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Masterthesis

Management of a thrombotic event in an acute heparin induced thrombocytopenia with thrombosis (HITT) patient : case report and narrative

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Abstract

Background: **Heparin-induced thrombocytopenia (HIT) is a severe immune-mediated complication of heparin therapy, potentially leading to life-threatening thrombotic events, referred to as HIT with thrombosis (HITT).** ***Case Presentation:*** **We report the case of a 46-year-old female patient with severe peripheral arterial occlusive disease who developed acute HITT after undergoing a covered endovascular reconstruction of the aortic bifurcation (CERAB). Perioperative heparin was administered during CERAB procedure. Low-molecular-weight heparin (LMWH), initially prescribed postoperatively, was replaced by apixaban due to thrombocytopenia. Despite this, an intra-stent thrombosis occurred, requiring urgent surgical revascularization. Intervention and Outcome: HITT was confirmed by a positive anti-PF4/heparin antibody assay and validated by functional platelet activation tests. Surgery was performed under danaparoid anticoagulation. Thrombectomy was achieved using a Fogarty catheter. The patient's limb was reperfused, her platelet count normalized, and no hemorrhagic complications occurred. Postoperative management included a tailored anticoagulation regimen.** ***Conclusion:*** **Anticoagulation is the cornerstone of HITT. Vascular surgery in this pathology is particularly challenging due to the need for perioperative anticoagulation while avoiding heparin exposure. This case illustrates the importance of early recognition and personalized perioperative management in HITT. A multidisciplinary approach is essential to ensure optimal outcomes while mitigating both thrombotic and hemorrhagic risks.**

Keywords: **Heparin-induced thrombocytopenia, thrombosis, vascular surgical procedures, anticoagulants, perioperative care.**

Introduction

Heparin therapy remains a cornerstone in the management of thromboembolic events. It is also essential during vascular surgery, particularly when vascular clamping is required1. However, its use may be complicated by heparin-induced thrombocytopenia (HIT), a rare but potentially severe immune-mediated reaction that can evolve into thrombosis (HITT). This paradoxical prothrombotic state poses significant challenges in the perioperative setting, especially when surgical revascularization necessitates intraoperative anticoagulation[2-4].

While the literature extensively describes HIT and its implications for coagulation, the perioperative surgical and anesthetic management of patients with HITT undergoing emergency revascularization remains insufficiently standardized[3,5]. Managing such patients demands a delicate balance: avoiding all heparin exposure, ensuring effective anticoagulation, and minimizing hemorrhagic risks, particularly relevant during open vascular procedures[1,5,6].

In this context, we present the case of a patient with HITT requiring urgent revascularization, where both anticoagulation protocol and surgical technique had to be specifically adapted. We discuss therapeutic

alternatives and strategies aimed at optimizing the management of high-risk patients, drawing from a narrative review of the current literature.

Case report

The patient provided informed consent for the anonymous publication of her clinical case. Approval was also obtained from the Ethics Committee of Liège University Hospital (Comité d'Éthique Hospitalo-Facultaire Universitaire de Liège, number 707 - ref 2025/225).

A 46-year-old female patient (85 kg, 163 cm) was scheduled for a covered endovascular reconstruction of the aortic bifurcation (CERAB). Her medical history included severe peripheral arterial occlusive disease, non-insulin-dependent diabetes, and active smoking. She had previously undergone lumbar spinal fusion and intestinal resection for a benign tumor. Her chronic medications included bisoprolol, bumetanide, simvastatin, metformin, lormetazepam, paroxetine, mirtazapin, pregabalin, pramipexole, pantoprazole, and inhaled budesonide/formoterol and salbutamol.

Preoperative laboratory tests showed mild anemia (10.6 g dL^{-1}) and a normal platelet count (366 000 mm^{-3}). Coagulation parameters, as well as renal and liver function tests, were within normal limits.

The procedure was uneventful under general anesthesia. The patient was administered 100 IU kg^{-1} of unfractionated heparin (UFH) intraoperatively. The early postoperative course was unremarkable. Dual antiplatelet therapy with aspirin 80 mg once daily (oid) and clopidogrel 75 mg oid was initiated on postoperative day 2 (POD 2), and the patient was discharged.

