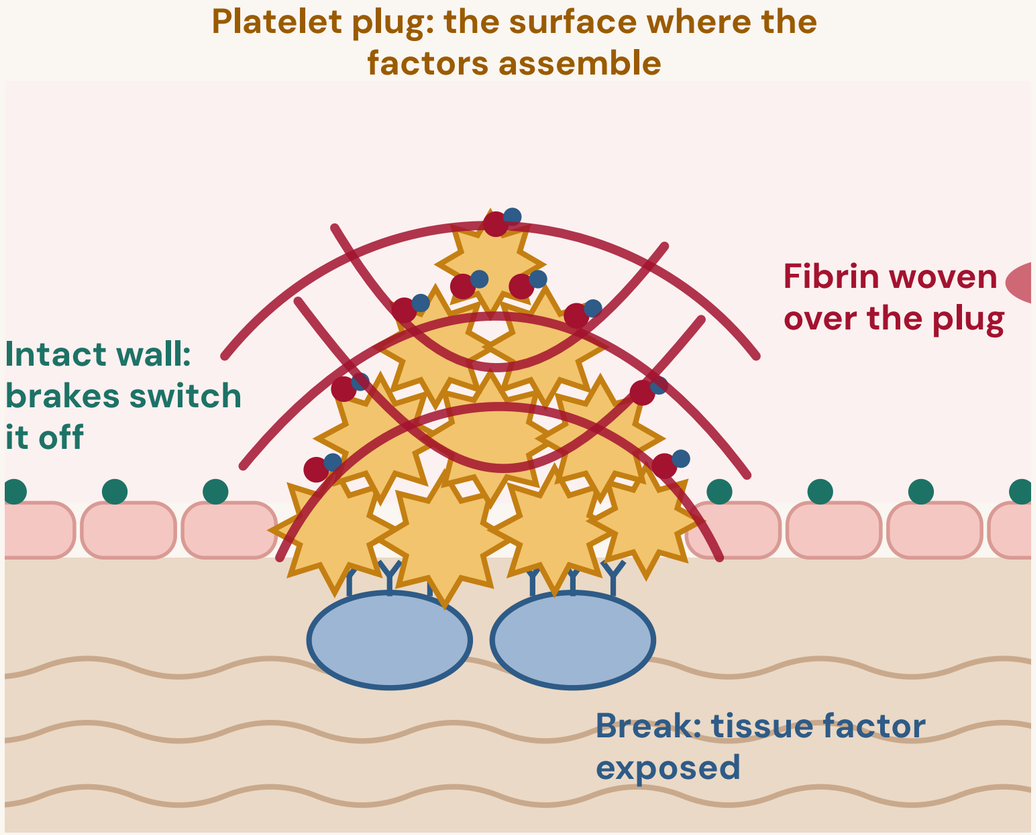
1 **Adhesion.** Von Willebrand factor (VWF) bridges platelet glycoprotein (GP) Ib to collagen.
Fails in: VWD, Bernard-Soulier

2 **Activation.** Granules and thromboxane call in more platelets.
Fails in: aspirin, for the platelet's life

3 **Aggregation.** GPIIb/IIIa links platelets through fibrinogen.
Fails in: Glanzmann thrombasthenia

SECONDARY HEMOSTASIS · MAKE

Where the clot is made: on the platelet plug



1. **Tissue factor is hidden** under the vessel wall. Only a break exposes it, so the cascade starts only there.

2. **The factors assemble on activated platelets** of the plug, held on their surface with calcium. Vitamin K gives II, VII, IX and X the anchor that binds it.

3. **A burst of thrombin** on the plug turns fibrinogen into fibrin, woven through the plug. Factor XIII cross-links it.

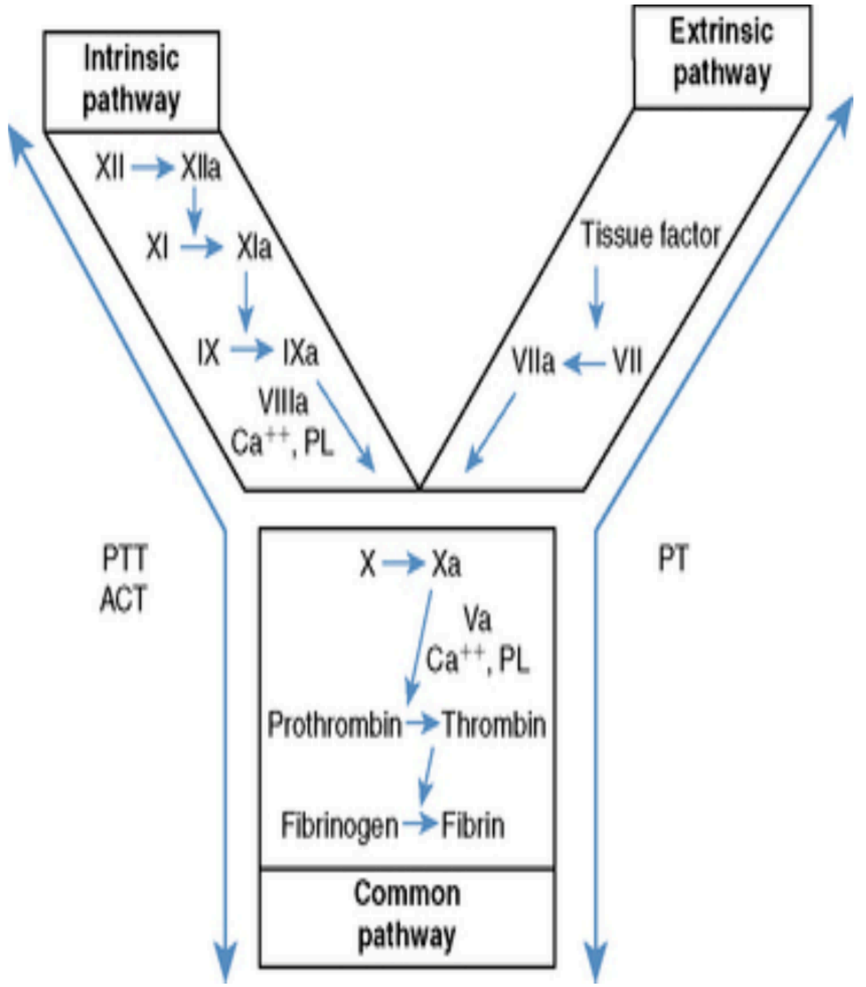
4. **Beyond the edge**, intact endothelium switches the cascade off. Blood stays liquid everywhere else.

