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OPIS PRZYPADKU
CASE REPORT

Left ventricular thrombus after acute myocardial infarction – case report and literature review

Skrzeplina w lewej komorze po ostrym zawale mięśnia sercowego – opis przypadku i przegląd literatury

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ABSTRACT

Left ventricular (LV) thrombus is a complication of acute myocardial infarction (AMI), especially in patients with anterior AMI and persistent LV systolic dysfunction. Regardless of the progress in invasive AMI therapy, there are literature data showing the frequency of LV thrombi ranging from 2.7% to 19.2% in patients with anterior ST elevation myocardial infarction (STEMI). LV thrombi formation is associated with endothelial injury, blood stasis, and hypercoagulability. Early diagnosis using transthoracic echocardiography (TTE) and cardiac magnetic resonance (CMR) imaging is crucial. Prevention strategies involve assessing the risk factors and considering anticoagulant therapy in exceptional cases. Treatment involves anticoagulation for up to 6 months in combination with antiplatelet therapy. The study presents the current state of knowledge on LV thrombi, provided with a relevant case report as a practical guideline.

KEYWORDS

thrombus, left ventricle, magnetic resonance imaging, anticoagulants, echocardiography, acute myocardial infarction

STRESZCZENIE

Skrzeplina w lewej komorze (*left ventricle* – LV) jest jednym z powikłań ostrego zawału mięśnia sercowego (*acute myocardial infarction* – AMI), zwłaszcza u chorych z AMI ściany przedniej z wtórną do zawału utrzymującą się dysfunkcją skurczową LV. Badania wskazują, że pomimo postępów w interwencyjnym leczeniu AMI częstość występowania skrzeplin waha się od 2,7% do 19,2% u pacjentów z AMI z uniesieniem odcinka ST (*ST elevation myocardial infarction* – STEMI). Tworzenie się skrzeplin w LV wiąże się z uszkodzeniem śródbłonka, zastojem krwi i nadkrzepliwością. Kluczowe znaczenie ma wczesna diagnostyka za pomocą echokardiografii przezklatkowej (*transthoracic echo-*

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cardiography – TTE) i rezonansu magnetycznego serca (*cardiac magnetic resonance* – CMR). Strategie zapobiegawcze obejmują ocenę czynników ryzyka i rozważenie leczenia przeciwzakrzepowego w wyjątkowych przypadkach. Leczenie obejmuje terapię przeciwzakrzepową przez okres do 6 miesięcy w połączeniu z terapią przeciwpłytkową. W pracy przedstawiono aktualny stan wiedzy na temat pozawałowych skrzeplin w LV, z opisem przypadku jako praktyczną wskazówką.

SŁOWA KLUCZOWE

skrzeplina, lewa komora, rezonans magnetyczny, antykoagulanty, echokardiografia, ostry zawał serca

INTRODUCTION

Epidemiology

Thrombus in the left ventricle (LV) may appear as a complication of acute myocardial infarction (AMI), especially after AMI of the anterior wall, delayed reperfusion therapy and secondary LV systolic dysfunction [1]. Over the years, the incidence of LV thrombi after AMI has decreased significantly, which is due to the implementation of percutaneous coronary intervention (PCI). Despite the use of reperfusion therapy, the incidence of LV thrombi remains considerable, especially in patients with extensive AMI.

One meta-analysis found the rate of LV thrombi to be 2.7% after all ST elevation myocardial infarctions (STEMI) and 9.1% among patients with anterior STEMI [2]. In a more recent study, the incidence of LV thrombi after all STEMI was 6.3% but 96% of them occurred in patients with anterior STEMI (incidence 12.2%). Considering only anterior STEMI with LV ejection fraction (LVEF) < 50%, the incidence of LV thrombi was even 19.2% [3].

As another study showed, among patients who developed LV thrombi, the vast majority were diagnosed with solitary (80.9%), protuberant thrombi (68.8%) of the apical region (90.4%) [4].

Pathomechanism

The blood stasis results from impaired LV function are associated with a decreased LVEF, large apical or anterior LV akinesis or dyskinesis, and abnormal systolic function [5]. Long-term ischemia causes damage to the subendocardial tissue with inflammatory changes. The hypercoagulable state is associated with increased levels of prothrombin, fibrinopeptide A and von Willebrand factor (vWf), in addition to a decreased level of the enzyme responsible for the cleavage of vWf – ADAMTS13 [6]. Local tissue damage combined with a low shear rate in the infarct zone activates the coagulation system, causing fibrin accumulation by cross-linking platelets and erythrocytes together to form a fresh clot. These three factors – endothelial damage, blood stasis and hypercoagulability – make up Virchow's triad. A newly formed thrombus may contribute to a persistent inflammatory reaction within the myocardium and itself generate a thrombus [7].

