LEFT VENTRICULAR THROMBUS – DIAGNOSIS AND MANAGEMENT BASED ON A CASE REPORT

Skrzeplina w lewej komorze serca – diagnostyka i postępowanie na podstawie opisu przypadku

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Abstract

Left ventricular thrombus is one of the complications of ST-elevation myocardial infarction. Its presence is associated with the risk of serious embolic complications; therefore, diagnosis and effective treatment of formed thrombi are necessary. Suggested treatment methods include anticoagulation, thrombolysis and surgery, but there is no consensus on the choice of treatment method. We present a case report of a 52-year-old patient diagnosed with post-infarction left ventricular thrombus complicated by an embolic event. Due to the ineffective anticoagulant therapy, the patient underwent surgical removal of the thrombus, which resulted in its complete elimination from the left ventricle.

Streszczenie

Skrzeplina w lewej komorze serca jest jednym z powikłań zawału mięśnia sercowego z uniesieniem odcinka ST. Jej obecność niesie za sobą ryzyko poważnych powikłań zatorowych, dlatego konieczna jest diagnostyka i skuteczne leczenie uformowanych skrzeplin. Sugerowane metody postępowania obejmują leczenie przeciwkrzepliwe, trombolityczne i chirurgiczne, nie osiągnięto jednak konsensusu co do wyboru sposobu leczenia. Poniżej przedstawiono opis przypadku 52-letniego pacjenta, u którego rozpoznano pozawałową skrzeplinę lewej komory serca powikłaną incydentem zatorowym. W związku z brakiem skuteczności leczenia przeciwkrzepliwego u pacjenta wykonano operacyjne usunięcie skrzepliny, które pozwoliło na całkowite jej wyeliminowanie z lewej komory serca.

Keywords: left ventricular thrombus; embolic complications; coronary artery disease; myocardial infarction; anticoagulant therapy

Słowa kluczowe: skrzeplina w lewej komorze serca; powikłania zatorowe; choroba niedokrwienna serca; zawał serca; leczenie przeciwkrzepliwe

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Introduction

Left ventricular thrombus (LVT) may develop as a complication of ongoing or past ST-segment elevation myocardial infarction (STEMI). The incidence of LVT is estimated at 4% in patients with STEMI, and about 10% in the subgroup of patients with anterior STEMI [1, 2]. The presence of a thrombus is associated with the risk of embolic complications, including ischaemic stroke or systemic embolism [3, 4]. We describe a case of a patient with LVT complicated by embolism of one of the renal segmental arteries with partial infarction of the kidney and infarction of the spleen.

Case report

A 52-year-old patient with a history of myocardial infarction (MI) managed with angioplasty of the anterior descending coronary artery (ADCA) 15 years earlier, hypertension (HT) (not treated with antihypertensives), gastric and duodenal ulcers, active smoking, and depressive disorders, reported to the hospital

emergency department due to a 30-minute episode of burning chest pain that had occurred the day before admission, followed by severe pain in the left lower abdomen. Physical examination showed mild tenderness in the left iliac fossa on deep palpation. Ultrasonography (US) and X-ray of the abdominal cavity showed no significant abnormalities. However, due to the episode of chest pain and elevated markers of myocardial necrosis (elevated high-sensitivity troponin T: 986 ng/L vs. normal up to 14 ng/L), the patient was transferred to the cardiac intensive care unit (CICU) with suspicion of acute coronary syndrome.

On admission, he was in a good general condition, with good circulatory and respiratory function, full verbal and logical contact. He reported constant pain in the lower abdomen. Electrocardiogram (ECG) showed a regular sinus rhythm of 85/min, normal cardiac axis, QS complexes in leads V1–V3, and negative T waves in leads V4–V5 (stable in subsequent recordings). Laboratory findings included elevated markers of myocardial necrosis, normal amylase and lipase activity, and no elevated inflammatory parameters. General urinalysis revealed mild proteinuria. Transthoracic echocardiography (TTE) showed enlarged LV and left atrium, segmental contractility disorders: akinesia of the apex, apical segments of all walls and the middle segment of the interventricular septum, globally impaired left ventricular ejection fraction (LV-EF) of 35%; no significant valvular defects, signs of right ventricular overload or pericardial effusion were found. A mobile structure measuring 21 × 21 mm was identified in the apical LV region. A thrombus was suspected. Due to severe pain, elevated D-dimers and the identified structure in the left ventricle, an angio-CT scan of the abdominal aorta was performed for embolic complications, revealing absence of contrast enhancement of approximately 21 mm in the LV, which could correspond to a hypodense thrombus. Additionally, thrombosis of one of the left renal segmental arteries with middle and inferior renal infarction was detected. An isolated, conical area of right renal infarction could not be excluded. Also, an infarct zone was evident in the abdominal part of the spleen. The patient was consulted by a vascular surgeon, who concluded that revascularisation of the left kidney was not possible as the symptoms persisted for over 12 hours. A continuous unfractionated heparin (UFH) infusion was started, monitored by the activated partial thromboplastin time (APTT). The patient was also put on dual antiplatelet therapy (acetylsalicylic acid [ASA] plus clopidogrel), a proton pump inhibitor, a statin, antihypertensives and analgesics.