Classification: Personal

Make the clot (old theory)

- Platelets plug is not enough.
- This stage happens at the platelets plug surface
- Activations of coagulation factors /co-factors
- **X:V activate prothrombin**
- **Prothrombin** (II)→ **Thrombin** (IIa)
- **Fibrinogen**(I) → **Fibrin** clot

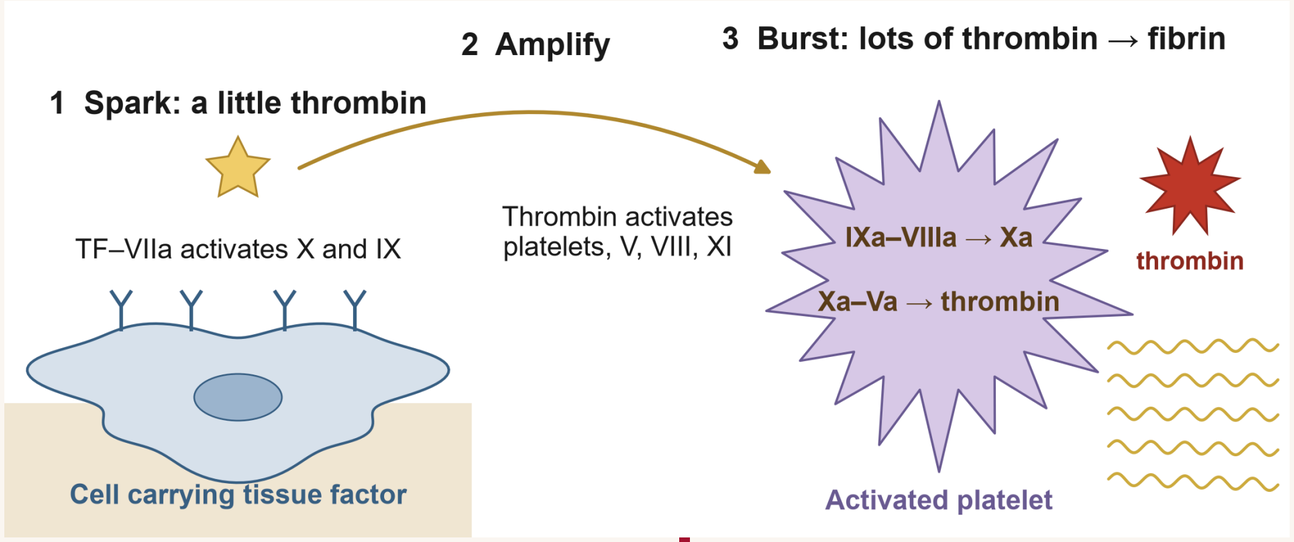
1. TF + FVII
2. TF:FVIIa activate X
3. X:V activate prothrombin
4. Thrombin convert Fibrinogen into fibrin clot



1. Contact system →XII+XI (12+11) → IX + VIII (8+9) → X:V
2. X:V (5;10)activate prothrombin
3. Thrombin convert Fibrinogen into fibrin clot (2:1)

SECONDARY HEMOSTASIS · MAKE

Extrinsic and intrinsic: one loop, not two pathways



The “extrinsic” spark (tissue factor with VIIa) makes a little thrombin. That thrombin switches on the “intrinsic” factors — VIII, IX, XI — on the platelet, and they make far more thrombin. In the body the two run together, as a loop.

In hemophilia the spark still happens, but without IXa–VIIIa there is no burst on the platelet: the loop breaks, and the clot is weak and late.

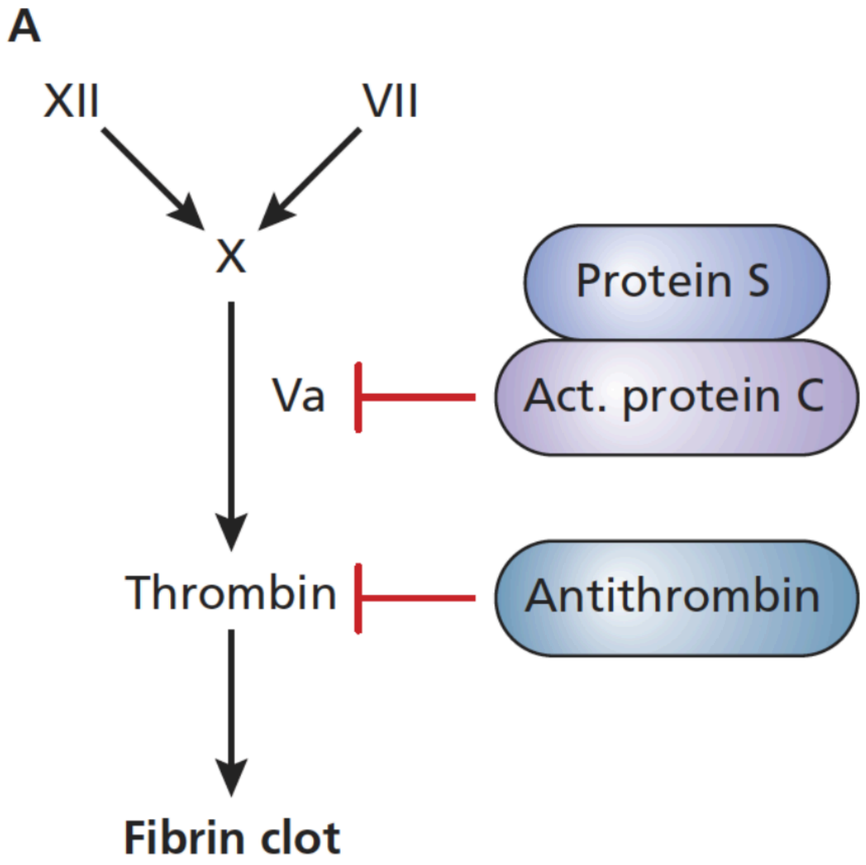
Classification: Personal

Stop (termination phase)

- Switch off the hemostasis cascade in order to limit the reaction to the site of injury

Natural anticoagulants :

1. TFPI (Tissue factor pathway inhibitor)
2. Protein S
3. Protein C
4. Anti thrombin



Clean (Fibrinolysis)

