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Case Report

Rapid Resolution of Left Ventricular Thrombus Post-STEMI on Rivaroxaban: Effective Lysis or Asymptomatic Embolic Event?

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ABSTRACT

Guidelines recommend the use of warfarin for the treatment of LVT, but Novel Oral Anticoagulants (NOACs) are being frequently used despite the lack of significant randomized trials in support of it. A 78-year-old male with an apical LVT two days post STEMI, demonstrated by Trans-Thoracic Echocardiogram (TTE), was treated with aspirin, clopidogrel and Rivaroxaban. Less than 72 hours later, a second TTE showed the absence of thrombus without symptoms of stroke or peripheral embolic event. A Trans-esophageal echocardiogram (TEE) corroborated the findings. It remains unclear if such rapid resolution of the LVT was due to thrombolysis or to asymptomatic embolism.

INTRODUCTION

Myocardial Infarction (MI) accounts for 15% of mortality worldwide every year [1]. It is the leading cause of morbidity and mortality worldwide and LVT is one of the complications that may arise post-MI [1]. The incidence of LVT has been estimated to be as high as 15% in patients with STEMI and up to 25% in patients with anterior MI [2]. Virchow's triad (stasis, hypercoagulability, and vessel wall injury) plays an important role in the development of left ventricular thrombus. Important factors contributing to left ventricular thrombus formation post-myocardial infarction include:

1. ST-elevation myocardial infarction results in localized dyskinesis or akinesis of the left ventricular chamber. As a result, areas of blood stasis can lead to the occurrence of thrombosis [3].

2. The inflammation and collagen exposure seen in subendothelial tissue injury serves as a focal point for platelet aggregation and clot formation.

3. Increased coagulation system activity present post-acute MI will further facilitate clot formation [3].

The role of NOACs in the management of LVT is evolving and has gained much interest in recent years. The NO-LVT trial is the first randomized clinical study

established to compare the effectiveness of Warfarin versus NOACs in the treatment of LVT [4].

CASE PRESENTATION

A 78 y-o Afro-Caribbean male, with a known history of hypertension, diabetes type 2, smoking, and medication non-compliance, presented to us after a complete extensive anterior MI with persistent ST elevation in anterior leads, and he was referred from another facility after more than 24 hours of observation and no specific treatment. He had intractable chest pain before coming to our hospital. His family history was not significant. The vital signs upon admission were within normal limits. Physical examination was unremarkable. The ECG showed persistent ST elevation in anterior leads with Q waves. Cardiac enzymes were elevated but were declining in the subsequent samples.

When he presented to us, he had no further chest pains or other signs of instability and we considered that the MI was completed. We performed a TTE as a routine for any patient with a late presentation MI to ensure no complications were present, like a new VSD or LVT. We found that the patient had compromised LV function (Ejection fraction (EF) 27%), with severe septal, apical, and anterior hypokinesia. Additionally, there was an apical left ventricular thrombus measuring 1.5cm x 2.3cm as shown in the exhibit (Figure 1.1).

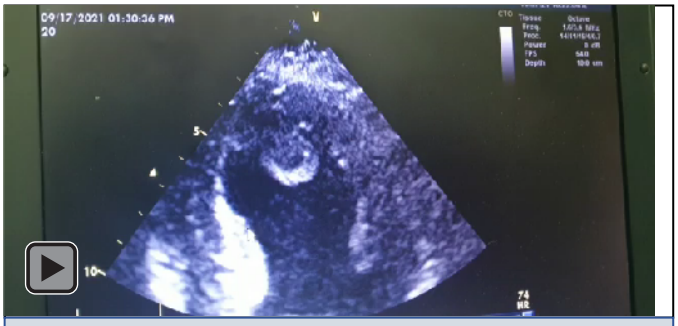

Figure 1.1 Video: An Apical 2 Chamber view with LVT.

The cardiac team discussed the feasibility of intervention vs. waiting for complete resolution of the LVT. The urgency for intervention was not granted to be a safe option in the presence of a friable thrombus that would be detached because of iatrogenic manoeuvres. Conservative strategy was decided as long as the patient continued stable. Triple therapy with Rivaroxaban 20mg OD, Aspirin 81mg OD, and Clopidogrel 75mg OD was instituted, in addition to the usual therapy for acute STEMI and sliding scale insulin. To monitor the progress of this LVT, a subsequent TTE was obtained 3 days later which confirmed the total disappearance of LVT, depicted in the following video [Figure1.2, 1.3].

