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Left Ventricular Thrombus in Old Myocardial Infarction

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ABSTRACT

Background. Left ventricular thrombus is a major risk factor for systemic thromboembolism, a complication of acute and chronic myocardial infarction. The high incidence of left ventricular thrombus in untreated acute myocardial infarction patients can be described in the pre-thrombolytic era. Left ventricular thrombus occurred in 7-46% of cases, especially in anterior or apical myocardial infarction. **Case presentation:** A 57-year-old man is reported with complaints of shortness of breath and a history of chest pain typical of angina, hypertension, diabetes mellitus, and smoking. On electrocardiographic examination, infrequent premature ventricular contraction and anterior extensive old myocardial infarction were found. Transthoracic echocardiography examination revealed protuberant thrombi at the apex of the left ventricle with a size of 14 x 15 mm, left ventricular ejection fraction of 20%, and akinetic at extensive anterior, anterior, anterolateral, broad apical, inferoseptal, and inferior segments. **Conclusion:** Untreated acute myocardial infarction has a high risk of developing left ventricular thrombus. Intravenous unfractional heparin and VKA in left ventricular thrombus patients with an increased risk of embolism respond well.

1. Introduction

Left ventricular thrombus is a major risk factor for systemic thromboembolism, a complication of acute and chronic myocardial infarction. The study by Velangi et al. (2019) showed that 3.7% of patients with left ventricular thrombus experienced systemic embolism during a 3-year follow-up in stroke infarction, transitory ischemic attack, and extracranial systemic arterial embolism.[1,2]

Left ventricular thrombus generally occurs 3 months after acute myocardial infarction. The risk for left ventricular thrombus has consistently been associated with infarct therapy, infarct size, severe infarct asynchrony (dyskinesis, akinesis), left ventricular aneurysm, and anterior myocardial infarction.[1,3]

The combination of blood stasis, endothelial damage, and hypercoagulability (Virchow's triad) are factors that predispose to thrombus formation. Akinesia and dyskinesia regional wall of the left ventricle after acute myocardial infarction will lead to blood stasis. Prolonged ischemia will cause subendocardial tissue injury accompanied by inflammation. Patients with the acute coronary syndrome are generally in a state of hypercoagulability, such as increased concentrations of prothrombin, von Willebrand factor, and decreased concentrations of ADAMTS12. Virchow's triad together causes the formation of a left ventricular thrombus consisting of fibrin, erythrocytes, and platelets.[1,3]

Transthoracic echocardiography is a widely used examination to assess the presence, shape, and size of a left ventricular thrombus. A left ventricular thrombus on TTE is defined as a discrete echo dense mass with clear boundaries to the endocardium during systolic

and diastolic phases. The left ventricular thrombus is located adjacent to the hypokinetic/akinetic wall. Thrombus is classified into protuberant thrombi that protrude into the left ventricular cavity and mural thrombi which are flat masses. Protuberant thrombi are mobile masses that are associated with a higher risk of ischemic stroke.[1,3]

Several studies have demonstrated the use of left ventricular thrombus biomarkers by postulating that factors involved in the coagulation cascade can be biomarkers to identify patients at increased risk of LV thrombus. Data from the European Society of Cardiology (ESC) show high concentrations of soluble tissue factor and d-dimer in the left ventricular thrombus.[1,3]

The efficacy of anticoagulant therapy is proven both as prevention of left ventricular thrombus formation and as a therapy. Management recommendations for left ventricular thrombus from the American College of Chest Physicians (ACCP), American Heart Association (AHA), and ESC recommend oral anticoagulants and vitamin K antagonists (VKA) as first-line. The basis of these guidelines is the study of Turpie et al. (1989) which emphasized the efficacy of parenteral heparin, and the meta-analysis of Vaitkus et al. (1993), which demonstrated that anticoagulants and VKA were effective in reducing the risk of left ventricular thrombus formation and embolism. Oral VKA therapy is more effective and practical than anticoagulants, so long-term parenteral anticoagulant therapy is not recommended. Parenteral anticoagulation should be discontinued when an effective therapeutic dose of warfarin has been reached (INR 2 - 3). To assess thrombus resolution, a repeat TTE after 3 months is recommended. Anticoagulants should be discontinued immediately after the resolution of the thrombus.[1,3–5]

2. Case Presentation

A man came with complaints of shortness of breath that had been felt since 2 months ago with paroxysmal nocturnal dyspnoea and dyspnoea on the effort. There was a history of chest pain typical of angina 3 months ago, but the patient did not seek treatment. The patient has a history of hypertension, diabetes mellitus, and smoking.

