Treatment of left ventricular thrombus by rivaroxaban: An echocardiographic case report

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Summary

Treatment of left ventricular thrombus is challenging work for the clinician. Current guidelines recommend vitamin K antagonists for thrombus resolution, however there is a growing evidence that rivaroxaban can be used in selected cases. In the paper, we report a 47 year-old-man with a large left ventricular thrombus (60x70mm) which was treated with 10 month 15mg rivaroxaban twice per day. The outcome was total resolution of left ventricular thrombus. During the treatment course, there was an episode of stroke which was completely recovered. The patient was followed up for further recurrence of LV thrombus after stopping rivaroxaban at 10-month.

Keywords: Left ventricular thrombus, vitamin K antagonists, rivaroxaban.

1. Background

Left ventricular thrombus (LVT) is most frequently observed in patients with ischemic or non-ischemic dilated cardiomyopathy [1], [2]. Patients with LVT are at high risk for cardioembolic events like stroke and systemic thromboembolism [3]. The current guidelines recommend vitamin K antagonists over direct oral anticoagulants as a treatment to prevent distal systemic embolization or resolve thrombi [4]. Rivaroxaban has been used for stroke prevention in atrial fibrillation [5], however, its indication for LVT is under debate.

2. Case presentation

A 47-year-old man was referred to the Department of Cardiology for dyspnea. Physical examination revealed normal neurologic status and pale skin; blood pressure, 85/60mmHg; and heart rate, 78 beats per minute. Blood tests revealed increased serum N-terminal brain natriuretic peptide and D-dimer. The protein S and C levels were in normal range. Anti-beta 2-glycoprotein 1 antibody and anti-cardiolipin antibody tests were negative. Renal creatinine clearance was 91mL/min and liver function was normal.

Echocardiography revealed global dyskinesia and dilated ventricles with an end-diastolic diameter of 71mm, end-systolic diameter of 59mm, and ejection fraction of 31%; a massive, nonmobile 60 × 70mm thrombus located at the apex of the left ventricle and two other smaller, mobile thrombi (12 × 10 and 13 × 9mm) located at the septum (fig. 1; supplemental video 1). Coronary angiography showed a normal coronary system. Patient was diagnosed dilated cardiomyopathy complicated by LV thrombus.

Standard care for heart failure was initiated with furosemide, sacubitril-valsartan, beta-blocker, mineralocorticoid antagonist, and oxygen therapy and oral rivaroxaban (15mg twice per day) for thrombus resolution and stroke prevention. Follow-up echocardiography showed a gradual resolution from inside of the intracardiac thrombi which defragmented into small parts and achieved complete resolution at 10 months (fig. 2-6, supplemental videos 2-6). However, during the course of treatment patient left hemiplegia and CT imaging infarcted lesion in right frontal lobe and

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cerebellum. He was diagnosed cardio-embolic stroke and recovered completely without serious complications.

