INTRODUCTION

Thromboembolic events are complications that are feared related to left ventricular apical thrombus formation. Left ventricular apical thrombus formation is based on the presence of Virchow's triad, include endothelial injury, hypercoagulability, and stasis of blood flow. The phenomenon increased in patients with reduced ejection fraction mainly due to anterior myocardial infarction, chronic heart failure (CHF), and dilated cardiomyopathy (DCM). Identification of thrombus by echocardiography, management strategies use anticoagulant, and the follow up to the patient must be applied to prevent poor outcomes.

CASE PRESENTATION

A 56 year old man was admitted to emergency room because of dyspnea since two weeks and getting worse. Swelling lower extremities. No complaint of chest pain. The patient had a history of uncontrolled hypertension. The physical examination showed the blood pressure 140/90 mmHg, heart rate 87/min, respiratory rate 36/minute, no elevated of JVP, pulmonary crackles was in both basal lungs. Edema was on both extremities. The ECG showed sinus rhythm with old anterior myocardial infarction. Echocardiography examination revealed the marked enlargement of all four cardiac chamber. EF 29%, global hypokinetic with regional wall motion abnormalities. An immobile 4.09 cm x 0.99 echo dense mass was found in the left ventricle apical suggestive of a large left ventricular apical thrombus. The patient was treated with 5000 units intravenous bolus heparin, continued with titration of 450 IU/ hour, intravenous furosemide 2x40 mg, ramipril 1x5 mg, spironolactone 1x25 mg, and bisoprolol 1x1.25 mg. The patient was discharged after the condition improved, and continued with outpatient care by giving warfarin 1x3 mg. Furthermore, monitoring INR and echocardiography examination will be performed to evaluate side effects of anticoagulant and resolution of the thrombus.

DISCUSSION

Left ventricular apical thrombus was diagnosed when echocardiography showed an echo dense mass clearly border from the left ventricle endocardium attached to the apical wall that was identified in at least two different views. Poor systolic function and regional wall motion abnormalities result in obstruction of blood in the left ventricle. The events play important role in the formation of left ventricular apical thrombus. Myocardial infarction, chronic heart failure (CHF) with low ejection fraction, and enlargement of all four cardiac chambers are the precipitating factors for left ventricular apical thrombus with high risk for thromboembolic complications. Risk assessment for thromboembolism can be observed from thrombus mobility. The more mobile a thrombus is the greater the risk of thromboembolism. Based on echocardiography examination in this case the thrombus was less mobile. Delayed PCI associated with extensive infarction is an independent predictor of left ventricular thrombus. Therefore, the best way to reduce the formation of left ventricular thrombus is by immediate revascularization. Intravenous heparin in hospitalization was followed by vitamin K antagonist (warfarin) during outpatient care was recommended for this patient to reduce the risk of thromboembolism. In addition, the patient was given furosemide 2x40 mg, ramipril 5 mg, spironolactone 25 mg, and bisoprolol 1.25 mg as therapy for CHF. Furthermore, it will be evaluated by echocardiography to confirm thrombus resolution after 30 days of thrombus detection. Vitamin K antagonist (warfarin) is as a standard therapy for left ventricular thrombus in the ESC guidelines. Similar recommendation is found in the ACC/AHA guidelines with a target of INR 2.5 at least 3 months in patients without high risk of bleeding. Based on the guidelines, the optimal duration of anticoagulation is 3-6 months. But there is no prospective data to support the recommendation. In a study of patients diagnosed left ventricular thrombus, longer administration of anticoagulant was recommended depend on the absence of thrombus and improvement of apical wall motion. In this patient with heart failure and low ejection fraction was given vitamin K antagonist as anticoagulant therapy during outpatient treatment as recommended by the guidelines and applied in several studies. From some studies conducted that the efficacy of vitamin K antagonist was similar to DOAC in thrombus resolution and preventing thromboembolic events. DOAC was recommended in certain conditions if the patient was intolerant of vitamin K antagonist. Vitamin K antagonist can be an alternative therapy if the thrombus persist with DOAC therapy while monitoring factors that affect the value of INR (i.e. food and drugs).

CONCLUSION

Maintenance of anticoagulant and medication for heart failure with low ejection fraction by monitoring their effectiveness and safety are needed for left ventricular apical thrombus in heart failure with low ejection fraction to prevent thromboembolic complications.

REFERENCES


Keywords:
Left ventricular apical thrombus, Low EF, Thromboembolic events.