On POD 4, the patient was readmitted due to a localized collection at the right inguinal incision site. Cultures were obtained, and empirical antibiotic therapy with cefazolin 2g three times daily (tid) was started. Thromboprophylaxis with low-molecular-weight heparin (enoxaparin 40 mg oid) was prescribed the following day, as the patient was advised to remain on bed rest.

Four days after LMWH initiation, a routine blood test revealed significant thrombocytopenia (54 000 mm^{-3}). The 4T score (Table I) was calculated at 4, prompting the immediate discontinuation of LMWH. An enzyme-linked immunosorbent assay (ELISA) for anti-PF4 antibodies was markedly positive (99.65 U mL^{-1}; normal < 1 U mL^{-1}) the following day, and apixaban 2.5 mg twice daily (bid) was initiated.

Two days later, the patient developed bilateral lower limb paresthesia. Angio-CT imaging revealed extensive intra-stent thrombosis, prompting emergency surgical revascularization. A Fogarty embolectomy under radioscopic guidance was

Table I. — Adapted from the 4Ts score proposed by Warkentin et al. (with author's permission) (15).

	2 points	1 point	0 point
Thrombocytopenia	Platelet count decrease > 50% AND a nadir >= 20 AND no surgery within preceding 3 days	Platelet count decrease > 50% BUT surgery within preceding 3 days OR 30-50% platelet count decrease OR nadir between 10-19	< 30% platelet count decrease OR nadir < 10
Timing	Platelet count decrease day 5-10 after initiation Of heparin therapy OR Platelet count decrease within 1 day after initiation Of heparin therapy AND exposure to heparin within past 30 days	Consistent with Platelet count decrease day 5-10 but not clear OR Platelet count decrease within 1 day after initiation Of heparin therapy AND exposure to heparin within past 31 to 100 days. OR Platelet count decrease after day 10	Platelet count decrease day 4 without exposure to heparin
Thrombosis or other sequelae	Confirmed new thrombosis veinous or arterial OR Skin necrosis OR Systemic reaction to IV heparin bolus	Extension of an existing thrombosis or recurrence of a thrombo-embolic event on anticoagulant therapy. OR suspected thrombosis OR Erythematous skin lesions	Thrombosis not suspected
OTher causes for thrombocytopenia	None	Possible	Probable

selected, enabling real-time stent monitoring and intraoperative arteriographic control. Limb reperfusion was successfully achieved.

A perioperative anticoagulation protocol was developed in collaboration with the hematology team and tailored to renal function. It began with an intravenous bolus of danaparoid (3000 IU), followed by a continuous infusion: 400 IU h^{-1} for 4 hours, then 300 IU h^{-1} for 4 hours, and finally 150–200 IU h^{-1} until POD 5 according to anti-Xa levels. These were measured 8 hours after initiation and then daily, with a target range of 0.5 and 0.8 IU mL^{-1}. Danaparoid was subsequently transitioned to subcutaneous administration (4500 IU bid) for four days, and then to fondaparinux (7.5 mg oid).

The platelet count returned to baseline 15 days after LMWH cessation. The patient was discharged 12 days after the second procedure. Unfortunately, she developed dry necrosis of the right hallux and the distal phalanx of the second toe, necessitating minor amputation at a later stage. She remained on dual antiplatelet therapy (aspirin and clopidogrel) and fondaparinux. Clopidogrel was discontinued after one month, while aspirin and fondaparinux were continued for three months. She was then switched to a combination of aspirin and rivaroxaban 2.5 mg bid, and later to a vitamin K antagonist, with a target INR of 2–3, following the diagnosis of antiphospholipid syndrome during postoperative evaluation.

Discussion

Pathophysiology of HIT

We present a case of surgical management of an extensive thrombosis of the aorto-iliac bifurcation resulting from a HIT.

HIT is a complication of heparin therapy and comprises two distinct types:

- Type I HIT is a non-immune, benign reaction that typically arises within 1-2 days after the initiation of heparin. It is transient, resolves spontaneously even with continued heparin exposure, and is likely due to the direct pro-aggregatory effect of heparin on platelets[2,3].
- Type II HIT, which is the focus of this report, is an immune-mediated disorder involving IgG antibodies against platelet factor 4 (PF4)/heparin complexes. This results in the formation of neo-complexes, which in turn bind to and activate Fcɣ platelet receptors (FcɣRIIa) and monocyte receptors (FcɣRI), leading to thrombin generation and a hypercoagulable state[2,4] (Figure 1).

