HEMATOLOGY · PHARMACOLOGY · NCLEX 2026

Heparin-Induced *Thrombocytopenia* (HIT)

A life-threatening, immune-mediated reaction to heparin where the body forms antibodies that paradoxically **drop platelets AND cause clotting** — sometimes called "white clot syndrome."

⚠ Life-Threatening | High-Yield NCLEX | Clinical Judgment | Diane's NGN NCLEX 2026 Series

1 Definition & Overview

What HIT is and why it matters

- **HIT** = an adverse, immune-mediated drug reaction to heparin causing a drop in platelets **AND** abnormal clot formation.
- Occurs in patients receiving **unfractionated heparin (UFH)** or **low-molecular-weight heparin (LMWH, e.g., enoxaparin)**.
- Despite the LOW platelet count, the danger is **thrombosis (clotting)** — not bleeding.

Two Types — Know the Difference

Type I (Non-immune): Mild, transient platelet drop within 1–2 days. Resolves on its own. Heparin can be continued.

Type II (Immune-mediated): SERIOUS. Onset 5–10 days. STOP heparin immediately. This is the HIT tested on NCLEX.

2 Pathophysiology — Simplified

Why platelets drop AND clots form simultaneously

1. Patient receives **heparin** (UFH, LMWH, or even heparin flushes/coated catheters).
2. Heparin binds to **platelet factor 4 (PF4)** on the platelet surface, forming a heparin-PF4 complex.
3. The body sees this complex as foreign and produces **IgG antibodies** against it (typically 5–10 days later).
4. Antibody-coated platelets become **HYPER-activated** → release pro-coagulant material → widespread clot formation.
5. **Result:** Platelets are consumed by clotting (count drops) AND massive thrombosis occurs (DVT, PE, stroke, MI, limb ischemia).

3 Signs & Symptoms

Recognize the paradox: low platelets + clots

⏱ EARLY (Days 5–10)

- **Platelet count drops >50%** from baseline
- Platelet count typically falls below **150,000/mm³**
- Skin lesions/necrosis at heparin **injection sites**
- Erythematous plaques, painful skin reactions
- Low-grade fever, chills after IV heparin bolus

🚨 LATE / SEVERE (Thrombosis)

- **DVT:** unilateral leg swelling, warmth, pain
- **PE:** sudden dyspnea, chest pain, ↓ SpO_2
- **Stroke:** facial droop, slurred speech, weakness
- **MI:** chest pain, ECG changes
- **Limb ischemia:** cold, pale, pulseless extremity
- Skin **necrosis** (gangrene) at injection site

🚨 **RED FLAG: A patient on heparin whose platelet count drops by 50% or more from baseline = SUSPECT HIT until proven otherwise. The bleeding risk is LOW; the clotting risk is HIGH.**

4

Priority Nursing Interventions

ABCs · Safety · Stop the offending agent

🛑 IMMEDIATE ACTIONS

- **STOP all heparin sources** immediately — IV heparin, SQ heparin, LMWH, heparin flushes, AND heparin-coated catheters/lines.
- **Notify the HCP** of suspected HIT and the platelet trend.
- **Start a non-heparin anticoagulant** (argatroban, bivalirudin, or fondaparinux) — patient still needs anticoagulation because of clotting risk.
- **Draw HIT antibody panel** (PF4-heparin ELISA, serotonin release assay).
- **Document HIT** prominently in chart, allergy band, and EHR — patient must avoid heparin **for life**.

📊 ONGOING MONITORING

- Monitor **platelet count daily** (or more often if unstable).
- Assess for new **thrombosis**: peripheral pulses, calf swelling, neuro checks, SpO_2, chest pain.
- Inspect **injection sites** for necrosis, redness, painful plaques.
- Monitor **aPTT** for argatroban; anti-Xa for fondaparinux.
- Assess for bleeding (gums, urine, stool) once on alternative anticoagulant.
- Apply **medical alert bracelet** documenting heparin allergy.

✅ **PRIORITY ORDER (NCLEX): 1️⃣ Stop heparin → 2️⃣ Start non-heparin anticoagulant → 3️⃣ Notify HCP → 4️⃣ Draw labs → 5️⃣ Document & educate**

5 Pharmacology

Avoid these · Use these instead

✅ ALTERNATIVE ANTICOAGULANTS (Use These)

DRUG	MECHANISM	SIDE EFFECTS	NURSING CONSIDERATIONS
Argatroban Direct Thrombin Inhibitor	Directly inhibits thrombin (factor IIa)	Bleeding, hypotension, dyspnea	Hepatic clearance — **use with caution in liver disease**. Monitor **aPTT** (1.5–3× baseline).
Bivalirudin Direct Thrombin Inhibitor	Directly inhibits thrombin	Bleeding, back pain, nausea	Renally cleared — **adjust in kidney disease**. Often used during PCI.
Fondaparinux Factor Xa Inhibitor	Selectively inhibits factor Xa	Bleeding, thrombocytopenia (rare), injection site reaction	SQ injection. **Renally cleared** — avoid in CrCl < 30. Monitor **anti-Xa levels**.
Warfarin Vitamin K Antagonist	Inhibits vitamin K–dependent clotting factors	Bleeding, skin necrosis (early)	⚠ **NEVER start alone in acute HIT** — causes paradoxical thrombosis. Add only AFTER platelets recover (>150K) and overlap with non-heparin agent.

❌ DRUGS TO AVOID IN HIT

All forms of heparin: unfractionated heparin (UFH), LMWH (enoxaparin / Lovenox, dalteparin), heparin flushes, heparin-bonded catheters · **Warfarin alone (initially)** · **Platelet transfusions** (will fuel further clotting!)

