EXPERT MEDICAL REPORT
FOR THE COURT

Prepared by

Dr Jordan Burgess
Consultant Haematologist
FRCPath, MRCP, MBChB (Hons), M.Phil (Cantab), M.Math, M.Phys
Queen Elizabeth University Hospital, 1345 Govan Road, Glasgow, G51 4TF

Mr A

v

Large UK Tertiary Teaching Hospital

Date of report:	26 April 2026
Author:	Dr Jordan Burgess
Specialist field:	Haematology
Instructed on behalf of the Pursuer:	Mr A
On the Instructions of:	Instructing Solicitors
Subject matter:	Heparin-induced thrombocytopenia with thrombotic complications

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Appendices

Report

1. Introduction / Executive summary

1.01. The Expert

I am Dr Jordan Burgess, Consultant Haematologist at Queen Elizabeth University Hospital, Glasgow. I am on the GMC Specialist Register for Haematology. My clinical practice includes the diagnosis and management of thrombosis, haemostasis and anticoagulation-related disorders. Full details of my qualifications and experience are set out in Appendix 1.

1.02 Summary background of the case

The case concerns Mr A, a 66-year-old male (date of birth: 10 April 1960), who underwent an elective right-sided total hip replacement on 10 January 2026. He received post-operative thromboprophylaxis with heparin (a “blood thinner” to prevent blood clots).

He subsequently developed severe thrombocytopenia and thrombosis, including pulmonary embolism and intra-abdominal venous thromboses. The central issue in this report is whether these events were due to heparin-induced thrombocytopenia with thrombosis (HITT).

A technical summary outlining HITT in further detail can be found in Appendix 5.

1.03 Summary of my conclusions

In summary, I am of the opinion that Mr A developed heparin-induced thrombocytopenia with thrombosis between 15 and 16 January 2026. In my opinion, a reasonably competent consultant haematologist would have considered HIT as a leading diagnosis when Mr A presented with new thrombosis, severe thrombocytopenia and recent heparin exposure. The failure

to consider HIT, stop heparin, avoid platelet transfusion in the absence of major bleeding, and commence non-heparin anticoagulation represented a departure from accepted haematology practice. In my opinion, those departures materially contributed to the subsequent progression of thrombosis.

1.04 Technical terms and explanations

I have indicated any technical terms in **bold type**. I have defined these terms when first used and included them in a glossary in Appendix 4.

References to published works are marked with a superscript number (e.g., reference[1]), and are listed in Appendix 3.

Direct quotes from records are indicated in *italic script*.

2. The issues to be addressed and a statement of instructions

2.01 The purpose of the report

I have been instructed by the instructing solicitors to write a medical report on Mr A.

I have interpreted the instructions as follows:

1. On the balance of probabilities, did Mr A develop heparin-induced thrombocytopenia and thrombosis (HITT), and if so, when did this occur?

2. Was the management of Mr A's condition in accordance with a reasonable standard of care?
    - If not, please identify **any specific acts or omissions** that fell below that standard, and when.

3. If the standard of care was not met, did it **cause or materially contribute** to:
    - The progression of thrombosis (including pulmonary embolism and intra-abdominal thrombosis)

I have confined my opinion to haematological diagnosis, investigation, anticoagulation, platelet transfusion, and causation from a haematology perspective. I do not offer an opinion on the standard of care expected of Emergency Medicine, Intensive Care Medicine, General Surgery or Radiology, except insofar as their actions provide the factual background to the haematology opinion.

2.02 Materials considered

I have examined the following documents:

- GP records
- Orthopaedic admission notes, 10–14 Jan 2026
- ED notes, 16 Jan 2026
- ICU notes, 16–25 Jan 2026
- Haematology advice entries, 16–17 Jan 2026
- Laboratory results, including platelet counts, coagulation, HIT antibody
- Radiology reports: CTPA, CT abdomen
- Operation note, 18 Jan 2026
- Discharge summary, 20 Feb 2026

3. My investigation of the facts

3.1. Chronology

3.01 Mr A was born on 10 April 1960. He has a past medical history of severe **osteoarthritis** of the right hip, which was diagnosed in October 2023. He was placed on the waiting list for a total hip replacement in November 2023.

3.02 On 10 January 2026, Mr A was admitted to the Queen Elizabeth University Hospital (QEUH). At approximately 08:30, Mr A underwent an elective total hip replacement performed by Dr O. The procedure was completed at approximately 11:00. No intraoperative complications were documented. A pre-operative platelet count (08:00) was 245 × 10^9/L (normal range: 150 – 450).

3.03 Between 10 and 14 January 2026, Mr A remained an inpatient. He received **thromboprophylaxis** with **enoxaparin** 40 mg **subcutaneous injections** once daily. The first dose was administered at approximately 20:00 on 10th January 2026.