Type II HIT occurs in fewer than 0.1% to 7% of patients exposed to heparin, with incidence varying by the type and duration of heparin exposure, being more frequent with bovine or unfractionated heparin than with porcine heparin or LMWH. It is also more common in medical than in surgical patients[3,5,6]. Female sex and advanced age are considered as predisposing factors[7].

Diagnostic approach

Thrombocytopenia (defined as a platelet count <150 000 μL^{-1}) is typically the first indicator raising suspicion for type II HIT. Key diagnostic features include the extent of the platelet drop, its timing in relation to heparin administration, the presence of thrombotic complications, and the exclusion of alternative causes of thrombocytopenia. In HIT, thrombocytopenia is generally moderate, characterized by a 30–50% reduction from baseline. A nadir below 20 000 μL^{-1} should prompt reconsideration of the diagnosis. The baseline refers to the highest platelet count recorded immediately prior to HIT suspicion[3,7-9]. The platelet count typically decreases between days 5 and 14 following the initiation of heparin. However, "early-onset HIT" can develop within 24 hours in patients previously exposed to heparin within the past 100 days. Conversely, rare cases of "delayed-onset HIT", in which thrombocytopenia and/or thrombosis occur several days after stopping heparin, have also been described[10,11].

Thrombocytopenia is not uncommon in the perioperative setting. Differential diagnoses include pseudothrombocytopenia, hemodilution, intraoperative platelet consumption or bleeding, transfusion-related alloimmunization, and drug-induced thrombocytopenia, which may be triggered by agents such as cephalosporins or amiodarone[9,12].

In all patients who develop thrombocytopenia during or after heparin exposure, the 4T score (Table I) should be calculated[13]. This clinical scoring tool estimates the probability of HIT based on four criteria: the degree of thrombocytopenia, the timing of platelet count decline, the occurrence of thromboembolic events, and the presence or absence of alternative explanations[14,15]. Importantly, asymptomatic thrombotic events, particularly deep vein thrombosis of the lower limbs, should be ruled out using Doppler ultrasound before completing the 4T score[3,13]. The score ranges from 0 to 8. A low probability (0–3) usually rules out HIT, and no further testing is required. Intermediate (4–5) and high (6–8) scores warrant further investigation, immediate discontinuation of heparin, and initiation of a non-heparin anticoagulant[6,14]. Heparin-coated catheters should also be removed when present[10].

The first-line confirmatory test is the semi-quantitative enzyme-linked immunosorbent assay

