CASE REPORT

MULTIPLE RIGHT VENTRICULAR THROMBI FORMATION FOLLOWING ACUTE MYOCARDIAL INFARCTION: A CASE REPORT

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Abstract

A 64 year old gentleman presented with features of acute coronary syndrome. Urgent electrocardiogram showed evidence of acute infero-posterior and right ventricular ST segment elevated MI (STEMI). Trans-thoracic echocardiogram (TTE) on admission revealed marked RV and inferior wall hypokinesia with poor RV function. TTE on day three revealed multiple thrombi attached to the RV wall near the ventricular apex. Echocardiogram on the following day revealed coalesced thrombi forming a large mass. This case report illustrates the occurrence of *multiple* RV thrombi following an episode of acute infero-posterior and right ventricular STEMI. Timely performed TTE can be utilized as a safe, non-invasive and cost-effective diagnostic modality as well as a monitoring tool of the therapeutic outcome of such patients.

Introduction

Right ventricular (RV) thrombi could originate either from the right ventricle itself or migrate from the peripheral circulation. Mobile RV thrombi leading to pulmonary embolism is a common phenomenon, whereas the occurrence of RV thrombus following acute MI is less frequently reported and it is even rarer to have *multiple* RV thrombi formation [1,2,3].

Case Report

A 64 year old gentleman presented with an acute severe anginal episode that lasted over one hour with autonomic symptoms. He was a 20 pack-year smoker without any other modifiable cardiovascular risk factors.

Urgent electrocardiogram on admission showed ST segment elevation in leads L_{II}, L_{III}, aVF and V_{4R} with reciprocal changes in L_I and aVL. Tall R with ST depressions were noted in V_1 to V_3 **(Figure 1a)**. Cardiac troponin I become positive subsequently with very high titers. Diagnosis of an acute infero-posterior and right ventricular STEMI was made. Since primary percutaneous coronary intervention facilities were not available, streptokinase was administered (Intravenous infusion of 1,500,000 IU

within 60 min) after excluding contraindications. It was followed by subcutaneous Low Molecular Weight Heparin (LMWH) in a dose of 1mg/Kg, twice daily. Additionally, he was started on dual anti-platelet therapy (Aspirin 150 mg daily and Clopidogrel 75 mg daily) as well. Since the patient had marginally low blood pressure (100/80 mmHg), a fluid challenge of 200 ml of normal saline followed by 1ml/kg/hr infusion was started. Following the cautious fluid resuscitation he became haemodynamically stable after few hours.

Trans-thoracic echocardiogram (TTE) on admission revealed marked RV and inferior wall hypokinesia with poor RV function. Left ventricular function was relatively preserved. TTE on day three revealed three hyper-echogenic mobile masses measuring 12.7x16.5 mm, 12.9x15.5 mm and 7.8x9.1 mm respectively, attached to the RV wall near the ventricular apex. These were not found in the initial 2-Dimentional (2D) TTE **(Figure 1b)**. There was no evidence of aneurismal dilatations in either ventricle. All masses demonstrated free intra-cavitatory motion in systole and diastole. Echocardiogram on the following day revealed coalesced thrombi forming a larger mass **(Figure 1c)** while the RV function remained poor. During the course of the illness, he was extensively investigated for primary thrombophilic conditions (Protein C and S deficiency, Anti thrombin III deficiency and factor V Leiden variant), which were found to be negative. In addition to that he was found to have a negative autoimmune screen (Negative Antinuclear antibodies, VDRL and Antiphospholipd antibodies). His homocystine levels were also normal.

He was started on warfarin with a target International Normalized Ratio (INR) of 2.5 on top of LMWH. After five days of bridging of warfarin with LMWH, the target INR was achieved. Subsequently, aspirin was withdrawn after one month and clopidogrel was continued since the occurrence of gastric haemorrhage is less with clopidogrel. He was followed up with repeated echocardiograms for the evaluation of the RV thrombi. After three weeks of warfarin, repeat echocardiogram showed disappearance of RV thrombi with improvement of RV function **(Figure1d).** Though the thrombi were no longer visualized, warfarin was continued for six months with a target INR of 2.5. In his follow-up visit six months later, he was not found to have any major cardiovascular events or significant bleeding episodes.

Discussion

According to the Virchow's triad; stasis of blood, endothelial injury and hyper coagulability are considered as essential prerequisites for *in-vivo* thrombus formation. Pathology of thrombus formation after an acute MI could be described by the components of this triad. Re-circulating blood flow under low shear stresses which leads to stasis of blood is known to predispose to intra-cardiac thrombus formation[4].

