Letter to the Editor

# Right Ventricular Thrombosis in<br/>a Patient with Arrhythmogenic<br/>RightRightVentricular<br/>Ventricular<br/>CardiomyopathyCardiomyopathyPrecluding<br/>IntracardiacIntracardiacDefibrillator<br/>Implantation Initially

Dear editor,

A 52-year-old man was admitted to a hospital with the chief complaint of palpitation. Electrocardiography revealed right ventricular outflow tract (RVOT) ventricular tachycardia, which was subsequently treated by direct-current shock after no responses to amiodarone infusion. Selective coronary angiography demonstrated a normal coronary artery. The patient was referred to our tertiary referral center for further evaluation. Electrocardiography demonstrated incomplete right bundle branch block, slurred S wave in the precordial lead, T-wave inversion in V1-V4 leads, and epsilon wave, especially in V<sub>2</sub> (Figure 1). Transthoracic echocardiography demonstrated normal left ventricular size with global hypokinesia and moderate systolic dysfunction (ejection fraction=40%). The RVOT was dilated: its diameter in both parasternal long- and short-axis views was 43 mm (24 mm/ m<sup>2</sup>). The RV was also dilated with severe systolic dysfunction (fractional area shortening=20%), and the RV free wall was akinetic with aneurysm formation at the base of the RV free

wall. Additionally, a thrombus was noted at the base of the RV free wall (43×25 mm) (Figure 2A). According to these findings, a diagnosis of arrhythmogenic right ventricular cardiomyopathy (ARVC) was established, and the patient was scheduled for intracardiac defibrillator (ICD) implantation. The procedure was, however, postponed because of the large thrombus and the risk of embolism generated by its possible manipulation during ICD implantation. The patient was given amiodarone and sotalol for the suppression of his arrhythmia and the beneficial effect of beta-blockers (sotalol). Nonetheless, 10 days of a continuous intravenous infusion of heparin failed to cause any change in the size of the thrombus, and the patient was given rivaroxaban and discharged home. After 5 months of rivaroxaban consumption, he underwent follow-up transthoracic echocardiography, which illustrated the resolution of the RV thrombosis (Figure 2B). As a result, ICD implantation was performed and rivaroxaban was continued.

We established a definite diagnosis of ARVC in this case based on RV akinesia and aneurysm formation in the RV free wall, accompanied by dilated RVOT and reduced fractional area change, inverted T wave in  $V_1-V_3$ , and the presence of epsilon wave.<sup>1</sup> Moreover, the RV thrombus was formed in the "triangle of dysplasia" (ie, the RV apex, the RVOT, and below the tricuspid region).<sup>2</sup> RV wall aneurysms generally lead to RV dysfunction and provide a context for blood stasis and thrombosis formation. We believe that the formation of the thrombosis in our patient was the logical consequence of rivaroxaban consumption because his RV dysfunction continued and probably deteriorated. Further, the presence of a foreign body in this context could be a new origin for



Figure 1. Electrocardiography demonstrates an incomplete right bundle branch block, a slurred S wave in the precordial lead, a T-wave inversion in  $V_1 - V_4$  leads, and an epsilon wave, especially in  $V_2$  (arrows).



Figure 2. A) A thrombus (arrow) at the base of the right ventricular free wall is illustrated before therapy in the right ventricle-focused 4-chamber view of transthoracic echocardiography. B) The same echocardiographic view shows the resolution of the thrombosis after therapy. LA, Left atrium; LV, Left ventricle; RA, Right atrium; RV, Right ventricle

thrombosis formation and probable embolization.<sup>3</sup> RV thrombosis in the background of ARVC is a rare condition (2.8% of patients).<sup>3</sup> The rate of failure to resolve RV thrombi is 30%.<sup>2</sup>

In patients with ARVC, it is advisable that the RV be investigated for the presence of thrombosis because it can change the therapeutic plane and prevent such catastrophic events as pulmonary emboli in the background of significant RV dysfunction.

# To watch the following videos, please refer to the relevant URLs:

## https://jthc.tums.ac.ir/index.php/jthc/article/view/1440/982

Video 1. Transthoracic echocardiography demonstrates a dilated right ventricular outflow tract in parasternal views, and aneurysm formation at the base of the right ventricular free wall with thrombosis formation in the apical 4-chamber view and the subcostal view.

https://jthc.tums.ac.ir/index.php/jthc/article/view/1440/983 Video 2. Transthoracic echocardiography illustrates the resolution of the right ventricular thrombosis after therapy.

# References

 Marcus FI, McKenna WJ, Sherrill D, Basso C, Bauce B, Bluemke DA, Calkins H, Corrado D, Cox MG, Daubert JP, Fontaine G, Gear K, Hauer R, Nava A, Picard MH, Protonotarios N, Saffitz JE, Sanborn DM, Steinberg JS, Tandri H, Thiene G, Towbin JA, Tsatsopoulou A, Wichter T, Zareba W. Diagnosis of arrhythmogenic right ventricular cardiomyopathy/dysplasia: proposed modification of the task force criteria. Circulation 2010;121:1533-1541.

- Te Riele AS, James CA, Philips B, Rastegar N, Bhonsale A, Groeneweg JA, Murray B, Tichnell C, Judge DP, Van Der Heijden JF, Cramer MJ, Velthuis BK, Bluemke DA, Zimmerman SL, Kamel IR, Hauer RN, Calkins H, Tandri H. Mutation-positive arrhythmogenic right ventricular dysplasia/cardiomyopathy: the triangle of dysplasia displaced. J Cardiovasc Electrophysiol 2013;24:1311-1320.
- Akdis D, Chen K, Saguner AM, Stämpfli SF, Chen X, Chen L, Rao M, Haegeli LM, Tanner FC, Brunckhorst C, Song J, Duru F. Clinical Characteristics of Patients with a Right Ventricular Thrombus in Arrhythmogenic Right Ventricular Cardiomyopathy. Thromb Haemost 2019;119:1373-1378.

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