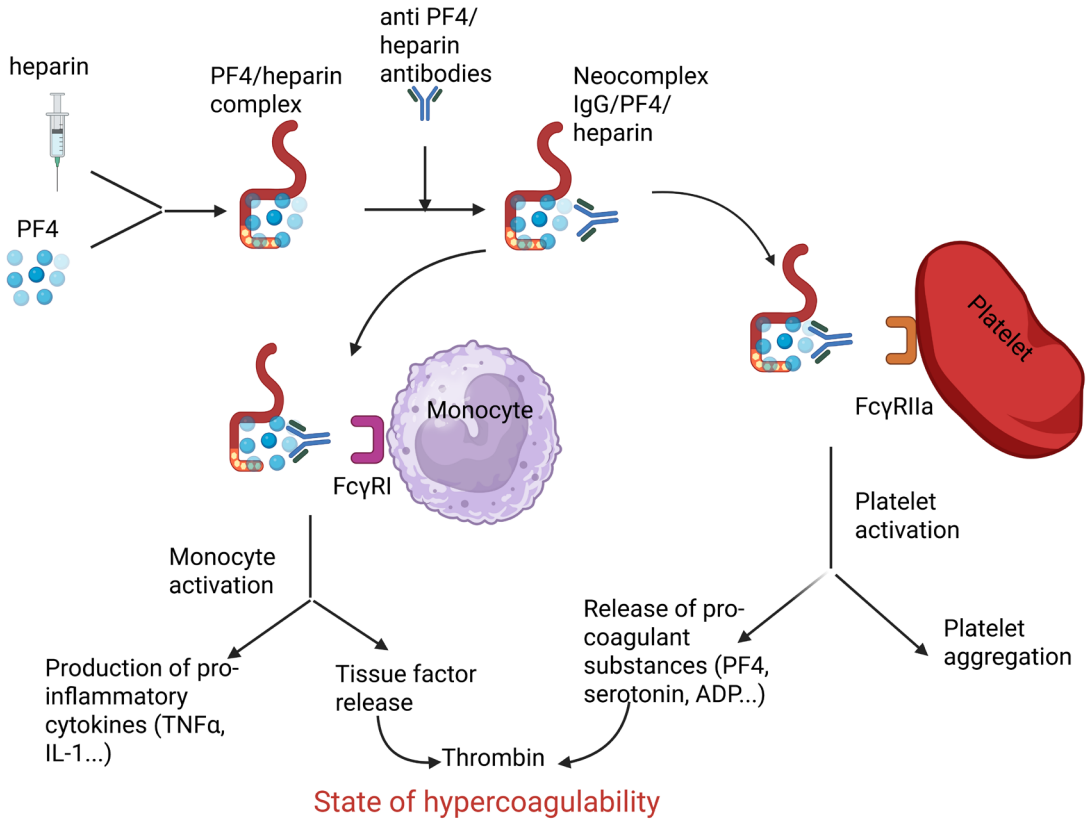

Fig. 1 — Type II HIT results from an immune-mediated response triggered by the formation of PF4/heparin complexes, recognized as neoantigens by the immune system. IgG antibodies bind to these complexes and engage FcγRIIa receptors on platelets, inducing their activation, further PF4 release, and thrombin generation. This feedback loop establishes a prothrombotic state despite thrombocytopenia. Monocyte activation via Fcγ receptors contributes to coagulation through tissue factor expression. This mechanism accounts for the paradoxical thrombosis risk in HIT and underlines the challenges in perioperative anticoagulation.

Created with BioRender.com. Reynkens, A. (2025) https://BioRender.com/mqi1c8o. Diagram of the pathophysiology of HIT.

(ELISA) for anti-PF4 antibodies. While it has a high negative predictive value (>99%), its specificity is limited (30–70%)[2,3]. Functional assays, such as the serotonin-release assay (SRA) and the heparin-induced platelet activation assay (HIPA), evaluate the ability of the antibodies to activate platelets. These are used as confirmatory tests due to their higher specificity (approximately 80%), but they are only available in specialized centers and often involve delays[4,6,10]. In cases with a high 4T score and a clearly positive ELISA, the probability of a positive functional test is high (83–95%), making further testing unnecessary[2,13].

Management Strategies

In this clinical case, the patient had a 4T score of 4 at the time thrombocytopenia was identified, and the anti-PF^4 antibody test returned strongly positive the following day. Based on this, heparin was discontinued and a direct oral anticoagulant was initiated.

Thromboembolic complications represent the most frequent (up to 64%) and severe manifestations of HIT[11]. Venous thromboses (17–55%) are more common than arterial thromboses (3–10%). In approximately 25% of cases, thrombotic events may even precede the onset of thrombocytopenia[10]. Therefore, HIT should be considered in any thrombotic event in a patient with recent or ongoing heparin exposure, regardless of platelet count. The mortality associated with HIT-related thrombosis ranges from 6% to 30%[16,17]. Additional complications may include venous limb gangrene, particularly when vitamin K antagonists are used during acute HIT, and hemorrhagic adrenal necrosis due to adrenal vein thrombosis[3,8,11,18]. Hemorrhagic manifestations remain rare despite thrombocytopenia[10].

Once HIT is confirmed, therapeutic anticoagulation with a non-heparin agent should be initiated or continued, irrespective of the presence of thrombosis[13,16]. Several anticoagulants are available, and those compatible with time-sensitive surgery requiring short-term intraoperative anticoagulation are discussed below.

It is important to note that HIT does not confer immune memory. Anti-PF4/heparin antibodies are transient and typically disappear within 40 to 100 days following an acute episode[9,19]. Five successive clinical phases have been described, each associated with distinct risks of HIT

reactivation and thrombosis in the event of heparin re-exposure[10,19,20]:

- Suspected HIT: the phase during which clinical findings raise suspicion.
- Acute HIT: the diagnosis is confirmed (typically via functional testing such as SRA), but the platelet count has not yet returned to normal. Recovery generally occurs within seven days of discontinuing heparin.
- Subacute HIT A: after the platelet count has returned to baseline but the functional test remains positive (median of 50 days), indicating that antibodies are still detectable and active.
- Subacute HIT B: the functional test has turned negative, but the immunologic test remains positive (median of 85 days), meaning the antibodies remain detectable but inactive.
- Remote HIT: Anti-PF4/heparin antibodies are no longer detectable by immunologic testing[1,5,7].

