

Acute myocardial infarction due to coronary embolism originating from left ventricle thrombus in a patient with dilated cardiomyopathy and sinus rhythm

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Coronary artery embolism causing acute myocardial infarction (AMI) is more common than previously thought. Prizel *et al.* observed coronary artery embolic infarcts in 55 out of 419 patients, which comprised 13% of autopsy-studied infarcts [1]. The large diversity of etiologies makes the precise diagnosis challenging.

A 46-year-old male cigarette smoker was admitted to the emergency department due to sudden severe chest pain. He had a history of arterial hypertension, hypercholesterolemia and heart failure in New York Heart Association (NYHA) functional class III due to post-inflammatory dilated cardiomyopathy, diagnosed in 2007. He was previously treated with loop diuretics, angiotensin-converting-enzyme inhibitor (ACEI) and B-adrenolytic. Laboratory tests on admission revealed significantly elevated markers of myocardial necrosis (high sensitivity troponin T level up to 1766 ng/l) and a high level of plasma B-type natriuretic peptide (1801 pg/ml). Electrocardiography (ECG) showed left bundle branch block (LBBB). Transthoracic echocardiography (TTE) examination revealed a significant reduction of left ventricular ejection fraction (LVEF) of 14% with diffuse global hypokinesis, an akinetic anterior wall and the presence of a mobile thrombus in the left ventricle (Figure 1 A). Urgent coronary angiography showed distal occlusion of the left anterior descending coronary artery (LAD) by a well-organized thrombus and no atherosclerotic lesions in the coronary arteries (Figure 1 B). Because of the very distal location of the thrombus, manual aspirational thrombectomy was not feasible. Percutaneous coronary intervention (PCI) was also rejected due to the small diameter of the vessel. The patient was qualified for conservative treatment. Low molecular weight heparin (LMWH) was administered followed by oral an-

ticoagulant until the international normalized ratio (INR) was in the therapeutic range. The patient also received acetylsalicylic acid and standard heart failure treatment.

During hospitalization, dynamic changes in the shape and mobility of the gradually resorbing thrombus were observed in the echocardiographic images. After 6 weeks of anticoagulation therapy TTE showed the absence of a thrombus in the LV and revealed the improvement of LVEF from 14% at baseline to 23% (Figure 1 C).

In many cardiac conditions the risk of thromboembolic events is a reason for concern. The most common causes of coronary artery embolism are valvular heart disease (40%), dilated cardiomyopathy (29%), chronic atrial fibrillation (24%) and coronary atherosclerosis (16%) [1, 2]. Dilated LV offers a suitable terrain for chronic thrombus formation. The examination of 45 patients with dilated cardiomyopathy with mild to moderate systolic dysfunction, who were in sinus rhythm and without anticoagulation therapy, revealed left ventricular thrombus in 13.3% and left atrial appendage thrombus in 68.9% [3]. The features that may predict a higher likelihood for thrombus embolization are: protrusion into the LV, mobility, size and pedunculated appearance [4].

The consequences of coronary embolism depend on both the size of the embolus and the size of the artery in which it becomes impacted. Emboli lodging distally in normal coronary arteries most often cause small but transmural myocardial infarction [1].

Many researchers indicate that there is still a need for a prospective, randomized clinical trial to assess the risks and benefits of long-term anticoagulation in patients with dilated cardiomyopathy. We are looking forward to having clear guidelines for management of

