




Case Report

Conservative Management of Left Ventricular Thrombus Patient with Decreased Systolic Function: A Case ReportI Kadek Herry Hermawan^{1*} , Ken Christian Kawilarang¹ , Febriyanti Hartono²¹Intern General Practitioner, dr. Ramelan Navy Hospital, Surabaya, Indonesia.²Cardiologist, dr. Ramelan Navy Hospital, Surabaya, Indonesia.

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ABSTRACT

Case Summary. A 35-year-old male complained shortness of breath for 1 month, had worsened in the last 3 days. When walking 3-5 steps, the patient already felt dyspneu. When sleeping, the patient must be in a semi-sitting position, and sometimes still feels tight. The patient also complained of occasional chest pain. The patient is known to have a history of heart disease, and a history of taking drugs such as candesartan, ISDN and bisoprolol.

Discussion. Left ventricular thrombus (LVT) is a frequent and potentially dangerous complication of acute myocardial infarction and is associated with increased risk of systemic embolization. Incidence of LVT following acute MI has decreased, probably due to improvement in patient care as a result of better and earlier reperfusion techniques. Management of LVT is primarily based on studies before the widespread use of potent pharmacological and interventional therapies such as primary percutaneous coronary intervention, especially in the setting of acute myocardial infarction. Though advances in diagnostic technology have improved detection of LVT, clinicians face several uncertainties in the management of LVT in daily practice.

Highlights:

1. It provides insight into the management of LVT.

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Introduction

With the improvement of interventional therapy and pharmacotherapy, patients with cardiovascular diseases live longer despite suffering from myocardial disease. Left ventricular thrombosis (LVT) can complicate left ventricular (LV) systolic dysfunction in ischemic and non-ischemic cardiomyopathy and can lead to thromboembolic. There are, however, several uncertainties in the management of LVT. Most studies on treatment of LVT occurred before the era of rapid percutaneous coronary intervention (PCI) and without concomitant use of potent dual anti-platelet.

Case Presentation

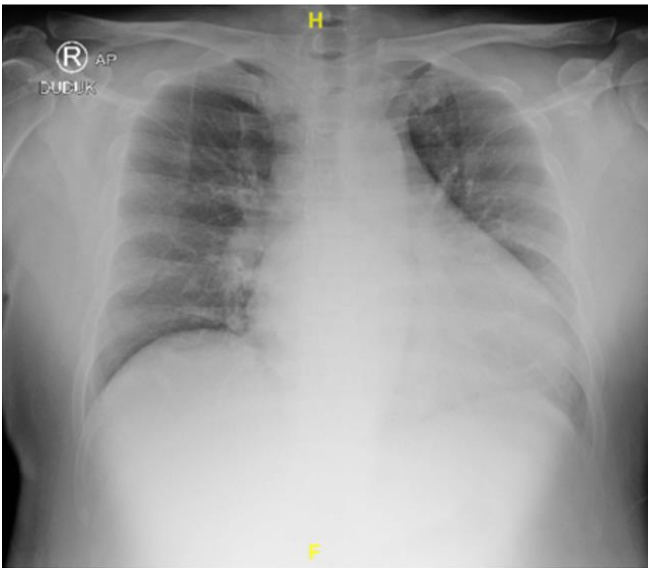


Figure 1. Chest X-ray

A 35-year-old male complained shortness of breath for 1 month, had worsened in the last 3 days. When

walking 3-5 steps, the patient already felt dyspneu. When sleeping, the patient must be in a semi-sitting position, and sometimes still feels tight. The patient also complained of occasional chest pain. The patient is known to have a history of heart disease, and a history of taking drugs such as candesartan, ISDN and bisoprolol.

The patient came in a conscious state of compost mentis, vital signs were monitored blood pressure 135/98 mmHg, pulse 99 beats/minute, temperature 36.9°C, respiratory rate 24 breaths/minute. Physical examination revealed pitting edema in both lower extremities. In laboratory tests, there was a slight increase in leukocytes, and an increase in SGOT & SGPT levels. Chest X-ray examination revealed cardiomegaly (figure 1), and electrocardiography (ECG) was sinus 78 bpm, left axis deviation (LAD), small R wave in V3-V6 (figure 2). Transthoracic echocardiography (TTE) was performed and found ejection fraction (EF 36% biplane), mild mitral regurgitation (MR), LA (left atrium) and LV (left ventricle) dilatation, decreased LV diastolic function grade III, LV eccentric hypertrophy, thrombus at apical wall LV (2.0-2.4 x 2.5-3.3 cm), akinetic anterior (M-A), Lateral (M-A), hypokinetic septal (A), anteroseptal (B-M), inferoseptal (B-M) (figure 3).