The highest risk of heparin re-exposure occurs during the acute and subacute A phases and declines substantially in the remote phase, though data remain limited. Therefore, whenever possible, surgical procedures should be postponed until the patient reaches the remote phase, when antibodies are no longer detectable[1,6,13]. In such cases, intraoperative heparin use may be considered safe, while alternative anticoagulation should be employed during the pre- and postoperative periods[6,13].

When surgery cannot be delayed, three perioperative anticoagulation strategies are available:

- Use of a non-heparin anticoagulant intraoperatively
- Pre- or intraoperative plasmapheresis followed by heparin anticoagulation
- Heparin anticoagulation combined with antiplatelet agents (such as cangrelor or tirofiban)[10,19].

The choice between these strategies largely depends on available resources, clinician expertise, and cost considerations[1,13]. It is essential to involve both hematologists and vascular surgeons in perioperative planning to tailor the safest and most effective management approach[16].

In this case, urgent surgical intervention was required during the acute HIT phase, when the patient's platelet count had not yet normalized. Therefore, the intraoperative use of a non-heparin anticoagulant was preferred to minimize the risks associated with heparin exposure.

Alternative Anticoagulants

Among the available alternatives, bivalirudin (Angiox®), a direct thrombin inhibitor derived from hirudin, is a key option. Current guidelines recommend bivalirudin as the anticoagulant of choice in patients with HIT undergoing percutaneous coronary interventions (PCI) and cardiac surgery. Its metabolism is predominantly enzymatic (80%), with renal elimination accounting for the remaining 20%[6,9,17,21]. For off-pump vascular and cardiac surgery, the standard dosing regimen includes an initial bolus of 0.75 mg kg^{-1}, followed by a continuous infusion of 1.75 mg kg^{-1} h^{-1}. The target is an activated clotting time (ACT) greater than 300 seconds[1,21]. Bivalirudin offers the advantage of a short half-life (20–30 minutes), allowing rapid adjustment. However, caution is advised in patients with renal impairment or during cardiocirculatory arrest with hypothermia, where blood stasis may reduce circulating drug concentrations[1,21,22]. Clinical studies have demonstrated that bivalirudin provides a safety and bleeding profile comparable to heparin in cardiac surgery, regardless of HIT status[21].

Argatroban (Arganova®), another viable option, is a synthetic direct thrombin inhibitor derived from L-arginine, administered intravenously. It is especially recommended in the medical management of HITT and is preferred for patients with renal impairment due to its hepatic elimination[6,10,21]. In vascular surgery, it may be administered as a continuous infusion (2 to 5 µg kg^{-1} min^{-1}, initiated 30 to 60 minutes preoperatively) or as a bolus of 100 µg kg^{-1} followed by an infusion under 2 µg kg^{-1} min^{-1}, with a target ACT of 200–300 seconds[1,11]. Despite a relatively short half-life (40–50 minutes), it is contraindicated in severe hepatic dysfunction. It is currently unavailable in Belgium[5,6,12,15,21].

In this case, danaparoid (Orgaran®) was selected in consultation with the hematology team. Danaparoid is a heparinoid mixture that primarily exerts its effect through anti-Xa activity, with some anti-IIa properties[6,10]. It inhibits PF4/heparin complex formation and is renally eliminated[17]. Indicated for both the prophylaxis and treatment of thromboembolic events in HIT, danaparoid can be administered subcutaneously or via continuous infusion. The latter requires a weight-based loading dose (1250 U if under 55 kg; 2500 U for 55–90 kg; 3750 U if over 90 kg), followed by a stepped infusion: 400 U h^{-1} for two hours, then 300 U h^{-1} for two hours, and a maintenance dose of 200–150 U h^{-1} [6]. Anti-Xa activity should be monitored with a target range of 0.5–0.8 IU mL^{-1}, particularly in patients with renal impairment or those weighing over 90 kg[6,16]. Although its long half-life (~24 hours) and limited thrombin inhibition in clot-bound environments may restrict its use in surgical contexts, it was deemed appropriate in our patient due to stable renal function (GFR 45 mL min^{-1})

and the unique advantage of allowing continuous perioperative anticoagulation through both intravenous and subcutaneous routes[10,11,13].