LV thrombi are usually visualized between days 2 and 11 (median 6) after the clinical onset of AMI in 40% of patients with anterior STEMI [7]. It was determined that LV boundary thrombus is a common symptom in patients after anterior STEMI. Interestingly, by providing mechanical support to AMI, a mural thrombus may protect against LV rupture and allow a greater degree of LV function.

We present a case of an LV thrombus diagnosed during a routine check-up after STEMI as the basis for further considerations regarding diagnostic and therapeutic management.

CASE REPORT

A 47-year-old male with a history of nicotinism and hypertension, treated continuously with ramipril and amlodipine, was admitted urgently to the emergency department due to suspected anterior wall STEMI. Earlier that day, the patient had been reporting severe chest pain. While in transport by the emergency medical service, there was an incidence of ventricular fibrillation (VF), resulting in sudden cardiac arrest (SCA) requiring a single 200 J defibrillation to obtain a hemodynamically viable rhythm and consciousness.

On admission, a standard 12-lead electrocardiogram (ECG) showed sinus rhythm, 88 bpm, ST-segment elevation in leads aVL, aVR, V1–V4, with reciprocal ST-segment depression in leads II, III, aVF, V5 and V6. In transthoracic echocardiography (TTE), an apical akinesis was found with LVEF estimated at 42%. Other findings included uniform concentric LV hypertrophy (interventricular septum thickness: 12 mm, posterior wall thickness: 10–11 mm), and mild mitral regurgitation (MR).

The patient was transferred immediately to the catheterization laboratory. The coronary angiography performed via radial access revealed a left anterior descending artery (LAD) occlusion, which was successfully opened with stent (3 × 20 mm Promus ELITE™ drug-eluting stent) implantation. The procedure was accompanied by four episodes of VF, otherwise without complications. In the remaining arteries, parietal changes were observed, requiring no intervention. Laboratory testing revealed a maximum troponin T level of 4.58 pg/mL, and creatinine concentration of 1.2 mg/dL (estimated glomerular filtration rate [eGFR] 55 mL/min/1.73 m^2).

In a routinely performed control TTE, apical and septal hypokinesis with accompanying hyperkinesis of the remaining walls were seen and LVEF estimated at 60%. Particular attention was paid to the presence of

a previously absent hyperechoic, round thrombus in the apex, 22 × 18 mm in its largest dimension. These findings required treatment modification. After a short period of in-hospital low-molecular-weight heparin (LMWH) treatment, an oral anticoagulant was administered. Before the final decision of medicine prescription, the risks of in-stent thrombosis and hemorrhagic complication were analyzed. The risk of in-stent thrombosis was assessed as high (LAD reperfusion, long stent), and the bleeding risk was low (HAS-BLED score = 1).

The patient was discharged home with a recommendation of regular check-ups at a cardiologist and general practitioner, heart rate and blood pressure measurements, as well as laboratory tests: complete blood count, lipid profile, serum creatinine, glucose, and electrolytes. Lifestyle changes were indicated with a balanced diet limiting simple sugars, saturated fats, and salt, together with regular physical activity and smoking cessation. Additionally, follow-up TTE was scheduled in 3 months' time. The patient was prescribed and instructed on the use of the following medications: acetylsalicylic acid (ASA) 1 × 75 mg and clopidogrel 1 × 75 mg for 12 months, dabigatran 2 × 150 mg for 3 months (a continuation based on TTE verification of LV thrombus), bisoprolol 2 × 2.5 mg, ramipril 2 × 5 mg, eplerenone 1 × 25 mg, rosuvastatin 1 × 40 mg, pantoprazole 1 × 20 mg, and sublingual nitroglycerin on demand.

DISCUSSION

Diagnosis

As the most widely used imaging method, TTE is characterized by high sensitivity (95–98%) and relatively low specificity (21–35%) [8]. These factors can be improved with the use of a contrast agent [9]. LV thrombus appears as an echo-dense mass, distinct from the surrounding endocardium, particularly often located in the apical region, therefore limiting the possibilities of using transesophageal echocardiography (TEE) [10]. The 2023 European Society of Cardiology (ESC) Guidelines recommend routine echocardiography in all patients during the hospital stay to assess regional and global LV function, to detect mechanical complications, and exclude LV thrombus [I, C] [11]. Similarly, according to the 2013 American College of Cardiology Foundation/American Heart Association (ACCF/AHA) Guidelines, LVEF should be measured in all patients with STEMI, but without indicating the predominance of any of the imaging modalities [I, C] [12]. Thus, in the described patient already the first routine follow-up TTE after successful PCI revealed the presence of an LV thrombus of a typical location (Figure 1).