3.04 Serial platelet counts were monitored throughout his admission:

Date	Platelet count
10th January 2026	245 × 10^9/L
11th January 2026	210 × 10^9/L
12th January 2026	180 × 10^9/L
13th January 2026	155 × 10^9/L

3.05 He recovered as expected from the procedure. He worked with the physiotherapists and mobilised without assistance on 13 January 2026. No thrombotic complications were documented during this admission. No formal assessment for heparin-induced thrombocytopenia was documented.

3.06 On 14 January 2026, at approximately 11:00, Mr A was discharged home. A platelet count was not checked on the day of discharge. He was prescribed

prophylactic enoxaparin 40 mg once daily subcutaneously for 6 weeks. I do not express an opinion on whether a platelet count should have been checked on discharge, as this was not necessary for my conclusions.

16 January 2026

3.07 At approximately 14:20, Mr A presented to the QEUH Emergency Department (ED) with **pleuritic chest pain** and shortness of breath. Observations included heart rate 122 bpm, oxygen saturations 92% on air and blood pressure 110/75 mmHg. Blood tests (15:00) demonstrated a platelet count: 26 × 10^9/L (normal range 150 – 400). A **CT pulmonary angiogram** performed at approximately 16:30 demonstrated **bilateral segmental and subsegmental pulmonary emboli with no evidence of right heart strain.**

3.08 Mr A was reviewed at approximately 17:00 by Dr E, the consultant in charge of the ED. On review of the medical notes, I was unable to identify any documentation regarding the considered causes of the thrombocytopenia or investigations deemed necessary.

3.09 At approximately 18:00, Mr A was transferred to the Intensive Care Unit. The rationale for this was to *"Transfer to ICU to commence an **unfractionated heparin infusion** due to high risk of bleeding as a consequence of thrombocytopaenia"* as documented by Dr E in the medical notes.

3.10 Mr A was reviewed by the intensive care unit (ICU) consultant (Dr ICU) at 18:20. Dr ICU documented in the medical notes: *"Recent total hip replacement. Post-surgical pulmonary embolism with no radiological evidence of right heart strain, and a normal troponin. Very high risk of bleeding due to thrombocytopaenia. Discuss with haematology with a view to commence unfractionated heparin infusion."*

3.11 The resident doctor in ICU contacted the on-call haematologist, Dr X at 18:30. Dr X advised: "*Give the patient 2 pools of platelets and commence an unfractionated heparin infusion to reduce the risk of bleeding since unfractionated heparin has a short half-life and can be easily reversed. An urgent blood film*

should also be requested to exclude TTP". At approximately 18:30, an unfractionated heparin infusion was commenced. The patient received one **pool of platelets** at approximately 19:00 and a further pool at 20:00.

3.12 On review of the medical notes, I was unable to identify any consideration of the cause of the thrombocytopaenia, including a **4T score** or equivalent HIT risk assessment, by Dr X, the on-call haematologist.

17 January 2026

3.13 At approximately 09:10, Mr A developed sudden and severe central abdominal pain. Observations: Heart rate: 120 bpm, Blood pressure: 95/60 mmHg. Blood tests (09:30): Platelet count: 24 × 10^9/L and lactate: 4.2 mmol/L. A CT abdomen at approximately 11:30 demonstrated: **splenic vein thrombosis, superior mesenteric vein thrombosis, and features consistent with bowel ischaemia.** The **APTT** was within the documented therapeutic range for the unfractionated heparin infusion.

3.14 At approximately 12:15, the ICU team referred the patient to the general surgeon, who advised that they would review the patient as soon as possible.

3.15 At approximately 13:00, the ICU team contacted the on-call haematologist, Dr B. Dr B calculated a "4T score" for HIT as 8/8. He advised that this was *"consistent with a high probability of heparin-induced thrombocytopenia with thrombosis (HITT)."*

3.16 Dr B advised to immediately stop the unfractionated heparin infusion and to commence an argatroban infusion. The rationale for this, as documented in the notes, was *"high risk of HITT in the context of critical illness and anticipated surgical requirement with significant bleeding risk".* Dr B advised sending urgent samples to the laboratory for HIT testing, and to exclude alternative causes of thrombocytopaenia. Dr B requested several other blood tests, including a coagulation screen, fibrinogen, D-dimer, blood film, and B12 and folate. Dr B advised that further platelet transfusions were contraindicated unless the patient were to have a major haemorrhage.

3.17 At approximately 13:15, the unfractionated heparin infusion was stopped, and the patient was commenced on an argatroban infusion.