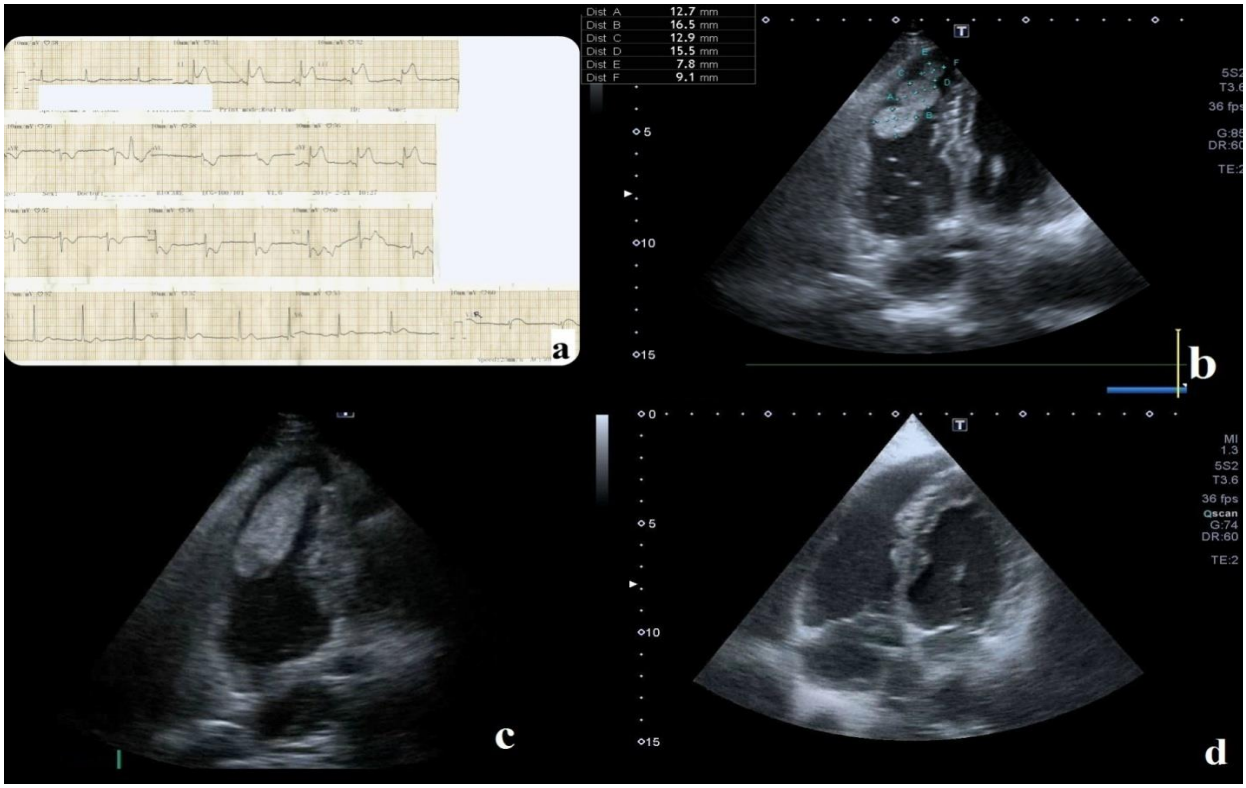

Figure 1. Electrocardiogram and sequential 2D Echocardiograms of the patient

Furthermore, akinesia and hypokinesia following MI, recognized by two dimensional echocardiogram, may result in the stasis of blood within the RV.

Endothelial injury following prolonged ischemia and inflammation would be crucial in initiating platelet plug formation, and would have been an important step in thrombus formation in our case [5,6]. Also, patients presenting with acute MI could be in a hyper coagulable state, due to increased concentration of thrombotic factors and reduced anti-thrombotic factors [7]. Therefore, the presence of conditions indicated in the Virchow's triad would have increased the thrombotic tendency following an acute MI in our patient.

Two-dimensional (2D) echocardiography can be used as an important cost effective investigation tool in the diagnosis of intra-ventricular thrombi following acute MI. Several studies have highlighted the importance of 2D echocardiogram as a simple, non-invasive, bed side test that could be used as a key examination modality in the detection of RV thrombi [1,3].

The timing of the echocardiogram also plays an important role in the diagnosis of intra ventricular thrombi. Thrombi formation could happen within 24 hours following a MI and more than half are visualized within 48 hours[5,7].The incidence of LV mural thrombi formation is higher during day three to five following an acute MI[8]. Even

though our patient had RV thrombi, the time of presentation is in accordance with the above data.

Although the complications of RV thrombi demands early treatment, the treatment of choice still remains controversial. A recent meta-analysis had shown that efficacy of heparin, thrombolytic agents and surgical embolectomy are similar and they all enhance the survival rate of patients having RV thrombi and pulmonary embolism[10]. This analysis as well as other published reports provides evidence to support that drug therapy should be the first option for patients with RV thrombus progressing to pulmonary embolism while surgical embolectomy remains the classical treatment modality for patients with contraindications for such therapy or ineffective thrombolysis with such therapy [1,9,11].

Conclusions

Our case report illustrates the association of *multiple* RV thrombi formation following an acute infero-posterior and right ventricular ST segment elevated myocardial infarction. This highlights the importance of periodic and careful echocardiographic assessment of the RV chamber following an acute MI with marked RV dysfunction. It may help in the early identification of RV thrombi and in the prevention of serious complications such as embolization of RV thrombi. In our opinion, anticoagulation is the most suitable treatment option for patients with intra-ventricular thrombi following acute MI irrespective of the area of involvement.

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