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# Heparin-induced thrombocytopenia in a 72 years old patient with myocardial infarction without ST-segment elevation

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## Abstract

The study presents a case of a 72-year-old patient who developed heparin-induced thrombocytopenia during hospitalization for non-ST elevation myocardial infarction. The coexistence of both conditions poses a significant problem regarding treatment in such patients. The authors discuss the management used and current standards of care in the course of this disease.

Key words: heparin-induced thrombocytopenia, HIT, acute coronary syndrome, NSTEMI

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## Introduction

Heparin-induced thrombocytopenia is a serious complication of pharmacotherapy with the use of heparins and is associated with a hypercoagulable state, less commonly with haemorrhagic complications, and results in limitations to current therapy [1, 2]. This paper discusses the case of a 72-year-old woman who was hospitalised for a non-ST elevation myocardial infarction (NSTEMI) and developed heparin-induced thrombocytopenia (HIT) during hospitalisation.

## Case report

A 72-year-old woman was admitted to the cardiology clinic for symptoms of acute coronary syndrome without ST-segment elevation. On physical examination, there were signs of stasis in the pulmonary circulation. Otherwise, there were no other lesions observed on physical examination.

On the day of admission, electrocardiography revealed a regular sinus rhythm of 88/min, intermediate axis, ST-segment depressions in I, aVL, $V_2$–$V_6$, negative T waves in I, II, aVF, $V_5$, $V_6$, pathological Q waves in III, aVF (Figure 1). Echocardiography showed severe left ventricular systolic dysfunction with left ventricular ejection fraction: 10–15%, moderate/severe mitral regurgitation (proximal isovelocity surface area: 8 mm), moderate tricuspid regurgitation (tricuspid regurgitation velocity: 3.0 m/s, maximum gradient: 35 mm Hg), enlargement of both atria, left ventricular muscle with signs of hypertrophy. Lung ultrasonography revealed B-lines over the pulmonary fields. Laboratory tests found an increase in markers of myocardial damage (dynamic increase in troponin TnT from 1141 ng/L to 1984 ng/L). Coronary angiography showed stenosis of the left anterior descending artery — approximately 50% from the ostium, as well as closure of the proximal segment after the diagonal branch, critical stenosis of the diagonal branch, closure of the proximal part of the circumflex artery

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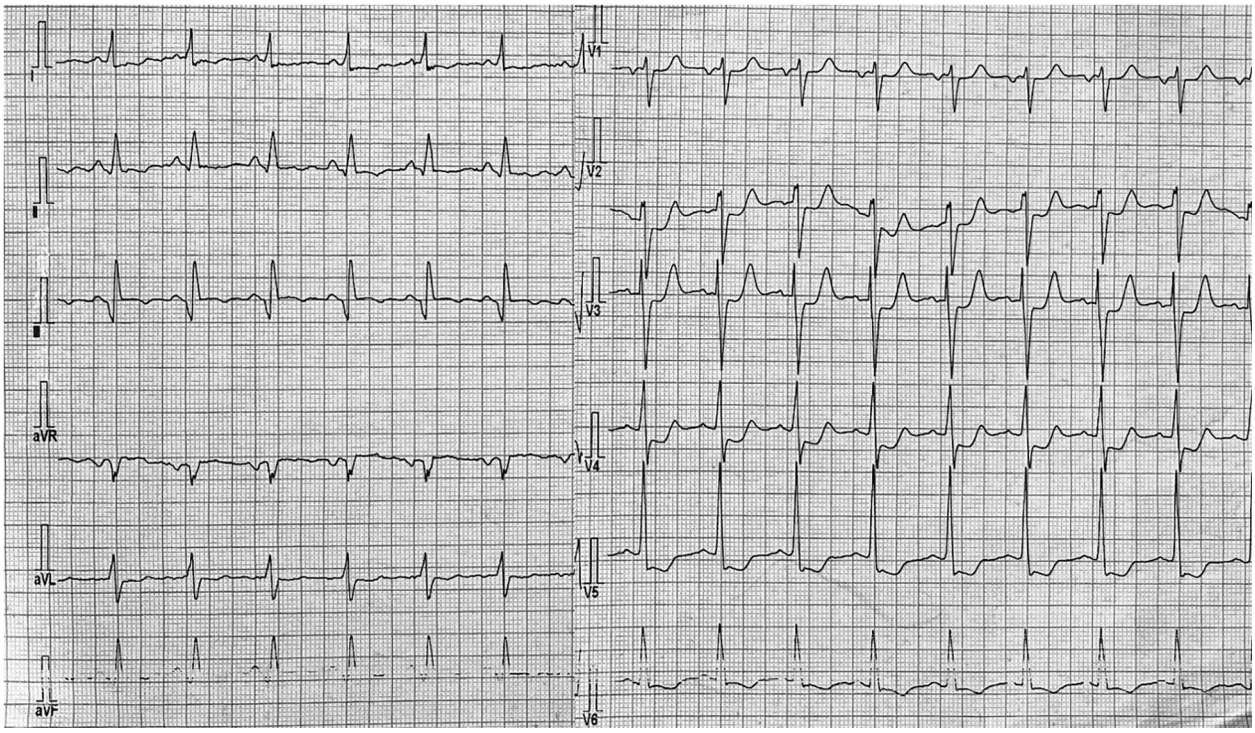


