

# Multiple cardiovascular complications in a patient with missed small apical myocardial infarction caused by a coronary artery thrombus of uncertain origin

Liczne powikłania sercowo-naczyniowe u chorego z niezpoznanym niewielkim zawałem koniuszkowym spowodowanym przez skrzeplinę w tętnicy wieńcowej o nieznanym pochodzeniu

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An 82 year-old hypertensive male with no concomitant diseases was admitted because of missed myocardial infarction (MI), 3 days after chest pain onset. On admission, the patient was pain free with stable haemodynamics and normal physical examination. The ECG (Fig. 1) showed atrial fibrillation (AF) and ischaemic changes with its resolution at follow-up. During coronarography (Fig. 2), the presence of a thrombus in the apical part of the left anterior descending (LAD) artery, atherosclerotic plaques with lack of culprit lesion clearly connected to the thrombus, and insignificant myocardial bridging (MB) were demonstrated. The patient was followed conservatively. Troponin and CK-MB elevation with decreasing consecutive values was observed. Transthoracic echocardiography (TTE) delineated left atrial (LA) enlargement, akinesia limited to the left ventricular apex with preserved global ejection fraction of 60% as well as a recent, partially movable, apical left ventricular mural thrombus (LVMT) (Fig. 3A). Warfarin therapy was introduced. On day 2, he developed acute ischaemic stroke (AIS). Carotid artery disease was excluded. On day 6, a systolic murmur demonstrated by echocardiography as an apical ventricular septal rupture (VSR) with left-to-right shunt and a thrombus partially occluding the defect which was smaller and less movable was diagnosed (Fig. 3B). The cardiothoracic surgeon recommended delaying surgical treatment. The patient's haemodynamic status worsened gradually with signs of exclusive right ventricular failure. An intra-aortic balloon pump was not used due to lack of patient co-operation. On day 18, the family requested that he be discharged home where he finally died. It is most likely that atherosclerotic plaque was involved in the thrombus formation and coronary occlusion, although an AF related embolism is quite possible. These hypotheses could not be verified because there was no culprit lesion and no LA thrombus confirmation. Less probably, MB could have given rise to coronary thrombosis with subsequent MI. Finally, tako-tsubo cardiomyopathy, coronary vasospasm or MB can be a primary cause of apical akinesia, blood stagnation, LVMT development and secondary coronary embolism. LVMT and VSR are strongly associated with extensive anterior MI. In this case it was small and limited to the apex, but proximal LAD occlusion with downstream thrombus migration is possible. Moreover, we speculate that ST elevation in inferior leads might be related to dominant LAD and that ST depression in precordial leads might be a reciprocal change. A coincidence of LVMT and VSR has rarely been documented. Moreover, we observed the partial occlusion of the defect by the thrombus which might delay its recognition and slow the natural evolution. The most probable explanation for AIS is embolisation related to recent LVMT or AF.

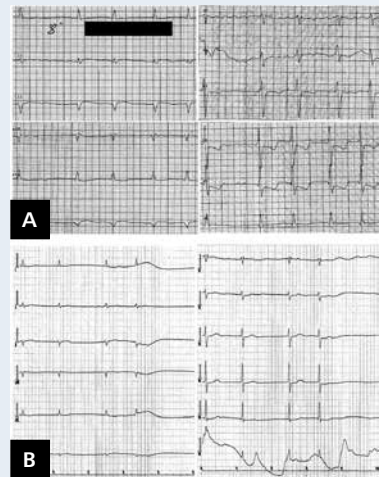


Figure 1. A. Admission ECG with pathological Q waves in lateral leads and ST-T changes in anterolateral and inferior leads (B). Follow-up ECG with resolution of ST-T changes. Paper speed 50 mm/s

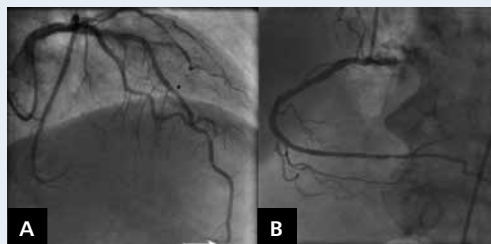


Figure 2. A. Left coronary angiogram with insignificant lesions in the main LAD lumen, a recent thrombus (arrow) in its distal part, MB (asterisk) and significant stenoses in diagonal branches; B. Right coronary angiogram

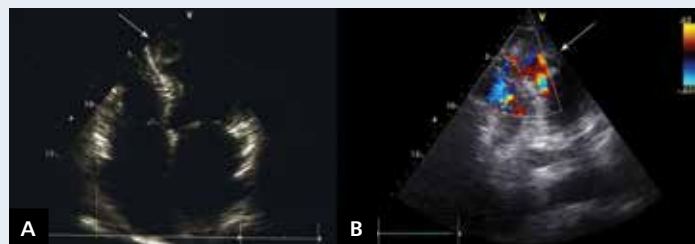


Figure 3. TTE. A. Recent LVMT (arrow) in the apex and the adjacent apical septum; B. VSR with a left to right shunt and a thrombus (arrow) that is smaller in size

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**Conflict of interest:** none declared