

Chronic Coronary Syndrome with Left Ventricular Apicoseptal Aneurysm

Yudie Tanta^{1*}, Taufik Indrajaya²

¹ Fellow of Cardiovascular Division of Internal Medicine Department, Faculty of Medicine, Universitas Sriwijaya/ Mohammad Hoesin General Hospital, Palembang, Indonesia

² Head of Cardiovascular Division of Internal Medicine Department, Faculty of Medicine, Universitas Sriwijaya/ Mohammad Hoesin General Hospital, Palembang, Indonesia

ARTICLE INFO

Keywords:

Left Ventricular Aneurysm
Anterior STEMI
Stable Angina Pectoris

Corresponding author:

Yudie Tanta

E-mail address:

tanta7an7a@yahoo.com

All authors have reviewed and approved the final version of the manuscript.

<https://doi.org/10.37275/JRP.v2i2.22>

ABSTRACT

The left ventricular aneurysm is one of the worst complications of acute myocardial infarction episodes. Fibrous tissue replacing dead myocardial cells in an infarcted area can't perform contraction and herniate outward during systole. In turn, a left ventricle aneurysm will present devastating clinical outcomes such as heart failure, ventricular arrhythmias, even sudden cardiac death. A 65 year-old man came to our hospital complaining of worsened chest pain 5 days before admission, located on the left side of his chest. It was described to have a squeezing character. The symptom was induced when he doing some mild activities and relieved with a short time rest. He had this symptom for one year. In the past, this symptom was induced by heavier activities. He also complains of having shortness of breath for the past 2 months, the symptom was also relieved by resting. He had been admitted to the hospital for a heart attack twice before. He has diabetes and hypertension, thus he consumed metformin 500 mg 3 times a day, and amlodipine 5 mg daily. He also had a history of kidney stone and prostate hypertrophy operation on March 2021. No remarkable abnormality was found in physical examination. On laboratory workup, there was a slight elevation of serum creatinine concentration. The troponin level was in the normal range. ECG and Echocardiography finding supported the ventricular aneurysm as one of the complications of acute myocardial infarction.

1. Introduction

The left ventricular aneurysm is one of the worst complications of acute myocardial infarction episodes. Fibrous tissue replacing dead myocardial cells in an infarcted area can't perform contraction and herniate outward during systole. In turn, a left ventricle aneurysm will present devastating clinical outcomes such as heart failure, ventricular arrhythmias, even sudden cardiac death. Left ventricular aneurysm's ECG pattern with ST-elevation and pathological Q wave potentially raised a false alarm for acute myocardial infarction management. Klein and colleagues propose criteria to differentiate left ventricular aneurysm ECG pattern and acute anterior myocardial infarction.^{1,2} Here we

presented a case of a middle-aged male with stable angina pectoris symptom and mid apical-antero-septal left ventricular aneurysm. The ECG tracing showed QS waves in v1 to v4 with slight domed ST elevation. Lesions found on LAD and LCx on coronary angiography. Angioplasty was done only in LCx lesions because there was only minimal benefit in performing angioplasty in chronic total occlusion LAD lesion. Conclusions LV aneurysm often presents as ST-elevation activating false alarm, while there is a minimal benefit on doing reperfusion in this area because it consists mainly of fibrotic tissue.

2. Case Presentation

A 65 years old man came to ER complaining of worsened chest pain 5 days before admission. The chest pain is located on the left side of the patient's chest. It was described to have a squeezing character. The symptom was induced when the patient doing some mild activities and relieved with a short time rest. The patient has already had this symptom for one year. But in the past, this symptom was induced by heavier activities. The patient also complains of having shortness of breath for the past 2 months, the symptom was also relieved by resting.

The patient had been admitted to the hospital for a heart attack twice before. At the time, the patient refused to undergo coronary angiography. The patient has diabetes and hypertension, which he consumed metformin 500 mg 3 times a day, and

amlodipine 5 mg daily. The patient also had a history of kidney stone and prostate hypertrophy operation on March 2021.

The patient was in stable condition and fully conscious, blood pressure 140/80, heart rate 80x/minute regular, respiratory rate 20x/minute, temperature 36,5 C. No remarkable abnormality was found in physical examination.

On laboratory workup, there was a slight elevation of serum creatinine concentration 1,52 mg/dl. The troponin level was in the normal range. On ECG recording there are QS waves with domed slight ST elevation in v1-v4. Further analysis showed the ratio between the summation of T amplitude (V1 to V4) divided by the summation of QRS amplitude (V1- to V4) is less than 0,22, suggesting the ST elevation is caused by LV aneurysm rather than STEMI.