- Activation of plasminogen
- plasmin
- Fibrin → FDP

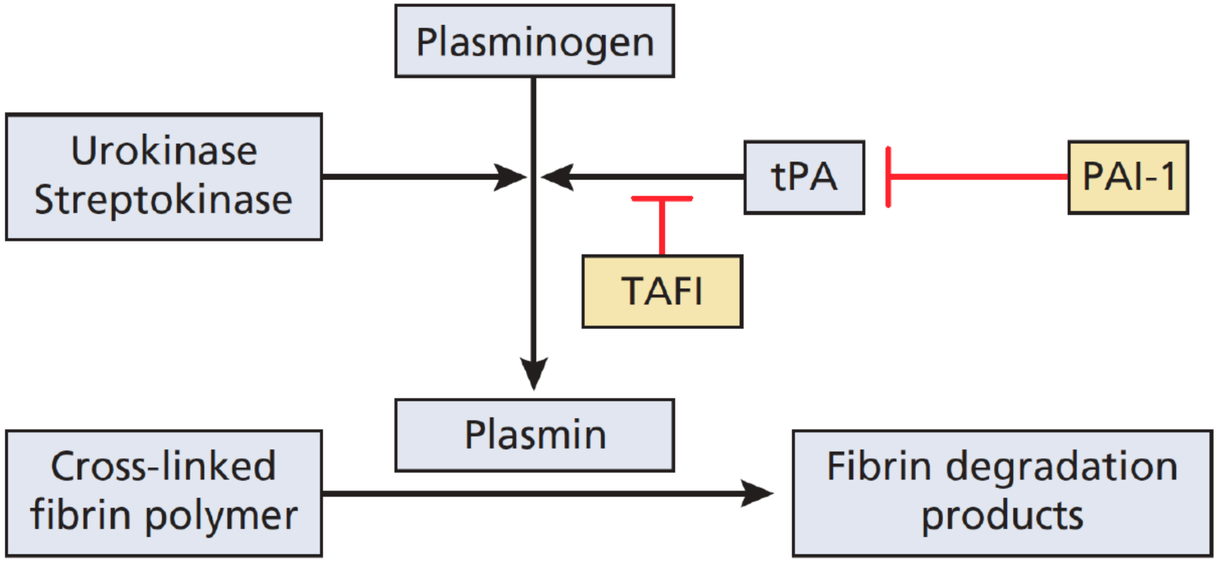


Figure 9-4 Fibrinolysis. TAFI, thrombin-activatable fibrinolysis inhibitor; tPA, tissue plasminogen activator.

02

Four questions at the bedside

Is it a bleeding disorder? Primary or secondary? Inherited or acquired? What does the first panel show?

QUESTION 1

Local bleed, or a bleeding disorder?

One site: look for the lesion

- A nosebleed on its own
- Blood in the urine
- Vomiting or coughing blood

Usually a vessel, an ulcer, a tumour or a stone. Investigate with endoscopy or imaging, not a clotting screen.

A pattern: think of a disorder

- More than one site
- Out of proportion to the injury
- Failed challenges: birth, circumcision, teeth, surgery, childbirth
- A family history

QUESTION 2

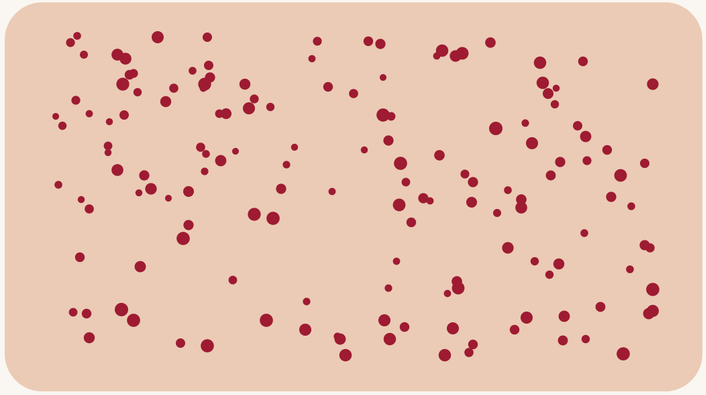
Primary or secondary: which arm failed?

Feature	Primary · platelets, VWF	Secondary · coagulation factors
Site	Skin and mucosa	Deep tissue: joints, muscles
Onset after injury	Immediate	Delayed, hours to days
Petechiae	Common	Rare
Bruises	Small, superficial	Large, deep, palpable
Joint bleeds	Rare	Common in severe disease
Small cuts	Bleed for long	Usually stop
Bleeds again after stopping	Uncommon	Common
Examples	ITP, VWD, aspirin	Hemophilia, warfarin, liver disease

ITP: immune thrombocytopenia · VWD: von Willebrand disease. Severe VWD (type 3) and type 2N can bleed like a secondary disorder, because VWF carries factor VIII.

EXAMINATION

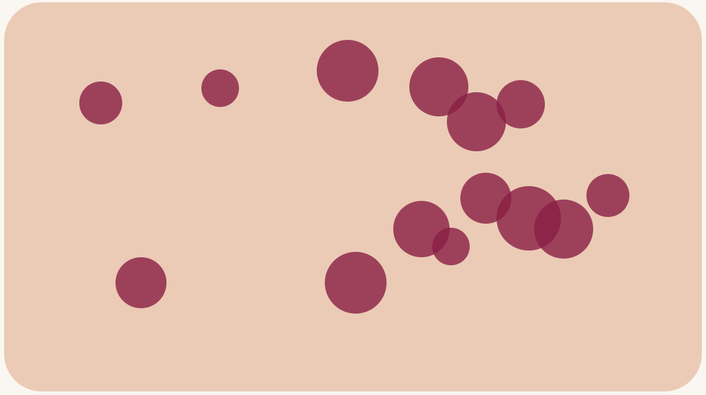
Skin signs: primary or secondary?



Petechiae

Under 2 mm; do not blanch

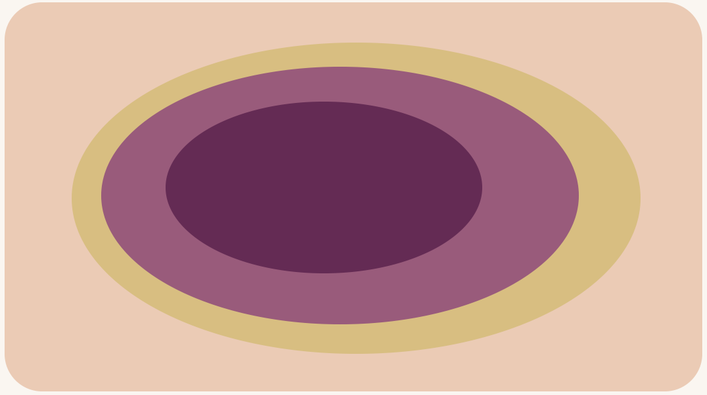
Primary: low or dysfunctional platelets



Purpura

2 to 10 mm; raised and palpable in vasculitis

Primary: platelets, or the vessel wall



Ecchymosis

Over 1 cm; large bruises, no petechiae

Secondary: coagulation factors

> Many petechiae point to platelets. Big bruises without petechiae point to a coagulation defect.