6 NCLEX Test Traps ⚠

Where students lose points

❌ WRONG **"Platelets are low — give a platelet transfusion."**
Platelet transfusion fuels further clotting and is contraindicated in HIT (unless active life-threatening bleeding).

✅ RIGHT **STOP heparin and start a non-heparin anticoagulant.**
The danger is clotting, not bleeding. Treat the thrombotic state.

❌ WRONG **"Switch from UFH to enoxaparin (Lovenox)."**
LMWH still triggers HIT antibodies — cross-reactivity. NEVER use as a substitute.

✅ RIGHT **Use argatroban, bivalirudin, or fondaparinux.**
These bypass the heparin-PF4 antibody pathway.

❌ WRONG **"Start warfarin right away to thin the blood."**
Warfarin alone in acute HIT depletes protein C/S first → paradoxical clotting and skin necrosis.

✅ RIGHT **Wait until platelets recover (>150K), then bridge with non-heparin anticoagulant.**
Always overlap warfarin for at least 5 days.

❌ WRONG **"Continue heparin flushes for the IV line — it's just a small amount."**
ANY heparin exposure perpetuates the immune reaction.

✅ RIGHT **Remove ALL heparin — flushes, coated catheters, SQ, IV.**
Use saline flushes only.

7 Patient & Family Education

Lifelong heparin avoidance

🪪 Identification

- Wear a **medical alert bracelet** stating "Heparin Allergy / HIT"
- Carry a wallet card
- Tell **every** provider (dentist, surgeon, ER)

🚫 Avoid Forever

- All forms of heparin (UFH, Lovenox, flushes)
- Heparin-coated catheters
- Bridging with LMWH for surgery

👀 Watch For

- **Clot signs:** leg swelling, sudden chest pain, shortness of breath, weakness
- **Bleeding signs:** bruising, dark stool, bleeding gums (while on alt. anticoag)
- Painful skin lesions

Teach-back focus: "If you ever go to the ER or have surgery, the FIRST thing you tell them is that you cannot have heparin or Lovenox in any form."

8 Memory Boosters & Mnemonics

Pattern recognition for fast recall

🧠 The 4 T's of HIT (Diagnostic Score)

- **T** **Thrombocytopenia** — platelet drop >50% from baseline
- **T** **Timing** — onset 5–10 days after starting heparin (sooner with prior exposure)
- **T** **Thrombosis** — new clot, skin necrosis, or anaphylactoid reaction
- **T** **oTher causes** ruled out (sepsis, DIC, drug-induced thrombocytopenia)

💡 H · I · T

- **H** **Halt** heparin (all forms — flushes too!)
- **I** **Initiate** alternative anticoagulant (argatroban / bivalirudin / fondaparinux)
- **T** **Track** platelets daily and watch for thrombosis

🩸 "White Clot Syndrome"

Clots in HIT are **platelet-rich** (not red-cell rich), so they appear pale/white in pathology — hence the nickname.

Pattern: Patient on heparin × 5–10 days → platelets crash → leg swells → think HIT, NOT bleeding.

🔑 The Paradox to Remember:

In HIT, platelets are LOW but the patient is NOT at risk for bleeding — they're at risk for **CLOTTING**. So the answer is NEVER "give platelets" — it's always "stop heparin and anticoagulate with something else."

9 NCJMM · Clinical Judgment Scenario

Six-step Next-Generation NCLEX walkthrough

Scenario: *A 68-year-old client is on day 7 of IV unfractionated heparin for a DVT. The nurse reviews morning labs: platelets 78,000/mm³ (down from 220,000/mm³ on admission). The client now reports new right calf pain and swelling, and a small, dusky lesion is noted at the SQ heparin injection site on the abdomen. Vitals: BP 138/82, HR 96, SpO_2 95%, T 99.4°F.*

STEP 1

Recognize Cues

Relevant: Day 7 of heparin · platelet drop >50% (220K → 78K) · new calf pain/swelling · dusky skin lesion at injection site · low-grade fever. **Less relevant:** BP and SpO_2 within acceptable limits.

STEP 2

Analyze Cues

The cues meet the **4 T's** of HIT: Thrombocytopenia (50%+ drop), Timing (day 7), Thrombosis (calf pain + skin lesion), and no other obvious cause. The new calf swelling suggests **extension of DVT or a new thrombus** — the paradox of HIT.

STEP 3

Prioritize Hypotheses

#1 (most likely & life-threatening): Type II HIT with new thrombosis. **#2:** Heparin-induced skin necrosis. **#3 (less likely):** Sepsis-related thrombocytopenia or DIC. HIT must be addressed FIRST given the timing pattern.

STEP 4

Generate Solutions

Stop ALL heparin (IV drip, SQ, flushes, coated lines) · Notify HCP STAT · Anticipate orders for argatroban or fondaparinux · Draw HIT antibody panel (PF4 ELISA) · Hold platelet transfusion · Apply medical alert · Document HIT prominently.

STEP 5

Take Action

FIRST action: Stop the heparin infusion and remove any heparin flushes. Then notify the provider, draw labs, and initiate the alternative anticoagulant once ordered. Educate client about lifelong heparin avoidance.

STEP 6

Evaluate Outcomes

Expected: Platelets begin to recover within 4–7 days · No new thrombosis · Skin lesion does not progress to necrosis · aPTT therapeutic on argatroban · Client correctly verbalizes need to avoid heparin and wears alert bracelet.