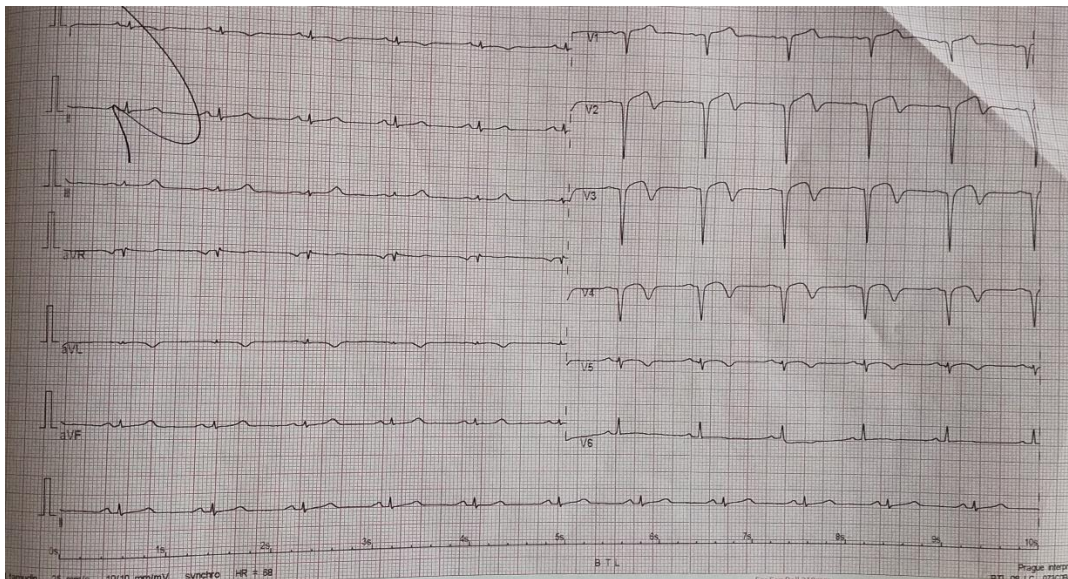


Figure 1. ECG recording showed QS pattern with slight domed ST elevation from v1 to v4 and inverted T in v5,v6, avL suggesting anterior infarction with lateral ischemia.

Thoracic echocardiography examination showed 29,1% ejection fraction with LV aneurysm in apico septal and apical cap region. Segmental wall motion

abnormality was also found in the mid anterior region which appears akinetic.



Figure 2. Echocardiography showed 29,1% EF, apicoseptalventricular aneurysm, and akinetic mid-anteroseptal segment.

Patient was treated with candesartan 1x8mg, gliquidone 2x15 mg, clopidogrel 1x75mg, atorvastatin 1x 20 mg, and spironolactone 1x25 mg. Coronary angiography was then performed.

Concerning the slight increase of creatinine level, the non-iodine low osmolar contrast was chosen for the procedure. The maximum volume of contrast was not to exceed 3 ml/kg BW. The patient has a bodyweight of 65 kg, so the maximum contrast volume for the patient was around 200 cc.³

Corangiography study showed ostial chronic total occlusion lesion in Left Anterior Descending (LAD) coronary artery gaining collateral perfusion from Right Coronary Artery's (RCA)left posterior

descending branch. There was also 80% stenotic lesion in proximal and distal Left Circumflex (LCx) Coronary Artery.

Angioplasty was not performed in LAD because the high-risk characteristic of the lesion and the area it perfuses already showed aneurysm indicating minimal benefit gained from reperfusion. It was decided to perform angioplasty in LCx lesions.

After predilation with 1.0 and 2.0 diameter balloon, the diameter of the distal LCx lesion were still less than 2,5 mm. A lesion which diameters under 2,5 mm is relatively contraindicated for the stent implantation procedure. It was decided to perform *stenting* only in proximal LCx lesions.

