Does right ventricular involvement increase risk of thrombus formation in post-partum cardiomyopathy?

Dear Editor,

We have read the interesting article of Dr. Koç et al.[1] about the development of biventricular apical thrombus formation and cerebral embolism in a young woman with post partum cardiomyopathy (PPCMP). The patient was 21 years old and developed congestive heart failure (CHF) symptoms 3 months post partum. In our clinic, a 32-year-old patient with hypertension, post-partum cardiomyopathy, CHF, and AICD was diagnosed with a left atrial and large apical thrombus 18 months postpartum. The patient's home medications included carvedilol 12.5 mg bid, furosemide 40 mg, lisinopril 10 mg, spirinolactone 25 mg and pantaprazole 40 mg. She experienced persistent nausea one month prior to admission with intermittent vomiting following meals. She was diagnosed with cholelithiasis and underwent cholecystectomy, with no improvement in her symptoms after surgery. On admission she also experienced worsening of CHF symptoms with dyspnea on exertion, orthopnea, and lower extremity edema (NYHA, class III). Physical examination revealed significant jugular venous distension, lower extremity edema and hepatomegaly. Her lungs were clear to auscultation. Laboratory tests revealed a hemoglobin level of 10 g/dl, creatine 1.1 mg/dl, albumin 3.2 g/dl, cholesterol 68 mg/dl, total bilirubin 4.7 mg/dl, direct bilirubin 1 mg/dl, ALT 24 U/L, AST 20 U/L, ALP 20 U/L, BNP 9049 pg/ml and INR of 1.52. Chest X-ray showed cardiomegaly with no congestion. After admission she had an esophagogastroduodenoscopy which was unremarkable, a hepatitis screen was

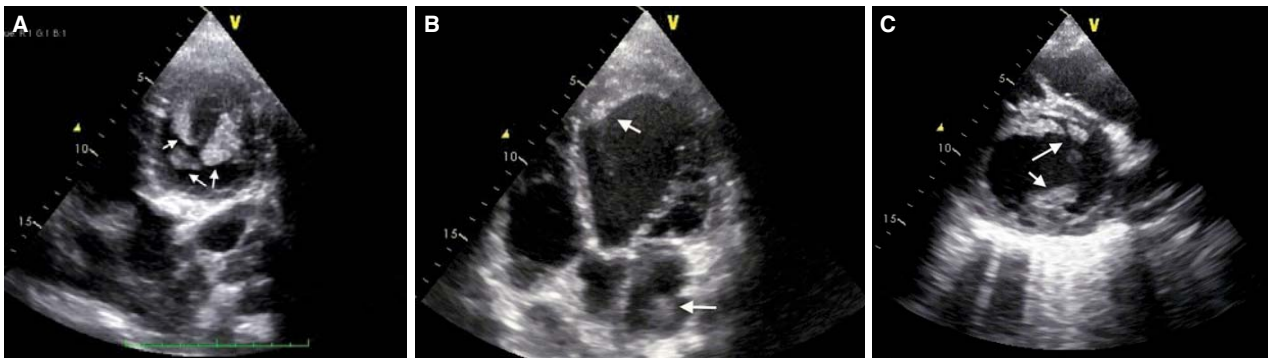

Figure 1. **(A)** Apical thrombus in foreshortened apical 4 chamber view. Apical clot with mobile extensions (arrows). **(B)** Apical thrombus, Left atrial thrombus in apical 4 chamber view of transthoracic echocardiogram. Apical LV thrombus (small arrow), LA thrombus (Big arrow). **(C)** LV clot in short axis view of echocardiogram. LV clot (arrows) in short axis view.

negative and her abdominal ultrasound showed ascites with hepatomegaly which measured about 20 cm with pulsatile venous flow in the main portal vein with prominent hepatic venous flow suggestive of cardiac congestion and her gastrointestinal symptoms were attributed to massive hepatomegaly. An echocardiogram (ECHO) showed EF of 20% with enlarged left (LV) and right ventricle (RV) with severe global hypokinesis of the RV and LV and several large, mobile thrombi in the LV extending from the apex (Fig. 1a-c); there was also a thrombus in the left atrium (Fig. 1). There was mild/moderate tricuspid regurgitation and moderate mitral regurgitation and dilated inferior vena cava. The patient was started on a Heparin drip and warfarin. On day three of the hospital stay, the patient suffered an acute stroke with slurring of speech, right sided weakness and aphasia. The patient's CT angiogram was unremarkable. The patient was continued on a heparin drip and warfarin with resolution of aphasia but continued to have mild weakness in her right upper and lower extremity. Her CHF medications were optimized and she was discharged home on warfarin. Upon 1 month follow up, her liver size was normalized in the ultrasound and she did not have any new neurological symptoms.

In several case reports[1,2] and in our case, significant RV involvement was present with large thrombus formation in the LV. Our patient had thrombus formation almost 18 months after the diagnosis of post-partum cardiomyopathy while she was having worsening of RV function as evidence by GI symptoms secondary to hepatomegaly with increased biluribin and INR. In addition to her hypercoagulable state of the peripartum period, severe ventricular dysfunction resulting in blood stasis can be blamed for the formation of ventricular thrombi in patients with PPCMP.[2] In a review by Goland et al.,[3] four out of forty-six patients with major adverse events had a thromboembolism. LV function (EF <25%) was a predictor of major adverse event. There are no available reports of RV involvement or RV dysfunction as a predictor of thromboembolism or major adverse outcome in PPCM. Decline of RV function could contribute to worsening of hemostasis in the LV in patients with severe LV dysfunction. Although there are no recommendations for prophylactic anticoagulation in patients with PPCMP, anticoagulation should be considered in patients with worsening LV and RV failure, especially if they have neurological symptoms.

Sincerely yours.

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