Clinical photos: DermNet — purpura, 10 photo cases. Drawing made for this lecture.

QUESTION 3

Inherited, or acquired?

Past challenges

- Circumcision, birth
- Teeth out, tonsils out
- Surgery, childbirth
- Periods since menarche

Family

- X-linked: brothers, the mother's brothers
- Autosomal: both sexes
- Are the parents related?

Drugs and illness

- Antiplatelets, anticoagulants, NSAIDs, herbal
- Liver, kidney, cancer, pregnancy, infection, travel

Lifelong points to inherited. **New, in an adult**, points to acquired until proved otherwise.

Tired, feverish and bruising? Think of the marrow — count and film today.

About 56% of Saudi marriages were between relatives in a national survey (2004–05): recessive bleeding disorders are not rare here. NSAIDs: nonsteroidal anti-inflammatory drugs.

A GIRL, AGED 14

“How are your periods?” “Fine.”

Tired and pale. Examination: pallor only.

Test	Result
Hemoglobin	97 g/L — low
Mean cell volume	70 fL — low
Platelets	331 $\times 10^9$/L — normal

Asked for details: each period lasts 3 to 4 weeks; she changes protection between every class.

> Many girls do not know what a normal period is. Ask for numbers, not adjectives.

WHAT NORMAL LOOKS LIKE

Heavy menstrual bleeding (HMB)

Normal

- 7 days or fewer
- Cycles of 21 to 45 days
- A few hours between changes

Red flags

- Large clots, gushing
- Soaking through, changing at night
- Iron deficiency

Causes in a teenager

- Anovulatory cycles (commonest)
- Bleeding disorders — VWD first
- Pregnancy, infection, drugs
- Structural causes (rare)

> Heavy since the first period, with other bleeding or a family history: ask the bleeding questions.

Riyadh schoolgirls: 16.7% (330 of 1,980) reported heavy periods (Owaidah 2018, self-report).

A STRUCTURED INTERVIEW

Score the history: ISTH-BAT

The International Society on Thrombosis and Haemostasis Bleeding Assessment Tool (ISTH-BAT) scores each bleeding symptom by severity and adds them into one total.

Low pre-test probability

Mild bruising, no family history, passed every challenge.

Normal score: a bleeding disorder is unlikely.

Intermediate or high

A relative with VWD, iron deficiency, failed challenges.

Test anyway: a normal score does not rule it out.

ASH/ISTH/NHF/WFH 2021 guideline on VWD diagnosis (James et al.).

BLEEDING ASSESSMENT TOOL

ISTH-BAT: the score sheet

14 symptoms, each scored 0 to 4

Skin and mucosa	Challenges and deep bleeds
Nosebleeds	Tooth extraction
Skin bruising	Surgery
Minor wounds	After childbirth
Mouth and gums	Muscle hematoma
Gut	Joint bleeds
Blood in urine	Brain
Heavy periods	Other bleeding

How one symptom is scored: nosebleeds

Score	Nosebleeds
0	None, or trivial
1	More than 5 a year, or lasting more than 10 minutes
2	Needed a doctor’s visit only
3	Packing, cautery or tranexamic acid
4	Transfusion, factor replacement or desmopressin

Add the 14 scores. Abnormal total: **men 4 or more · women 6 or more · children 3 or more**.

Rodeghiero et al., J Thromb Haemost 2010 (the tool); Elbatarny et al., Haemophilia 2014 (normal ranges). Each symptom has its own wording for scores 1 to 4.

03

The first panel

Platelet count, film, PT, aPTT and fibrinogen: the patterns, and when to refer.

QUESTION 4

Five tests, two steps

Platelet count

How many platelets

Blood film

Clumps, fragments, blasts, platelet size

PT (INR)

Factor VII + common path

aPTT

XII, XI, IX, VIII + common path

Fibrinogen

The substrate; falls in DIC

Amber tests look at primary hemostasis. **Crimson** tests look at secondary hemostasis.

Normal ranges depend on the reagent and analyser: use your laboratory's range.

FOUR PATTERNS

Reading the PT and aPTT together

	aPTT normal	aPTT long
PT normal	**Both normal** Still bleeding? VWD, platelet function disorder, a mild factor deficiency, factor XIII deficiency, vascular causes **A factor low enough to bleed may not lengthen the tests: convincing history, normal screen, special tests.**	**aPTT alone** Intrinsic factors: hemophilia A or B, heparin, acquired hemophilia
PT long	**PT alone** Factor VII: early warfarin, vitamin K deficiency, early liver disease	**Both long** Common path or many factors: DIC, severe liver disease, warfarin excess, massive transfusion

Low platelets? Read the film first: ITP, marrow disease, TTP, DIC. An unexplained long PT or aPTT: refer.

KEY POINT

A normal panel does not rule out a bleeding disorder.

Von Willebrand disease

The commonest inherited bleeding disorder

Platelet function

Drugs, uremia, Glanzmann

Mild factor deficiency

Enough factor for the tube, not for surgery

Factor XIII

Umbilical stump and brain bleeds

Vascular causes

Scurvy, connective tissue disorders

If the history is convincing, refer — even with a normal panel.

04

The disorders, step by step

Platelet disorders, von Willebrand disease, hemophilia and acquired causes.

PRIMARY HEMOSTASIS · THE PLUG

Patient B: a woman, aged 24

- Three weeks of bruising on her legs
- Gums bleed when she brushes
- Two long nosebleeds; a much heavier period
- A viral illness a month ago; no medicines
- Petechiae; blood blisters in the mouth
- No nodes, no big spleen, no bone tenderness

RUN THE QUESTIONS

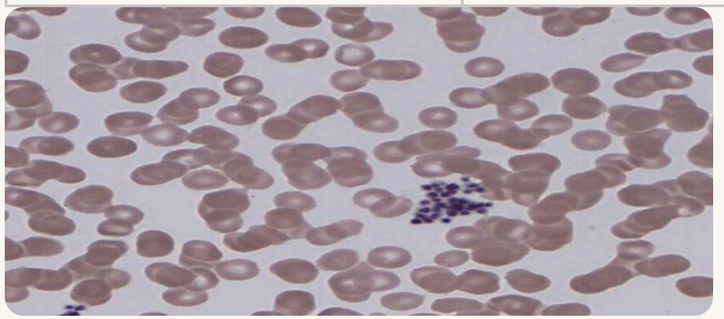
1. **A disorder?** Many sites at once: yes.
2. **Which arm?** Skin, mucosa, petechiae: primary hemostasis.
3. **Inherited?** Three weeks, no past bleeding: acquired.
4. **First panel?** Count and film.