3.18 At approximately 14:00, HIT antibody testing was sent. At approximately 18:30, the laboratory called the ICU registrar to advise that the result was strongly positive: 68 U/mL (measured via the AccuStar analyser). The additional blood tests requested by Dr B (coagulation screen, fibrinogen, D-dimer, and B12 and folate) were all normal. A blood film demonstrated *"genuine thrombocytopaenia with no red cell fragmentation".*

3.19 At approximately 15:30, Mr A developed worsening **hypoxia.** He required escalation to high-flow nasal oxygen (FiO_2 0.6). A repeat CTPA at approximately 16:00 demonstrated a **saddle pulmonary embolism.**

3.20 At approximately 18:00 he developed cardiovascular instability with a blood pressure of 75/45 mmHg. At approximately 18:15, **thrombolysis with alteplase** was administered.

18 January 2026

3.21 At approximately 03:00, Mr A developed worsening abdominal pain, bloody diarrhoea and a lactate level rising to 6.1 mmol/L. The general surgeons immediately reviewed him, and a clinical examination demonstrated a **peritonitic abdomen.**

3.22 At approximately 10:00, Mr A underwent an emergency **laparotomy.** He was found to have an **ischaemic small bowel secondary to a mesenteric thrombosis.** He underwent a **small bowel resection and formation of an ileostomy.** Argatroban was discontinued at approximately 06:00 (4 hours pre-operatively) and recommenced at approximately 18:00 (6 hours post-operatively). Mr A had no significant intraoperative or postoperative bleeding.

18th January – 25th January 2026 (Immediate Post-operative Period).

3.23 Mr A remained in the Intensive Care Unit following the emergency laparotomy on 18 January 2026. He required ongoing organ support, including **high-flow nasal oxygen** and **vasopressor support for haemodynamic instability** for approximately 48 hours post-operatively. Blood tests during this period demonstrated: ongoing thrombocytopenia, with platelet counts ranging between 40–90 × 10^9/L, and gradual biochemical improvement in lactate. An argatroban infusion was continued throughout this period. He remained nil by mouth and received **parenteral nutrition.**

25th January – 5th February 2026 (Step-down from ICU).

3.24 On 25 January 2026, Mr A stepped down from Intensive Care to a high-dependency surgical ward. At this stage, oxygen requirements had reduced to low-flow nasal oxygen, and haemodynamic status was stable. Argatroban was continued. There was a gradual recovery of platelet count, and on the 28 January the platelet count had normalised (165 × 10^9/L). **Enteral feeding** was introduced on 03 February 2026. **Stoma** function was established.

5th February – 20th February 2026 (Ward-based Recovery):

3.25 Mr A remained an inpatient on the surgical ward. He underwent physiotherapy and mobilisation. Anticoagulation was transitioned from argatroban to an oral anticoagulant (apixaban 5mg bd). He required ongoing input from the surgical team, haematology and stoma care nurses.

3.26 Mr A reported ongoing shortness of breath on exertion. He did not require supplemental oxygen at rest. However, he had significantly reduced exercise tolerance compared to his pre-morbid baseline. An **echocardiogram demonstrated features consistent with moderate pulmonary hypertension.**

3.27 Mr A was discharged home on 20th February 2026. At discharge:

- He was independently mobile with reduced exercise tolerance (only able to walk approximately 50 metres without rest)

- He remained on anticoagulation (minimum 3 months required)
- He had a functioning ileostomy
- Follow-up was arranged with: Haematology, Cardiology, and Colorectal surgery

3.2 Interview and examination

I have not interviewed or examined Mr A.

My opinion is based on the medical records and documents listed in Appendix 2.

4. My opinion

On the balance of probabilities, did Mr A develop heparin-induced thrombocytopenia (HIT), and if so, when did this occur?

4.01. On the balance of probabilities, Mr A developed heparin-induced thrombocytopaenia with thrombosis (HITT).

4.02. A technical description of HITT is provided in Appendix 5. I summarise below the features relevant to my opinion.

4.03. The diagnosis of HITT is supported by four features: new thrombosis, severe thrombocytopenia, timing more than five days after first heparin exposure, and absence of a more plausible alternative explanation.

4.04. The HIT antibody test was strongly positive at 68 U/mL. This test detects antibodies against the PF4–heparin complex. The test is sensitive but not fully specific. A positive result, therefore, needs to be interpreted alongside the clinical probability. In this case, the strongly positive result materially increases the likelihood of true HIT when combined with the high 4T score.

4.05. The likelihood of HITT with a positive antibody screen increased further when associated with a high pre-test probability. In my calculation, the 4T score is 8/8: thrombocytopenia >50% fall with nadir ≥20 gives 2 points; timing day 5–10 gives 2 points; thrombosis gives 2 points; and no other apparent cause gives 2 points.

4.06. Alternative causes should, of course, be considered and excluded. Dr B, the on-call consultant haematologist, requested a panel of investigations, which were all normal. These results made alternative diagnoses such as DIC, TTP, B12/folate deficiency and pseudo-thrombocytopenia less likely.