Danaparoid remains a recommended treatment for HITT and facilitates the transition into postoperative management. However, in patients with renal dysfunction, bivalirudin would be the preferred agent[10,16]. Despite promising clinical data, bivalirudin remains challenging in practice. Although activated partial thromboplastin time (aPTT) is widely used to monitor its effect, the correlation between aPTT and anticoagulant activity can be unreliable, especially in perioperative inflammation where factor VIII levels are often elevated[21,22]. Transfusions and hypothermia can further distort aPTT readings[22]. While bivalirudin's short half-life enables rapid dose adjustments, ACT may offer a more accurate monitoring method during surgery[21].

Antiplatelet Strategies

Antiplatelet agents such as cangrelor and tirofiban have been reported in specific cardiac surgical settings. Their primary goal is to inhibit platelet activation prior to heparin administration, thereby reducing the formation of PF4/heparin antibody complexes and the associated thrombotic risks[23,24].

Cangrelor, a reversible P2Y12 receptor antagonist, features a short half-life (3–6 minutes), rapid dephosphorylation clearance, and restoration of platelet function within two hours of discontinuation. Platelet function can be monitored using platelet reactivity units (PRU) testing, although defined target values are lacking[23,24]. In the protocol described by Seider et al., cangrelor was administered as a 30 $\mu g\ kg^{-1}$ bolus 10 minutes before heparin, followed by a continuous infusion at 4 $\mu g\ kg^{-1}\ min^{-1}$ [23].

Tirofiban, a GPIIb/IIIa receptor antagonist, also prevents platelet aggregation. However, its renal clearance and longer half-life (~2 hours) increase the risk of bleeding, especially in patients with renal impairment[23,24]. In the same case report, tirofiban was administered as a 10 $\mu g\ kg^{-1}$ bolus 15 minutes prior to heparin, followed by a 0.15 $\mu g\ kg^{-1}\ min^{-1}$ infusion. Heparin was introduced once 80% receptor blockade was achieved, as confirmed using the VerifyNow IIb/IIIa assay. The tirofiban infusion was discontinued one hour before weaning from cardiopulmonary bypass, and heparin was reversed with protamine[23].

We did not pursue this therapeutic option, as anticoagulation was required in our case, and antiplatelet therapy was deemed impractical in our center due to the limited availability of the Multiplate® analyzer in the perioperative setting.

To assist clinicians in choosing the most appropriate strategy, Table II provides a comparative overview of the main anticoagulant and antiplatelet agents available for perioperative use in HIT patients.

Additional Strategies

Other therapeutic strategies include plasmapheresis and intravenous immunoglobulins. Plasmapheresis is primarily indicated in urgent cardiac surgeries to remove circulating anti-PF4/heparin antibodies, thereby allowing limited intraoperative use of

Table II. — Comparative overview of perioperative anticoagulants used in the management of HIT patients undergoing urgent surgery.

Anticoagulant	Mechanism of Action	Half-life	Elimination	Monitoring	Notes
Danaparoid	Anti-Xa/ Anti IIa	~ 24h	Renal	Specific anti-Xa activity	Easier post-operative transition (IV → SC)
Bivalirudin	Direct thrombin inhibitor	20 –30 min	80% enzymatic/ 20% renal	aPTT or ACT	1st-line in PCI & cardiac surgery (guidelines)
Argatroban	Direct thrombin inhibitor	40 –50 min	Hepatic	aPTT or ACT	Not available in Belgium
Cangrelor	P2Y12 receptor inhibitor (antiplatelet)	3–6 min	Enzymatic	PRU	In association with UFH
Tirofiban	GPIIb/IIIa receptor inhibitor (antiplatelet)	~2 h	Renal	VerifyNow IIb/IIIa	In association with UFH

ACT : activated clotting time - aPPT : activated partial thromboplastin time - IV : intravenous - PCI : percutaneous coronary intervention - PRU : P2Y12 reaction units - SC : subcutaneous - UFH : unfractioned heparin.

heparin[17]. However, in cases of intraoperative hemodynamic instability, it may also be initiated during surgery. In such situations, supplemental intravenous heparin (0.4 U mL^{-1} per mL plasma removed) is required to maintain anticoagulation[25].

Intravenous immunoglobulins (IVIG) inhibit platelet activation through modulation of Fcγ receptors. Their efficacy may vary depending on FcγRIIa polymorphisms. A dose of 2 g kg^{-1} has been shown to significantly reduce HIT-related platelet activation[9].