Patient B: the panel and the film

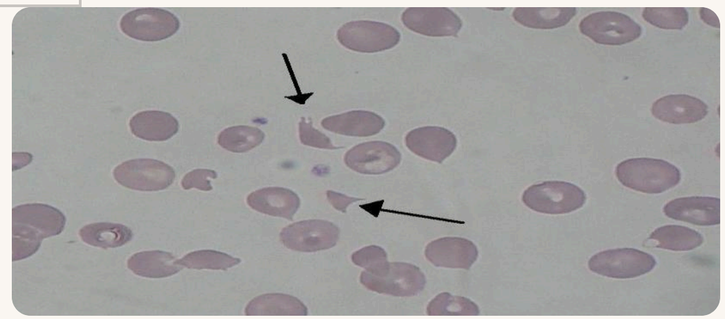
Test	Result
Platelets	8 ×10⁹/L
Hemoglobin	118 g/L
White cells	Normal
PT, aPTT	Normal
Film	Few, large platelets

Normal film apart from few, large platelets.

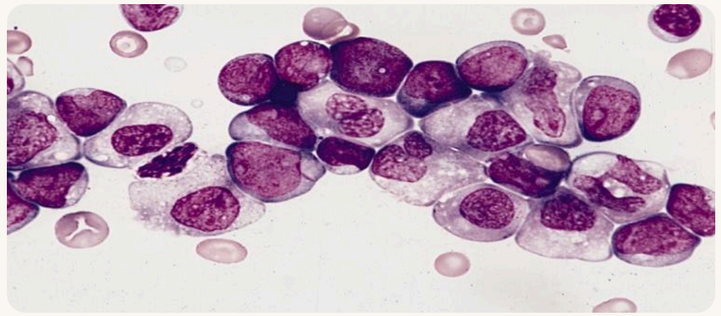
No clumps, no fragments, no blasts.



Clumps: the count is falsely low



Fragments: think TTP or DIC

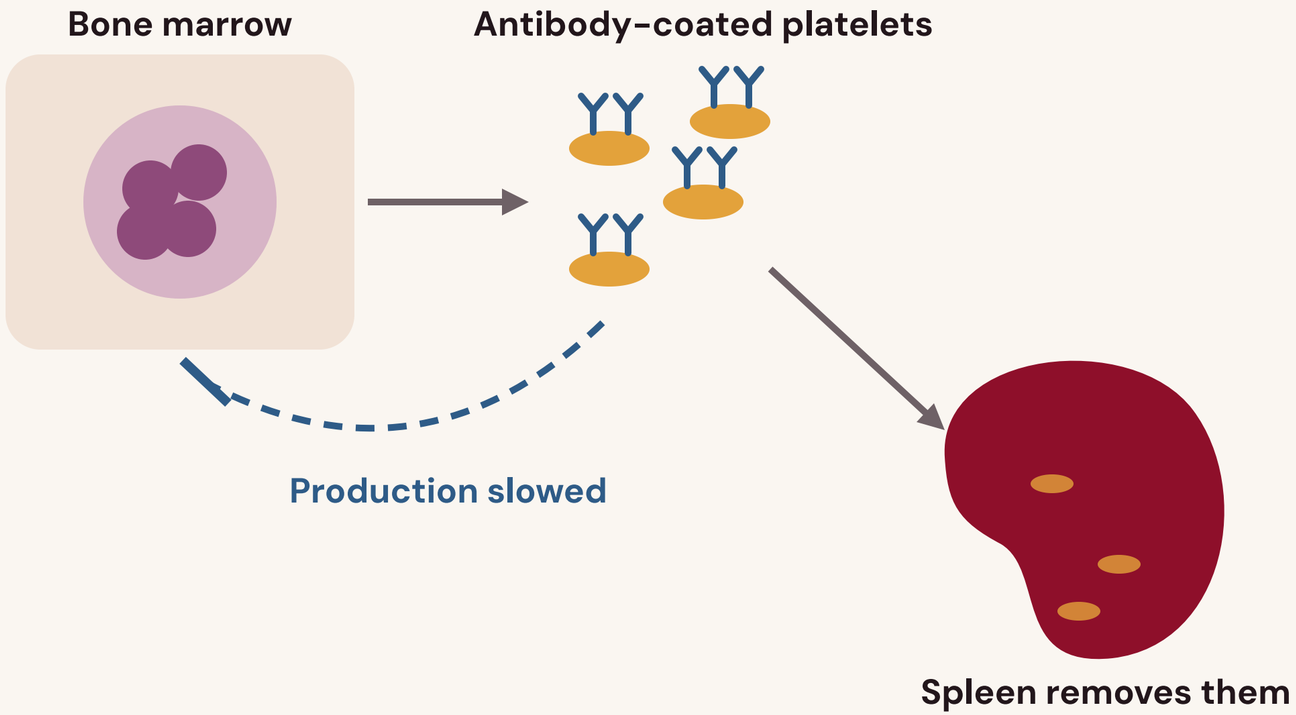


Blasts: marrow disease

Films: Erhabor Osaro, CC BY-SA 4.0; Paulo Henrique Orlandi Mourão, CC BY-SA 3.0; AFIP, public domain — via Wikimedia Commons.

PRIMARY HEMOSTASIS · THE PLUG

Why the count falls in ITP



1. Autoantibodies coat the platelets

2. Spleen macrophages remove them

3. The same antibodies slow production in the marrow

> Large platelets on the film: the marrow is working hard to keep up.

Immune thrombocytopenia (ITP)

Exclude first

- Clumping (film)
- Marrow disease (other counts, nodes, spleen)
- TTP and DIC (fragments, PT, aPTT)
- Heparin-induced thrombocytopenia

Then

- A well patient with a low count and nothing else — no test proves ITP
- Send HIV, hepatitis C, pregnancy test
- Marrow only if something does not fit

First-line care

- Treat the bleeding, not the number
- Corticosteroids first line
- Intravenous immunoglobulin (IVIG) for a fast rise
- Children with mild bleeding: often watch

Wet purpura or any serious bleed: hematology the same day.