4.07. On review of the medical records and laboratory notes, I could not find evidence that antiphospholipid syndrome (APLS) was considered. Testing for this would have involved checking for a Lupus Anticoagulant and anti-cardiolipin antibodies. Although this remains within the differential diagnosis, I am of the opinion that HIT was the far more likely explanation given my reasoning in paragraphs 4.03 – 4.05. APLS would not explain the classic temporal relationship with heparin exposure as well as HIT does.

4.08. With regards to the timing of the onset of HITT, I am of the opinion that Mr A developed HIT between 15 January and 16 January 2026. The initial mild platelet fall is most consistent with a non-immune, transient post-operative thrombocytopenia, which is commonly observed.

4.09. Immune-mediated HIT typically develops 5–10 days after starting heparin. Mr A was first exposed to heparin on 10 January 2026. He was admitted to the hospital with severe thrombocytopaenia and thrombosis on 16 January 2026. Therefore, taking into account the pathophysiology of HIT, it is most probable that he developed this syndrome either on the day of admission (16 January) or the day prior (15 January).

Was the management of Mr A's condition in accordance with a reasonable standard of care? If not, please identify any specific acts or omissions that fell below that standard, and when.

4.10. On admission (16 January 2026), Mr A was identified to have a thrombosis (pulmonary embolism) with a significant thrombocytopaenia, in the context of recent heparin exposure. These findings are strongly suggestive of HITT.

4.11. The British Society for Haematology guideline[1] recommends that where HIT is clinically suspected, all heparin should be stopped and a non-heparin anticoagulant commenced while appropriate laboratory testing is undertaken.

4.12. Neither the emergency department consultant, Dr E, nor the intensive care consultant, Dr ICU documented any documentation regarding the cause of the thrombocytopenia or appropriate investigations. I could not identify documentation that HIT was considered.

4.13. My expertise is in haematology, and I therefore do not offer an opinion on whether the actions of the Emergency Medicine or Intensive Care teams met the standards expected within their respective specialties; this would be a matter for experts in those fields.

4.14. The initial on-call haematologist consulted, Dr X advised to "*Give the patient 2 pools of platelets and commence an unfractionated heparin infusion to reduce the risk of bleeding. An urgent blood film should also be requested to exclude TTP".* I have treated this as the material haematology advice on which the subsequent management was based. On review of the medical records, I could find no evidence that HITT was considered as part of the differential diagnosis.

4.15. At this point, with the clinical and laboratory information available, HITT should have been strongly considered. A reasonably competent

consultant haematologist would have been expected to recognise HIT as a leading diagnosis, calculate or apply the principles of the 4T score, stop all heparin exposure, avoid platelet transfusion unless there was major bleeding, send urgent HIT testing, and commence a non-heparin anticoagulant.

4.16. In my opinion, the failure to consider HIT and to apply a 4T score, or equivalent structured assessment, represented a departure from the standard expected of a reasonably competent consultant haematologist.

4.17. As a consequence, Dr X advised starting the patient on an unfractionated heparin infusion, which in my opinion, represents a departure from the standard expected of a reasonably competent consultant haematologist, because further heparin exposure can amplify the immune-mediated platelet activation that drives HIT and thereby increase the risk of thrombosis.

4.18. Dr X also advised administering two pools of platelets. Platelet transfusions are relatively contraindicated in HITT. In HIT, platelet transfusion can provide additional platelets for antibody-mediated activation and may worsen thrombosis. In the absence of major or life-threatening bleeding, platelet transfusion would not usually be recommended. This represents a further action which fell below the standard of care.

4.19. I would also have expected a reasonably competent haematologist to request a coagulation screen, fibrinogen and D-dimer to assess for DIC. This omission is less central to my causation opinion than the failure to recognise and treat suspected HIT.

If the standard of care was not met, did it cause or materially contribute to: the progression of thrombosis (including pulmonary embolism and intra-abdominal thrombosis)?

4.20. HIT causes an extremely **hypercoagulable state**. If untreated, HIT is associated with a high risk of thrombosis[2], reported in some series as up to approximately 50%.

4.21. Large observational data demonstrate that patients with HIT have a significantly increased in-hospital mortality[3], approximately 10%. This figure is included to illustrate clinical seriousness; my causation opinion in this case rests primarily on the timing, mechanism and subsequent thrombosis.

4.22. Failure to recognise that HIT was the leading diagnosis in this case resulted in the commencement of an unfractionated heparin infusion and administration of two pools of platelets – both of which are contraindicated in HIT.

4.23. In HIT, the immune system reacts to heparin and causes platelets to become overactive. Instead of preventing clots, this leads to an increased risk of clot formation.

4.24. Giving unfractionated heparin at this stage worsens the reaction. It creates more targets for the immune system, which leads to further platelet activation and more clotting.

4.25. Giving platelet transfusions can also make the condition worse. The transfused platelets can become activated in the same way, effectively adding fuel to the process.