A follow-up echocardiography (ECHO) 7 days post admission showed no reduction in the size of the structure in the left ventricle (29 × 22 mm) despite continued anticoagulant therapy. A decision was made to perform a cardiovascular magnetic resonance (CMR) imaging to differentiate the lesion from a myxoma. The CMR revealed an akinetic aneurysm involving the mid-segment of the anteroseptal wall, apical segments of the anterior and lateral walls, the interventricular septum, as well as the apex. Additionally, there was a global reduction in left ventricular systolic function with LVEF of 36%. Cine images (a type of MRI sequence acquired to capture motion) showed a spherical hypointense structure near the LV apex. Early gadolinium enhancement (EGE) imaging showed an 18 × 12 mm hypointense structure, suggesting absence of contrast uptake, which is typical of a thrombus (fig. 1).

Late gadolinium enhancement (LGE) sequences revealed areas of subendocardial necrosis involving <50% of the myocardial thickness in the mid-inferoseptal wall segment, >50% of the myocardial thickness in the mid-anteroseptal wall segment, and nearly full-thickness necrosis in the apical segments of the anterior and lateral walls, the interventricular septum, and the apex, as well as a small, punctate area of full-thickness necrosis at the border of the basal segments of the inferior and inferolateral walls (fig. 2).

The examination showed ischemic necrosis with lack of viability in the region supplied by ADCA. During hospital stay, coronary angiography was also performed, showing 60% ADCA stenosis, and only mural lesions in the remaining coronary arteries.

Due to the presence of an LV apical thrombus complicated by an embolic event and not responding to anticoagulant therapy, the patient was qualified for surgical retrieval of the thrombus. Furthermore, the 60%

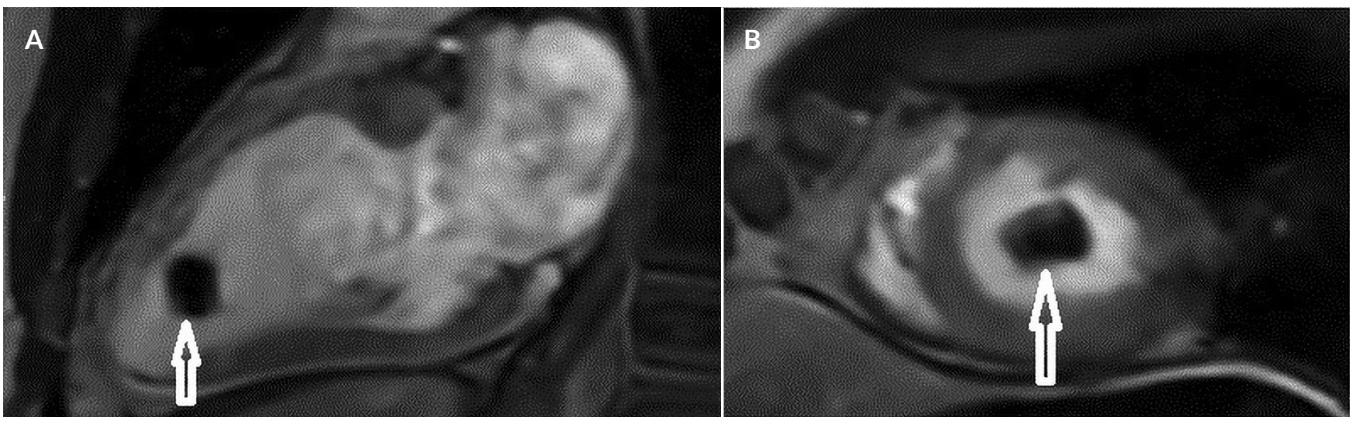

Figure 1. Early-enhancement sequences, **A.** dual chamber view, **B.** short axis view at the level of apical segments. White arrows indicate a hypodense thrombus