ASH 2019 guidelines for immune thrombocytopenia.

PRIMARY HEMOSTASIS · THE PLUG

When platelets are present but do not work

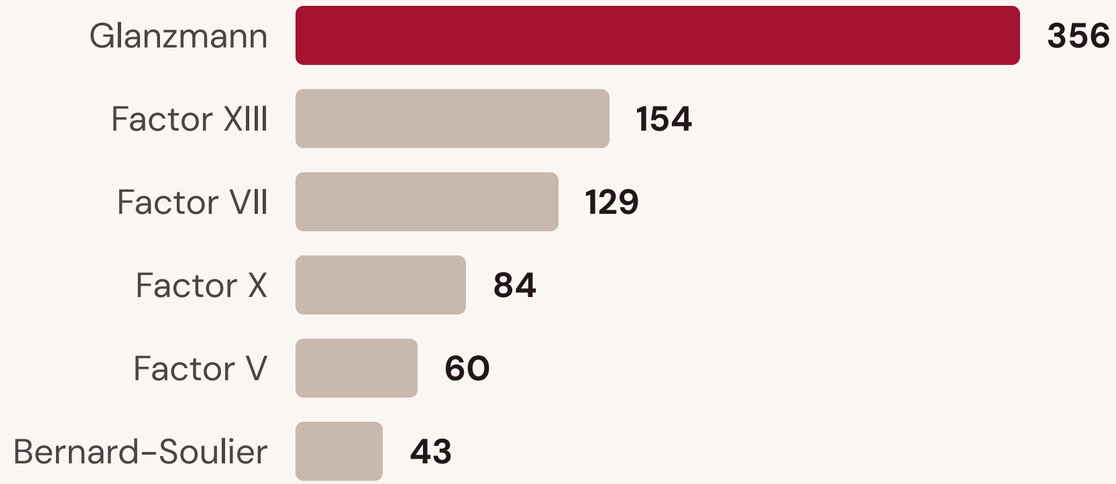
Acquired — common

- Aspirin (7 to 10 days), P2Y12 blockers, NSAIDs
- Kidney failure (uremia), liver disease

Inherited — rare, but not here

- **Glanzmann:** no GPIIb/IIIa; normal count and size
- **Bernard-Soulier:** no GPIb; large platelets, count a little low

Rare bleeding disorders registered in Saudi Arabia



Glanzmann is the largest group: think of it in heavy periods from menarche with a normal count.

World Federation of Hemophilia (WFH) Global Survey 2023 — registry counts, not prevalence.

PRIMARY HEMOSTASIS · THE PLUG

Patient D: a woman, aged 19

- Heavy periods since her teens
- Easy bruising, nosebleeds
- Bled for days after wisdom teeth
- Mother has heavy periods too
- Platelets, PT and aPTT: all normal

RUN THE QUESTIONS

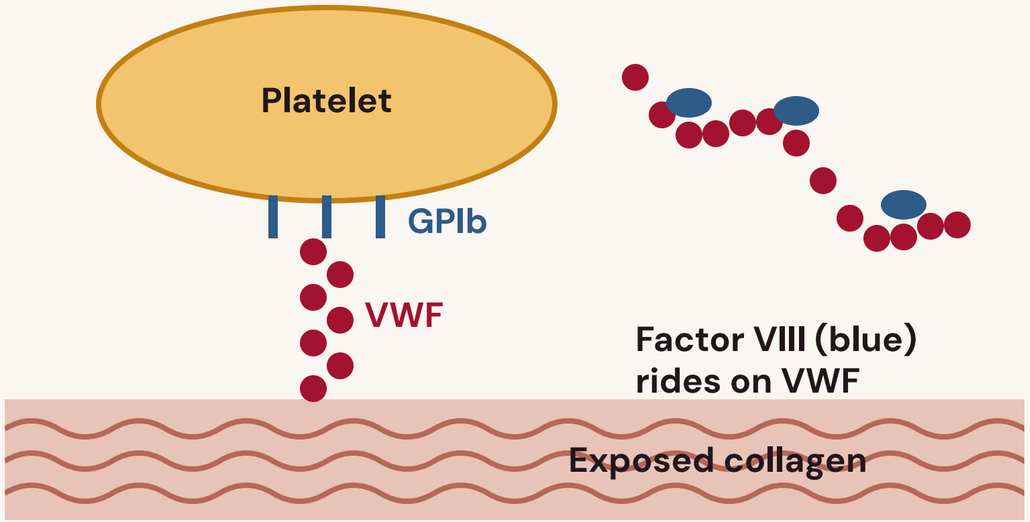
1. **A disorder?** A lifelong pattern, failed challenge: yes.
2. **Which arm?** Skin and mucosa: primary hemostasis.
3. **Inherited?** Since menarche, family history: yes.
4. **First panel?** Normal — so test VWF next.

PRIMARY · AND SOMETIMES SECONDARY

Von Willebrand factor has two jobs

1 · Glues platelets to the wound

2 · Carries factor VIII



PATIENT D'S RESULTS

Test	Result
VWF antigen	Low
VWF activity	Low, in proportion
Factor VIII	Low–normal

> Too little VWF that works normally: **type 1 VWD**.

Very little VWF → factor VIII is cleared fast → joint bleeds, as in type 3.

Cut-offs: ASH/ISTH/NHF/WFH 2021 guideline on VWD diagnosis.

PRIMARY HEMOSTASIS · THE PLUG

Von Willebrand disease: three types

1

Too little VWF

Most patients; usually mild

2

VWF that does not work

2B: low platelets too
2N: mimics hemophilia A

3

Almost no VWF

Severe; factor VIII very low; joint bleeds

> The commonest inherited bleeding disorder: about 1 in 100 people have low VWF on testing; far fewer bleed enough to need care.

Saudi Arabia: 651 registered with VWD (WFH Global Survey 2023), a registry count.

PRIMARY HEMOSTASIS · THE PLUG

Von Willebrand disease: first-line care

1. **Tranexamic acid** — mouth, nose, heavy periods, dental work
2. **Desmopressin** releases stored VWF — most type 1 (after a trial)
3. **Hormonal therapy** for heavy periods, plus iron
4. **VWF concentrate** — type 3, major surgery, desmopressin failure

Avoid

- Aspirin and NSAIDs
- Intramuscular injections in severe disease

Plan surgery and childbirth with hematology.