4.26. For these reasons, continuing heparin or giving platelets in HIT can increase the risk of new thrombosis or extension of existing thrombosis.

4.27. Mr A was commenced on an unfractionated heparin infusion on 16 January at 18:30. He also received one pool of platelets at approximately 19:00 and a further pool at 20:00. The unfractionated heparin infusion was continued until 17 January 13:15. During this period, approximately 9

hours after commencement of the unfractionated heparin infusion, Mr A developed acute abdominal pain with subsequent imaging demonstrating splenic vein thrombosis, superior mesenteric vein thrombosis, and features consistent with bowel ischaemia.

4.28. The development of new abdominal venous thrombosis during ongoing heparin exposure and shortly after platelet transfusion is temporally and biologically consistent with propagation of HIT-associated thrombosis.

4.29. I cannot say that correct treatment would have eliminated the risk of further thrombosis, because HIT itself is a strongly prothrombotic condition. However, in my opinion, earlier recognition and correct management would more likely than not have materially reduced the risk of the subsequent splenic and superior mesenteric vein thromboses and bowel ischaemia.

4.30. Following consultation with Dr B, Consultant Haematologist, the unfractionated heparin was stopped at 13:15 on 17 January. Mr A was then commenced on an argatroban infusion. At approximately 15:30, Mr A developed worsening hypoxia and a repeat CTPA at approximately 16:00 demonstrated a **saddle pulmonary embolism,** which shortly thereafter developed into cardiovascular instability requiring resuscitation.

4.31. Although the patient was on an argatroban infusion at the time, it had only been commenced approximately 2 hours prior to deterioration. I have not identified coagulation monitoring results demonstrating therapeutic argatroban effect before the deterioration. However, argatroban typically takes 2 - 6 hours to achieve therapeutic anticoagulation. In my opinion, the progression from bilateral segmental/subsegmental pulmonary emboli to saddle pulmonary embolism was temporally consistent with, and probably materially contributed to by, the preceding period of ongoing heparin exposure in the context of unrecognised HIT.

5. Declarations

Statement of compliance

I understand my duty as an expert witness is to the court. I have complied with that duty and will continue to comply with it. This report includes all matters relevant to the issues on which my expert evidence is given. I have given details in this report of any matters which might affect the validity of this report. I have addressed this report to the court. I further understand that my duty to the court overrides any obligation to the party from whom I received instructions.

Declaration of Awareness

I confirm that I am aware of the requirements of Part 35 and Practice Direction 35, and the Guidance for the Instruction of Experts in Civil Claims 2014.

Statement of conflicts

I confirm that I have no conflict of interest of any kind, other than any which I have already set out in this report. I do not consider that any interest which I have disclosed affects my suitability to give expert evidence on any issue on which I have given evidence and I will advise the party by whom I am instructed if, between the date of this report and the trial, there is any change in circumstances which affects this statement.

Statement of truth

I confirm that I have made clear which facts and matters referred to in this report are within my own knowledge and which are not. Those that are within my own knowledge I confirm to be true. The opinions I have expressed represent my true and complete professional opinions on the matters to which they refer.

Signature…*J. Burgess*…… Date: 26/04/2026

Appendix 1: My experience and qualifications

I am a **Consultant Haematologist** with experience in the diagnosis and management of a broad range of haematological conditions, including disorders of thrombosis and haemostasis, anticoagulation, bleeding disorders, haemoglobinopathies, transfusion medicine, and haematological malignancies.

My clinical practice includes the assessment and management of complex patients, interpretation of laboratory investigations, and multidisciplinary working within a tertiary care setting. I have particular experience in the evaluation and management of thrombotic disorders and anticoagulation, which are directly relevant to the issues in this case.

I am Chair of the sector thrombosis multidisciplinary team (MDT), where I lead regular discussions on the diagnosis and management of complex thrombotic and anticoagulation-related cases.

I am the lead author of the regional thrombophilia testing guidelines.

Current Appointment

- Consultant Haematologist
- Queen Elizabeth University Hospital, Glasgow

Qualifications

- MBChB (Hons)
- MRCP (UK)
- FRCPath
- M.Math
- M.Phys
- M.Phil (Cantab)

Professional Registration and Memberships

- Member of the Royal College of Physicians (MRCP UK)

- Fellow of the Royal College of Pathologists (FRCPath)
- General Medical Council (GMC) – Full registration with licence to practise (GMC number 7518948)

Medico-Legal Training

- Bond Solon Expert Witness Certificate, University of Aberdeen (in progress)

Appendix 2: List of documents that I have examined, with copies of important extracts

I have examined the following documents:

- GP records
- Orthopaedic admission notes, 10–14 Jan 2026
- ED notes, 16 Jan 2026
- ICU notes, 16–25 Jan 2026
- Haematology advice entries, 16–17 Jan 2026
- Laboratory results, including platelet counts, coagulation, HIT antibody
- Radiology reports: CTPA, CT abdomen
- Operation note, 18 Jan 2026
- Discharge summary, 20 Feb 2026

All factual information, documented above and used in the investigation of the facts, has been provided to me by my instructing solicitors. I have not obtained facts from any other sources.

