Case report

Bridging haematology and cardiology: recurrent ST-segment elevation myocardial infarction in a patient with multiple myeloma

Wiktoria Michnowska[1], Aleksandra Liżewska-Springer[1, 2], Agata Tyczyńska[3], Marta Marcinkowska[4], Agnieszka Mickiewicz[4], Ewa Lewicka[1, 2]

[1] *Department of Cardiology and Electrotherapy, Faculty of Medicine, Medical University of Gdańsk, Poland*

[2] *Cardio-Oncology Clinic, University Clinical Center, Gdańsk, Poland*

[3] *Department of Haematology and Transplantology, Medical University of Gdańsk, Poland*

[4] *I Department of Cardiology, Faculty of Medicine, Medical University of Gdańsk, Poland*

Correspondence:
Wiktoria Michnowska, MD
Department of Cardiology and Electrotherapy, Faculty of Medicine, Medical University of Gdansk
80-402 Gdańsk, ul. Kliniczna 5/67, Poland
Phone: +48 728 575 405
email: wmichnowska@uck.gda.pl

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ABSTRACT

We present case of a 49-year-old man with arterial hypertension, hypercholesterolemia, and nephrotic syndrome was diagnosed with multiple myeloma, who underwent first-line treatment with the DVTd regimen. This case highlights the complex interplay between multiple myeloma, prothrombotic anticancer therapy, and severe dyslipoproteinemia in accelerating coronary atherosclerosis and recurrent ischemic events.

Key words: autologous stem cell transplantation, lipoprotein(a), multiple myeloma, myocardial infarction, ST-segment elevation

A 49-year-old man with arterial hypertension, hypercholesterolemia, and nephrotic syndrome was diagnosed with multiple myeloma (MM). In February 2023, he initiated first-line treatment with the DVTd regimen (daratumumab, bortezomib, thalidomide, dexamethasone). One month later, he presented with chest pain. Electrocardiography confirmed an anterior ST-segment elevation myocardial infarction (STEMI). Emergency coronary angiography revealed a critical 95% stenosis of the proximal left anterior descending artery (LAD), which was successfully treated with percutaneous coronary intervention and drug-eluting stent implantation.

Echocardiography demonstrated increased left ventricular (LV) wall thickness (fig. 1A), left atrial enlargement (53 mL/m^2), preserved LV ejection fraction (65%) with apical and mid-lateral wall abnormalities (fig. 1B), raising suspicion for cardiac amyloidosis. Cardiac magnetic resonance imaging excluded hypertrophic cardiomyopathy and AL amyloidosis.

Figure 1A. 2D-Echocardiography: parasternal long axis view. Increased left ventricular wall thickness: interventricular septum – 17 mm, posterior wall – 14 mm.

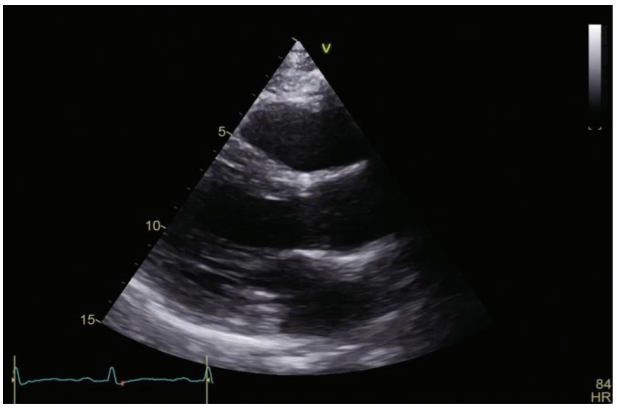
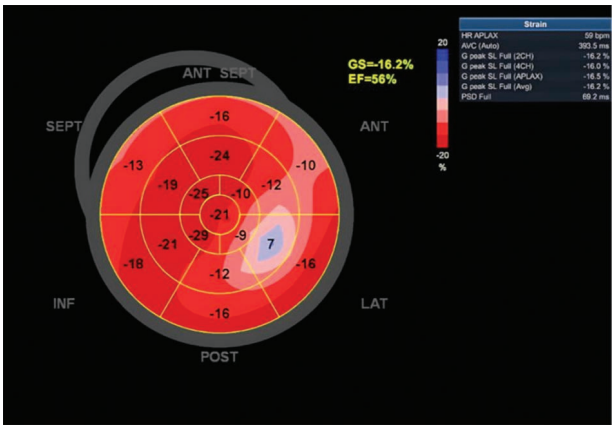


Figure 1B. Speckle tracking echocardiography (STE) bullseye plot representing left ventricular longitudinal strain demonstrating akinetic and hypokinetic apical and mid-lateral wall segments with preserved apical contractility.