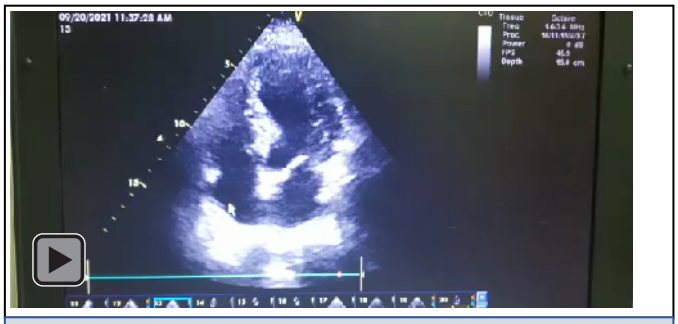

Figure 1.2 Video: An apical 4 chamber view on TTE demonstrating spontaneous echo contrast and no LVT three days after initiation of triple therapy.

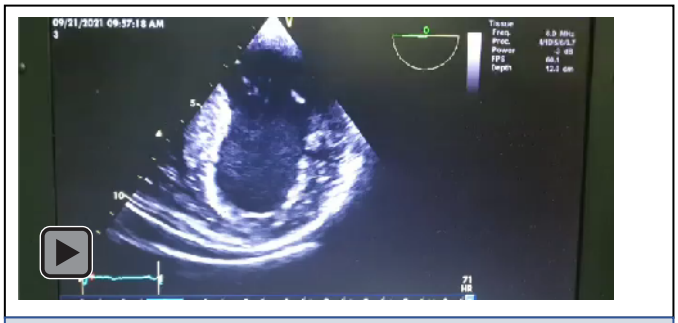

Figure 1.3 Video: A Transesophageal Echo (TEE) confirming disappearance of LVT.

The initial plan was to assess viability after the complete resolution of the thrombus and perform elective intervention later, if needed. Rivaroxaban was given as a long-term option and a safer alternative to warfarin, as the patient was frail and could not monitor his INR frequently. The rapid resolution of the left ventricular thrombus was not expected, and suggested either lysis of the thrombus with the use of Rivaroxaban and dual antiplatelet therapy, or a silent embolization event. There have been no clinical manifestations of embolism (central nervous system, coronaries, kidney, abdominal organs or limbs). We decided to continue with the triple therapy of dual anti-platelet and NOACs, and TTE follow up. A computed tomography scan (CT) of the Brain could not be performed due to the financial constraints of the patient. A Coagulation profile (Protein C, S, anti-Phospholipid antibody) was normal. The last follow-up visit took place in November 2022. The last TTE was done in September 2022 which

demonstrated an improvement of the overall LV function with remaining apical akinesis, and absence of residual or new thrombus.

DISCUSSION

LVT may lead to systemic embolism with devastating consequences, which may increase morbidity and mortality post-myocardial infarction [5]. The incidence of LVT formation has been reduced since the advent of revascularization strategies for ST-elevation myocardial infarction. 2017 European Society of Cardiology guidelines for the management of acute MI proposed considering short-term (3 months) anticoagulation in patients with apical akinesis or large anterior wall MI as a class IIb level of evidence [6]. The first-line treatment for LVT should be vitamin K antagonists (VKAs). A cohort by Bass ME et al. suggested that NOACs and warfarin provided comparable effectiveness in reducing thromboembolic complications [7]. Warfarin is being replaced with direct oral anticoagulants (NOACs) in treating LVT. However, the effectiveness and safety of NOACs vs. VKAs are still debatable, but NOACs have better predictable dosing and no need for frequent monitoring. A recent meta-analysis that studied a total of 2334 patients from 12 cohort studies and three randomized controlled trials showed that NOACs have lower bleeding incidence and non-inferiority in the time of thrombus resolution, stroke, thromboembolic events, and all-cause mortality [8]. The reason for our case report is that it is known that warfarin or heparin infusion is the commonly used approach in treating such patients, with little data about the feasibility of using NOACs in the case of LVT.

Another multicentre cohort by Robinson AA et al. showed that NOACs was associated with a higher risk of stroke or systemic embolism compared with warfarin [9]. The debate about the higher stroke rate in the NOACs group may arise "theoretically" because NOACs could have a faster mechanism of action that would detach the thrombus before complete dissolution, which might lead to dislodgment and distal embolization. More research should be conducted about the mechanism of higher stroke and embolic rates of NOACs in LVT to clarify the possibilities of developing distal embolization, especially that NOACs proved to be a better alternative in the case of LAA thrombi in patients with non-valvular Atrial Fibrillation (AF) as indicated by the guidelines. In our case, the chance of dislodgement of the thrombus with no major incident is potentially real.

