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CASE REPORT

Unexpected Course of Left Ventricular Thrombus after COVID-19 Infection in a Woman with Peripartum Cardiomyopathy Single Shield Against Double Shock

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Abstract

Peripartum cardiomyopathy (PPCMP) is defined as heart failure that develops in the last trimester of pregnancy or in the first few months after delivery without an underlying cause. Altought it is seen rarely, it can lead to thromboembolic events and can be life-threatening. Similarly, COVID-19, which is a viral pneumonia agent, is known to cause thrombogenesis. In this case report, the unexpected course of left ventricular thrombus developing in a patient with peripartum cardiomyopathy accompanied by COVID-19 infection is presented.

Keywords: Peripartum cardiomyopathy, Cardiac thrombus, COVID-19

1. Introduction

Peripartum cardiomyopathy (PPCMP) is defined as heart failure that develops in the last trimester of pregnancy or in the first few months after delivery without an underlying cause [1]. Heart failure symptoms that mimic a normal pregnancy can delay in diagnosis and treatment. PPCMP can have a wide range of worse outcomes such as persistent heart failure, arrhythmias, and thromboembolic events [2].

Since the day it appeared, COVID -19 has become a major health concern worldwide. Although death occurs in approximately 2% of cases, this rate may increase in cases with cardiac involvement [3]. Acute heart failure, arrhythmias, acute coronary syndrome, myocarditis, pericarditis, and thromboembolic events are among the well-known cardiovascular complications. Many cases of thromboembolism associated with COVID-19 with different treatment approaches and different outcomes have been published [3].

This case report describes the unexpected course of left ventricular thrombus that developed in a patient with peripartum cardiomyopathy during infection with Covid −19.

2. Case

A 25-year-old female patient with no known history of chronic disease admitted to the emergency department with a 3-day history of fever, cough, and shortness of breath. It was notable that she had given birth by vaginal delivery one month ago.

At the physical examination she had heart rate of 110 bpm, respiratory rate of 20/min, body temperature 37.8 °C, oxygen saturation 89%. Auscultation of lung and heart revealed bilateral 1+ pretibial edema, bilateral basilar crackles and S1, S2, apical 3/6 systolic murmur on cardiac auscultation.

ECG showed sinus tachycardia, loss of R wave progression in anterior leads, and left ventricular strain pattern.

In Laboratory examinations; pH: 7.36, PO2: 82 mmHg, Lactate 4.9 mmol/L, Hb: 12.2 g/dl, WBC:

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6500 (30% lymphocytes), ALT: 1581 U/L , AST: 2316 U/L, INR: 1.94, D-dimer: 13620 mcg/L FEU, CRP: 54.7 mg/L, Procalcitonin 1.05 mcg/L, ferritin >450 mcg/L, BNP: 957 ng/L, troponin I: 224 ng/L was detected.

On transthoracic echocardiography (TTE) LVEF was 25%, left ventricular end-systolic and end-diastolic diameters were increased and there was a surprising a mobile mass consistent with a thrombus: 2.7 × 2.6 cm in size , attached to the apex of the left ventricle, hypoechoic in the center and hyperechoic at the edges, with smooth borders (Fig. 1).

A filling defect in the left ventricle was detected in the Thorax CT of the patient whose COVID-19 PCR test was positive but no findings in favor of COVID-19 pneumonia were observed (Fig. 2).

The patient was started on Bromocriptine 2.5 mg 1 × 1 and Enoxaparin 4000 ANTI-XA IU/0.4 ml 2 × 1 dose in addition to standart heart failure treatment. In the control TTE; performed on the 5th day of hospitalization, it was observed that the thrombus in the left ventricle completely disappeared (Fig. 3).

During this period, the patient had no clinical signs of central or peripheral embolism. The patient, who was compensated for heart failure, was discharged with warfarin treatment. It was planned to continue bromocriptine 2.5 1 × 1 treatment for another 6 weeks. No newly developed thrombus was observed in the control TTE of the patient in the first and sixth month after discharge. Unfortunately, LVEF remained at %30 at sixth month.