**Figure 1.** A 12-lead electrocardiographic recording performed on admission to hospital

(Cx) and closure of the proximal segment of the right coronary artery. A diagnosis of multivessel disease was made. The patient was consulted by a heart team and disqualified from coronary artery bypass grafting due to the very high risk of surgery (EuroScore: 11 points; EuroScore II: 7.66%), and she was qualified for palliative percutaneous coronary intervention (PCI) of the Cx.

The patient was started on pharmacotherapy including unfractionated heparin (day 1 and 2 of hospitalisation), acetylsalicylic acid, atorvastatin, furosemide, lisinopril, eplerenone, empagliflozin, and allopurinol. Clopidogrel was added to the treatment due to the high risk of bleeding (estimated using the CRUSADE Score for Post-MI Bleeding Risk). From hospital day 3, the patient received subcutaneous enoxaparin in a therapeutic dose (80 mg twice daily). On hospital day 10, low-molecular-weight heparins were discontinued and palliative PCI Cx with drug-eluting stent was performed (Figure 2). On hospital day

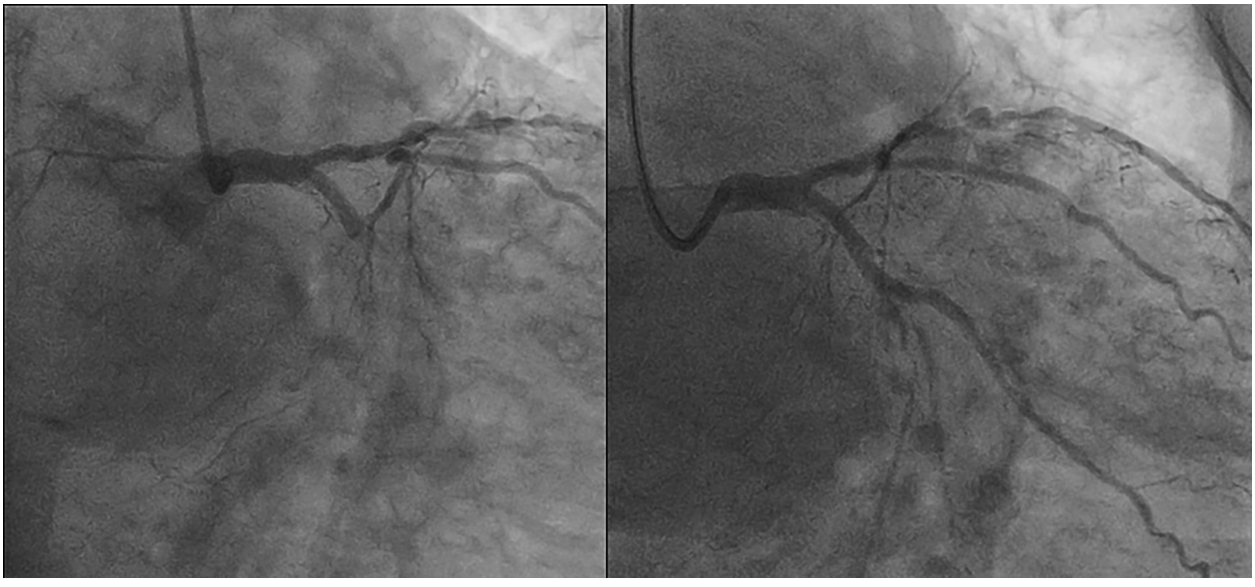

**Figure 2.** Angiography of the left coronary artery, right oblique view, before PCI Cx (left) and after PCI Cx (right); Cx — circumflex artery; PCI — percutaneous coronary intervention

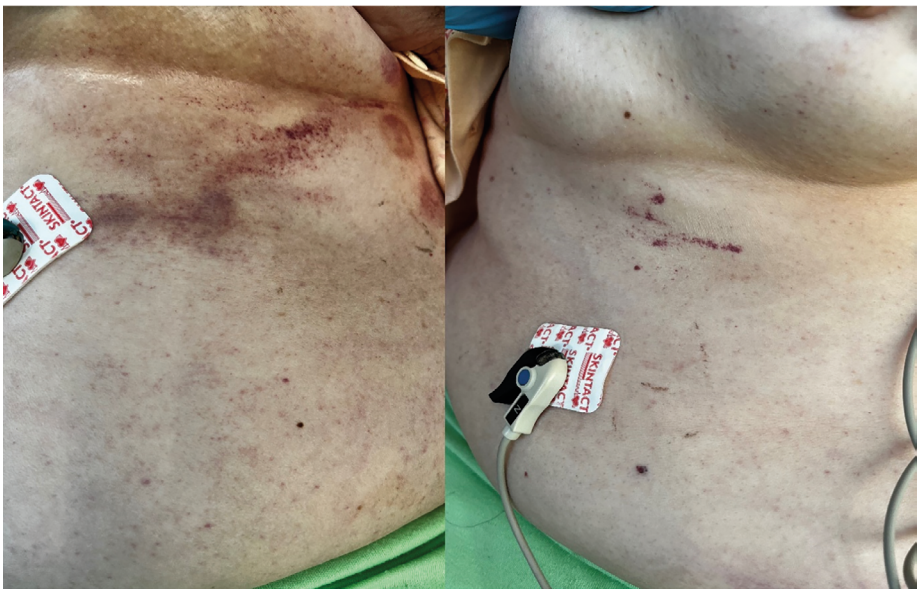

