

# SEVERE THROMBOCYTOPAENIA IN A PATIENT AFTER ACUTE MYOCARDIAL INFARCTION WITH INFLUENZA

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## Authors' contribution:

A. Study design/planning • B. Data collection/entry • C. Data analysis/statistics • D. Data interpretation • E. Preparation of manuscript • F. Literature analysis/search • G. Funds collection

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## ABSTRACT

This paper presents the case of 59-year-old patient who presented to the clinic with weakness, shortness of breath at rest, fever, and dry cough. Based on laboratory tests and physical examination, the patient was diagnosed with pneumonia. On the third day of hospitalization the patient presented resting, burning pain in the chest. Electrocardiogram (ECG) showed signs of STEMI. The percutaneous coronary intervention (PCI) attempt was not therapeutically successful, and the patient received heparin. Progressive thrombocytopenia was found after myocardial infarction. Based on this, an assumption of heparin-induced thrombocytopenia was made. Any clinical suspicion of heparin-induced thrombocytopenia requires that treatment should not be withheld pending the result. This paper pays attention to this problem.

**Key words:** thrombocytopenia, heparin, acute myocardial infarction, heparin-induced thrombocytopenia.

## INTRODUCTION

Heparin-induced thrombocytopenia (HIT) is a serious and increasingly common side effect of heparin use. Heparin discontinuation alone is not sufficient for treatment; untreated or incorrectly treated HIT is associated with high mortality. Receiving heparin involves the risk of developing antibodies in patients of any age, no matter the type and dose of heparin. It is important to recognize that not every patient will develop a clinical syndrome of HIT [1].

## CASE DESCRIPTION

A 59-year-old woman after bone marrow transplantation caused by acute lymphoblastic leukaemia in 2010 was admitted to hospital due to weakness, shortness of breath at rest, fever, and dry cough. The patient did not have medical documentation regarding oncological treatment and further therapeutic procedures.

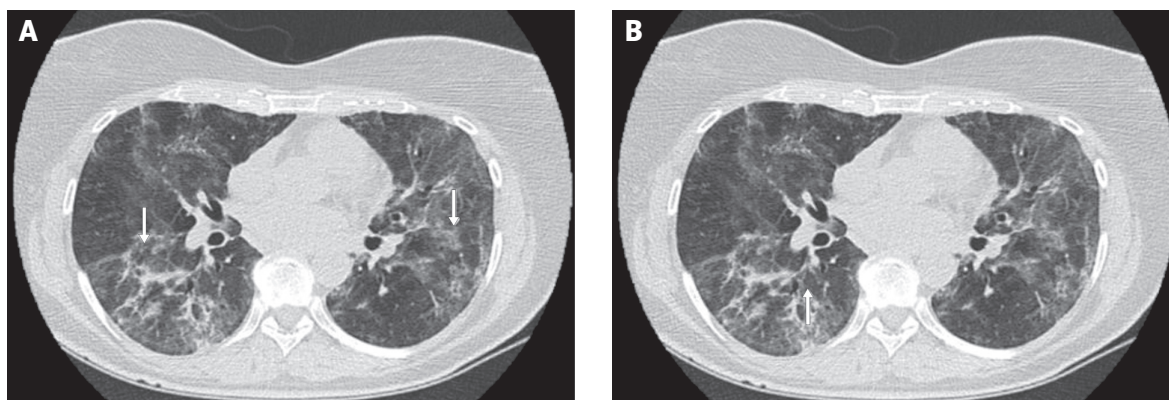
At the time of admission, the patient was in average general condition. Physical examination revealed dyspnoea at rest without cyanosis or oedema. Respiratory system: percussion on both sides, sub-basal mute, bilateral crackles to the angles of the scapulae, with rales on the left. Heart rate: 80 bpm, regular. Medium-high tones, clean. Blood pressure (BP) 90/60 mmHg, and blood saturation 85%. Peri-