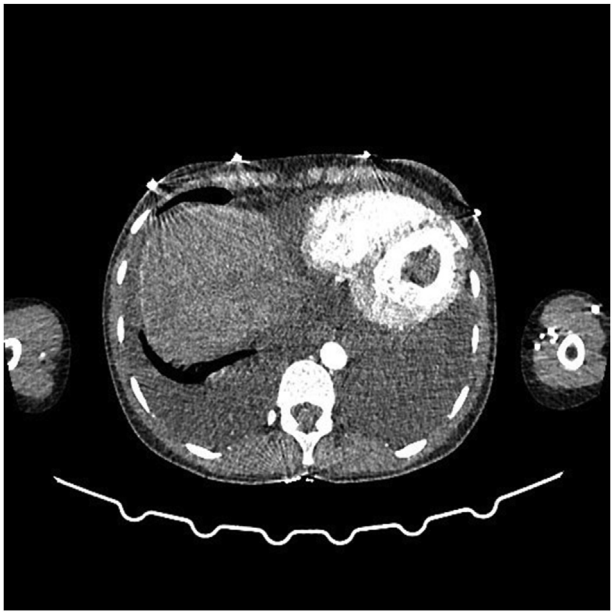

Fig. 2. Thorax computed tomography reveals right-sided pleural effusion and filling defect compatible with thrombus in the left ventricle.

3. Discussion

Peripartum cardiomyopathy is a rare but potentially fatal pregnancy-related heart failure [1,2]. Although the etiopathogenesis is not fully understood, viral myocarditis, autoimmunity, inflammatory cytokines, genetics, oxidative stress and prolactin are blamed [1,4]. Death can occur with heart failure, arrhythmias, and thromboembolic complications; 1-year mortality can range from 4 to 96% [1].

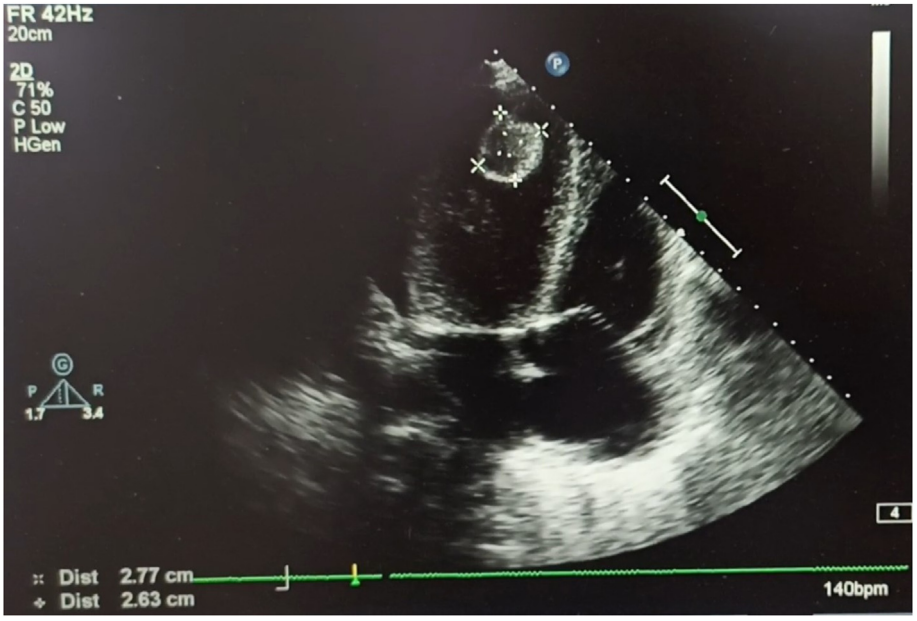

Fig. 1. Apical 4 chamber echocardiographic view showing a mass consistent with a thrombus attached to the apex of the left ventricle, hypoechoic in the center and hyperechoic at the edges, with smooth borders.

