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Thrombus in the left ventricle in patient after anterior wall myocardial infarction treated with percutaneous coronary intervention and dual antiplatelet therapy

Zorganizowana skrzeplina w lewej komorze powstała po zabiegu przezskórnej angioplastyki wieńcowej mimo stosowanej podwójnej terapii przeciwpłytkowej

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Abstract

Myocardial infarction is an acute condition which can lead to many complications in a postinfarct period (MI, myocardial infarction). Left ventricular thrombus formation after successful primary percutaneous coronary intervention (PCI) with stenting and treating with dual antiplatelet therapy nowadays is a less common finding especially in absence of severe left ventricular dysfunction associated with MI. Case of 70-year-old male who was admitted to the Emergency Department with severe MI treating with successful primary PCI with stenting and dual antiplatelet therapy with a new unexpected finding of thrombus in the left ventricular apex observed in control of the echocardiography examination during hospitalisation despite dual antiplatelet therapy.

Key words: DAPT, myocardial infarction, left ventricular thrombus, echocardiography

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Case report

A 70-year-old male was admitted to the Emergency Department because of severe chest pain of one hour duration and ST-segment elevations on electrocardiogram (ECG). The patient had history of hypertension and chronic renal insufficiency. Physical examination revealed: the blood pressure 140/90 mm Hg, the pulse 100 beats per minute, body temperature was normal, the oxygen saturation 90% and bilateral crackles over the lung fields. Creatine kinase myocardial-bound (MB) fraction was elevated to 300.30 ng/mL (reference range 0.00 to 6.73), as well as troponin T was 8.05 ng/mL (reference range < 0.01). In the laboratory examination we confirmed the symptoms of inflammation process: the increase value of WBC 14.8 × 10^3/μL (reference range 4.0–10.0) and increased level of C-reactive protein (CRP) 40.42 mg/L (reference range 0.00–5.00). A 12-lead ECG showed both sinus rhythm at 100 beats per minute with ST-segment elevations of 2 to 4 mm in anterior leads V1–V4 and concomitant T-wave inversion in leads I aVL, V5–V6.

The patient was immediately qualified to urgent coronarography, which revealed isolated critical stenosis 99% in 6th segment of left anterior descending (LAD), 90% stenosis in middle segment 1st marginal artery (OM1) of left circumflex artery (Cx) and also 80% lesion in postero-lateral branch of right coronary artery (RCA) (Figure 1A). Simultaneous percutaneous coronary intervention (PCI) of

