**CASE REPORT** 

# Giant ventricular pseudoaneurysm and associated eccentric severe mitral regurgitation: Surgery or follow-up?

# Dev ventriküler psödoanevrizma ve ilişkili egzantrik ciddi mitral yetersizliği: Cerrahi mi, takip mi?

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Summary- A ventricular pseudoaneurysm develops mainly after myocardial infarction complicated by a ventricular free wall rupture contained by localized pericardial adhesions. The risk of rupture in untreated pseudoaneurysms is approximately 30%-45%, and the mortality rate is 50%. Although there is no clear evidence of treatment in the literature, the main suggestion is to perform surgical treatment without delay. However, the age of the patients, additional comorbidities, and the accompanying severe mitral regurgitation and left ventricular systolic dysfunction considerably increase the mortality rate of the surgical procedure. The treatment of left ventricular pseudoaneurysm accompanied by severe mitral regurgitation has not been clarified in the literature, and patient-based individual approaches vary. In this case report, the clinical course of the three patients was explained with different treatment approaches, and we tried to create a resource for treatment approaches in light of the literature.

A left ventricular pseudoaneurysm forms when Cardiac rupture is contained by adherent pericardium, and it contains no endocardium or myocardium.<sup>[1]</sup> The most common cause is myocardial infarction (MI), particularly inferior wall infarction. <sup>[2,3]</sup> The other common causes are mitral valve surgery, congenital heart surgery, and trauma.<sup>[2,3]</sup> Risk factors for the occurrence of left ventricular pseudoaneurysms include female sex, older age, history of hypertension, and lack of collateral circulation after MI.<sup>[4]</sup> The most common symptoms associated with pseudoaneurysm are heart failure, dyspnea, and angina. In the literature, patients presented with sudden cardiac death, syncope or arrhythmia, rup-

Özet- Ventriküler psödoanevrizma çoğunlukla miyokart enfarktüsünden sonra lokalize perikardiyal adezvonlar ile sınırlanan ventriküler serbest duvar rüptürü nedenivle olusmaktadır. Tedavi edilmeyen psödoanevrizmalarda rüptür riski yaklaşık %30-45 ve mortalite oranı %50'dir. Literatürde tedavisine dair net bir kanıt olmamasına rağmen temel öneri cerrahi tedavinin gecikmeden yapılmasıdır. Ancak hastaların yaşı, ek komorbiditeleri, eşlik eden ciddi mitral yetersizliği ve sol ventrikül sistolik fonksiyon bozukluğu cerrahi işlemin mortalite oranını önemli ölçüde artırmaktadır. Ciddi mitral yetersizliğinin eşlik ettiği sol ventrikül psödoanevrizmasının tedavisi ile ilgilide literatürde net öneriler bulunmamakta ve hasta bazlı bireysel yaklaşımlar uygulanmaktadır. Bu olgu sunumunda ventriküler psödoanevrizması ve ciddi mitral yetersizliği olan üç hastanın farklı klinik seyir ve tedavileri anlatılmış, literatürdeki veriler incelenmiş ve bundan sonraki benzer hastalara tedavi yaklaşımları için kaynak oluşturulmaya çalışılmıştır.

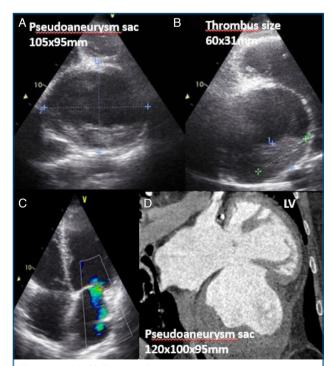
ture, cardiac tamponade, and asymptomatic clinical presentation have also been reported.<sup>[2,3]</sup> Severe mitral valve regurgitation and left ventricular systolic dysfunction because of pseudoaneurysm sac are the main factors that make both clinical and surgical management difficult. The treatment of left ventricular pseudoaneurysm accompanied by severe mitral regurgitation has not been clarified in the literature, and patient-based individual approaches vary. In this report, we present the detailed clinical courses of these three patients with different treatment approaches to share the experience for this rare complex pathology. Informed consent forms were obtained from all the patients.