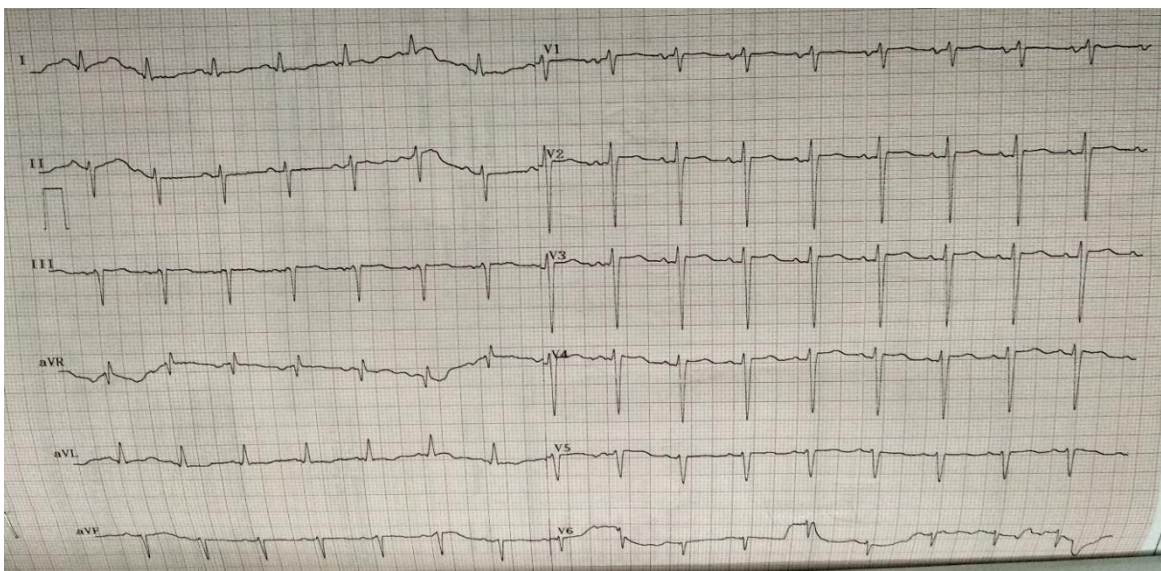


Figure 2. Electrocardiography

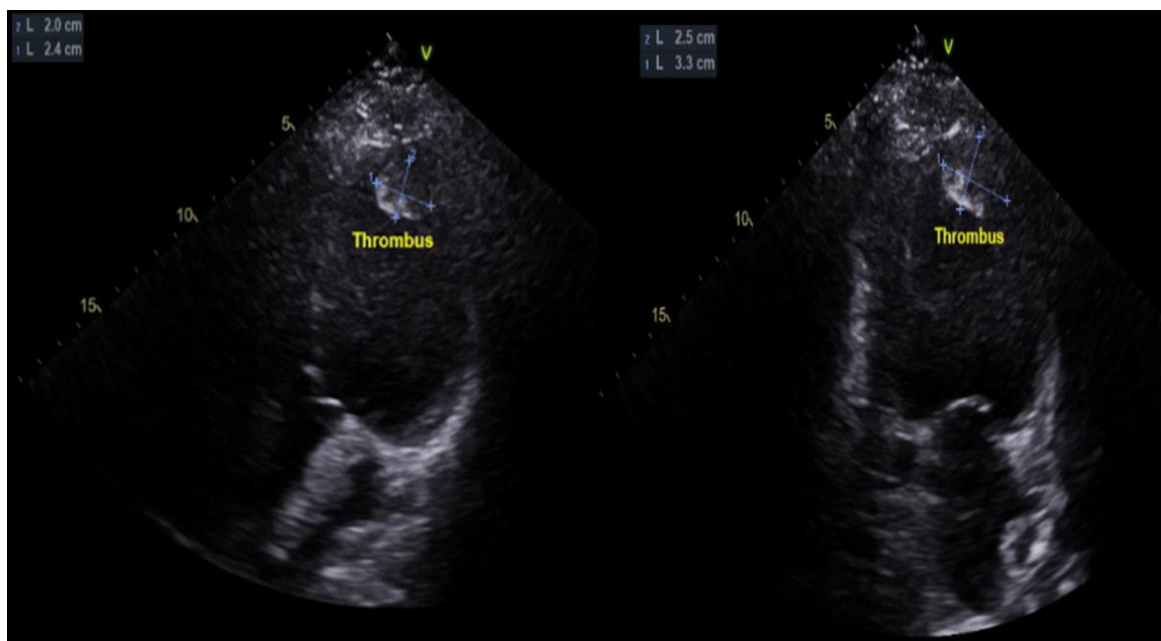


Figure 3. Transthoracic Echocardiography (TTE)

The conclusion of the TTE examination was ischemic cardiomyopathy and left ventricular thrombus. Subsequently, patient consulted to a cardiothoracic surgeon regarding left ventricular thrombus and the answer was not to perform surgery

(surgical thromboectomy) but conservative therapy with anticoagulants. The patient then received therapy anticoagulant warfarin 2 mg related to his LVT.