SECONDARY HEMOSTASIS · THE CLOT

Patient A: a boy, aged 2

- His knees swell and hurt, again and again
- A large bruise after a small fall
- No petechiae
- Ask: did he bleed at circumcision?
- Ask: does any man on his mother’s side bleed?

RUN THE QUESTIONS

1. **A disorder?** Two sites, again and again: yes.
2. **Which arm?** Joints, no petechiae: secondary hemostasis.
3. **Inherited?** A toddler, a boy: X-linked.
4. **First panel?** Next slide.

SECONDARY HEMOSTASIS · THE CLOT

Patient A: the panel

Test	Result
Platelet count	Normal
PT	Normal
aPTT	Long
Factor VIII	Below 1%

Pattern: **a long aPTT alone** → an intrinsic factor.

The factor assay names it: **VIII, not IX**.

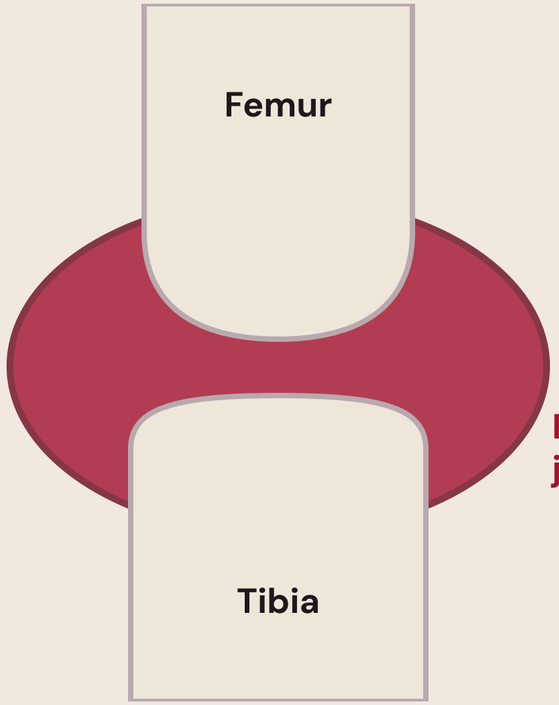
> Below 1%: **severe hemophilia A**.

SECONDARY HEMOSTASIS · THE CLOT

Hemophilia: the factor level sets the pattern

< 1%	**Severe:** bleeds with no injury — joints, muscles
1–5%	**Moderate:** bleeds after minor injury
5–40%	**Mild:** bleeds with surgery or major injury
50–150%	**Normal** factor VIII or IX level

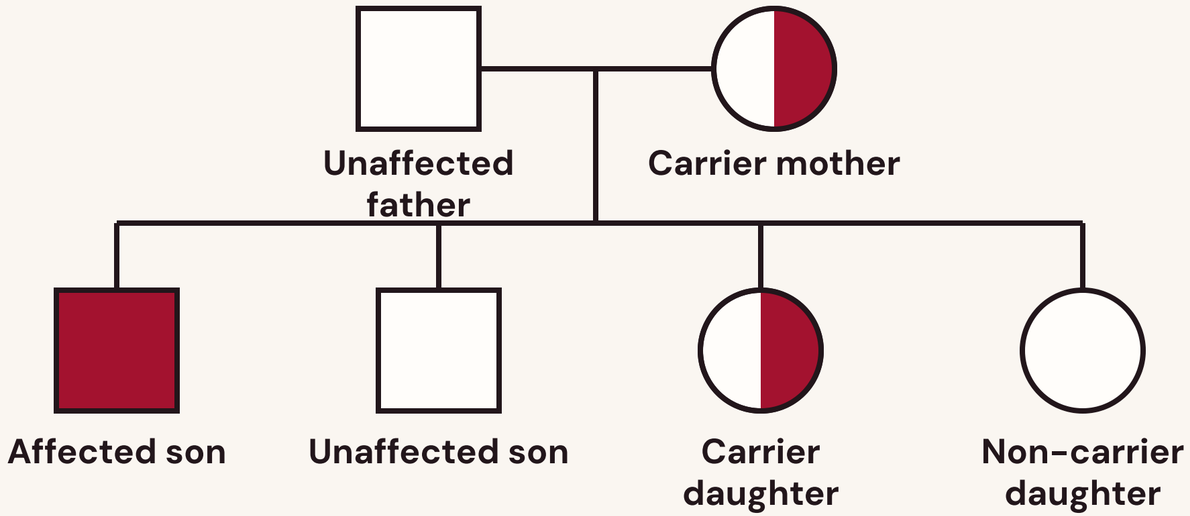
Fear head and retroperitoneal bleeds.



Target joints → lasting damage. Radiopaedia: hemophilic arthropathy

Saudi registry: 1,595 people with hemophilia (A 1,281, B 306) — WFH Global Survey 2023, registry counts.

How hemophilia is inherited



Each son: 1 in 2 affected. Each daughter: 1 in 2 a carrier.

X-linked recessive, for both A and B

An affected father: all daughters are carriers, no son is affected

About a third have no family history (new variant)

> Carrier women can bleed: ask about heavy periods and check their level.

SECONDARY HEMOSTASIS · THE CLOT

Hemophilia: first-line care

Replace

- Factor VIII or IX concentrate
- Severe disease: regular prophylaxis, not just on demand
- Emicizumab: a weekly-to-monthly injection for hemophilia A

Protect

- No intramuscular injections
- No aspirin or NSAIDs
- Tranexamic acid for mouth bleeds

Plan

- Comprehensive care centre
- Joint protection, physiotherapy
- Screen carriers in the family

A head injury or a big joint bleed: give factor first, then investigate.

WFH Guidelines for the Management of Hemophilia, 3rd edition (2020).

SECONDARY HEMOSTASIS · THE CLOT

Patient C: acquired hemophilia

A man, aged 70. Sudden, large bruises. Never bled before — teeth out and surgery without trouble.

- Long aPTT alone
- PT and platelets normal

Think of it

An autoantibody against factor VIII. Older adults, or a woman after childbirth; both sexes; not inherited.

What to do

Call hematology the same day. No intramuscular injections, no unnecessary procedures.