The rate revascularization, the patient continued to experience angina, described as a burning retrosternal pain, predominantly mornings. In October 2023, he was readmitted with severe chest pain. Acute coronary syndrome was ruled out. A treadmill exercise test showed no ST-T segment changes, whereas myocardial perfusion scintigraphy revealed reversible ischemia involving the apex, interventricular septum, and anterior wall (26% of LV myocardium). Repeat coronary angiography demonstrated 50–60% stenosis in the mid and distal LAD and 95% in-stent restenosis, which was treated with balloon angioplasty under intravascular ultrasound guidance (fig. 2).

Figure 2. Coronary angiography demonstrating 95% in-stent restenosis in the left anterior descending coronary artery (LAD). The mid and distal segments of the LAD show 50–60% stenosis.



Lipid-lowering therapy resulted in a significant reduction in LDL cholesterol from 130 to 69 mg/dL. Pharmacological treatment included ezetimibe, low-dose rosuvastatin (limited by hepatotoxicity related to hematologic therapy), ticagrelor, and acetylsalicylic acid. Reduced-dose apixaban was added due to the increased thrombotic risk associated with MM and thalidomide therapy [1].

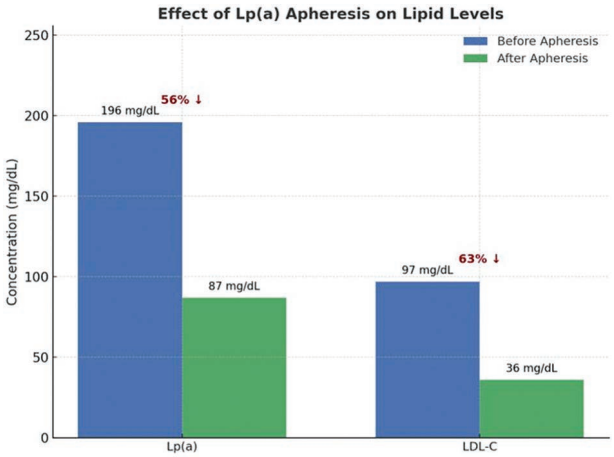
In December 2023, the patient underwent autologous stem cell transplantation (ASCT), and thalidomide was discontinued. However, thalidomide was reintroduced during maintenance therapy and it was followed by another STEMI. Coronary angiography revealed a new lesion distal to the previously implanted stent and a 75% stenosis of the diagonal branch; balloon angioplasty was performed, and thalidomide was permanently withdrawn. Subsequent testing revealed markedly elevated lipoprotein(a)

levels (196 mg/dL), considered a major contributor to rapid progression of coronary atherosclerosis [2].

Due to an unsatisfactory response to the first ASCT and recurrent ischemic events, lipoprotein apheresis was initiated in February 2024 (fig. 3). No further ischemic episodes occurred. A second ASCT was performed, and MM remission is currently maintained. Given the presence of extreme cardiovascular risk factors – including recurrent myocardial infarction, documented coronary atherosclerosis, persistently elevated LDL-cholesterol despite therapy, markedly elevated lipoprotein(a), and chronic kidney disease (eGFR <60 mL/min/1.73 m^2) the patient is currently being evaluated for intensified lipid-lowering therapy with a PCSK9 inhibitor or inclisiran within the national treatment program [3].

This case highlights the complex interplay between MM, prothrombotic anticancer therapy, and severe dyslipoproteinemia in accelerating coronary atherosclerosis and recurrent ischemic events. Markedly elevated lipoprotein(a), together with thalidomide-associated thrombogenicity and chronic kidney disease, contributed to rapid disease progression despite optimal revascularization and standard lipid-lowering therapy. The case underscores the importance of early recognition of high-risk lipid profiles [4], cardiovascular monitoring during hematologic treatment [5], and consideration of advanced lipid-lowering strategies in patients at extreme cardiovascular risk.

Figure 3. Effect of lipoprotein(a) apheresis on the lipid profile, demonstrating a 56% reduction in its concentration (from 196 mg/dL to 87 mg/dL) and a 63% reduction in LDL-cholesterol concentration (from 97 mg/dL to 36 mg/dL) immediately after the procedure.



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All authors contributed equally to the work.

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The paper complies with the Helsinki Declaration, EU Directives and harmonized requirements for biomedical journals.