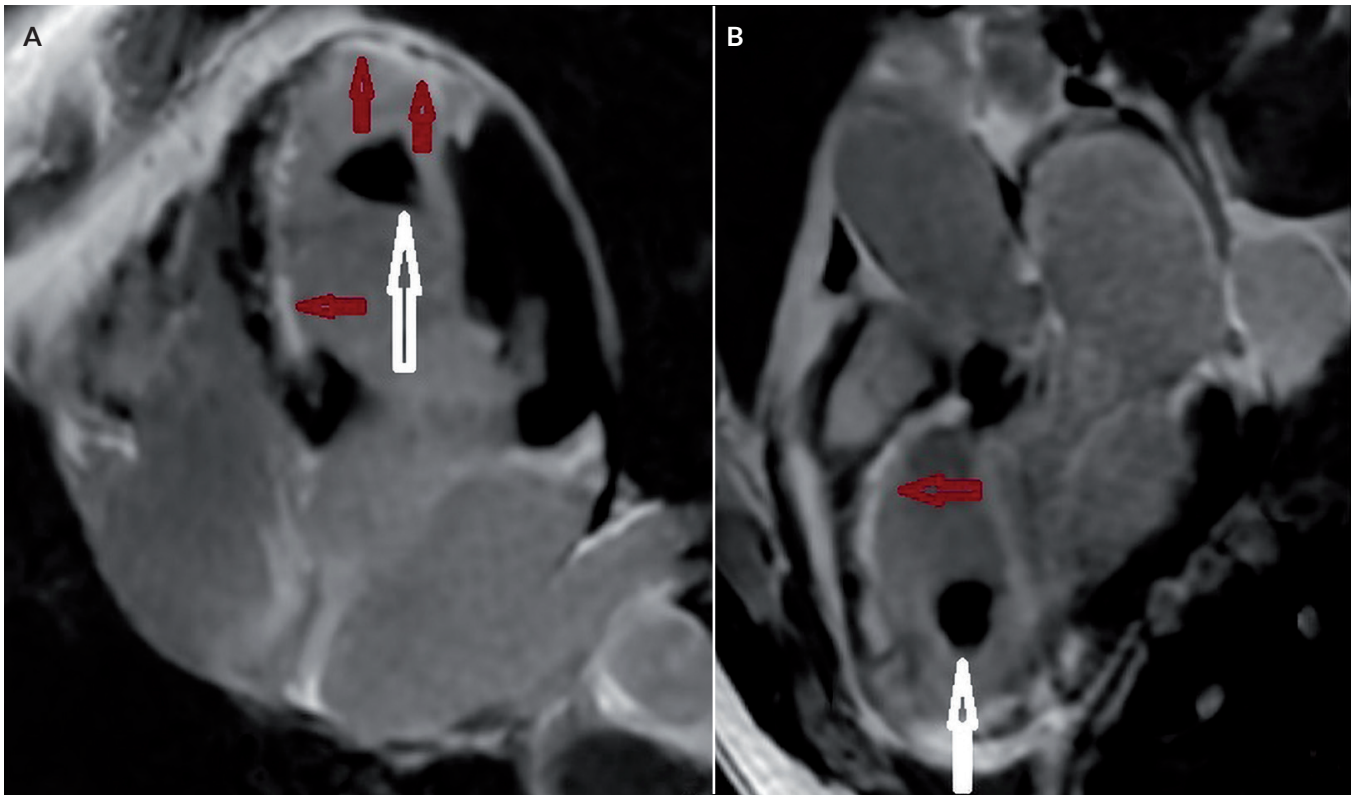

Figure 2. Early-enhancement sequences, **A.** four-chamber view, **B.** three-chamber view. White arrows indicate an LV thrombus, red arrows indicate examples of ischemic necrosis

ADCA stenosis identified in coronary angiography prompted a decision to perform a simultaneous coronary artery bypass grafting (CABG). The procedure was performed through sternotomy, using extracorporeal circulation. The LVT was retrieved through the left atrium accessed via the right atrium and interatrial septum, and the obtained specimen was sent for histopathological examination. This was followed by aortocoronary bypass grafting between the left internal mammary artery and ADCA. Histopathology confirmed the diagnosis of an organised thrombus, and the postoperative follow-up ECHO confirmed its total removal from the left ventricle.