**Figure 3.** Haemorrhagic petechiae under both breasts

**Table 1.** Changes in platelet count (PLT) on subsequent hospital days compared to current patient management

| Days of hospitalization | 1 | 3 | 10 | 13 | 14 | 15 | 18 | 19 | 22 |
|---|---|---|---|---|---|---|---|---|---|
| PLT [1000/μL] | 203 | 226 | | 6 | 3 | 8 | 36 | 105 | 177 |
| Notes | UHF started | UHF discontinued, LMWH started | LMWH discontinued | ASA discontinued, GCS started, and PC transfused | Clopidogrel discontinued, PC transfused | PC transfused | Clopidogrel started | ASA started | |

ASA — acetylsalicylic acid; GCS — glucocorticosteroids; LMWH — low molecular weight heparin; PC — platelet concentrate; UHF — unfractionated heparin

13, the patient had thrombocytopenia — platelet count (PLT) of 25 000/μL in two consecutive blood draws. The citrated platelet count was 6000/μL. Both interview and physical examination revealed no signs of bleeding or haemorrhagic diathesis in the patient. After haematology consultation, it was decided to discontinue acetylsalicylic acid, intravenous dexamethasone was started twice daily at 8 mg and 2 packs of platelet concentrate (PC) were transfused. Despite the discontinuation of heparin and the management implemented, there was a further decrease in PLT — 3000/μL, and in citrated platelet count: 4000/μL. The patient was consulted by a hematologist — treatment was modified as recommended: clopidogrel was temporarily discontinued, intravenous dexamethasone was continued twice daily, consecutively 8 and 4 mg, and 1 pack of PC was transfused daily on consecutive days.

