Case report: 66-year-old man with a history of mental disorder, presented with acute pulmonary edema and pneumonia. ECG indicated signs of previous anterior wall myocardial infarction. Based on patient history we concluded that it had occurred approximately one month before he was admitted. Echocardiography identified mildly dilated left ventricle with moderately reduced ejection fraction with anterior wall akinesia (Figure 1). Apical akinetic segments were filled with thrombus. In accordance with the existing guidelines, we introduced three months of anticoagulation therapy with warfarin. Follow up echocardiography showed large apical pseudo aneurysm (probably in the inferoapical segment) partially filled with thrombus (Figure 2). With contrast echocardiography (SonoVue) we tried to identify the site of entry into the pseudoaneurysm. However, we could not confirm clear communication. The patient was offered immediate hospital admission for additional MRI diagnosis and operative treatment, but the patient refused all further diagnostic and surgical procedures. The patient is treated with heart failure drugs. Anticoagulation treatment was stopped due to the size of the pseudoaneurysm.

Conclusion: Cardiac pseudoaneurysms are rare but clinically significant lesions. Although often challenging to diagnose, advances in non-invasive imaging (echocardiography, MRI, CT) have improved our ability to distinguish cardiac pseudoaneurysms from other pathologies. Most pseudoaneurysms, particularly if acute or associated with symptoms, require surgical repair or percutaneous exclusion to reduce the risk of rupture and potential for thromboembolism. In patients who have a high risk for surgical (or percutaneous) intervention, particularly when a pseudoaneurysm is chronic in nature, conservative management may be prudent. In our patient, a pseudoaneurysm was detected 6 months after myocardial infarction, which is not usual. We assume that the thrombus in the apex temporary closed the initial rupture of the left ventricle. Introduced anticoagulation therapy may have been an additional factor in the aneurysm development, because it probably contributed to thrombus dissolution.

**KEYWORDS:** pseudoaneurysm, myocardial infarction, echocardiography.


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**LITERATURE**