Discussion

Risk factors for post-MI LV thrombus include, among others, anterior MI, impaired LV contractility and low LVEF (especially <35%), LV aneurysm, and apical akinesia [1, 3, 5]. The presence of thrombus is associated with the risk of embolic complications, which is particularly high if the blood clot is large, balloting and mobile [2, 6–8]. The significantly increased risk of a serious complication in the form of systemic embolism manifested by ischemic stroke, ischemia of organs or limbs, and even sudden death, prompts careful monitoring of patients for thrombus formation. TTE and CMR are used for this purpose. Studies comparing these two imaging tools have reported that compared to CMR, TTE shows 21–24% sensitivity, 95–98% specificity, as well as that CMR has a lower intra- and interobserver variability in thrombus detection than TTE [9]. Although these findings support the superiority of CMR over TTE in the diagnosis of LVT, ECHO is much more available in everyday clinical practice. Therefore, it seems that MRI is more appropriate in situations when TTE raises the suspicion of a thrombus, but is not fully diagnostic or leaves doubts [2]. CMR can also be used to differentiate a thrombus from a neoplastic lesion [10, 11]. Unlike tumours, thrombi are avascular and therefore characterised by a lack of enhancement in first-pass perfusion CMR imaging, and a hypointense signal in EGE sequences, which persists in LGE sequences.

Treatment options for LVT include anticoagulation, thrombolytic therapy, and surgical retrieval. However, there is no consensus on the most optimal approach or choice of treatment method [7, 8]. Current anticoagulation guidelines suggest the use of vitamin K antagonists (VKAs) for 3–6 months with a target international normalised ratio (INR) between 2 and 3 [2, 12, 13]. The length of therapy should also be individualised due to the risk of bleeding. Currently, attempts are being made to replace VKAs with novel oral anticoagulants (NOAC). No need to monitor the anticoagulant effect with frequent INR measurements, which significantly improves the quality of life of patients, is one of their benefits. According to the available data, it is justified to use NOACs instead of VKAs, as they offer a similar therapeutic effect and are equally safe [2, 12–14]. They are particularly beneficial in patients in whom the therapeutic level of INR cannot be achieved or frequent INR measurements are a significant burden. In both cases, ECHO should be used to assess treatment efficacy.

Cases described by Agarwal et al. and Liao et al. are examples of effective use of anticoagulation therapy [15, 16]. Agarwal et al. described a case of a patient with a history of coronary artery disease (CAD), status post stenting of the anterior descending and circumflex arteries, who presented to hospital with a suspicion of MI [15]. Coronary angiography revealed three-vessel disease and the patient was referred for CABG. Preoperative TTE showed an apical LVT and an LV aneurysm. The patient was started on a continuous UFH infusion and ASA. CABG was then performed. Postoperatively, the patient complained of severe pain in the right leg, accompanied by pallor and absent pulse in both lower limbs. CT of the abdomen and lower limbs showed a large thrombus in the aortic bifurcation, occluding the right common iliac artery and nearly occluding the left common iliac artery, as well as a thrombus in the right popliteal artery. The presence of embolic material was considered a complication of the LV thrombus. The patient underwent thrombectomy with postoperative UFH, clopidogrel, followed by warfarin. Another TTE showed a decrease in the size of LVT. No new embolic complications were observed during hospital stay, and the patient was discharged home with a recommendation to continue warfarin, clopidogrel, and ASA.

Anticoagulant therapy was also effective in the case of a 37-year-old woman with a history of long-term oral contraceptives, silent MI and anteroseptal hypokinesia, as well as an LV thrombus, bilateral renal infarction and occlusion of both popliteal arteries, described by Liao et al. [16]. The implemented anticoagulant and antiplatelet therapy (ASA, clopidogrel, intravenous heparin later switched to oral warfarin) resulted in complete thrombus dissolution, as confirmed during a follow-up after 2 months of treatment.

However, anticoagulant therapy not always leads to thrombus reduction or dissolution. This was the case described by Wejner-Mik et al., where a 5-day heparin infusion failed to reduce the size of a ventricular thrombus [17]. This can be partly explained by the relatively short treatment duration.

Fibrinolytic therapy seems to be an alternative to anticoagulant therapy, although there is insufficient evidence for its efficacy and safety [2, 18]. For this reason, it is not recommended as a first-line treatment.