On physical examination, there was an increase in jugular venous pressure, cardiomegaly, and grade III systolic murmur with a maximum punctum at the apex radiating to the left axilla and pitting edema in the extremities. Infrequent premature ventricular contractions and extensive anterior OMI were found on the ECG (Figure 1).

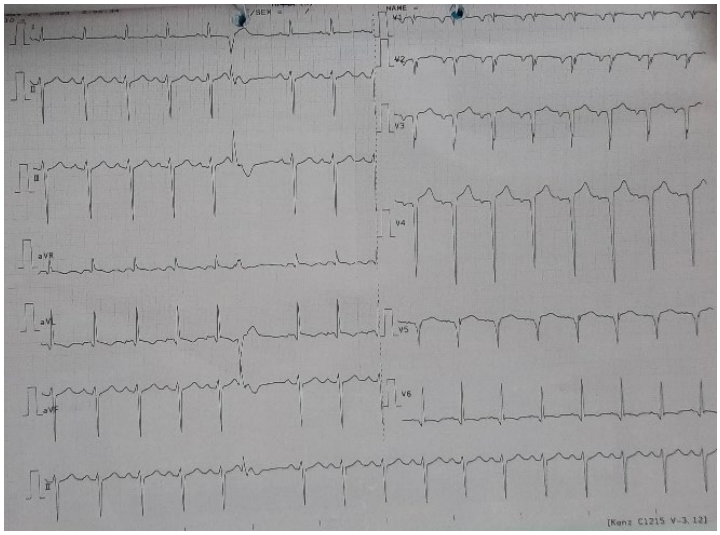

Figure 1. Electrocardiography with infrequent PVC and extensive anterior OMI

On laboratory examination, platelets were 181,000/mm3, HbA1C 7.8%, PT 11.4 seconds, aPTT 30.4 seconds, INR 0.56, d-dimer 2002 ng/ml, total cholesterol 174 mg/dl, HDL 33 mg /dl, LDL 117 mg/dl, and triglycerides 118 mg/dl. On TTE examination found spontaneous echo contrast in the left ventricle, thrombus at the apex of the left ventricle with a size of 14 x 15 mm, a moderate degree of mitral regurgitation, left ventricular eccentric hypertrophy, and decreased global systolic function with an LVEF of 20%, extensive anterior segment akinetic, anterior, anterolateral, broad apical, inferoseptal, and inferior (Figure 2).

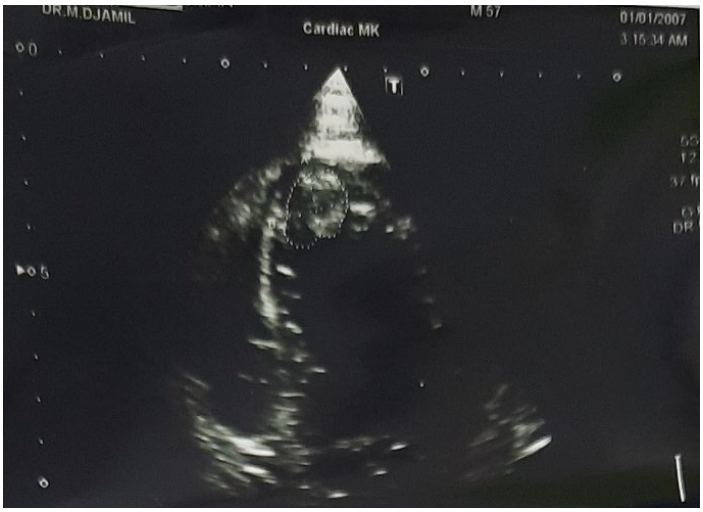

Figure 2. Transthoracic echocardiography with protuberant thrombi at the apex of the left ventricle

The patient was diagnosed with heart failure with reduced ejection fraction (HFrEF) NYHA functional class III stage D with left ventricular thrombus, extensive OMI, infrequent PVC, type 2 diabetes mellitus, hypertension, and dyslipidemia.