3. Discussion

A ventricular aneurysm is defined as thinning of some ventricle wall segments, which has a wide neck and protrudes during systole and diastole. Generally, the incidence of the ventricular aneurysm was around 12% of the total acute myocardial infarction incidence. *Single vessel disease*, proximal LAD stenosis, higher end-diastolic pressure were found to be a risk factor for the development of left ventricle aneurysm after acute myocardial infarction. ⁴

The ventricular aneurysm is related to a significant increase in mortality compared with the absence of it in acute myocardial infarction. Thrombus formation in aneurysm cavities increased the embolic incidence. Besides, a ventricular aneurysm can cause an extensive remodeling process despite optimal medical therapy, leading to progressive heart failure. ^{4,5}

In the left ventricular aneurysm, the force created by the healthy myocardium was not simultaneously balanced by the opposite myocardium that undergone infarction. This, in turn, caused increased wall stress in the infarcted area. Moreover, the intraventricular dyssynchrony caused a reduction in stroke volume. Clinically, the process will manifest as a symptom of heart failure such as *dyspnea on effort*. In this patient, the process caused symptoms of shortness of breath during activity.⁶

Classically, a left ventricular aneurysm should be suspected from ECG recording if persistent ST elevation is found weeks after acute myocardial infarction event. Thus, potentially left ventricular aneurysm caused *false alarm* for an acute condition such as STEMI where reperfusion should be performed immediately.⁷

Lead	T	QRS	Ratio
V1	2mm	6mm	0,33
V2	3mm	17mm	0,17
V3	2,5mm	18mm	0,13
V4	1,5mm	10mm	0,15

Figure 3. T/QRS ratio in v1 to v4

Smith proposed criteria to distinguish between acute anterior myocardial infarction and apical left ventricular aneurysm. In Smith criteria, it is said that if the summation of T amplitudes from V1 to V4 divided by the summation of QRS amplitudes from V1 to V4 (T_{V1-V4}/QRS_{V1-V4} ratio) were more than 0,22 then the finding suggests toward anterior

STEMI⁸. Klein then perfected Smith criteria with additional criteria, which said if the T/QRS ratio from one of the leads. v1 to v4 is more than 0,36 then the finding also suggests toward anterior STEMI. ²

The T_{v1-v4}/QRS_{v1-v4} ratio from the patient is 0,17. While the biggest T/QRS ratio was in v1 which is 0,33. Thus, the ECG tracing of this patient

suggests a left ventricle aneurysm rather than anterior STEMI.

On echocardiography, the left ventricle aneurysm is recognized from thinning of myocardial wall segments forming a protrusion with a wide neck, which has a dyskinetic movement on systole. The dyskinetic movement itself is defined as the paradoxical movement of the myocardial wall segment, where the segment moves outward during systole.^{9,10}

It is important to distinguish between a true ventricular aneurysm and pseudoaneurysm. Pseudoaneurysm is a ruptured ventricular wall compensated with thrombus formation and pericardial inflammation, thus preventing

tamponade. The character of pseudoaneurysm from echocardiography is a very localized protruding myocardial segment with a narrow neck. Pseudoaneurysm is a critical condition that needs immediate surgical correction.⁹

Echocardiography in this patient showed a wide neck protruding segment with dyskinetic movement in apical caps and apico-septal area. The aneurysm area was demonstrated on Parasternal Long Axis, Apical 4 Chamber, and Apical 3 chamber views. The oxygen demands of the aneurysm region in this patient were supposedly supplied from the Left Anterior Descending artery which was occluded.¹¹



Figure 4. Angiographic study showed ostial CTO in LAD with collateral from posterior descending branch of RCA, 85% stenotic lesion in proximal LCx and bifurcation lesion in 2nd Obtuse Marginal branch with 70% stenosis.

Three lesions were found in this patient from coronary angiography. The first lesion Total Occlusion (CTO) in LAD. CTO lesions were classified as type C lesions from ACC-AHA lesion classification.

Type C lesion has a lower success rate (61%) compared to other types of lesions with a higher incidence of procedural complication (21%) should intervention performed.

The decision for performing angioplasty in this lesion type should undergo a very thorough consideration of its risk and benefit. In this case, since the region supplied by LAD already progressed to an aneurysm that majorly consisted of fibrotic tissue, there is almost no benefit obtained from performing angioplasty. It is decided not to perform

angioplasty in LAD.¹²

Two lesions were found in the Left Circumflex artery. The proximal LCx lesion has an 85% narrowing, with discrete (less than 5mm), eccentric, non-angulated, and non-calcified characteristics. This lesion can be classified as a type A lesion that has the highest success rate and minimal complication incidence (92% success rate and < 2% complication incidence). The distal lesion in 2nd obtuse marginal branch has a 70% narrowing. It was a diffuse (more than 20 mm), bifurcation lesion (medina 0,0,1). This lesion can be classified as a type B lesion with a 76% success rate and less than 10% complication incidence. It was decided to perform angioplasty in these two lesions.¹²

PCI on the 2nd OM lesion begins with crossing double wire to the main vessel and the significant *side branch*. Only percutaneous balloon angioplasty (POBA) performed in this lesion since optimal dilatation for stent crossing was not obtained in this location.¹³ PCI on the proximal LCx can be executed well. After pre dilatation with a 2.0 diameter balloon, stenting was performed with 2,25/12mm *Drug-Eluting Stent* expanded to 2,75 mm. A TIMI 3 flow without residual stenosis on proximal lesion was obtained.¹²

4. Conclusion

The ventricular aneurysm is one of the complications of acute myocardial infarction. The ECG pattern of the ventricular aneurysm with ST-elevation and pathological Q waves often caused false alarm in STEMI emergency response system. Although there is little benefit in performing revascularization in the aneurysm area, medical, surgical, or interventional treatment of ventricular aneurysm might prevent further progression of the left ventricle remodeling process.