Cardiac magnetic resonance (CMR) imaging is considered the most valuable for the diagnosis and assessment of LV thrombus with its high sensitivity (82–88%) and specificity (100%) compared to other imaging methods [13]. As it does not show gadolinium uptake, thrombotic material contrasts well with vascularized myocardial tissue [9]. The use of additional sequences, such as late gadolinium enhancement (LGE), can even improve diagnostic accuracy [14]. Despite its high efficiency, CMR remains a relatively rarely used tool owing to its high cost and yet low availability in clinical practice. In accordance with the 2023 ESC Guidelines, it should only be considered as a preferable alternative in patients with equivocal echocardiographic images or in cases of a high clinical suspicion of LV thrombus [IIa, C] [11]. Therefore, it was not necessary in the described patient as the echocardiographic image was clear and sufficient to implement appropriate management.

Although not yet standardized for the detection of LV thrombi, other imaging methods under consideration include computed tomography (CT) and positron emission tomography (PET) [15,16]. They seem to be promising options, providing even more detailed information than TEE, the first of which, although inferior to CMR in terms of diagnostic accuracy, has a much lower cost and shorter procedure time.

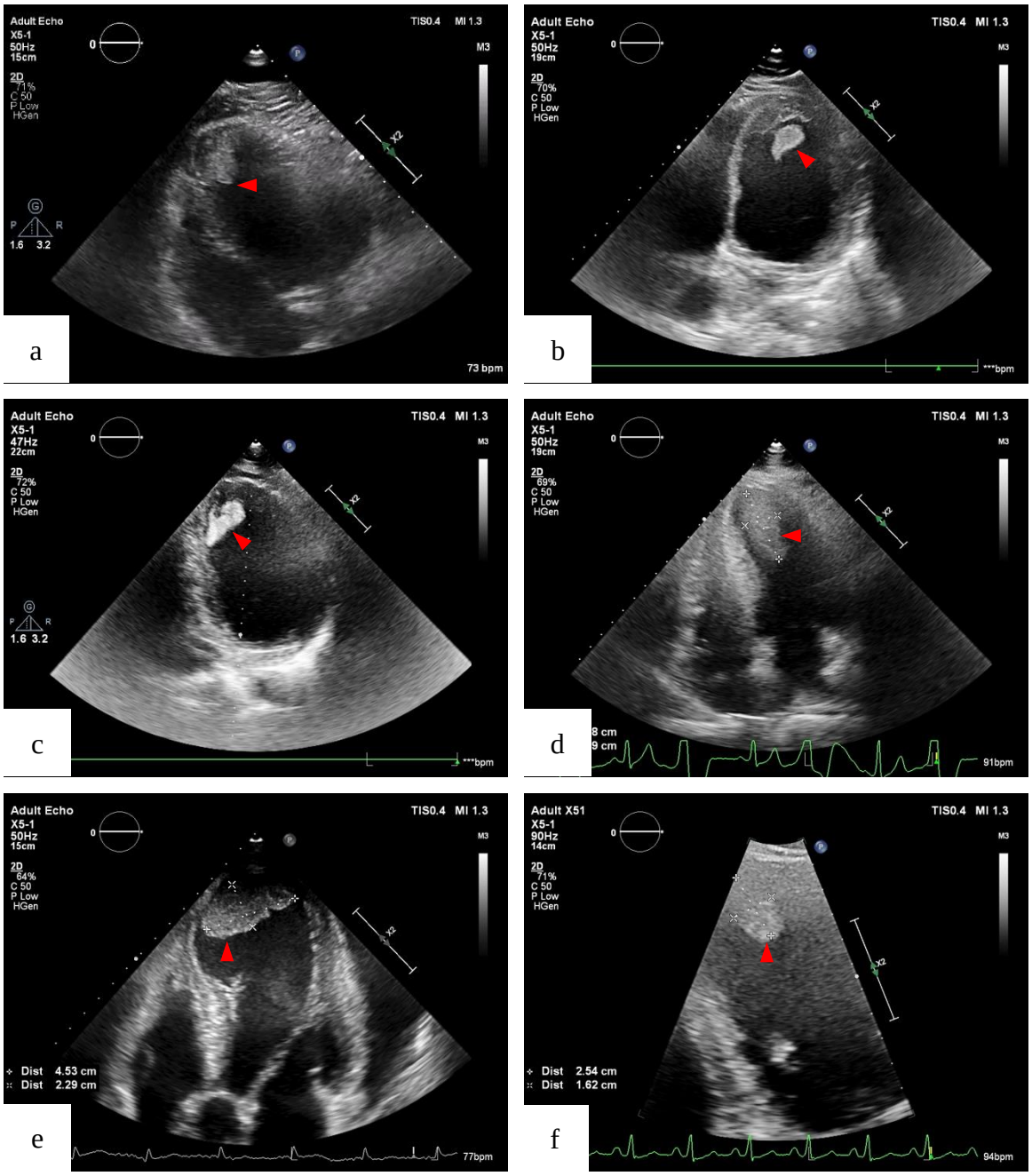

Fig. 1. Transthoracic echocardiography (TTE) performed in reported patient (a) and five others (b-f) revealing left ventricular (LV) thrombi of different shapes and sizes (red arrows). Although giving clear picture of location and morphology, this imaging method does not allow one to assess nature of the mass, and thus its etiology. (Authors' own material).
Ryc. 1. Echokardiografia przezklatkowa (TTE) wykonana u opisanego pacjenta (a) i pięciu innych (b-f) uwidoczniła skrzepliny w lewej komorze (LV) o różnych kształtach i rozmiarach (czerwone strzałki). Ta metoda obrazowania, choć daje wyraźny obraz lokalizacji i morfologii, nie pozwala na ocenę charakteru zmiany, a tym samym jej etiologii. (Materiał własny autorów).

Prevention

The 2023 ESC Guidelines provide no recommendations for prophylactic anticoagulant therapy in patients with severe LV dysfunction after AMI. An AHA scientific statement based on more recent literature review presents few data that support routine anticoagulation in the selected group of patients [17]. The risk factors for thrombus formation, bleeding risk secondary to the combined antiplatelet and anticoagulant therapy and patient preference, have been proposed to be considered when deciding on whether to initiate prophylactic anticoagulation. A few independent factors have been identified that increase the risk of LV thrombus formation in patients with AMI (Table I). Since they are unmodifiable, the assessment

of their presence allows one to at least estimate the risk of its occurrence and to take appropriate steps to avoid or reduce further complications.

Table I. Independent risk factors for left ventricular (LV) thrombus formation in patients with acute myocardial infarction (AMI; based on [17])
Tabela I. Niezależne czynniki ryzyka powstania skrzepliny w lewej komorze (LV) u chorych z ostrym zawałem mięśnia sercowego (AMI; na podstawie [17])

