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References

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Two Cases with Similar Pseudoaneurysms but Different Outcomes

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The pseudoaneurysm is a rare cardiac pathology, in which the left ventricular free wall ruptures and the pericardium surrounds the rupture in combination with thrombus and inflammation and thus prevents the development of a hemopericardium. Left ventricular pseudoaneurysm may remain silent unless it gives rise to cardiac tamponade, collapse, and finally sudden death. In this case report, we present two cases with left ventricular pseudoaneurysms in the same area. One of them had a stable outcome and the other had a fatal outcome.

Key words: ventricular pseudoaneurysm, ventricular rupture, myocardial infarction, cardiac tamponade, sudden death

Introduction
Pseudoaneurysm is a rare cardiac pathology, in which the left ventricular free wall ruptures and the pericardium surrounds the rupture in combination with thrombus and inflammation and thus prevents the development of a hemopericardium. In pathological specimens of a pseudoaneurysm, there is neither an endocardial nor a myocardial tissue but only inflammated pericardial tissue, fibrin, and thrombus.

It can develop either as an early or as a late complication of myocardial infarction (MI). Unlike causes of mortality following MI, left ventricular pseudoaneurysm may remain silent unless it gives rise to a hemopericardium. However, once the hemopericardium develops, it may result in cardiac tamponade, collapse, and finally sudden death.

In this report, we present 2 patients with similar left ventricular pseudoaneurysms in the same area but with different clinical outcomes.

Case 1
A 70-year-old male patient, who underwent a coronary artery bypass grafting (CABG) operation (LIMA-LAD, Ao-OM2, Ao-RCA) 6 y ago, was admitted to our clinic with a typical angina of 2-mo duration. Ventriculography had not been performed previously due to the absence of an MI history and the presence of renal failure. No procedure had been applied to determine ventricular function at his follow-up after CABG. The patient had been asymptomatic until 2 mo previously. On physical examination, his blood pressure and heart rate were 135/85 mmHg and 82 beats/min, respectively. His heart rhythm was regular, without murmurs, rubs, or gallops. The electrocardiogram showed a normal sinus rhythm with no ischemic changes or MI signs of note. An echocardiography revealed cardiac wall thinning and akinesis in the apical region of his left ventricle. No wall motion abnormality was detected in the other segments, and the global ejection fraction was 55%–60%. Pseudoaneurysm, thrombus, valve disease, and pericardial effusion were not observed. A subsequent coronary angiography identified 3-vessel disease and severely stenosed bypass grafts. Although the patient had a diffuse vessel disease, none of the coronary arteries were found to be occluded. Finally, a left ventricular pseudoaneurysm measuring 7 × 5 mm was detected in the apical region on his ventriculography (Figure 1). The patient underwent an aneurysmectomy and redo bypass surgery and is doing well, 5 mo after his operation.

Case 2
A 66-year-old male patient was admitted to another hospital with chest pain that had started 5 h previously. ST-segment elevation had been detected in the anterior leads on the electrocardiogram (ECG), and thrombolytic therapy had been done. As a result, the severity of chest pain and the level of ST-segment elevations was decreased. After 2 d, chest pain had recurred, and ST-segment elevations were detected again in the inferior leads.

Thrombolytic therapy was repeated with a poor response, and the patient was immediately referred to our hospital for a percutaneous intervention. On admission to our hospital, the heart rate was 86 beats/min, and the blood pressure was 105/70 mmHg. Cardiac auscultation revealed a grade 2/6 systolic murmur, best heard at the apex. The ECG study showed a normal sinus rhythm, together with pathological Q waves and ST-segment elevations in the anterior leads.
the anterior and inferior leads. His coronary angiography revealed 90% and 50% stenosis of the proximal left anterior descending coronary artery and the left circumflex artery, respectively. There was a left ventricular pseudoaneurysm measuring 13 × 8 mm in the apex area on ventriculography (Figure 2). During echocardiographic examination, the pseudoaneurysm ruptured and the patient went into cardiopulmonary arrest with an electromechanical dissociation. Despite emergency transfer to the operating room for immediate surgery, he could not be saved.

Discussion

Pseudoaneurysm generally develops in patients having their first MI and hypertension usually accompanies the condition. Although the most common predisposing factor is MI, chest trauma, cardiac surgery (valve operation, correction of congenital heart disease, aneurysm repair), infective endocarditis, and rheumatismal diseases are commonly known to cause pseudoaneurysm.\textsuperscript{5,6}

Inferior and posterolateral MI are responsible for 82% of the pseudoaneurysm developing after MI.\textsuperscript{6} In most of these cases, pseudoaneurysm is located inferoposteriorly due to stenosis of the circumflex artery. It can also be found in the apical region due to anterior MI. Anterior MI rarely results in pseudoaneurysm formation because ruptures in the anterior wall generally leads to hemopericardium or tamponade.\textsuperscript{7,8} In both of the presented cases the pseudoaneurysm was located in the apical region. The etiology in the first case was not clear, but it is highly possible that pseudoaneurysm resulted from a silent MI that developed during the perioperative or postoperative period. In the second case, anterior MI was the etiology of formation of a pseudoaneurysm.