This report is a hypothetical example prepared for training purposes. All details have been anonymised and do not relate to any real patient, clinician, organisation, or legal case.

Appendix 3: Details of any literature that I have examined

1. Arachchillage DJ, Laffan M, et al. Diagnosis and management of heparin-induced thrombocytopenia: third edition. *Br J Haematol*. 2024;204(2):459–475.

2. Gallo T, Curry SC, Padilla-Jones A, Heise CW, et al. A computerized scoring system to improve assessment of heparin-induced thrombocytopenia risk. *J Thromb Haemost.* 2019 Feb;17(2):383-388.

3. Devlin M, Movahed MR, Hashemzadeh M. Age, gender, and ethnicity are associated with higher all-cause mortality in hospitalized patients with heparin-induced thrombocytopenia: a nationwide analysis. *J Hematop.* 2025 Nov 7;18(1):51.

Appendix 4: Glossary of technical terms

- **APTT** — A blood test used to monitor some blood-thinning treatments, including unfractionated heparin.
- **Bilateral segmental and subsegmental pulmonary emboli with no evidence of right heart strain** — Blood clots in smaller blood vessels in both lungs, without signs that the right side of the heart was under major pressure.
- **CT pulmonary angiogram** — A special CT scan used to look for blood clots in the lungs.
- **Echocardiogram demonstrated features consistent with moderate pulmonary hypertension** — An ultrasound scan of the heart suggested raised pressure in the blood vessels of the lungs.
- **Enoxaparin** — A low molecular weight heparin blood thinner, usually given by injection under the skin.
- **Enteral feeding** — Feeding through the gut, either by mouth or through a feeding tube.

- **Heparin** — A commonly used blood thinner used to prevent or treat blood clots.
- **Heparin-induced thrombocytopenia with thrombosis (HITT)** — A rare immune reaction to heparin where the platelet count falls but the risk of dangerous blood clots increases.
- **High-flow nasal oxygen** — Oxygen delivered through tubes in the nose at a high flow rate to support breathing.
- **Hypercoagulable state** — A condition where the blood is more likely than normal to form clots.
- **Hypoxia** — A low level of oxygen in the body or blood.
- **Ischaemic small bowel secondary to a mesenteric thrombosis** — Part of the small bowel was damaged because a blood clot blocked its blood supply.
- **Laparotomy** — An operation where the abdomen is opened to examine or treat internal problems.
- **Osteoarthritis** — Wear-and-tear arthritis causing joint pain and stiffness.
- **Parenteral nutrition** — Nutrition given directly into the bloodstream through a drip.
- **Peritonitic abdomen** — A clinical finding suggesting serious inflammation or infection inside the abdomen, often requiring urgent surgery.
- **Pleuritic chest pain** — Sharp chest pain that is worse on breathing in, often associated with problems affecting the lungs or their lining.
- **Pool of platelets** — A transfusion of platelets, which are blood cells involved in clotting.
- **Saddle pulmonary embolism** — A large blood clot lodged at the main branching point of the blood vessels going to the lungs.
- **Small bowel resection and formation of an ileostomy** — Surgery to remove part of the small bowel and bring the bowel out onto the abdomen to form a stoma.
- **Splenic vein thrombosis, superior mesenteric vein thrombosis, and features consistent with bowel ischaemia** — Blood clots in abdominal veins, with signs that part of the bowel had reduced blood supply.

- **Stoma** — An opening of the bowel onto the surface of the abdomen, allowing waste to pass into a bag.
- **Subcutaneously** — Given by injection under the skin.
- **Thrombocytopenia** — A low platelet count.
- **Thrombolysis with alteplase** — Treatment with a powerful clot-dissolving drug.
- **Thromboprophylaxis** — Treatment given to reduce the risk of blood clots.
- **Thrombotic complications** — Medical problems caused by blood clots.
- **Unfractionated heparin infusion** — A form of heparin blood thinner given continuously through a drip into a vein.
- **Vasopressor support for haemodynamic instability** — Medicines given through a drip to support blood pressure when circulation is unstable.

Appendix 5: Technical description of HITT

Heparin-induced thrombocytopenia with thrombosis (HITT) is a rare but serious immune-mediated complication of heparin therapy.

Heparin itself is a commonly used anticoagulant (“blood thinner”) given to prevent or treat blood clots. It is widely used in hospital settings—for example, after surgery, during hospital admissions in patients with reduced mobility or other risk factors for thrombosis, or in the treatment of conditions such as deep vein thrombosis or pulmonary embolism.