ON THE WARD

Acquired causes in adults

Cause	First panel
Antiplatelets, NSAIDs	Count and screen normal
Kidney failure (uremia)	Count and screen normal; platelets work poorly
Warfarin	PT (INR) long first
Heparin	aPTT long
Direct oral anticoagulants (DOACs)	Variable; no routine monitoring test
Liver disease, vitamin K deficiency	PT long, then both
DIC	Both long, fibrinogen falling, platelets falling

BOTH ARMS · ACQUIRED

Patient E: in intensive care with sepsis

- Oozing from cannula sites
- One toe has turned dusky

Test	Result
Platelets	40 ×10⁹/L
INR	1.6
Fibrinogen today	1.8 g/L
Fibrinogen 2 days ago	4.2 g/L

Why does she bleed and clot at once?

Why is a “normal-looking” fibrinogen of 1.8 worrying?

BOTH ARMS · ACQUIRED

Disseminated intravascular coagulation (DIC)

Trigger: sepsis, cancer, obstetric, trauma, snakebite

↓

Tissue factor in the whole circulation

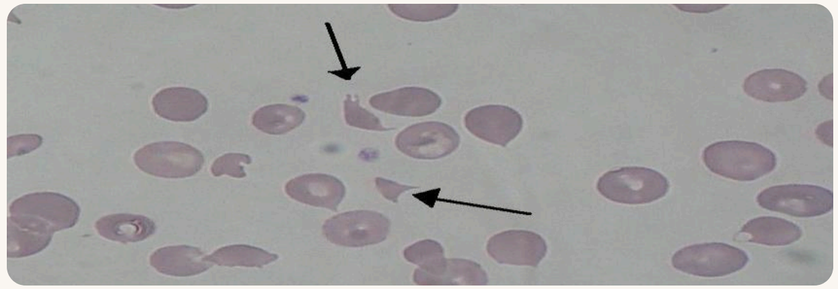
↓

Thrombin everywhere

↙ ↘

Microthrombi block small vessels → organs starve: **the dusky toe**

Factors and platelets used up → **oozing** from every line



Panel: PT and aPTT long, fibrinogen and platelets falling, D-dimer high, fragments on the film.

> **Treat the cause.** If bleeding: platelets, cryoprecipitate or fibrinogen, plasma.

Schistocytes: Paulo Henrique Orlandi Mourão, CC BY-SA 3.0, via Wikimedia Commons. Repeat the tests: the trend makes the diagnosis.

DO NOT CONFUSE

Fragments and low platelets: three emergencies

	TTP	HUS	DIC
Cause	Severe ADAMTS13 deficiency	Shiga toxin, or complement	Clotting switched on by another illness
Patient	Adult, often a young woman	Child after bloody diarrhea	Sepsis, cancer, trauma, obstetric
Organ	Brain and heart	Kidney	Variable: bleeding and clotting
PT, aPTT, fibrinogen	Normal	Normal	Long; fibrinogen falling
Urgency	Hematology today	Supportive care	Treat the cause

TTP: thrombotic thrombocytopenic purpura · HUS: hemolytic uremic syndrome.

WARFARIN AND CIRRHOSIS

Patient F: a long INR in two settings

On warfarin, with a nosebleed

INR above target; liver healthy.

- Warfarin blocks vitamin K: II, VII, IX, X not finished
- Liver healthy: the INR corrects with vitamin K

Major bleed: stop warfarin, intravenous vitamin K and prothrombin complex concentrate (PCC)

Cirrhosis, before paracentesis

INR raised; not bleeding.

- The liver makes fewer factors — and fewer brakes
- The balance is reset, not tipped
- Vitamin K does not fully correct it

The INR does not measure her bleeding risk. Do not "correct" it before routine procedures.

MANAGEMENT IN ONE SLIDE

The first-line toolbox

Tranexamic acid

Mucosal bleeding, heavy periods, VWD, hemophilia mouth bleeds

Desmopressin

Most type 1 VWD; mild hemophilia A

Factor concentrates

Hemophilia A or B; VWF concentrate for severe VWD

Vitamin K + PCC

Warfarin excess or bleeding; vitamin K deficiency

Corticosteroids, IVIG

Immune thrombocytopenia

Platelets, cryoprecipitate, plasma

DIC with bleeding, massive transfusion

> Avoid in every bleeder: aspirin, NSAIDs and intramuscular injections.

Regional points

Ask about	Where	Why
Snakebite	Asir	Some venoms cause a coagulopathy: ask in anyone who bleeds without a clear cause
Alkhurma fever	Najran, Makkah, Jizan	Tick-borne viral hemorrhagic fever: nosebleeds and gum bleeding in a Najran series of 28 (Alzahrani 2010); severe cases occur
Dengue	Red Sea coast: Jeddah, Makkah	Fever, travel and low platelets

Good Saudi data are few, and most come from Riyadh, Madinah and the Eastern Province. These are reasons to ask, not local rates.

WORK-UP SUMMARY

The whole approach

1

A pattern?

One site → local lesion. A pattern → go on.

2

Primary or secondary?

Mucosa and petechiae, or joints and muscles.

3

Born or acquired?

Challenges, family, drugs, illness.

4

First panel

Count, film, PT, aPTT, fibrinogen.

Low count: **read the film**

Long PT or aPTT: **match the pattern, refer**

All normal but convincing: **VWF, platelet function, XIII**

SOLVED

Our six patients

	Findings	Diagnosis
A	Boy, 2 · joints, delayed · long aPTT, factor VIII below 1%	Severe hemophilia A
B	Woman, 24 · skin and mucosa, new · platelets 8, clean film	Immune thrombocytopenia
C	Man, 70 · new, severe · long aPTT · never bled before	Acquired hemophilia
D	Woman, 19 · lifelong mucosal bleeding · normal panel · low VWF	Type 1 VWD
E	Sepsis · oozing and a dusky toe · fibrinogen falling	DIC
F	Long INR on warfarin, and in cirrhosis	Warfarin effect; rebalanced hemostasis in cirrhosis

TAKE-HOME

Five things to take home

1. A bleeding disorder is a pattern, not a single site.
2. Mucosa and petechiae = primary. Joints and muscles = secondary.
3. Lifelong = inherited. New in an adult = acquired until proved otherwise.
4. Read the first panel as a pattern — and always read the film.
5. A normal panel does not rule out VWD.

Read next

- **Hematology handbook:** chapters 20–25, 29
- **Casebook:** Case 53, The Bleeding Patient; Case 51, section 6 (DIC)
- **Question bank:** hematology items 46–59
- **Free book:** Bloody Easy Coagulation Simplified, 3rd edition (2026)

Coming later

How the lab works up a long PT or aPTT: sample problems, mixing studies and factor assays.

Thrombosis: handbook chapter 26; venous thromboembolism again in Med-2.

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