# **CASE REPORT**

#### **Case report 1**

A 72-year-old male patient with type II diabetes mellitus underwent primary percutaneous coronary intervention (PCI) in January 2018 for inferior ST-elevation MI. The procedure was terminated with thrombolysis in myocardial infarction (TIMI) 2 flow. The patient was admitted to our clinic after one week with shortness of breath; transthoracic echocardiography revealed pericardial effusion, and cardiac computed tomography angiography (CCTA) was performed with a pre-diagnosis of cardiac rupture. CCTA showed ventricular aneurysm formation in the left ventricular posteroinferior region and self-limited rupture. The right coronary artery was also found to be occluded. Angiography was not considered again because the patient did not have chest pain, and one week had passed after acute MI. At the same time, ventriculography was not considered because CCTA is more sensitive than ventriculography in the diagnosis of ventricular pseudoaneurysm. The cardiac surgery team preferred close follow-up instead of surgery because of the high risk of emergent surgical



**Figure 1. (A-C)** Echocardiographic evaluation shows an aneurysm of  $105 \times 95$  mm and a thrombus of  $60 \times 31$  mm in a patient with severe eccentric mitral regurgitation. **(D)** Computed tomography angiography image shows an enlarged aneurysm sac ( $120 \times 100 \times 95$  mm), and thrombus is present.

operation. In such patients, the failure rate in early surgery may be high owing to the risk of failure of the Teflon patch, and the mortality

#### Abbreviations:

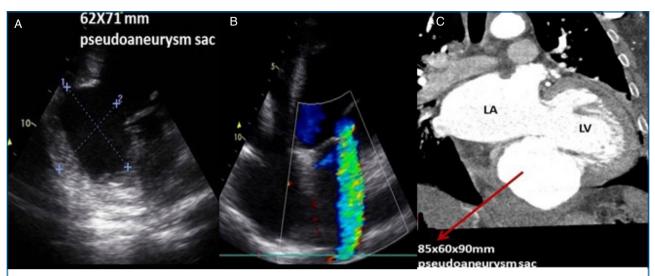
CCTA	Cardiac computed tomography angiography
MI	Myocardial infarction
NYHA	New York Heart Association
PCI	Percutaneous coronary intervention
TIMI	Thrombolysis in myocardial
	infarction

rate would also be high. The patient was monitored with daily echocardiography, and a control CCTA was performed after two weeks. The CCTA revealed that pericardial tissue appeared to limit rupture, and ventricular pseudoaneurysm formation of 50×60 mm size was detected. The transthoracic echocardiography showed the pseudoaneurysm sac to be located in the posteroinferior region disrupting the mitral valve posteromedial papillary muscle structure and causing eccentric severe mitral regurgitation. In addition, spontaneous echo-contrast and thrombus formation were observed to start in the pseudoaneurysm sac.

The clinical status of the patient was discussed in the cardiovascular surgery and cardiology council, and the patient was advised surgical pseudoaneurysm resection, mitral valve repair, and coronary artery bypass surgery. However, the patient did not give consent for the surgery. Therefore, the patient was discharged and followed up as an outpatient under medical treatment. In the echocardiography performed monthly, the pseudoaneurysm sac and thrombus were observed to be enlarged. The size of the aneurysm was 105×95 mm, and the thrombus size was 60×31 mm in the last control echocardiography, and the patient had severe eccentric mitral regurgitation (Figure 1A-C). The patient was re-evaluated with CCTA, which showed that the aneurysm sac grew (120×100×95 mm), and thrombus was present (Figure 1D). After two years of follow-up, the patient died because of pulmonary edema and cardiogenic shock.