There are two main types of heparin. The first is unfractionated heparin (UFH**)**, an older form that is usually given through a drip into a vein in hospital and requires close monitoring with blood tests to ensure the dose is safe and effective. The second is low molecular weight heparin (LMWH), a newer and more commonly used form that is given as a small injection under the skin (for example, enoxaparin). LMWH has a more predictable effect in the body, meaning it usually does not require the same level of monitoring. Both types work to reduce

clotting, but they differ in how they are given, how closely they need to be monitored, and their associated risks and side effects.

In most patients, heparin works by enhancing the activity of natural anticoagulant proteins in the blood, thereby reducing the ability of blood to clot. However, in a small proportion of individuals, heparin can trigger an abnormal immune response. This condition is referred to as heparin-induced thrombocytopenia (HIT). “Thrombocytopenia” means a reduced platelet count. Platelets are small blood cells that play a key role in forming clots to stop bleeding. In HIT, the platelet count typically falls significantly, usually 5–10 days after starting heparin, although it can occur earlier in patients previously exposed to heparin.

The underlying mechanism involves a protein called platelet factor 4 (PF4), which is released naturally from platelets. Heparin binds to PF4, forming a complex. In some patients, the immune system mistakenly recognises this PF4–heparin complex as foreign and produces antibodies against it. These antibodies then bind to the PF4–heparin complex on the surface of platelets. Rather than simply destroying platelets, this process paradoxically activates them.

This platelet activation is central to the condition. Activated platelets release further PF4, amplifying the cycle, and also promote the generation of thrombin, a key enzyme in clot formation. As a result, platelets are both consumed (leading to a falling platelet count) and excessively activated (leading to clot formation). This dual effect explains why HIT is paradoxical: despite a low platelet count, the main clinical risk is not bleeding but thrombosis (clotting).

When thrombosis occurs in the context of HIT, the condition is termed HITT (heparin-induced thrombocytopenia with thrombosis). This occurs in a subset of patients with HIT. The clots can form in veins (e.g. deep vein thrombosis or pulmonary embolism) or arteries (e.g. stroke, myocardial infarction, or limb ischaemia). These thrombotic complications can be severe and potentially life-threatening if not promptly recognised and treated.

In summary, HITT is an immune-mediated adverse reaction to heparin in which antibodies against the PF4–heparin complex activate platelets. This leads to a reduction in platelet count alongside an increased risk of abnormal clot formation. Early recognition, cessation of heparin, and initiation of alternative anticoagulation are critical to reducing morbidity and mortality associated with this condition