Although there is almost no goal from revascularizing aneurysm area, the area that surrounds it might still gain benefit from revascularization. Thus, the assessment of myocardial viability using single photon emission computed tomography, or dobutamine stress echocardiography, should be considered. Guo and colleagues in their study found that, although there is no difference of cardiac death prevalence between CTO patients receiving optimal medical therapy only and CTO patients who underwent revascularization, there are significant reduction of Major Adverse Cardiovascular Event (MACE) and Target Vessel Revascularization (TVR) in patients who underwent CTO revascularization.^{14,15}

5. References

1. Sattar Y, Alraies MC. Ventricular Aneurysm. In: StatPearl (Internet). Treasure Island (FL): StatPearls Publishing; 2021.

2. Klein LR, et al. Electrocardiographic criteria to differentiate acute anterior ST-elevation myocardial infarction from left ventricular aneurysm. *Am J Emerg Med.* 2015; 33 (6): 786-90.
3. Kern JM. Introduction to Catheterization Laboratory. In: Kern JM. *The Cardiac Catheterization Handbook* 5th ed. Elsevier Saunders Publishing; 2011. P16-17.
4. Yazdani SK, Ladich E, Virmani R. Pathology of Myocardial Ischemia, Infarction, Reperfusion, and Sudden Death. In : Fuster V, Walsh RA, Harrington RA. *Hurst's The Heart* 13th ed. The McGraw Hill Companies; 2011. P 1310-1311.
5. Schmidt T, Frerker C, Thielsen T, et al. New evidence for favourable effects on haemodynamics and ventricular performance after Parachute implantation in humans. *Eur. J. Heart Fail.* 2014; 16: 1112-1119.
6. Galli A, Lombardi F. Postinfarct Left Ventricular Remodelling: A Prevailing Cause of Heart Failure. *Cardiol Res Pract.* 2016;2016:2579832. doi: 10.1155/2016/2579832. Epub 2016 Feb 18. PMID: 26989555; PMCID: PMC4775793.
7. Goldberger AL, Goldberger ZD, Shvilkin A. *Goldberger's Clinical Electrocardiography A Simplified Approach* 8th ed. Elsevier Saunders Publishing; 2013. P82.
8. Smith SW. T/QRS ratio best distinguishes ventricular aneurysm from anterior myocardial infarction. *Am J Emerg Med.* 2005. 23.(3):279-87.
9. DeMaria AN, Blanchard DG. Echocardiography. In: Fuster V, Walsh RA, Harrington RA. *Hurst's The Heart* 13th ed. The McGraw Hill Companies; 2011. P 458-459.

10. Ryding A, Newton J, Zielke S. Essential Echocardiography 2nd ed. Elsevier Saunders Publishing; 2013. P 41.
11. Connolly HM, Oh JK. Echocardiography. In Bonow R, Mann DL, Zipes DP, Libby P. Braunwald's Heart Disease 9th ed. Elsevier Saunders Publishing; 2012. P 224.
12. Kern MJ. The Basics of Percutaneous Coronary Interventions. In Kern MJ, Sorajja P, Lim MJ. The Interventional Cardiac Catheterization Handbook 4th ed. Elsevier Publishing; 2018. P40-42.
13. Brikalis ES, et al. Treatment of Coronary Bifurcations. In Kern MJ, Sorajja P, Lim MJ. The Interventional Cardiac Catheterization Handbook 4th ed. Elsevier Publishing; 2018. P200-203.
14. Wiefels, C., Beanlands, R.S.B., and Chong, A.Y. Imaging in CTO : Should you look before you open? J Nucl Cardiol. 2021. 28, 2609–2612.
15. Guo L, Wu J, Zhong L, Ding H, Xu J, Zhou X, Huang R. Two-year clinical outcomes of medical therapy vs. revascularization for patients with coronary chronic total occlusion. Hellenic J Cardiol. 2020. 61(4):264-271.