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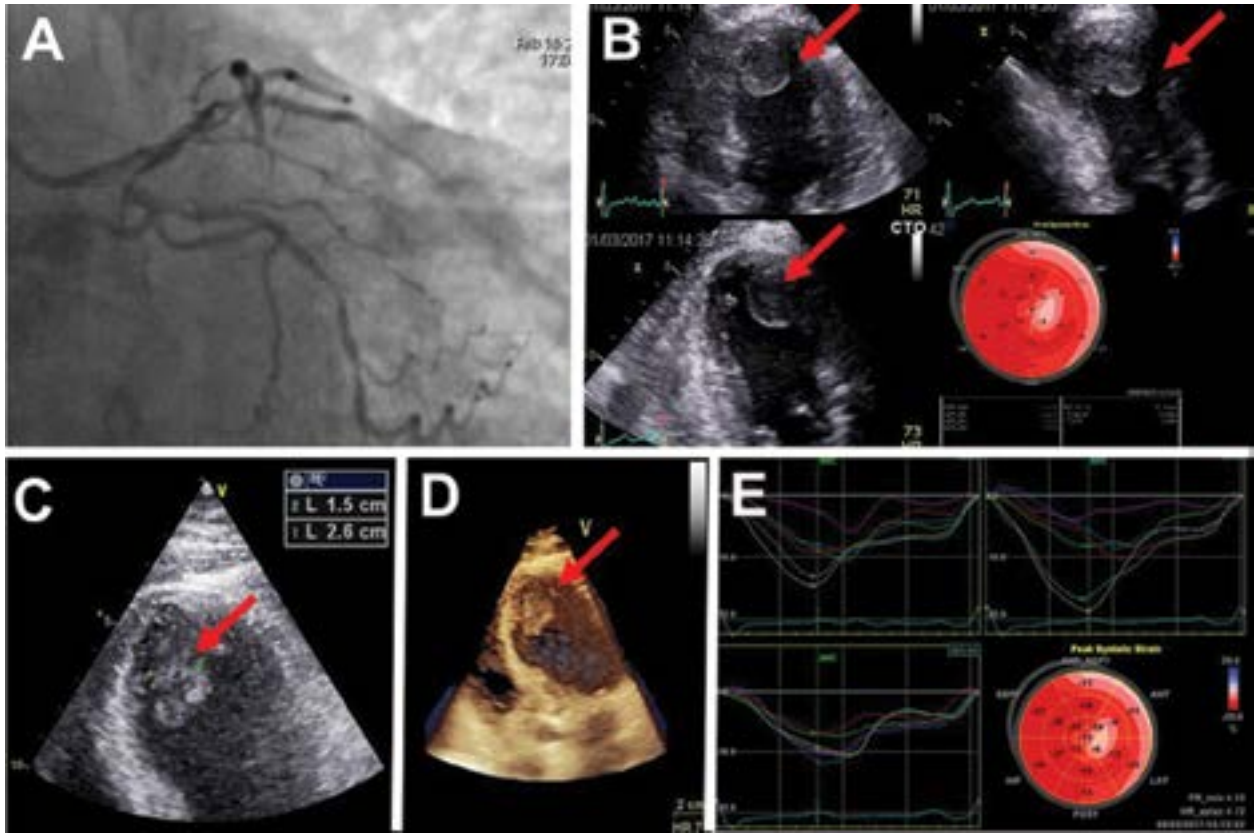

Figure 1A. Coronary angiography: critical stenosis in left anterior descending (LAD) and circumflex artery (Cx); **B.** Triplane view with visible thrombus in the apex of the left ventricle (red arrow), bull's eye-impaired contractility of the anterior and lateral wall, with global left ventricular strain of –15.5; **C.** Apical four chamber wall — diameter of the thrombus 1.5 × 2.6 cm; **D.** Three-dimensional (3-D) echocardiography with visible thrombus; **E.** Bull's eye with strain curves, impaired contractility of the anterior and lateral wall

LAD was performed with the implementation of everolimus drug-eluting stent (DES) Promus Premier 3.5 × 28 mm. After the procedure, the infusion of eptifibatide was continued for 12 hours. Dual antiplatelet therapy (DAPT) by ticagrelor (2 × 90 mg) and acetylsalicylic acid (75 mg) was initiated. Echo examination conducted on the next day revealed segmental wall motion abnormality; hypokinesis in the anterior and lateral wall of left ventricle (LV), with LV ejection fraction 51% (Figure 1E). Five days later, the second stage of angioplasty with implantation of everolimus eluting stent Synergy 2.5 × 16 mm was performed in OM1 artery with optimal angiographic result. The procedure was complicated with haematoma of the left forearm. The patient was consulted by surgeon, vascular surgeon and dermatologist, and according to their recommendations the accurate treatment was administrated (forearm elevation, gentamycin compress, low-molecular weight heparin [LMWH] 1 × 40 mg s.c.). During the hospitalisation, the evaluation of haematoma was observed from oedema, different stages of blisters to necrotic superficial changes. Because of clinically suspected pneumonia, the empirical antibiotic therapy by ceftriaxon and further ciprofloxacin was initiated. On the 13th day of hospitalisation, the control echo examination showed unchanged segmental wall motion abnormalities of LV (ejection fraction [EF] 48%), with a new unexpected finding of thrombus (size 14 × 27 mm) in the LV apex (Figure 1B–D). Notably, the thrombus formed despite dual antiplatelet therapy and LMWH treatment, and the infusion of unfractionated heparin (UHF) under control of the activated partial thromboplastin time (APTT) was instituted. In two subsequent echocardiograms continued two thrombi (17 × 9 mm and 25 × 24 mm) were still visible despite the intensification of treatment. During the hospitalisation, the patient's condition deteriorated and the level of CRP, white blood count (WBC) and procalcitonin increased. Antibiotic therapy was modified according to sputum culture and meropenem and levofloxacin treatment was initiated. Due to severe pneumonia and hypoxemic respiratory failure the artificial ventilation was started, but three days later the patient died because of respiratory failure and cardiac arrest.