pheral arterial pulse consistent with cardiac activity. Physical examination showed no changes within the abdominal cavity, urinary system, and genitals. Laboratory tests revealed high inflammatory markers (C-reactive protein [CRP] = 269.6 mg/l, procalcitonin [PCT] = 2.45 ng/ml), and chest X-ray showed bilateral inflammatory thickening. Influenza A test was positive, but COVID-19 test was negative. Cultures were taken (*A. baumannii*, *S. viridans*, and *Candida spp.* were found in the sputum; blood cultures were negative). Antibiotics and antiviral drugs were used empirically, and low-molecular-weight heparin was introduced as prophylaxis of thromboembolic events. Despite treatment, the patient's condition remained severe; dyspnoea at rest persisted.

On the third day of hospitalization, resting, burning pain in the chest occurred. Electrocardiogram (ECG) showed sinus rhythm, regular 76/min, levo-gram, trace R waves in leads: II, aVF, ST segment elevation in leads II, III, aVF, V2-V6. Urgent coronary angiography revealed a short occlusion in the middle segment of the left anterior descending artery (LAD). A percutaneous coronary intervention (PCI) attempt failed. Antiplatelet drugs were initiated, i.a. enoxaparin in therapeutic dose 0.5 mg/kg *i.v.* After surgery, symptoms of acute left ventricular heart failure occurred; the patient intermittently required non-invasive ventilation. Echocardiography after my-

ocardial infarction showed ascending aorta: 30 mm, LV: 45/30 mm, interventricular septum 12 mm, LA: 30 mm. The myocardium of the left ventricle concentrically thickened. Akinesis of the apical segment of

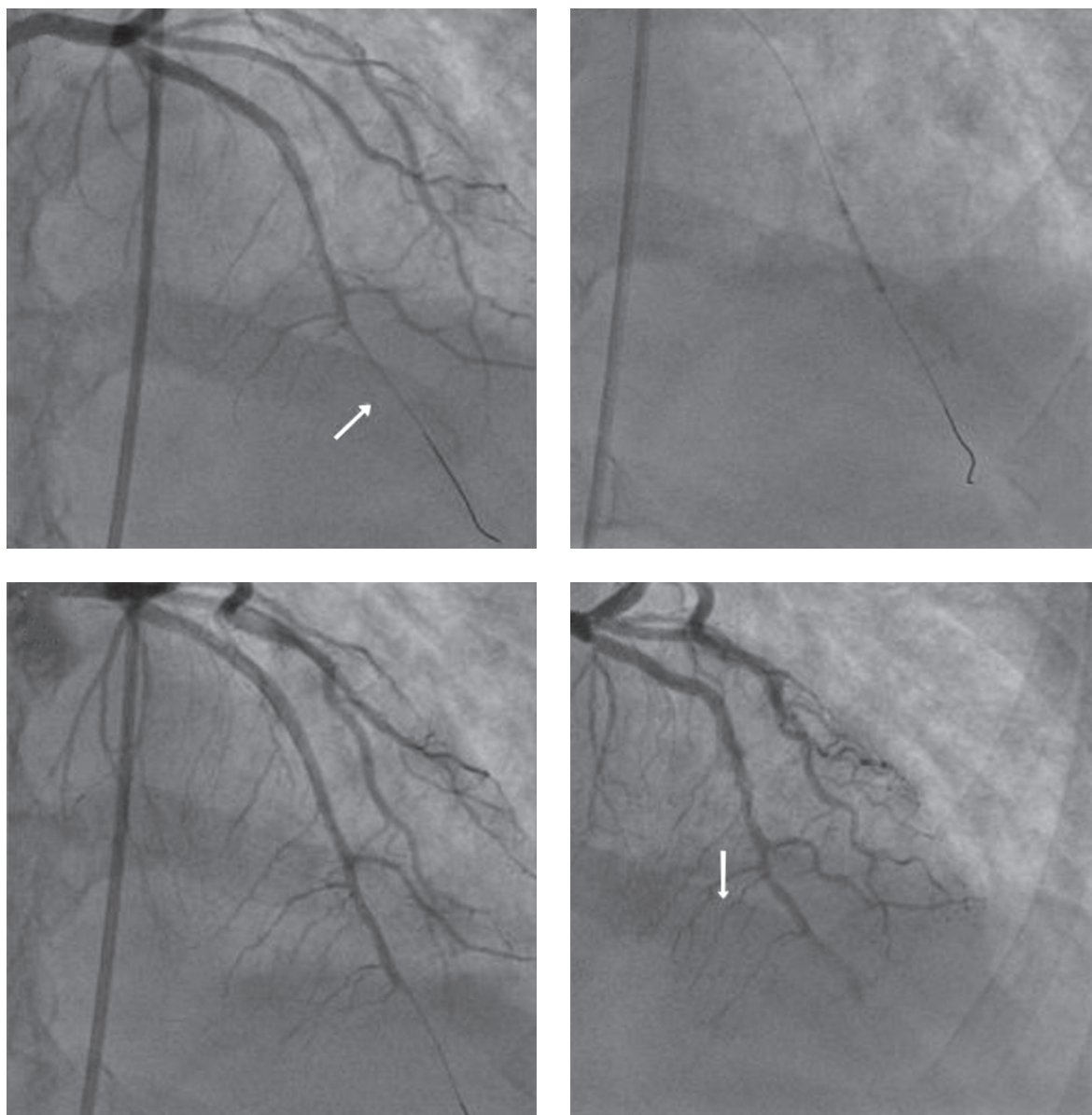
the interventricular septum and inferior wall, EF approximately 50%. The right ventricle and inferior vena cava collapsed. Pericardium unchanged. Right pleura contained a trace amount of fluid (1 cm).



