Folia Cardiologica 2021
vol. 16, no. 2, pages 148–150
DOI: 10.5603/FC.2021.0017

ISSN 2353–7752
e-ISSN 2353–7760

Myocardial infarction complicated by left ventricle thrombus: vitamin K antagonists as first-line treatment?

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Abstract

The article presents the case of a 59-year-old patient hospitalized due to myocardial infarction with ST-segment elevation, who developed a thrombus in the apex of the left ventricle in the early post-infarction period.

Key words: myocardial infarction, left ventricular thrombus, vitamin K antagonists

Folia Cardiologica 2021; 16, 2: 148–150

Introduction

The presence of thrombus in the left ventricle of the heart is one of the complications of acute coronary syndrome, associated with an increased risk of ischaemic stroke or systemic embolism [1]. The incidence of thrombus in cardiac cavities as a complication of myocardial infarction is not high, and it is estimated that it occurs in less than 1% patients [2[. Factors which significantly increase the risk of thrombus formation include: myocardial infarction of the anterior wall, left ventricular ejection fraction < 35%, dyskinesia of the apex or left ventricular aneurysm [3]. Treatment of patients with left ventricular thrombus consists in administering a vitamin K antagonist (VKA) for 3–6 months [4]. We present a clinical case report of a 59-year-old man hospitalized due to myocardial infarction complicated by a thrombus in the left ventricular apex.

Case study

A 59-year-old man was admitted to the cardiac intensive care unit due to acute coronary syndrome with ST-segment elevation (STEMI, ST-elevation myocardial infarction). Electrocardiography performed on admission revealed ST-segment elevation in the form of Pardee's wave in leads V1–V6 (Figure 1). Laboratory tests revealed hypoglycaemia 218 mg/dL (reference value 65–100 mg/dL), elevated concentration of myocardial necrosis markers: creatine kinase-myocardial bound (CK-MB) — 54 U/L (reference value 0–39 U/L), creatine phosphokinase (CPK) — 706 U/L (reference value 60–390 U/L) and troponin T — 453,1 ng/L (reference value 0.0–14.1 ng/L). The patient had a history of cardiovascular risk factors: long-term nicotine use (30 pack-years), type 2 diabetes and hypercholesterolemia [high-density lipoproteins (HDL) — 31 mg/dL, low-density lipoproteins (LDL) — 118 mg/dL, triglycerides — 119 mg/dL). The patient had not been receiving chronic treatment for his cardiovascular conditions and was managing diabetes with diet.

Transthoracic echocardiograph performed on admission revealed akinesis of the apex and the central and apical segments of the adjacent walls. Left ventricular ejection fraction was at 25%. Coronary angiography revealed a critical 90% stenosis in the proximal segment of the anterior descending branch of the left coronary artery. At the same time, percutaneous coronary intervention (PCI)

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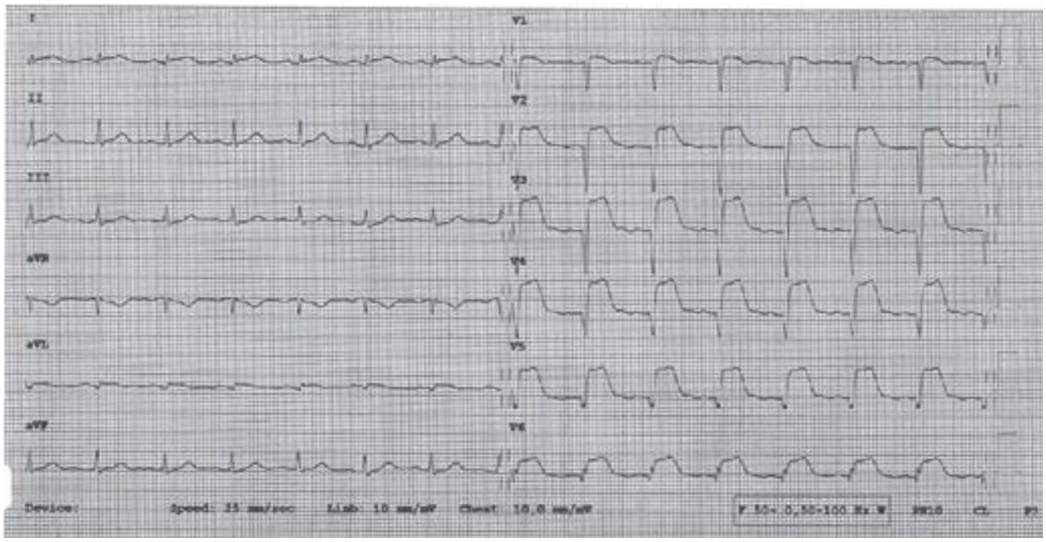

Figure 1. Electrocardiogram — sinus rhythm, regular, frequency of 88/min, normogram, ST-segment elevation in leads V1–V6