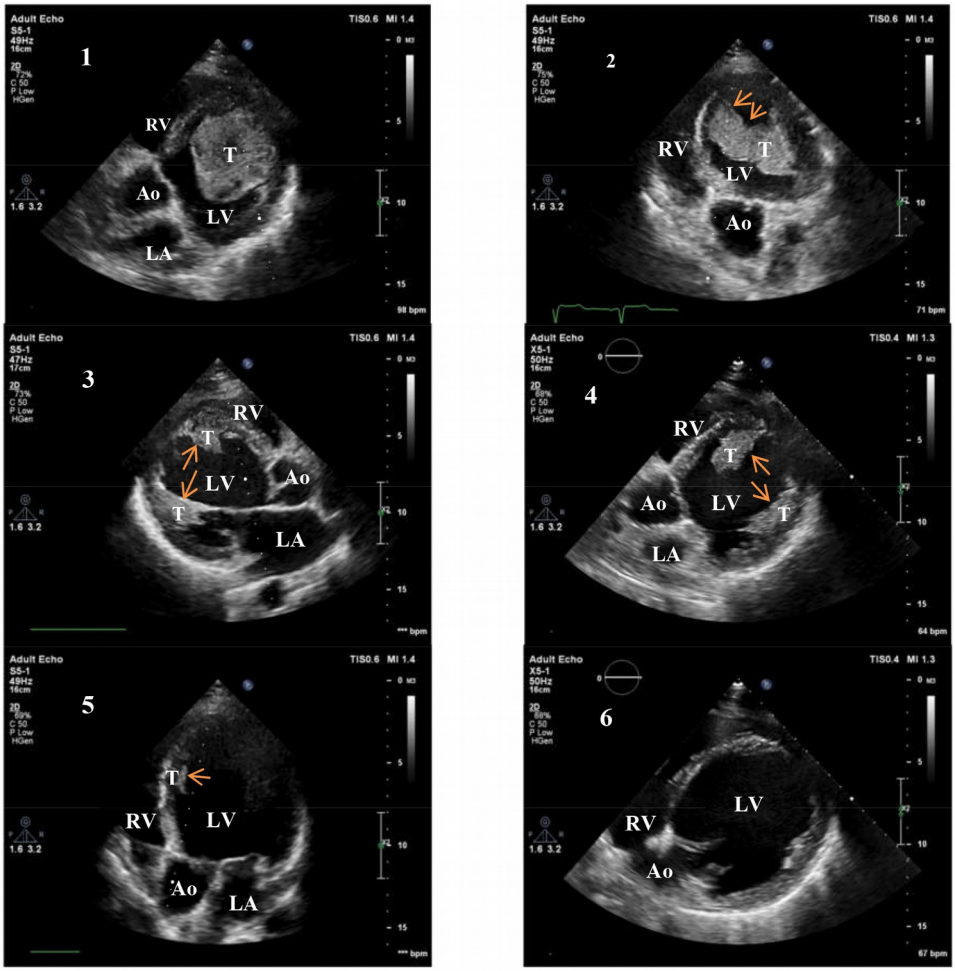

Figure 1.

(1) Apical 4-chamber view showed a large left ventricular thrombus (60 x 70mm) and two other small thrombi (12 x 10 and 13 x 9mm) at hospitalization. LV - left ventricle; RV- right ventricle; Ao - Aorta; LA - left atrium; T - thrombus (yellow arrows);

(2) Follow-up echocardiography showed that the intrinsic fibrinolysis of left ventricular thrombi may start from inside at 2 months.

(3) Thrombus defragmented into small parts at 3 months.

(4) Thrombi attached to septum and papillary muscle at 6 months.

(5) Thrombi were resolved almost completely, during this period patient got a stroke without serious complications at 9 months.

(6) Thrombus was completely resolved at 10 months.

3. Discussion

To the best of our knowledge, this is the first case showing how LVT was resolved by rivaroxaban. There is increasing evidence for the efficacy and safety of rivaroxaban for the treatment of LVT. Makrides et al. described three patients with post-STEMI LVT who were administered 15mg per day rivaroxaban plus antiplatelet therapy. All patients experienced thrombus resolution after about 2 - 4 weeks of treatment [6]. Similarly, Azizi et al. reported

thrombus resolution after 30 days of rivaroxaban in a patient with postinfarction LVT [7]. Smetana et al. reported successful resolution in five of seven patients with LVT with different causes (STEMI, active malignancy, and nonischemic cardiomyopathy) treated with rivaroxaban (15 or 20 mg daily); however, one patient was lost to follow-up and other had no reduction in thrombus size [8].

Robinson et al reported that treatment of LVT with direct oral anticoagulation was associated with a high risk of stroke or systemic events [9]; however, they were unable to explain the reason behind the high rate of embolic events. In this case, we performed echocardiographic follow-up after initial treatment with rivaroxaban. As a gradual resolution of LVT size from its core was observed, the patient continued rivaroxaban until the thrombi were completely resolved. The pathophysiologic mechanism of rivaroxaban, in this case was its ability to prevent clot formation, creating successful intrinsic fibrinolysis inside LVT. The therapeutic fibrinolysis has been dominated by tissue - type plasminogen activator (t-PA) which acts like urokinase or streptokinase. This case demonstrated that the process of fibrinolysis may start inside the thrombus, which may be the reason for the higher risk of embolism in comparison with vitamin K antagonists.

4. Conclusion

This case has demonstrated why treatment of LVT with rivaroxaban may be effective in clot resolution and it may increase thromboembolism. Further studies or case reports are required to understand the underlying pathologic mechanism.

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