#### Case report 2

A 59-year-old female patient was admitted to our outpatient clinic for decompensated heart failure and the New York Heart Association (NYHA) class 3-4 symptoms in February 2019. Her medical history revealed diabetes mellitus and coronary artery disease (with an unsuccessful PCI to the right coronary artery in 2013). The patient underwent implantable cardioverter-defibrillator implantation for heart failure in 2015, and transplantation was suggested owing



**Figure 2.** (**A**, **B**) Echocardiographic image of pseudoaneurysm sac and thrombus formation and distorted mitral valve posteromedial papillary muscle and eccentric severe mitral regurgitation. (**C**) Cardiac computed tomography angiographic image of the  $85 \times 60 \times 90$  mm pseudoaneurysm sac.



Figure 3. (A, B) Cardiac computed tomography angiographic image of the  $60 \times 48$  mm pseudoaneurysm sac.

to the development of systolic heart failure, ventricular pseudoaneurysm, and severe mitral regurgitation and was included on the transplant waiting list. Echocardiography revealed very low ejection fraction, restrictive type diastolic dysfunction, and the pseudoaneurysm sac. In addition, the inferoposterior region pseudoaneurysm sac caused mitral valve posteromedial papillary muscle structure distortion, and eccentric severe mitral regurgitation was detected (Figure 2A-B). CCTA was performed, and ventricular pseudoaneurysm formation of 85×60×90 mm size was detected (Figure 2C). The patient's symptoms of hypervolemia and heart failure were controlled with medical treatment, and she was discharged. The patient is still alive and waiting for cardiac transplantation.

### **Case report 3**

A 57-year-old male patient presented to our outpatient clinic with shortness of breath. His medical history was unremarkable, except for his current smoking. Echocardiography showed an ejection fraction of 45%, a 7-8 mm pericardial effusion in the lateral wall, and a  $50\times44$  mm pseudoaneurysm in the posterolateral myocardium. The pseudoaneurysm was observed to cause severe mitral regurgitation by damaging the posteromedial papillary muscle. A CCTA was performed, and the  $60\times48$  mm aneurysm in the posterolateral wall was confirmed (Figure 3A-B). Coronary angiography was performed, and it was observed that the thin calibrate obtuse marginal branch 2 was occluded and was filled by collateral vessels.

The patient was evaluated in the cardiology and cardiovascular surgery council, and surgery was recommended. As a result, the patient underwent pseudoaneurysm repair with a Teflon patch, mitral valve posterior leaflet retraction, papillary muscle reconstruction, 30-mm saddle rigid mitral annuloplasty ring implantation, and 32-mm three dimensional tricuspid annuloplasty ring implantation. The patient is now asymptomatic with a mild mitral regurgitation and an ejection fraction of 50%.

# DISCUSSION

Left ventricular pseudoaneurysms are rare entities that may occur after myocardial infarction. Enlargement of a pseudoaneurysm may lead to congestive heart failure by reducing forward ejection fractions and elevating end-diastolic pressure and ventricular volumes. The pseudoaneurysm sac in the posteroinferior region disrupts the mitral valve posteromedial papillary muscle structure and may cause eccentric severe mitral regurgitation.<sup>[5]</sup> The mitral valve has a complex anatomy, and optimum function depends on the synchronization of its components (i.e., the mitral valve leaflets, annulus, subvalvular apparatus, and left ventricle).<sup>[6]</sup>

There is no randomized clinical trial in the literature, but an analysis of cases of pseudoaneurysm has shown that untreated pseudoaneurysms have a 30% to 45% risk of rupture with medical therapy and mortality of almost 50% in one year.<sup>[7-9]</sup> Perioperative mortality is approximately 10% with existing techniques; surgery is thus the preferred therapeutic option.<sup>[10,11]</sup>