Discussion

Myocardial infarction is an acute condition, which can lead to many complications in a postinfarct period. LV

thrombus formation prevalence was as frequent as 60% of patients with acute myocardial infarction (AMI) before thrombolytic treatment was introduced [1, 2]. Nowadays, the LV thrombus formation after successful primary PCI with stenting and treating with DAPT is a less common (< 10%) finding, especially in the absence of severe left ventricular dysfunction [3]. The risk of LV thrombus formation is highest during the first three months following AMI. One study showed, that about 90% of thrombi occurred two weeks after the index event [4]. Delewi et al. [5] indicate, that the most common risk factors for the development of LV thrombus are: large infarct sizes, severe apical asynergy, LV aneurysm, anterior myocardial infarction. The combination of Virchow's triad (blood stasis, endothelial injury and hypercoagulability) is often considered as a prerequisite for *in vivo* thrombus formation. The study on one hundred patients with AMI treated with PCI of the LAD and dual antiplatelet therapy confirmed in echocardiography and/or MRI among 15% of patients thrombus formation within the first three months after AMI [5]. The patients with thrombus had higher levels of serum soluble tissue factor (TF) and D-dimer in comparison with these without thrombus. On the other hand, the levels of prothrombin fragment 1+2 (F1+2) and endogenous thrombin potential (ETP) were significantly lower in the thrombus group [6]. Hypercoagulability along with large infarct size and probably proinflammatory activity are the main predictive factors in the pathogenesis of LV thrombus formation among patients with AMI, who are treated with DAPT. Anticoagulation therapy can be considered in patients with large anterior infarction and elevated TF and D-dimer, because of increased risk of LV thrombus [6].

Our patient presented several risk factors of occurring LV thrombus formation such as anterior location of acute myocardial infarction, persistent segmental wall motion abnormality of LV in the echocardiography (ECHO) and severe pneumonia. The inflammatory process in the lungs resulting in patient's might stimulate the coagulation process, which resulted in thrombus formation despite the anticoagulation treatment.

In conclusion, we present a case of unexpected, progressive LV thrombus complicating anterior infarction despite early reperfusion, dual antiplatelet therapy and LMWH prophylaxis in a patient with borderline decreased ejection fraction. The key trigger of the thrombus was likely the severe systemic infection.

Conflict of interest(s)

None

Streszczenie

Zawał serca jest stanem ostrym, który może prowadzić do licznych powikłań we wczesnym okresie pozawałowym. Występowanie skrzepliny w lewej komorze u pacjentów po skutecznej pierwotnej przezskórnej angioplastyce (PCI) z implantacją stentu i zastosowaniu podwójnej terapii przeciwpłytkowej (DAPT) w obecnych czasach jest mniej powszechnym zjawiskiem, zwłaszcza u osób bez ciężkiej pozawałowej dysfunkcji mięśnia lewej komory. Zaprezentowano przypadek 70-letniego mężczyzny, przyjętego do kliniki z prezentacją ostrego zespołu wieńcowego leczonego skuteczną pierwotną PCI z implantacją stentu i zastosowaniem DAPT, u którego, mimo zastosowanej terapii, w kolejnych badaniach echokardiograficznych podczas hospitalizacji obserwowano skrzeplinę w koniuszku lewej komory.

Słowa kluczowe: DAPT, zawał serca, skrzeplina lewej komory, echokardiografia

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