Independent risk factors for LV thrombus formation in patients with AMI
• Severe LV systolic dysfunction
• High myocardial scar burden
• Apical wall motion abnormalities
• History of acute cardioembolic event
• LV aneurysm

Treatment

The appearance of LV thrombus in patients with AMI requires anticoagulation to be administered for 3–6 months [IIa, C], which can be indicated in addition to antiplatelet therapy; however, not with ticagrelor or prasugrel as part of a triple antithrombotic therapy [III, C]. The therapy should be monitored by repeated imaging, preferably by TTE [IIa, C]. A similar strategy has been suggested in patients with non-ischemic cardiomyopathies [17]. It can be assumed that in any case of diagnosed LV thrombus, and in the absence of clear contraindications, anticoagulation should be started immediately. Its duration should be continued until resolution of the lesion [18]. At the same time, in the 2023 ESC Guidelines, there are no recommendations regarding the preferred group of oral anticoagulants [11]. Since the morphology of the thrombus itself, the course of the underlying disease and the individual predisposition of the patient may affect the overall risk of systemic thromboembolism, when deciding on anticoagulant treatment it is worth evaluating their presence (Table II) [19]. All things considered, the presented patient was prescribed with triple antithrombotic therapy in the form of aspirin (75 mg), clopidogrel (75 mg), plus dabigatran (2 × 150 mg). The low risk of HAS-BLED was an indirect suggestion to administer a full non-vitamin K antagonist oral anticoagulant (NOAC) dose. In the case of patients with a high HAS-BLED score or comorbidities, e.g. renal impairment, the reduced NOAC doses would be considered safer. Additionally, a follow-up after 3 months was ordered to assess the need for further NOAC treatment.

Table II. Independent risk factors for systemic thromboembolism in patients with left ventricular (LV) thrombi (adapted from [19])
Tabela II. Niezależne czynniki ryzyka zatorowości systemowej u chorych ze skrzepliną w lewej komorze (LV; zaadaptowano z [19])

Independent risk factors for systemic thromboembolism in patients who developed LV thrombi
• Cardiac arrest
• Mobile/Multiple thrombi
• Female
• Prior cardiovascular accident
• Dilated cardiomyopathy
• Large LV end-diastolic dimension

CONCLUSIONS

The diagnosis and management of LV thrombus after AMI require special consideration. The clinical selection of patients with risk factors for LV thrombus formation as well as the appropriate imaging method are crucial for the diagnosis. TTE remains the main and widely available diagnostic method. In cases with a bad acoustic window, the use of contrast may improve LV visualization. Both in-hospital and after-discharge anticoagulation are standard therapy and should be administered in combination with antiplatelet treatment. Repeated TTE is necessary to re-evaluate the validity of the treatment.

Author's contribution

Study design – M. Razik, B. Basiaga, K. Mizia-Stec
Manuscript preparation – M. Razik, B. Basiaga, P. Bator
Literature research – M. Razik, B. Basiaga
Final approval of the version to be published – K. Mizia-Stec

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