CLINICAL IMAGE

Peripartum cardiomyopathy with advanced heart failure, complicated by thrombosis and pulmonary embolism with right lower lobe infarction: a therapeutic and diagnostic challenge

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A 27-year-old woman was admitted to the intensive cardiac care unit with advanced heart failure and generalized edema. The patient was 2 months after labor by cesarean section. It was her second pregnancy; the first pregnancy was extrauterine 2 years earlier. She did not have any other known risk factors for peripartum cardiomyopathy and pulmonary embolism. Laboratory tests revealed renal and hepatic failure, elevated D-dimer levels (103 457 ng/ml), and significantly increased levels of inflammatory markers. Transthoracic echocardiography (TTE) on admission showed thrombosis in both ventricles, an ejection fraction (EF) of 23%, a left ventricular end-diastolic diameter of 4.5 cm, a right ventricular diameter of 2.1 cm (body surface area, 1.2 m^2), tricuspid annulus plane systolic excursion (TAPSE) of 12 mm, and second to third degree mitral and tricuspid valve regurgitation, with a tricuspid valve regurgitation velocity of 355 cm/s, accompanied by pericardial fluid and pleural effusion (FIGURE 1A–1D). Chest X-ray showed pleural effusion in both cavities with possible inflammatory lesions in the right lower lobe area.

Treatment with unfractionated heparin was initiated, followed by administration of low-molecular-weight heparin, furosemide, dopamine and dobutamine infusions, and supplementary oxygen. Alternative treatment options were also considered, including the administration of alteplase and thrombectomy, considering the high risk of thrombolysis, multiorgan failure, and the time elapsed from the onset of symptoms (2 weeks). Due to the symptoms of heart failure in the postpartum period and the suspicion of peripartum cardiomyopathy (PPCM), treatment with bromocriptine was initiated (2.5 mg twice daily over 6 weeks). Because the symptoms of heart failure were still present, we administered levosimendan infusion, after which the patient's condition significantly improved. Once her renal function stabilized, a chest computed tomography angiography (CTA) was performed, which confirmed thrombi in both ventricles, as well as pulmonary embolism, with right lower lobe infarction (FIGURE 1E–1H). In addition, ultrasound examination revealed thrombosis of the right internal jugular, subclavian, and common iliac veins.

On control TTE, we observed an improvement of the left ventricular EF and a significant reduction of mitral and tricuspid valve regurgitation. The size of the thrombi in both ventricles was gradually reduced, until they resolved completely.

The patient was hospitalized for 7 weeks and was discharged with an EF of 41%. The treatment recommendation involved rivaroxaban, carvedilol, with low doses of furosemide, spironolactone, and cilazapril. At 7-month follow-up, a control TTE showed an EF of 55%, with normal systolic pulmonary artery pressure.

In 2010, the Working Group on PPCM of the European Society of Cardiology defined PPCM as "an idiopathic cardiomyopathy presenting with HF [heart failure] secondary to left ventricular (LV) systolic dysfunction towards the end of pregnancy or in the months following delivery, where