Despite this hypothesis proposed in our case, silent embolization is rare, and the reported cases are very few [10]. It has been established that the risk of embolization is highest during the first 1-2 weeks [11]. In the absence of systemic anticoagulation, the risk of embolization within three months among patients with MI complicated by mural thrombus is 10-20% compared with only 2.5% if triple therapy is instituted [5].

Abdelnabi M. et al [4] in the No-LVT trial showed that in comparison to dose-adjusted warfarin, rivaroxaban had a significantly higher LVT resolution at 1-month follow-up. However, there was no remarkable difference between both study groups at 3 months and 6 months. They further showed that Rivaroxaban had a significantly lower rate of stroke in comparison to warfarin at 6 months follow-up without an increased risk of major bleeding [4]. Surgery is usually not an option as the morbidity and mortality of this approach outweigh the benefits. The exception may be contemplated in patients with high embolic risk undergoing other open-heart surgery for other reasons [9].

Multiple studies have shown 18.5% bleeding events with prolonged triple antithrombotic therapy, but no bleeding events were found in short-term therapy (<1 month) [12].

There are increasing reports of fast resolution of LVT, especially fresh thrombus formation in females with peripartum cardiomyopathy. The earliest time between commencement of treatment and objective demonstration of thrombus disappearance was seen in a case report by Nishi I et al. [13] in a 23-year-old female with peripartum cardiomyopathy, in which resolution was proven after four days of intravenous heparin. Altuwaijri W.A et al. [14] described a similar case of a 25-year-old female with a history of peripartum cardiomyopathy treated with parenteral heparin. A TTE confirmed the resolution of the LVT after four days Shokr M. et al. [15]. Published a comprehensive literature review on the use of NOACs in the treatment of LVT and found that with the use of Rivaroxaban, the resolution of LVT post-STEMI was seen in most cases between 3 and 4 months after initiation of therapy. The follow-up interval in patients diagnosed with LVT varied greatly amongst different authors. The case series documented

by Shokr M. et al depicted this disparity. The follow-up interval ranged from 2months to 10months [15-19].

TTE is an inexpensive and readily available test for the diagnosis of LVT and remains the imaging modality of choice for most institutions. However, the recommended gold standard for diagnosis of LVT is Cardiac Magnetic Resonance (CMRI) when it is available [5]. Nevertheless, TTE is very efficient in diagnosing LVT, and facilities without CMRI can effectively use TTE to diagnose and monitor the resolution of LVT. It has been suggested that TTE be performed within 24 hours [2] of admission in those at high risk for apical LV thrombus. Contrast TTE or CMRI when available should be considered in the presence of any of the following on initial TTE:

1. When high apical wall motion scores are present (≥5 on non-contrast TTE).

2. LV apex is poorly visualized

3. Existence of abnormalities of anterior or apical wall motion [2,6].

Potentially there may be a role for routine early echocardiogram pre-discharge and even more frequent follow-up intervals. In our case report, we decided to obtain an early repeat of echocardiogram on the third day to monitor the progress of the LVT and the result showed disappearance LVT. Based on the extremely rapid disappearance of a thrombus of such size and configuration located in the LV apex, we leave the door opened to consider the unusual but not unlikely probability that the LVT resolution could occur as a result of a silent, asymptomatic, and sub-clinical embolic event.

CONCLUSION

The occurrence of LVT is increased in patients with anterior STEMI involving the apex with reduced EF. Triple regimen with dual antiplatelet drugs and NOACs has been demonstrated to be beneficial in the management of LVT. In this case report, the LVT resolution was proven to have happened within 72 hours of treatment. It is not clear if the vanishing of the apical LVT of such size and morphologic configuration could have occurred so early as a result of the therapy or if a silent asymptomatic embolic event could have taken place. However, no symptoms or signs of stroke were present, neither any clinical sign of peripheral embolic event. Our report adds to the growing body of literature demonstrating rapid resolution of LVT post MI in patients treated with NOACS and calls for further investigations to understand the mechanism for this occurrences.

CONSENT

Informed written consent was obtained from the patient for this case report.

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