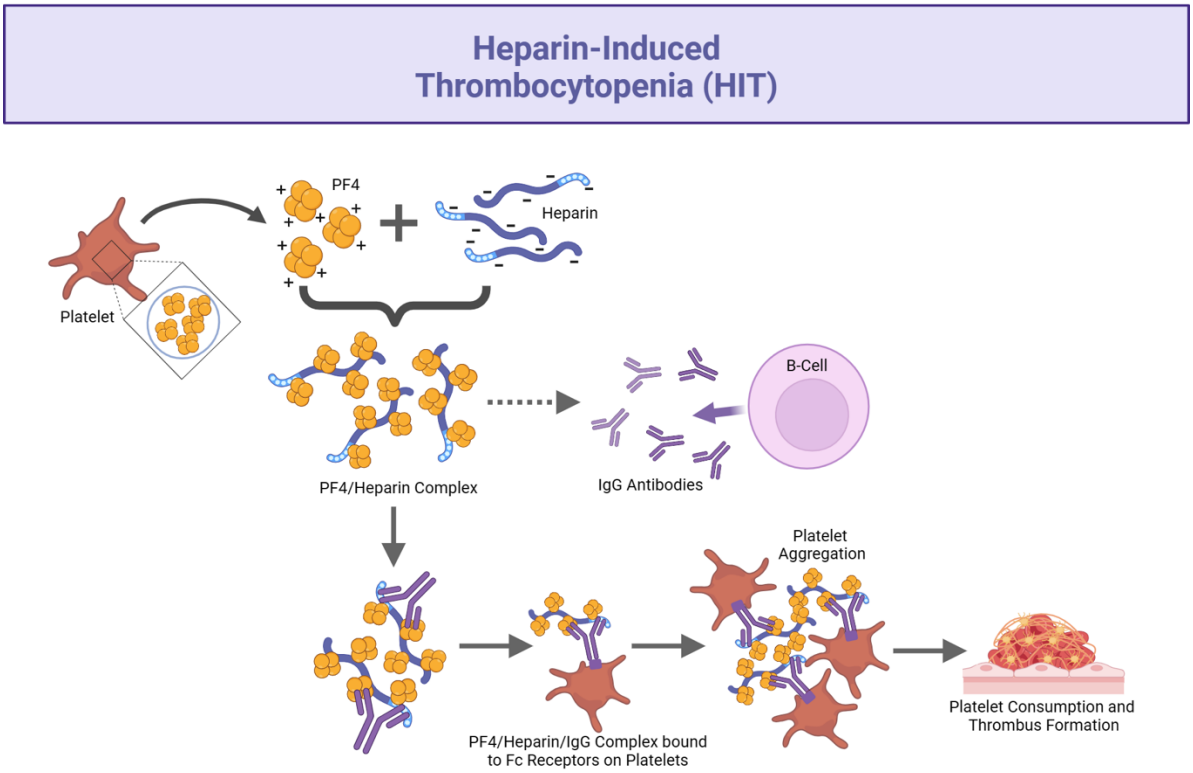

Figure 1: Diagram demonstrating the pathophysiology of heparin-induced thrombocytopaenia (HIT). Heparin can bind to a natural protein called platelet factor 4 (PF4), triggering the body to make antibodies. These antibodies can then bind to the PF4/heparin complex, resulting in platelet activation, causing them to form blood clots (thrombosis). Thrombocytopaenia (low platelet count) occurs due to the consumption of platelets.

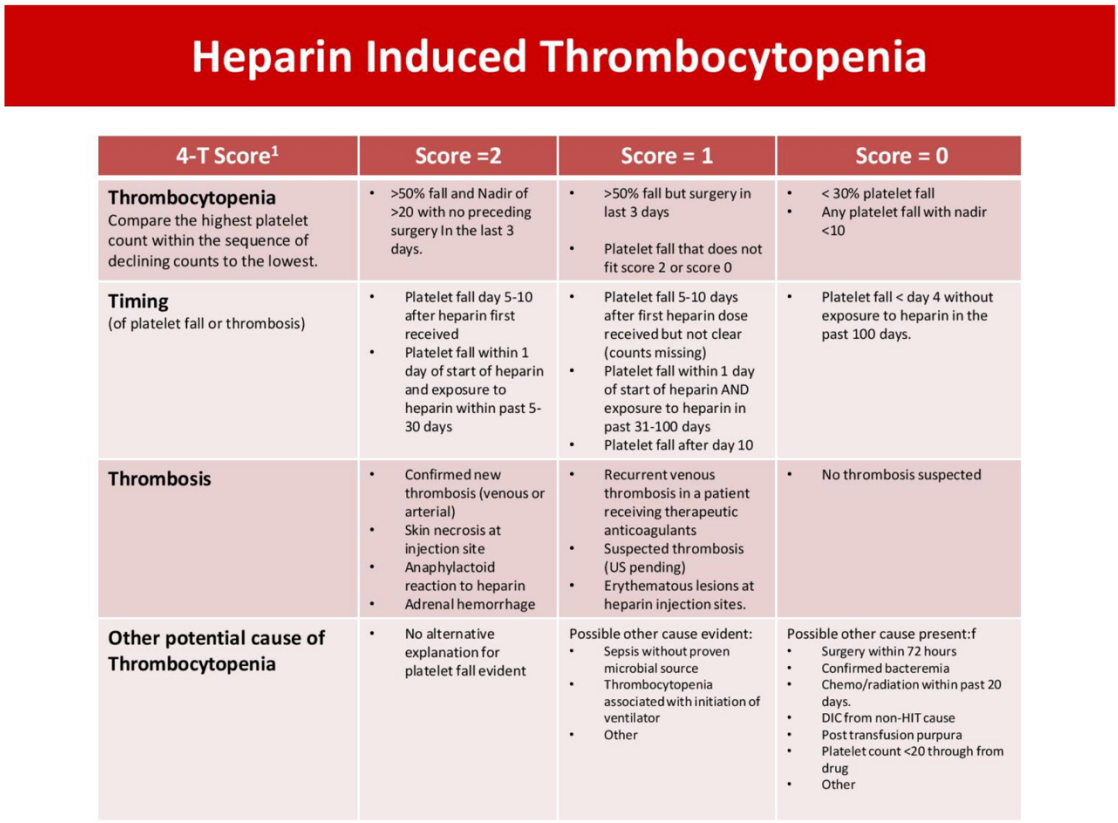

Figure 2: The 4T score is a simple clinical tool used by doctors to estimate how likely it is that a patient has heparin-induced thrombocytopenia (HIT). It considers four key features: the degree of thrombocytopenia (how much the platelet count has fallen), the timing of this fall in relation to starting heparin (typically 5–10 days), the presence of thrombosis (new blood clots), and whether there are other possible causes for the low platelet count. Each category is scored, and the total score indicates whether HIT is unlikely, possible, or likely. While the 4T score does not confirm the diagnosis, it is used to guide urgent clinical decisions, including whether to stop heparin, start alternative anticoagulation, and perform further specialised testing. If the pretest probability of HIT is ≥4, heparin should be stopped, and an alternative anti-coagulant started at therapeutic intensity while laboratory tests are performed.