In the largest series of cases with pseudoaneurysm, in which 290 patients were examined by Frances et al.<sup>[2]</sup> in 1998, high mortality rates (23%) were observed in patients who underwent surgery and in patients who were medically treated (48%). Frances et al.<sup>[2]</sup> also found high mortality rates in patients who did not undergo surgery; however, their research also indicated prolonged survival in certain patients who were treated conservatively. Small-scale trials have demonstrated that asymptomatic patients with an incidental diagnosis of a small aneurysm (<3 cm) and patients inappropriate for surgery are manageable medically.<sup>[12]</sup> Komeda and David<sup>[10]</sup> reported the results of surgical repair in 12 patients with left ventricular pseudoaneurysm in a further review. In three patients, left ventricular posteroinferior pseudoaneurysm was accompanied by severe mitral insufficiency, and mitral valve replacement and aneurysmectomy were performed. Unfortunately, all the patients died after mitral valve replacement.

It is very difficult to estimate an overall mortality rate for left ventricular pseudoaneurysm and severe mitral regurgitation after myocardial infarction from case reports and case series; however, in the analysis of all the left ventricular pseudoaneurysms performed by Frances et al.,<sup>[2]</sup> it was 23%; 25% in the series of Komeda and David;<sup>[10]</sup> 28% in the series of Prêtre et al.;<sup>[12]</sup> and 7% in Yeo et al.'s<sup>[3]</sup> series. In the published case series, different mortality rates from 7% to 30% have been published. In the experience of Komeda and David,<sup>[10]</sup> mortality was not associated with technical difficulties in pseudoaneurysm repair but with impaired left ventricular function or the need to replace the mitral valve.<sup>[2,10,12]</sup> In addition, Natarajan et al.<sup>[13]</sup> reported in a review of 35 studies published between 1984 and 1993 (66 patients) on the management of pseudoaneurysm after myocardial infarction that medical treatment of chronic pseudoaneurysm (>three months) was not associated with an increased risk of cardiac rupture. The data in this study have not included patients who were symptomatic and had rapidly enlarging pseudoaneurysms operated on in the first three months. In addition, the main cause of mortality in chronic left ventricular pseudoaneurysms is the developing heart failure with cardiac rupture being reported in a very limited number of patients.<sup>[2,3]</sup>

On the basis of the information mentioned above, acute pseudoaneurysms that enlarge very quickly have a mortality rate with medical treatment. Early surgical treatment should be recommended in young patients with less comorbidities, even if severe mitral regurgitation and left ventricular systolic dysfunction have developed. Patients with pseudoaneurysm-related ventricular tachycardia recurrent embolism and patients with other reasons for cardiac intervention, such as coronary artery bypass surgery, should be recommended to have surgery.<sup>[13]</sup> However, small (<3 cm) chronic pseudoaneurysms are often discovered with non-specific clinical symptoms or incidentally and can be treated conservatively. In older patients with comorbidities, surgical treatment risks may mask the benefit, and medical treatment may be an appropriate approach.

Severe left ventricular systolic dysfunction and severe mitral regurgitation may make surgical treatment challenging and significantly increase mortality. However, depending on the enlargement of the pseudoaneurysm sac in the later stages of the disease, papillary muscle distortion will increase in correlation with the enlargement of the pseudoaneurysm sac and the probability and severity of mitral regurgitation will increase. Although cardiovascular surgeons avoid early surgery in patients with new-onset myocardial necrosis and in whom the scar tissue has not developed, early surgery should be encouraged once the patients are clinically stable.

Mitral valve repair should be the preferred surgical procedure in these patients. According to case reports, mortality rates were very high in patients who underwent mitral valve replacement; however, the results appeared to be better in patients who underwent mitral valve repair.<sup>[10,14]</sup>

Left ventricular pseudoaneurysm and severe mitral regurgitation are very rare and fatal clinical conditions. In symptomatic patients with few comorbidities, surgery should be recommended. In patients with concomitant mitral regurgitation, mitral valve repair should be the preferred surgical procedure. In asymptomatic patients, small (<3 cm) chronic pseudoaneurysms can be treated conservatively. Patients with high surgical complications owing to associated medical conditions can also be treated conservatively.

**Informed consent:** Informed consent was obtained from the patient for the publication of the case report and the accompanying images.

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