In this case, plasmapheresis remained a possible option but was considered a second-line strategy due to the complexity of its implementation and limited institutional experience, despite supportive evidence in the literature.

Anesthetic and Perioperative Considerations

Neuraxial regional anesthesia is generally contraindicated in HIT patients due to the bleeding risk associated with non-heparin anticoagulants and the mandatory interruption periods that increase the thrombotic risk[20]. Currently, no specific guidelines exist for other regional anesthesia techniques; these should be evaluated on a case-by-case basis, weighing the potential benefits against bleeding and thrombotic risks.

Platelet transfusion is not recommended unless there is active bleeding, as transfused platelets may interact with circulating anti-PF4 antibodies, potentially leading to microthrombi formation[13]. No specific recommendations exist for red blood cell or plasma transfusion or for the management of hemorrhagic shock in the setting of HIT. Standard transfusion thresholds remain applicable.

Therapeutic anticoagulation should be continued for at least four weeks in isolated HIT and for a minimum of three months in cases of HITT[6]. As previously mentioned, when heparin is deemed acceptable for intraoperative use (subacute B and remote phases), it should be strictly limited to the intraoperative setting. Alternative anticoagulants should be used for pre- and postoperative periods. Postoperative monitoring of platelet counts is advised to ensure recovery and to detect potential late complications[13].

Postoperative Course

In our patient, intravenous danaparoid was transitioned to subcutaneous administration and subsequently to fondaparinux, primarily to enhance patient comfort due to its lower injection volume. Fondaparinux (Arixtra®), a synthetic pentasaccharide, has demonstrated efficacy in HIT. However, rare instances of in vitro cross-reactivity with anti-PF4/heparin antibodies have been reported[26].

During postoperative evaluation, our patient was also diagnosed with antiphospholipid syndrome (APS), in addition to confirmed HIT. Although this association is rare, both conditions are immune-mediated prothrombotic syndromes that share same pathogenic mechanisms, notably IgG-mediated platelet activation via Fcγ receptors[16,27]. Thrombotic events in HIT and APS can occur in both arterial and venous territories. In this case, the extensive intra-stent thrombosis may have resulted from a synergistic effect of both disorders, potentially triggered by vascular instrumentation. This so-called "second hit" phenomenon, described in the literature, could explain the severity of the clinical presentation[16,28]. A few reports have documented successful use of intravenous immunoglobulins in both HIT and APS, suggesting overlapping pathogenic pathways[16]. While no definitive causal link between the two conditions has been established, their coexistence underscores the importance of vigilant perioperative monitoring and the need for individualized anticoagulation strategies in high-risk patients[27,28].

Conclusions

This case highlights the complexity of managing HITT in the setting of urgent revascularization surgery. Clinicians should maintain a high index of suspicion for HIT in any thrombotic event following heparin exposure, regardless of the presence of thrombocytopenia. Diagnosis relies on a combination of clinical assessment, using the 4T score, and immunologic testing. Once HIT is suspected, all forms of heparin, including heparin-coated devices, must be immediately discontinued, and an alternative anticoagulant initiated.

In our case, danaparoid (Orgaran®) was used successfully as a perioperative anticoagulant. Its dual intravenous and subcutaneous administration routes facilitated continuous anticoagulation throughout the perioperative period, offering a practical and effective alternative while minimizing thrombotic and hemorrhagic risks. Platelet transfusion should be avoided unless active bleeding occurs, due to the prothrombotic potential of antibody-mediated platelet activation.

The absence of standardized guidelines for perioperative anticoagulation in HITT underscores the need for close multidisciplinary collaboration. This case highlights how close collaboration between vascular surgeons, anesthetists, and hematologists allows for the development of an individualised, structured strategy that contributes to a successful surgical outcome.

Future studies are needed to refine perioperative anticoagulation protocols for HIT patients undergoing urgent vascular procedures and to assess the potential role of newer antithrombotic agents and evolving surgical strategies in this high-risk population.

Ethics approval and consent to participate: The patient provided informed consent for the anonymous publication of her clinical case. Approval was also obtained from the Ethics Committee of Liège University Hospital (Comité d'Éthique Hospitalo-Facultaire Universitaire de Liège, number 707 - ref 2025/225).

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The remaining authors declare no conflicts of interest.

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