Discussion

In most cases, left ventricular thrombus is associated with acute myocardial infarction. However, with the growing use of percutaneous coronary intervention (PCI), the incidence of left ventricular thrombus after myocardial infarction has decreased (figure 4)^[1]. The overall prevalence of left ventricular thrombus in the general population is low. In a retrospective review of more than 80,000 medical records, the incidence of left ventricular thrombus was 7 per 10,000 patients. Eighty percent of these cases were related to ischemia, while the remainder were due to dilated cardiomyopathy (DCM) and stress-induced cardiomyopathy (8.15% and 4.8% respectively).^[2,4]

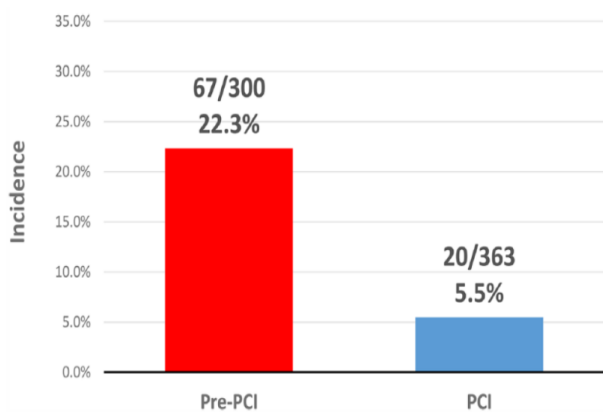


Figure 4. Cumulative incidence of LVT in studies reported in the pre-PCI era compared to PCI era (before and after 1995)

The presence of a left ventricular thrombus has the potential to cause thromboembolic complications, such as stroke. Thrombus formation reflects the presence of factors that represent Virchow's Triad in the ventricles, namely reduced wall motion, local

heart muscle (myocardial) injury, and hypercoagulability/blood flow stasis (Fig. 5).

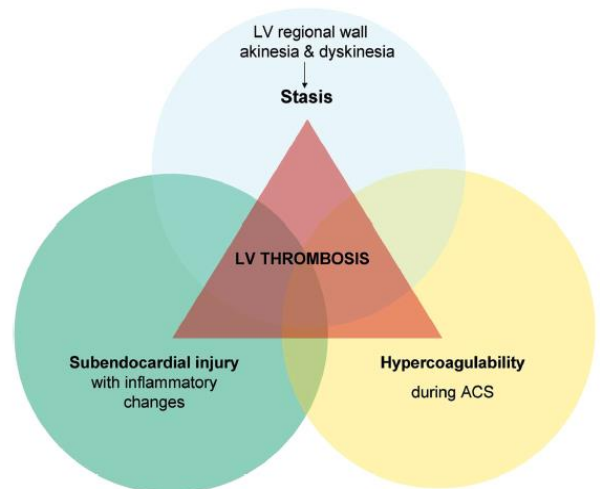


Figure 5. The three components of the Virchow's triad in left ventricular thrombus formation

Akinesia and regional wall dyskinesia of the left ventricle cause stasis of blood flow, often seen on two-dimensional echocardiography. Prolonged ischemia causes subendocardial tissue injury with inflammatory changes. Patients with acute coronary syndromes display a hypercoagulable state characterized by increased concentrations of prothrombin, fibrinopeptide A, von Willebrand factor, and decreased concentrations of the enzyme that breaks down von Willebrand factor (ADAMTS13).^[3]

The use of vitamin K antagonists (VKA) based on European and US guidelines is recommended as class IIa, treatment of left ventricular thrombus with VKA for a minimum of 3 to 6 months, with duration adjusted for individual patient's bleeding risk with

target international normalized ratio (INR) 2.5 (range 2-3). Re-imaging is recommended at the end of the treatment period to evaluate thrombus resolution as class IIa. There was a significant reduction in the risk of major cardiovascular events with anticoagulation for more than 3 months (48% decrease) and LVEF 35% (54% decrease). Resolution of left ventricular thrombus was achieved in patients receiving VKA and receiving additional dual antiplatelet therapy (DAPT).^[5,6]