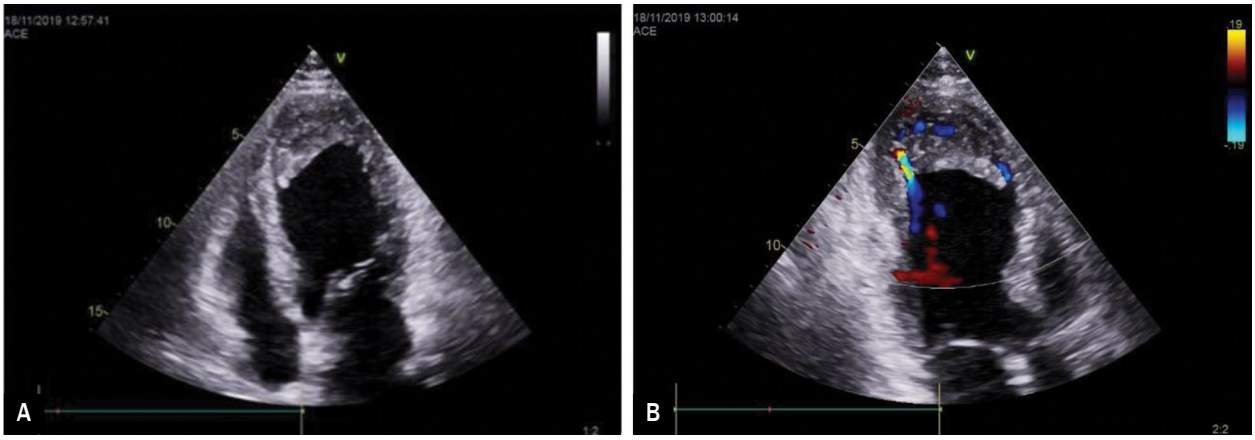

Figure 2A, B. Transthoracic echocardiography: thrombus in the left ventricular apex in four-chamber apical view and three-chamber apical view

with drug-eluting stent (DES) implantation was performed without complications.

The evolution of the myocardial infarction was observed in follow-up (ECG) conducted during the hospitalization. It revealed a thrombus in the lumen of the left ventricular apex (Figure 2), left ventricular ejection fraction was at 34%. The patient was discharged from the hospital 7 days after the infarction with the following recommendations regarding further treatment: acetylsalicylic acid 75 mg, clopidogrel 75 mg, warfarin 5 mg [controlling the international normalized ratio (INR), which should be at 2–2.5], bisoprolol 5 mg, ramipril 5 mg, atorvasterol 80 mg, eplerenone 50 mg, metformin 1,000 mg and empagliflozin 10 mg. The patient was instructed to report to follow-up echocardiography after 3 months.

Discussion

VKAs have been used to prevent and treat thromboembolic complications for over 50 years. Currently, they are increasingly often replaced with novel non-vitamin K antagonist anticoagulants (NOAC). NOACs are at least as effective VKAs and show a higher safety profile. NOACs are preferred for the treatment of patients with such diseases as atrial fibrillation and venous thromboembolism [5, 6]. However, VKA treatment remains the first-line therapy in patients with mechanical heart valves, atrial fibrillation with moderate or severe mitral stenosis as well as patients with left ventricular thrombus [4, 7].

Left ventricular thrombus formation may be a complication of myocardial infarctions, particularly those involving

the anterior wall, the apex and apical segments [8]. Other factors favouring the thrombus formation include left ventricular ejection fraction < 35% and apical dyskinesis or left ventricular aneurysm [3]. Due to the fact that thrombus may form after approximately 24 hours from the occurrence of acute coronary syndrome, it is important to perform follow-up echocardiography in days following the infarction. Two-dimensional transthoracic echocardiography is highly specific (85–95%) and sensitive (95%) in the detection of left ventricular thrombi. Thrombi present as separate masses with well-defined margins that are distinct from the endocardium. It can be visualized during both systole and diastole. According to studies, 90% of thrombi form within 2 weeks after infarction. In some patients, thrombi formation is observed after discharge from hospital as a result of deteriorating systolic function of the left ventricle [8]. Presence of thrombus in the left ventricle is associated with an increased risk of systemic embolism, for example ischaemic brain stroke. With anticoagulation treatment, the incidence of thromboembolic events can be significantly reduced [8].

In the patient in question, thrombus was found on the third day following the infarction. The electrocardiographic image revealed infarction of the anterior and lateral wall, which is associated with poor prognosis due to a higher incidence of thrombus formation in the left ventricle [9]. Due to the fact that VKA therapy is the first-line treatment in such cases, anticoagulant therapy with warfarin was implemented. Clinical experience with the use of NOACs in patients with post-infarction thrombus in the left ventricle is limited, although recently published data shed new light on the use of NOAC in this group of patients. An observational study involving 2,328 patients after myocardial infarction undergoing percutaneous coronary revascularization showed that thrombus in the left ventricle was observed in 101 patients. VKAs were given to 59.5% of them, while 40.6% received NOACs. Thrombus involution was faster in patients who received NOACs. In addition, the process was observed in a greater percentage of patients than in the group of individuals treated with VKAs. In addition, fewer bleeding complications were observed in patients treated with NOACs [10]. Further studies confirming the effectiveness and safety of NAOC therapy in patients with left ventricular apical thrombus are needed.

Summary

The treatment applied in the presented case consisted in a combination of two antiplatelet drugs and a vitamin K antagonist. There is numerous scientific evidence proving that combining antiplatelet drugs with NOACs in atrial fibrillation patients and patients after myocardial infarction/coronary revascularization is safe and effective. There are also reports indicating that such a combination can be used in patients in whom anticoagulant treatment should be introduced due to a left ventricular thrombus. It seems that this will be an increasingly common solution.

Conflict of interest

The authors declare no conflict of interest.

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