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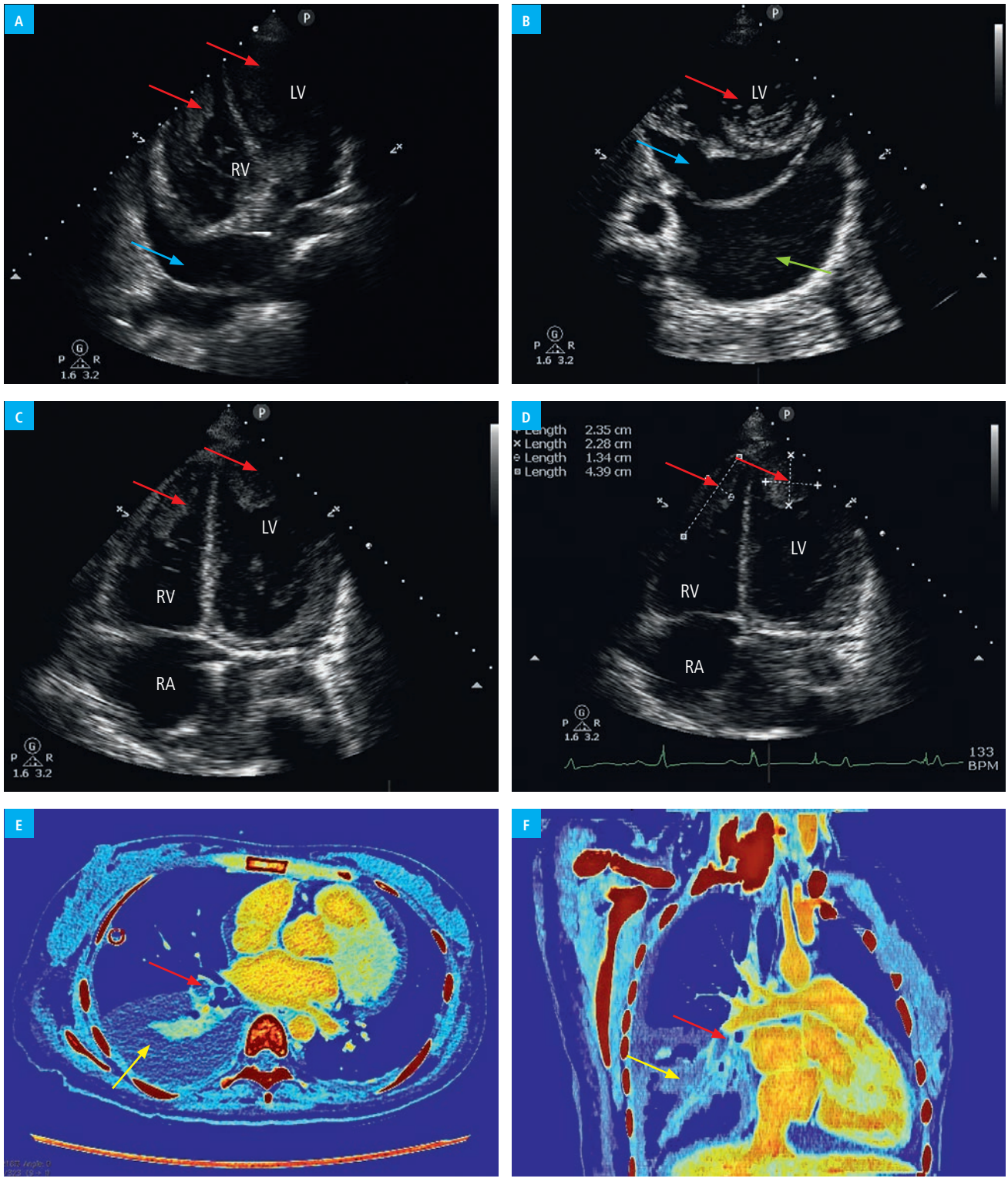
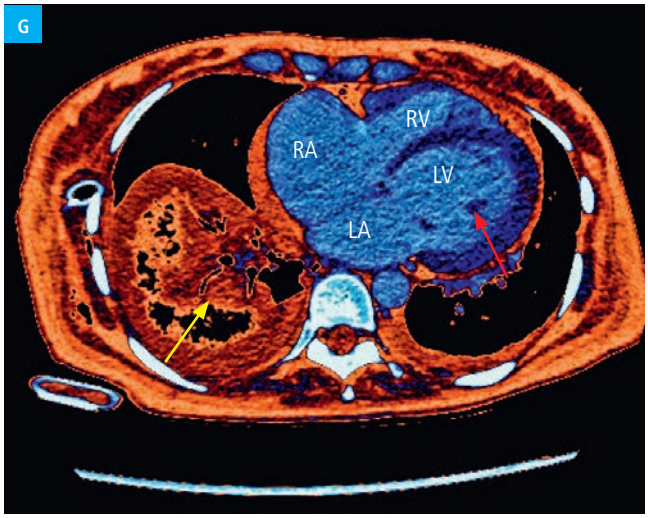


FIGURE 1 Imaging examinations: **A**, **B** – thrombosis in both ventricles (red arrows), pericardial fluid (blue arrow), pleural effusion (green arrow) (transthoracic echocardiography [TTE], subxiphoid modified view); **C**, **D** – thrombus (red arrows) in the left ventricular apex area, measuring 2.3×2.3 cm, and mural in the right ventricle, measuring 1.3×4.4 cm (TTE, apex view); **E**, **F** – reconstructed computed tomography angiography scans of the chest: thrombus inside the right lower lobe pulmonary artery (red arrows); infarction of the right lower lobe (yellow arrows);
Abbreviations: see on the next page



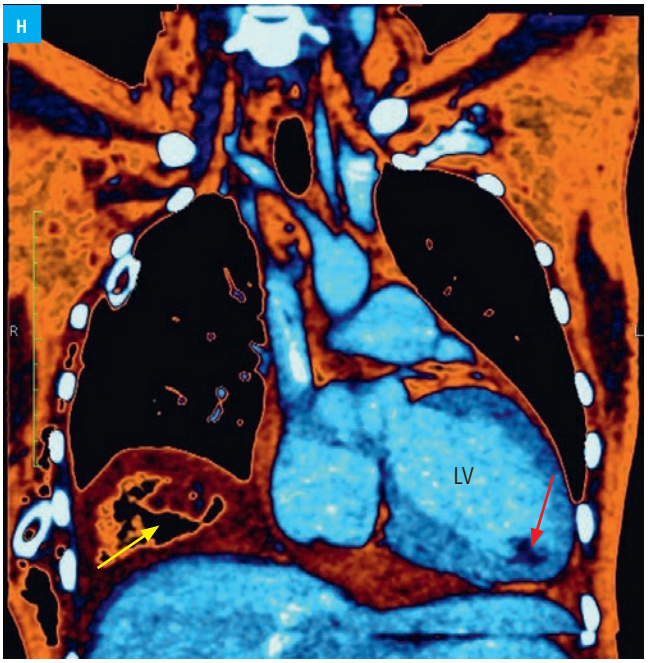


FIGURE 1 Imaging examinations: **G**, **H** – reconstructed chest computed tomography angiography scans: pulmonary infarction of the right lower lobe (yellow arrows); thrombus inside the left ventricle (red arrows) Abbreviations: LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle

no other cause of HF is found".[1] It is diagnosed by exclusion of other conditions. The etiology and precise pathophysiologic mechanisms of PPCM remain unknown. Several hypotheses suggest a multifactorial process including a combination of genetic factors, myocarditis, angiogenic imbalance, oxidative stress, and the prolactin theory.[1-4]

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