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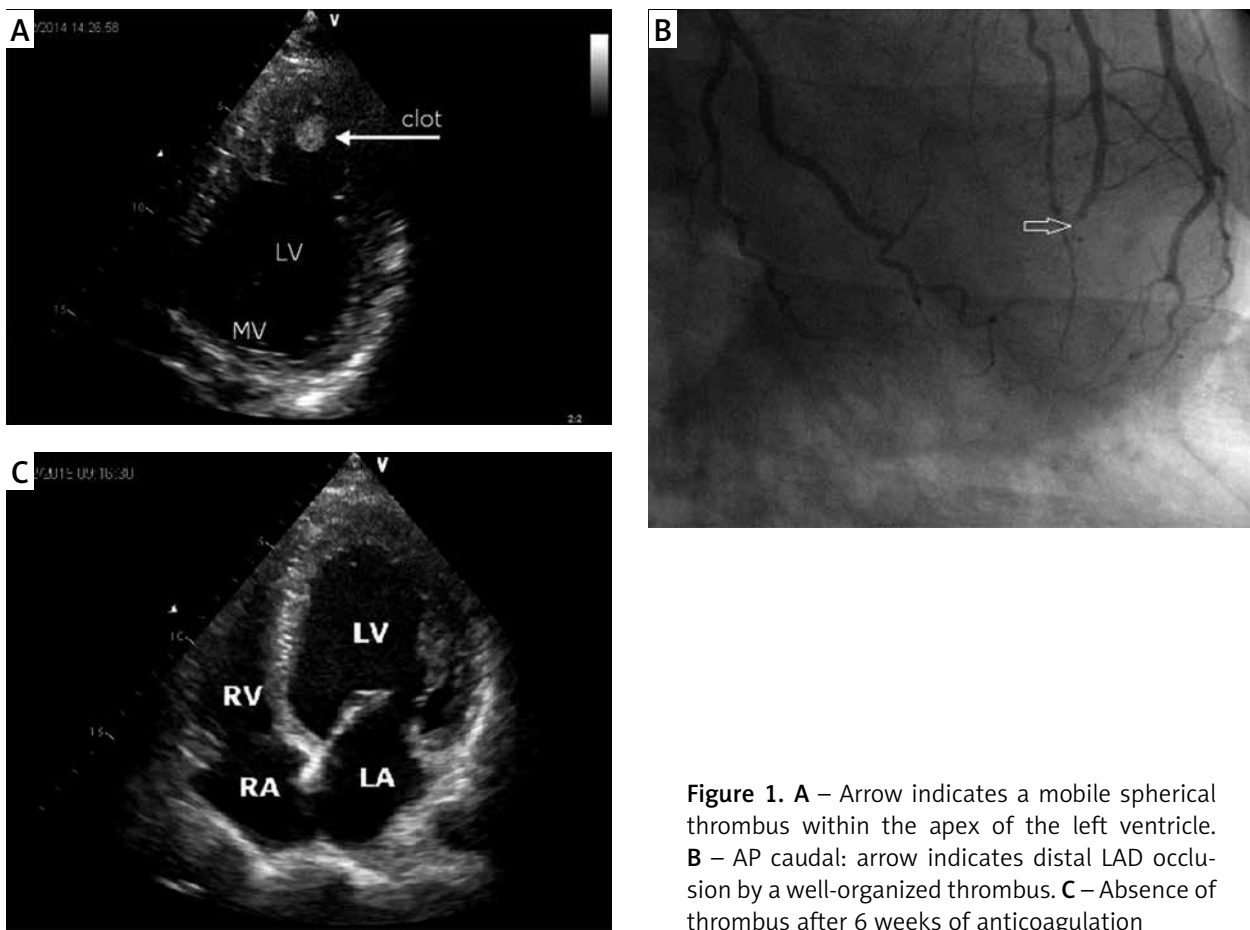


Figure 1. A – Arrow indicates a mobile spherical thrombus within the apex of the left ventricle. B – AP caudal: arrow indicates distal LAD occlusion by a well-organized thrombus. C – Absence of thrombus after 6 weeks of anticoagulation

such cases, hoping that new treatment strategies will be investigated.

Conflict of interest

The authors declare no conflict of interest.

References

1. Prizel KR, Hutchins GM, Bulkley BH. Coronary artery embolism and myocardial infarction. *Ann Intern Med* 1978; 88: 155-61.
2. Camaro C, Aengevaeren WRM. Acute myocardial infarction due to coronary artery embolism in a patient with atrial fibrillation. *Neth Heart J* 2009; 17: 297-9.
3. Bakalli A, Georgievska-Ismail L, Kocinaj D, et al. Prevalence of left chamber cardiac thrombi in patients with dilated left ventricle at sinus rhythm: the role of transesophageal echocardiography. *J Clin Ultrasound* 2013; 41: 38-45.
4. Glikson M, Agranat O, Ziskind Z, et al. From swirling to a mobile, pedunculated mass – the evolution of left ventricular thrombus despite full anticoagulation. Echocardiographic demonstration. *Chest* 1993; 103: 281-3.