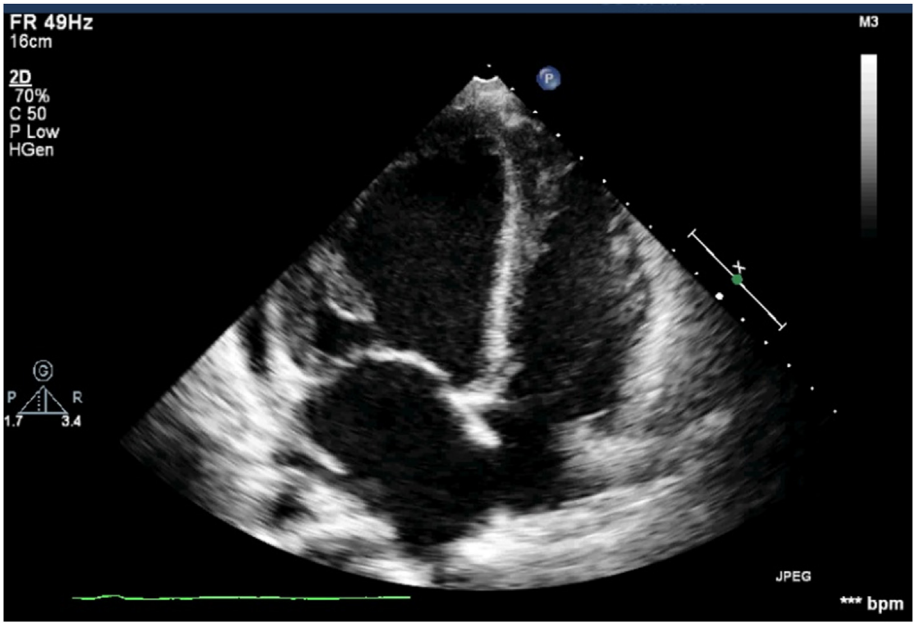

Fig. 3. In the apical 4-chamber trantrosaic echocardiography, it is noteworthy that the thrombus previously shown completely disappeared.

It has been reported that intracardiac thrombus is observed in 10–17% of peripartum cardiomyopathy cases and thromboembolic complications develop in 5–9% of the patients [5,6]. The thrombus is typically located at the left ventricle's apex. Due to the fact that late pregnancy and the postpartum period are linked to an increase in hypercoagulability, anticoagulation is recommended up to 6–8 weeks in the presence of significantly decreased left ventricular performance. (AHA; LVEF> 30%, ESC; LVEF $\leq$35). [1,5] Surgery, heparinization, or thrombolytic therapy has been used to treat LV thrombi and was successful in some cases whereas embolic consequences have appeared in others [7–10].

In the course of COVID-19 disease, increased thromboembolic complications are observed. The virus causes cell death by attaching to endothelial cells via ACE-2 receptors and increase in tissue factor, vWF, collagen, and other thrombotic factors as a result of endothelial damage stimulates coagulation from both intrinsic and extrinsic pathways. Increased shear stress and blood flow velocity causes morphological changes in vWF and activates platelets and causing thrombus development [9]

A number of cases of left ventricular thrombus have been reported in acute myocarditis and heart failure due to COVID-19. Numerous medical and surgical methods have also been tried, with a wide range of results, in the treatment of ventricular thrombi caused by COVİD-19 [10].

The so-called patient had PPCM and COVID-19 at the same time and because of cardiac decompensation she had a high risk for cardiac surgery, therefore she received enoxaparin medication. On the fifth day of treatment, echocardiogram showed the left ventricular thrombus had entirely resoled. The rapid medical treatment regressed of decompensation symptoms. Also the lack of pulmonary involvement and the lack of significant hypoxia of COVID-19 may have contributed to the patient's favorable outcome with LV thrombus. Interestingly, in this case, there were two different mechanisms with the potential to develop cardiac thrombus in the same patient. Although there are case reports describing left ventricular thrombi in PPCMP and COVID-19 separately in the literature, no other case involving these two disorders at once has been reported. There is no clear recommendation regarding the management of left ventricular thrombi that develop during peripartum cardiomyopathy and COVID-19, in the literature. Thus it would be acceptable to make treatment decisions based on the circumstances of each individual patient.

Author contribution

CONCEPTION: KEH, EÇŞ.
LITERATURE REVIEW: KEH, EÇŞ, GÇ.
METHODOLOGY: EÇŞ, CS.
SOFTWARE: KEH, EÇŞ, CS.
ANALYSIS AND/OR INTERPRETATION: KEH, EÇŞ, CS.
INVESTIGATION: KEH, EÇŞ.
DATA COLLECTION AND/OR PROCESSING: KEH, EÇŞ, GÇ.
WRITER-ORIGINAL DRAFT: KEH, EÇŞ.

WRITING- REVIEW & EDITING: KEH, EÇŞ, GÇ, CS.

VISUALIZATION: KEH.

SUPERVISION: EÇŞ, CS.

PROJECT ADMINISTRATION: EÇŞ, CS.

Conflict of interest

None declare.

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