Clinical risk factors for rupture of the myocardium, and thus, development of a pseudoaneurysm in the setting of acute MI are a relatively small first acute MI, age of the patient (>60 years), female sex, hypertension, pericarditis, postinfarct angina, high CK-MB levels, elevated peak serum C-reactive protein, decreased collateral flow, use of non-steroidal anti-inflammatory drugs (NSAIDs) or steroids during the acute phase of MI (interference with the healing process), and late thrombolytic therapy (after 7 h).\textsuperscript{3,10} The 2 presented patients were more than 60 y old, and the second case had experienced a reinfarction and failed thrombolysis.

Pseudoaneurysm may remain clinically silent and be discovered during routine clinical investigations. Both of the patients presented with chest pain. The most common presenting symptoms include heart failure, chest pain, arrhythmias, syncope, embolic events, and hemoptysis.\textsuperscript{3,7,11} The development of heart failure is related to an uncontracted and dyskinetic region and dilatation of the pseudoaneurysm sac during systole as the blood enters into the sac. So it impairs the anterograde ventricular ejection during systole. This hemodynamic impairment leads to ventricular dilatation. Accompanied mitral regurgitation and underlying heart disease accelerates the impairment of the ventricular performance.\textsuperscript{12} Pseudoaneurysm should be considered in the patients refractory to the medical treatment and in unexplained heart failure cases.

The physical examination and ECG are not of much help in the diagnosis. A physical examination may reveal decreased heart sounds, or systolic, diastolic, or to-and-fro murmurs related to blood flow across the narrow neck of the pseudoaneurysm during systole and diastole. The classical new-onset to-and-fro systolic murmur is absent in most of
these patients, and when present, it can be mistaken for a mitral regurgitation murmur, as in the second patient who had a grade 2/6 systolic murmur at the apex. The ECG findings are often unrevealing and only 20% show ST-segment elevation, sinus bradycardia, and tachycardia.\textsuperscript{8,10} Most of the patients have nonspecific ST-T wave changes. In the first case, ST-segment elevation or pathological Q waves were not present in any of the ECG leads. In the second case, ST-segment elevation was present in the area overlying the pseudoaneurysm.

Cardiac catheterization with left ventriculography remains the gold standard for pseudoaneurysm diagnosis; in addition, concomitant coronary angiography is often required for preoperative evaluation. Other diagnostic techniques with excellent visualization include cardiac magnetic resonance imaging, computerized tomography, and 3-Dimensional echocardiography.\textsuperscript{8,13}

Operation is emergent and the treatment of choice in symptomatic patients, but operative risk is high due to the underlying cardiac pathologies. Reported postoperative mortality ranges from 7%–29%.\textsuperscript{1,6} Pseudoaneurysms are usually stable under 3 cm, and if they are detected incidentally, they can be managed conservatively.\textsuperscript{12} A 1-y survey with conservative treatment is reported as 88% and a 4-y survey as 74.1%.\textsuperscript{1} Conservative treatment may also be considered in patients who carry a high risk for operation.\textsuperscript{1,6,14}

Fatal rupture, hemopericardium, and tamponade are among the most important complications of pseudoaneurysm. In 1 series, rupture was reported to occur in 30%–45% of patients.\textsuperscript{15} The risk of acute complications is higher in the first 3 mo after MI.\textsuperscript{1} The other causes of death are heart failure and arrhythmias.\textsuperscript{1,6}

Although our 2 patients developed their pseudoaneurysm in a similar region of the left ventricle, there were major clinical differences between them. The first patient did not have a clinically evident MI and a small akinetic area in the apical region. The pseudoaneurysm probably remained silent for a long time. The second patient with ruptured pseudoaneurysm had a large area of MI, recurrent and failed thrombolytic therapy, and larger pseudoaneurysm size compared with the first case. His pseudoaneurysm became complicated with a tamponade in a short time, which is consistent with the fact that the risk of tamponade being directly related to the size of the necrotic region and the myocardial wall stress.

Conclusion

Left ventricular pseudoaneurysm is a rare condition with a wide clinical spectrum ranging from silent chronic aneurysm and nonspecific symptoms to acute fatal rupture. The patients with high risk of rupture should undergo urgent surgery even if their general condition is assumed to be well. On the other hand, it is still not clear whether the patients with chronic pseudoaneurysm should undergo urgent surgery or be managed conservatively. Further studies and longer follow-ups are necessary to understand these challenging questions.

References