On hospital day 15, the patient had a small number of haemorrhagic petechiae under both breasts (Figure 3). In the following days, there was a gradual increase in PLT count (Table 1). A total of 4 packs of PC were transfused to the patient. The doses of glucocorticosteroids administered were gradually reduced. When PLT increased above 30 000/μL, the patient was started on clopidogrel and then acetylsalicylic acid was restarted. The patient had no additional thrombotic or haemorrhagic episodes during her hospitalisation. On hospital day 22, the patient was discharged from the clinic in a stable condition with PLT of 177 000/μL. There were no alarming signs at the patient's one-month follow-up.

## Discussion

Heparin-induced thrombocytopenia occurs in 0.1–5% of patients treated with heparins [3]. There are two types of this disease entity. Type 1 HIT has no immunological basis, the reduction in PLT is mild, usually occurs within the first 2–4 days of therapy, and there are usually no clinical sequelae. Type 2 HIT has an immunological basis and the decrease in PLT is greater (> 50%) [4]. The above diagnosis should be considered when the platelet count falls below 100 000/mL, usually 5–10 days after starting heparin treatment [5]. During the diagnostic evaluation of HIT, the determination of anti-heparin antibodies is recommended, however, this test is rarely used due to its low availability. Management of HIT includes discontinuation of heparins and implementation of alternative anticoagulant therapy. Argatroban, lepirudin, fondaparinux or non-vitamin K antagonist oral anticoagulant — preferably, rivaroxaban — are recommended [4]. In the case presented in this paper, the decrease in PLT started on hospital day 13, was rapid and worsened despite heparin withdrawal, indicating a likely immunological mechanism. Due to the very low PLT and the appearance of haemorrhagic petechiae, it was decided to transfuse PC and implement steroid therapy. An additional therapeutic challenge in the patient in question was the need for antiplatelet treatment associated with stent implantation. According to the recommendations, patients with a stent implanted in the setting of NSTEMI should receive dual antiplatelet therapy consisting of acetylsalicylic acid and P2Y12 inhibitor for up to 12 months [6]. The authors, taking into account the recommendations of the consulting haematologist, decided to discontinue antiplatelet drugs for a short period.

In the patient in question, alternative anticoagulant treatment was abandoned due to the appearance of signs of haemorrhagic diathesis in the form of petechiae. Currently, there are no detailed and clear rules for the management of the co-occurrence of acute coronary syndrome and heparin-induced thrombocytopenia. Therefore, individual decisions must be made on a case-by-case basis according to the evaluation of the clinical situation.

## Conflict of interest

None declared.

## References


1. Nicolas D, Nicolas S, Hodgens A. Heparin Induced Thrombocytopenia. StatPearls Publishing, Treasure Island (FL) 2022.
2. Ahmed I, Majeed A, Powell R. Heparin induced thrombocytopenia: diagnosis and management update. Postgrad Med J. 2007; 83(983): 575–582, doi: 10.1136/pgmj.2007.059188, indexed in Pubmed: 17823223.
3. Hogan M, Berger JS. Heparin-induced thrombocytopenia (HIT): Review of incidence, diagnosis, and management. Vasc Med. 2020; 25(2): 160–173, doi: 10.1177/1358863X19898253, indexed in Pubmed: 32195628.
4. Undas A, Zawilska K. Małopłytkowość wywołana przez heparynę. In: Gajewski P. ed. Interna Szczeklika. Mały podręcznik 2022/23. Medycyna Praktyczna, Kraków 2022: 449–452.
5. Collet JP, Thiele H, Barbato E, et al. 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. Eur Heart J. 2021; 42(14): 1289–1367, doi: 10.1093/eurheartj/ehaa575, indexed in Pubmed: 32860058.
6. Corrigendum to: 2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: The Task Force for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J. 2021; 42(23): 2298, doi: 10.1093/eurheartj/ehab285, indexed in Pubmed: 33983428.