**Figure 1.** Chest computed tomography visualized (arrows). A) Diffuse “ground glass opacities” areas; B) thickened interlobular septa



**Figure 2.** Coronary angiography. In picture number 3 occlusion was visualized within the anterior interventricular branch of LAD



**Figure 3.** PCI attempt – implantation of a balloon catheter at the occlusion site. Condition after an attempt to implant a balloon catheter. Persisting occlusion visualized with arrow in picture number 8

Progressive thrombocytopenia was found after myocardial infarction (platelet count [PLT] at admission 116 K/ $\mu$ l, on the day of myocardial infarction 101 K/ $\mu$ l, 2 days after myocardial infarction 32 K/ $\mu$ l, 9 days after myocardial infarction 8 K/ $\mu$ l); pseudothrombocytopenia was excluded. Therapeutic measures for thrombocytopenia were initiated, and so low-molecular-weight heparin and antiplatelet drugs were discontinued. No antiplatelet agents or LMWHs were used, but fondaparinux was initiated. The patient was transfused with 2 packages of irradiated leucocyte depleted platelet concentrate. In addition, treatment with methylprednisolone was applied. An increase in the platelet count to 54 K/ $\mu$ l was observed. The patient was discharged home in

good general condition. The main recommendation for the patient was Metypred 16 mg one tablet in the morning and at noon for the first 7 days, then one tablet in the morning and half a tablet at noon for the following days. The patient was recommended for an urgent follow-up at the haematology outpatient clinic.

## DISCUSSION

Thrombocytopenia is defined as a platelet count less than 150,000/ $\mu$ l. The causes of disease can be found in insufficient production of thrombocytes, excessive destruction of thrombocytes, or incorrect supply of thrombocytes. Reduced platelet production

is usually due to bone marrow damage, severe liver disease, or other causes such as vitamin B<sub>12</sub> or folic acid deficiency [2].

On the other hand, excessive destruction of thrombocytes may have an immunological basis and be related to the action of drugs such as antibiotics, non-steroidal anti-inflammatory drugs, or heparins. Destruction of platelets also occurs in such diseases as haemolytic uraemic syndrome or intravascular coagulation syndrome, then the survival time of these blood cells is shortened.

Heparin-induced thrombocytopenia is currently a significant complication of anticoagulant therapy. The most important risk group are patients after surgical procedures such as cardiac or vascular surgeries. HIT is more common in women than in men [3].

