



Heparin-Induced Thrombocytopenia

HIT · A prothrombotic disorder that happens to lower the platelet count

SCORE BEFORE YOU TEST

4Ts, not gestalt

SUSPECT IT *the paradox: they clot, they rarely bleed*

THE PATTERN

Platelet **fall of more than 50%** from baseline, typically to **50–70 ×10⁹/L** — rarely below 20. Onset **5–10 days** after starting heparin, or **within hours** if there was heparin exposure in the preceding 30 days (rapid-onset HIT).

THE DANGER IS THROMBOSIS

Antibodies to the **heparin–PF4 complex** activate platelets, generating thrombin. Consequences are **venous and arterial thrombosis**, limb ischemia, skin necrosis at injection sites, and anaphylactoid reactions. **Bleeding is unusual** despite the low count.

RISK IS NOT UNIFORM

Higher with **unfractionated** than low-molecular-weight heparin, higher after **surgery** (especially cardiac and orthopedic) than in medical patients, and higher in women. Fondaparinux essentially does not cause it.

SCORE IT FIRST *the 4Ts — guidelines specifically favor it over clinical impression*

Component	2 points	1 point / 0 points
Thrombocytopenia	Fall > 50% and nadir ≥ 20	Fall 30–50%, or nadir 10–19 / fall < 30% or nadir < 10
Timing	Clear onset days 5–10 , or ≤ 1 day with heparin in the last 30 days	Consistent but unclear, or onset after day 10 / fall at < 4 days with no recent exposure
Thrombosis	New confirmed thrombosis , skin necrosis, or anaphylactoid reaction after a heparin bolus	Progressive or recurrent thrombosis, or suspected but unproven / none
Other causes	None apparent	Possible / definite alternative cause
0–3 low · 4–5 intermediate · 6–8 high. With a low-probability score, avoid HIT laboratory testing and empirical treatment altogether — the pre-test probability is low enough that a positive immunoassay is far more likely to be a false positive than true HIT.		

ACT ON AN INTERMEDIATE OR HIGH SCORE *do not wait for the assay*

- Stop all heparin immediately** — infusions, subcutaneous prophylaxis, **line flushes**, and heparin-bonded catheters. Missed flushes are a classic reason the platelet count fails to recover.
- Start a non-heparin anticoagulant at therapeutic intensity.** Options include **argatroban**, **bivalirudin**, **danaparoid**, **fondaparinux**, or a **DOAC**. Stopping heparin alone is not enough — the thrombotic risk persists for weeks.
- Send the immunoassay** (anti-PF4 ELISA). It is **very sensitive but poorly specific**, so a positive result in a low-probability patient means little. Confirm with a **functional assay** such as the serotonin release assay where available.
- Image for thrombosis** — at minimum bilateral lower-limb Dopplers, since finding a clot changes the duration of anticoagulation and confirms the diagnosis clinically.
- With a high-probability score and a positive immunoassay, continue avoiding heparin and continue full-intensity non-heparin anticoagulation.** Do not step down to prophylactic dosing.

THE THREE THINGS THAT CAUSE HARM *each is a common error*

WARFARIN TOO EARLY

Do not start warfarin until the platelet count has recovered to at least 150 ×10⁹/L and the patient is established on a non-heparin agent. Early warfarin depletes protein C and causes **venous limb gangrene and skin necrosis**. If it was already started, **reverse it with vitamin K**. Overlap for at least 5 days.

PLATELET TRANSFUSION

Avoid routine platelet transfusion. Reserve it for **active bleeding or before a high-bleeding-risk procedure**. These patients are thrombotic, not hemorrhagic, and the count rarely falls low enough to bleed spontaneously.

UNDER-TREATING

Stopping heparin without substituting another anticoagulant leaves a **high ongoing thrombotic risk**. Also **document the diagnosis prominently** as an allergy or alert — future heparin exposure, including a cardiac bypass run, must be planned deliberately.

CHOOSING THE NON-HEPARIN AGENT *organ function usually decides*

Agent	Best when	Watch for
Argatroban	Renal impairment — hepatically cleared	Reduce the dose in hepatic impairment . Prolongs the INR , which complicates the warfarin transition.
Bivalirudin	Combined hepatic and renal impairment; cardiac procedures	Short half-life, partly enzymatic clearance. Often preferred around percutaneous intervention.
Fondaparinux	Stable patients, straightforward dosing	Renally cleared — avoid in significant renal impairment. No antidote.
DOAC	Clinically stable, absorbing enterally, once past the acute phase	Increasingly used and endorsed as an option; avoid where absorption or adherence is uncertain, or in severe renal impairment.

SPECIAL SITUATIONS *the questions that come up on call*

NEEDS CARDIAC SURGERY

If surgery is **urgent and HIT is acute**, options include **bivalirudin** for bypass or delaying until antibodies clear. If HIT is **historical and the antibody has cleared**, a brief intraoperative heparin exposure is often acceptable, with non-heparin anticoagulation before and after. **Always involve hematology and cardiac anesthesia in advance** — this is not a bedside decision.

OTHER SCENARIOS

Renal replacement therapy: circuits usually need heparin, so switch to a non-heparin circuit anticoagulant or citrate. **Pregnancy:** danaparoid or fondaparinux are generally preferred; warfarin and DOACs are avoided. **Autoimmune HIT** can occur with minimal or no heparin exposure, sometimes after surgery or infection, and may persist or worsen after heparin is stopped.

PEARLS & PITFALLS

- A low 4Ts score means do not test.** Testing low-probability patients generates false positives and unnecessary, expensive, bleeding-prone treatment.
- Stopping heparin is only half the treatment.** Always substitute a non-heparin anticoagulant at therapeutic dose.
- Never start warfarin while the platelets are low** — venous limb gangrene is a devastating, avoidable complication.
- Hunt down every heparin source**, including line flushes and coated catheters.
- Argatroban prolongs the INR**, which complicates the warfarin overlap — use a higher target INR band and confirm after stopping the infusion.
- Duration:** around 3 months if thrombosis was found, and roughly 4 weeks for isolated HIT while the thrombotic risk falls.

References: American Society of Hematology 2018 Guidelines for Management of Venous Thromboembolism: Heparin-Induced Thrombocytopenia (Blood Advances, 2018) · 4Ts score derivation and validation studies.

Educational aid; verify doses against local hematology protocol, renal and hepatic function, and patient factors.