A clot can also be retrieved surgically. However, such interventions are associated with a high risk of death and should therefore be reserved for special cases. Due to the high risk of systemic embolic complications, surgery seems to be beneficial in cases of a large, mobile, balloting LVT, as well as if anticoagulation is ineffective or not tolerated by the patient [2, 6, 7, 13]. Surgical thrombectomy can be performed via the left ventricular apex, left atrium or aorta when a concomitant aortic valve replacement is performed [7]. Thrombectomy via the LV apex was successfully performed by, among others, Pasli et al. and Wejner-Mik et al. [7, 17]. Pasli et al. described a 74-year-old female with a significantly reduced LVEF of 10–15% with an akinetic apex and a massive thrombus adjacent to the LV apex and interventricular septum complicated by acute ischemia of both lower limbs [7]. Due to her serious condition, the mobility of the thrombus and the need to prevent further embolic complications, a decision was made to perform surgical resection. The procedure was performed through a median sternotomy, with extracorporeal circulation and an incision of the LV apex. The thrombus was retrieved, and then the ventricle was cleaned and washed out. Postoperative ECHO confirmed that the left ventricle was completely free of thrombus. A similar solution was employed by Wejner-Mik et al. in a 65-year-old patient with extensive myocardial contractility disorders and apical akinetic aneurysm, as well as a large LV thrombus complicated by right common iliac artery embolism [17]. Initially, the patient was given a continuous UFH infusion, which did not bring any improvement in the form of reduced size of the thrombus after 5 days of treatment. It was considered that prolonging anticoagulation would probably prove ineffective and involve the risk of haemorrhagic complications. Additionally, given the high risk of embolic complications associated with the presence of thrombus, it was decided to retrieve it surgically. The procedure was performed by an incision of the aneurysmal LV apex. The thrombus was completely retrieved, and the aneurysm was managed with plasty according to Dora during LV wall closure. Successful removal of the thrombus from the left atrial access was reported by Tanaka et al. [8]. They described a case of a 37-year-old woman with a history of postpartum cardiomyopathy and multiple pulmonary embolism. Echocardiography revealed a deterioration of LV systolic function with LVEF of 10% and a large, mobile LV thrombus. Considering the size, thin pedicle of the thrombus and high embolic risk, it was decided that urgent surgical intervention was necessary. An incision of the left atrium was performed, and then the thrombus was exposed and retrieved through the open mitral valve. Postoperative echocardiography revealed a small, residual mural thrombus and an increase in LVEF of up to 40%. The patient was prescribed warfarin and no recurrence of embolic complications was observed during further follow-up. The authors of the paper pointed to the benefits of the transatrial approach. Atriotomy causes no damage to the LV wall resulting in deteriorated systolic function, which is often already significantly reduced in patients with a thrombus, or to the apex of the heart, making room for future implantation of an LV assist device. Additionally, such access allows for the removal of a larger thrombus than from the aortic access during simultaneous aortic valve surgery. However, less freedom of maneuver and limited possibilities of retrieving attached or thickly pedicled thrombi is a disadvantage compared to ventriculotomy. Transaortic access is rarely chosen, but its successful use has been described by Williamson et al., who found a large, mobile thrombus in the LV apex during transoesophageal echocardiography performed during aortic valve replacement [19]. After removal of the calcified valve, the thrombus was retrieved using forceps under video guidance. Postoperative follow-up showed no thrombus, and no new clots were detected during follow-up care.

To summarise, our patient had an LV thrombus detected in TTE, and later confirmed by CMR. The thrombus was complicated by occlusion of smaller arteries supplying the left kidney and the spleen. Anticoagulant treatment with UFH was initiated, which did not bring any improvement after 7 days of therapy. Due to the previous embolic events and the risk of their recurrence, it was decided that surgical approach was necessary. The clot was retrieved through the left atrium from the right atrial and interatrial septal access, achieving complete elimination of the thrombus with no recurrence in the postoperative follow-up.

Conclusions

The described case highlights the need to monitor patients after myocardial infarction due to the risk of a serious complication in the form of left ventricular thrombus. Patients suspected of MI complications should be regularly screened using TTE to detect abnormalities at an early stage, e.g. a forming thrombus, and receive treatment promptly enough to prevent serious embolic complications. Anticoagulants and surgical resection can be used for an organised thrombus. The choice of treatment approach should be adjusted to the patient's health status and based on the embolic–haemorrhagic balance, and the therapeutic decision should be made in consultation with the so-called heart team.

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