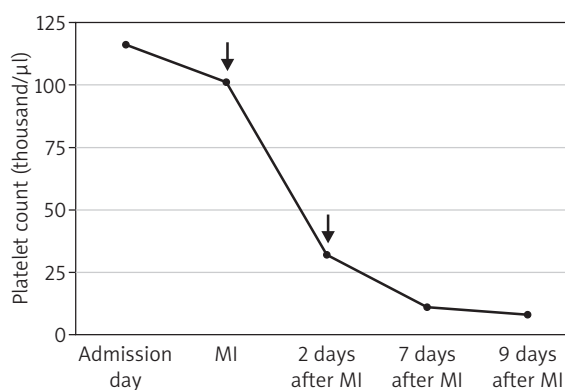
The pathogenesis of HIT is related to the presence of immunoglobulin G (IgG) autoantibodies formed as a response to the action of heparin, and more specifically against the PF4/heparin complexes [4]. This is a prothrombotic condition that involves a high risk of venous as well as arterial thrombosis. This is related to the activation of platelets because of the formation of immune complexes [3].

Warkentin has developed a clinical 4T index that allows you to assess the likelihood of HIT (thrombocytopenia, thrombosis, timing in the absence of their explanations, and lack of other causes of thrombocytopenia and thrombosis) [5].

The patient had indications for treatment in the form of low-molecular-weight heparin, as well as antiplatelet drugs. The observed thrombocytopenia as well as severe infection could be caused by their use in a patient after a previous bone marrow transplant. An increase in the level of D-dimer and acute myocardial infarction of potentially embolic aetiology may indicate intravascular coagulation. Due to the very high risk of venous and arterial thrombosis, despite thrombocytopenia, in such cases it is necessary to use anticoagulation methods other than heparins. Currently, 2 anticoagulants are recommended: argatroban and danaparoid. The disadvantages and high cost of these drugs are increasing the use of other non-heparin anticoagulants, e.g. fondaparinux and direct oral anticoagulants (DOACs) as off-label treatment options [4].

Patients treated with both unfractionated and low-molecular-weight heparin require careful monitoring due to their relatively unpredictable effects. Low-molecular-weight heparin has a more predictable effect. Therefore, it is advisable to monitor platelet counts in patients who are being treated with heparin as HIT prevention. This is an example of a severe complication of heparin therapy and is associated with a mortality rate of approximately 20-40% [6].

When differentiating similar symptoms reported by a patient, attention should be paid to such dis-



Platelet count	Day of treatment				
	Admission day	MI; AAT	2 days after MI	7 days after MI	9 days after MI
	116 K/μg	101 K/μg	32 K/μg	11 K/μg	8 K/μg

MI – myocardial infarction, AAT – anticoagulant and antiplatelet treatment

Figure 4. Medication history

eases as pneumonia and pleurisy, heart failure, acute coronary syndromes, exacerbation of asthma and chronic obstructive pulmonary disease, cardiogenic shock, ventricular septal rupture, cardiac tamponade, and aortic dissection.

The limitation of our case report is the lack of determination of antibodies. This diagnosis was mainly based on the clinical picture and other laboratory tests. The description of the case was made to indicate the importance of detecting and managing complications after heparin treatment in clinical practice.

## CONCLUSIONS

The character of this case report is interdisciplinary. Attention was paid to the individualization of medical services to the patient's health needs. Nursing diagnosis draws attention to the physical and mental state of the patient, the degree of their independence, and the degree of activity. Although the therapeutic team consists of many doctors of various specialties, the nurse spends the largest percentage of time with the patient performing care activities. Complementing these activities has a positive impact on the quality of life and increases the survival of patients with syndromes such as heparin-induced thrombocytopenia. The patient described in this case survived thanks to immediate discontinuation of heparin and measures to increase the platelet count. The importance of prompt identification of the cause of thrombocytopenia to avoid serious complications was also emphasized.

## Disclosure

The authors declare no conflict of interest.

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