Anticoagulant therapy is used to reduce the complications of left ventricular thrombus embolism while also improving cardiac function and innate

fibrinolytic mechanisms. Vitamin K antagonists such as warfarin have been used and their efficacy and safety have been evaluated in non-randomized studies. Table 1 summarizes the recommendations of the three expert associations in the treatment of left ventricular thrombus. While the ACCF/AHA guidelines describe the duration of anticoagulation warfarin as being up to 3 months, the ACCP guidelines divide patients with stented left ventricular thrombus in patients. ACCP guidelines divide patients with left ventricular thrombus in the presence or absence of a cardiac coronary stent and suggest 3 months of anticoagulation.^[7,8]

Table 1. Summary of recommendations of the American College of Cardiology (ACCF/AHA), American College of Chest Physicians (ACCP) and European College of Cardiology (ESC) for treatment of left ventricular thrombus (LVT)

ACCF/AHA 2013	Anticoagulation with warfarin is reasonable for patients with ST-elevation myocardial infarction (STEMI) and asymptomatic LV mural thrombi (class IIa, LOE C). Duration can be limited to 3 months in patients with or at risk for LVT*, whereas continuation of dual anti-platelet therapy (DAPT) as per ACS recommendations.
ACCP 2012	Patients with anterior myocardial infarction (MI) and LVT, or at high risk for LVT*: No stenting : warfarin plus low-dose aspirin 75–100 mg daily for the first 3 months over single anti-platelet therapy or DAPT (Grade 1B). Thereafter, recommend discontinuation of warfarin and continuation of anti-platelet therapy per the ACS recommendations. Bare-metal stent (BMS) placement : Triple therapy# for 1 month over DAPT; warfarin and single anti-platelet therapy for the second and third month; discontinuation of warfarin thereafter and resumption of DAPT for up to 12 months (Grade 2C). Drug-eluting stent (DES) placement : Triple therapy# for 3–6 months (Grade 2C); discontinue warfarin thereafter and continuation of DAPT for up to 12 months.
ESC 2012	Anticoagulation should be considered in patients with large anterior wall motion abnormalities, if they are at low risk of bleeding, to prevent the development of thrombi. Definite thrombus : Oral anticoagulant therapy with warfarin for up to 6 months. However, optimal duration of triple antithrombotic therapy is unknown and should take into account the relative risks of bleeding and stent thrombosis. Repeated imaging of the left ventricle after 3 months – consider discontinuation of anticoagulation earlier than 6 months, if no evidence of thrombus, particularly if there is recovery of apical wall motion.
(*)High risk for LVT is defined as patients with ejection fraction less than 40%, anteroapical wall motion abnormality. (#)Triple therapy includes warfarin (INR target 2–3), low-dose aspirin (75–100 mg) and clopidogrel 75 mg.	

In patients who are not stented, anticoagulant warfarin plus low-dose aspirin 75 mg-100 mg can be

given for the first 3 months, then it is recommended to discontinue warfarin and continue antiplatelet

therapy according to acute coronary syndrome guidelines. Meanwhile, in patients who had stenting, they received triple therapy, namely warfarin, low-dose aspirin 75 mg-100 mg and clopidogrel 75 mg for 1 month (patients received bare-metal stents) or for 3-6 months (patients received drug eluting stents). Then warfarin was discontinued and DAPT continued for up to 12 months^[7,8]. The ESC guidelines stipulate 6 months of anticoagulation warfarin. Re-imaging after 3 months and consider discontinuing anticoagulation earlier than 6 months, if there is no evidence of a thrombus, especially if there is restoration of apical wall motion.^[8]

Several case reports and one retrospective center reported similar efficacy between the use of vitamin K antagonist (VKA) and DOAC (Direct Oral Anticoagulant) therapy in left ventricular thrombus. There were several patients with persistent left ventricular thrombus who were treated with DOAC, and resolution occurred when switching to VKA with a higher target INR of 3-4. On the other hand, a retrospective multicenter cohort study showed that treatment with DOAC (apixaban, rivaroxaban, dabigatran) increased the risk of stroke and systemic embolism.^[9]

Surgical thrombectomy is an option for patients at high risk of embolism who are also planning another open heart surgery. The high morbidity and mortality of this operation outweigh the benefits of performing

surgery solely for the indication of a left ventricular thrombus.^[9]

Conclusion

The clinical prognosis of patients with left ventricular thrombus is poor with a very high risk of major cardiovascular morbidity and mortality. Improvements in antithrombotic management are needed to improve the clinical outcomes of these relatively young patients. Left ventricular thrombus is associated with a higher risk of cardiovascular events and death, and evidence suggests anticoagulation therapy for at least 3 months reduces this risk. Left ventricular thrombus should be considered as a marker of increased long-term thrombotic risk that may persist even after thrombus resolution.

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