For treatment of left ventricular thrombus, the patient was given a 5000 IU bolus of unfractional heparin (UFH) followed by continuous drip of heparin and warfarin 1 x 2 mg with PT/APTT/INR monitoring. On day 3 of heparinization, the patient's ECG showed no PVC, platelets 314,000/mm^3, PT 10.3 seconds, aPTT 52 seconds, INR 1.14, and complete resolution of left ventricular thrombus on TTE. The patient was discharged with warfarin dose increased to 1 x 4 mg.

3. Discussion

We report a 57-year-old male patient with HFrEF NYHA functional class III stage D with left ventricular thrombus, extensive OMI, infrequent PVCs, type 2 diabetes mellitus, hypertension, and dyslipidemia.

Heart failure with reduced ejection fraction in patients was directly related to the presence of untreated acute myocardial infarction based on a history of chest pain typical of angina, risk factors for diabetes mellitus, hypertension, smoking, and dyslipidemia with a Framingham score of 22 (10 years risk of cardiovascular disease >30%), extensive anterior OMI on ECG, and 20% LVEF, extensive anterior, anterior, anterolateral, broad apical, inferoseptal, and inferior segments of left ventricle akinetic on TTE. This is based on the pathophysiology of HFrEF associated with cardiomyocyte death, while diabetes, hypertension, smoking are associated with heart failure with preserved ejection fraction through systemic inflammation.[6]

Patients have a high risk of left ventricular thrombus due to the absence of treatment for acute myocardial infarction, extensive infarction involving the anterior region, and LVEF of 40%. The high incidence of left ventricular thrombus in patients with acute myocardial infarction can be described in the pre-thrombolytic era, where left ventricular thrombus

occurred in 7-46% of cases, especially in anterior or apical MCI. The current incidence is much lower with more aggressive therapy. Research by Solheim et al. (2010) and Mollet et al. (2002) found left ventricular thrombus in 5.4% and 7.1% of acute anterior myocardial infarctions that had primary percutaneous intervention (PCI). In the study of Chiarella et al (1998), anterior MCI had a higher incidence than other regions (11.5% vs. 2.3%, p<0.001) and was higher in LVEF <40% (10.5% vs. 4%, p <0.001). The incidence of left ventricular thrombus in patients with acute anterior myocardial infarction and LVEF 40% was 17.8%.[1,7–9]

Heart failure with reduced ejection fraction in these patients also contributes to the development of left ventricular thrombus. Heart failure is associated with a prothrombotic state and hypercoagulability, associated with hemostatic markers and platelet function. It is associated with decreased platelet lifespan, increased platelet volume, increased platelet reactivity and aggregation, high circulating fibrinogen levels, and increased d-dimer and von Willebrand factor. The patient found an increase in d-dimer (2002 ng/ml).[3]

The diagnosis of left ventricular thrombus was made based on the findings of a thrombus at the apex, which is an akinetic segment with a size of 14 x 15 mm. Transthoracic echocardiography has a sensitivity of 85 - 90% and a sensitivity of 95% compared to cardiac magnetic resonance (CMR), which is the gold standard for diagnosing left ventricular thrombus. Left ventricular thrombus in patients with a high risk of embolism, namely myocardial infarction in the anterior-apex region, extensive infarction with LVEF <40%, and protuberant thrombi. Premature ventricular contractions in patients caused by scarring of the myocardium after myocardial infarction also increase the risk of embolism, which in Agarwal et al. (2010) study found that patients with PVCs had a higher tendency to have a stroke (hazard ratio 1.72 (1, 14 - 2.59)). Therefore, although various clinical guidelines suggest the use of oral anticoagulants and VKA as first-line therapy, these patients were treated with high-dose intravenous UFH according to ESC 2021, where UFH is considered in large, mobile thrombi with a target aPTT of 60 seconds (50 - 70 seconds).[1,3,10,11]

In these patients, administration of UFH and intravenous warfarin showed significant results. On repeat TTE after 3 days of therapy, no left ventricular thrombus was found. Complete resolution of this thrombus was faster than in the study conducted by Heik et al. (1994) in which 19 of 23 patients left ventricular thrombus disappeared after 14 ± 4 days of UFH. Administration of VKA was continued for up to 3 months with dose adjustments until the target INR (2 - 3) and TTE were repeated at the end of therapy.[3,12]

4. Conclusion

Untreated acute myocardial infarction is at high risk for left ventricular thrombus. Intravenous unfractional heparin and VKA in left ventricular thrombus patients at high risk of embolism respond well.

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