CASE REPORT

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A Rare Case of Peripartum Cardiomyopathy Followed Up for Intracardiac Thrombus and Sudden Embolism of Right Coronary Artery: A Case Report and Review of the Literature

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ABSTRACT Peripartum cardiomyopathy (PPCM) is a rare disease with high mortality in which dilated heart failure occurs in the last month of pregnancy or within 5 months after delivery, without any other etiology or pre-existing heart disease. Sometimes, it may present with intracardiac thrombus and associated life-threatening embolic complications. Here, we will present the management of a 28-year-old patient who was diagnosed with PPCM and intracardiac thrombus immediately after delivery and was followed up in the intensive care unit, in which an inferior myocardial infarction was detected in the electrocardiogram taken for sudden onset chest pain. Although peripheral thromboembolism is occasionally detected in PPCM patients with intracardiac thrombus, coronary embolisms is not common. Presentation with coronary embolism in patients with PPCM has been described in only 4 cases in the literature, but to the best of our knowledge, this is the first case to describe embolism to the right coronary artery.

Keywords: Peripartum cardiomyopathy; intracardiac thrombus; coronary embolism; inferior myocardial infarction

Peripartum cardiomyopathy (PPCM) is a rare disease with high mortality and morbidity, manifested by congestive heart failure (HF) due to left ventricular systolic dysfunction, which usually occurs in the last month or five months after delivery in pregnant women without a previous history of heart disease.[1] The incidence of PPCM in pregnants ranges from 1/3,000 to 1/15,000.[2] Although its etiopathogenesis is not fully elucidated, prolactin hormone is held responsible.[3] Intracardiac thrombi are rarely observed in PPCM, and serious thromboembolic complications may develop as a result.[4] Although central, peripheral, and pulmonary thromboembolism cases have been described in the literature, only 4 cases of coronary embolism have been described, and to the best of our knowledge, this is the first case presenting with inferior myocardial infarction as a result of thromboembolization to the right coronary artery (RCA).[5-11]

CASE REPORT

A 28-year-old pregnant woman (gravida 1, parity 1) hospitalized in the obstetrics and gynecology clinic, who had no health problems before, had a cardiology consultation due to increased dyspnea after a term delivery. She had an uncomplicated delivery following the first pregnancy.

In the cardiology consultation, no problems were found in hematological and biochemical blood parameters. On physical examination, there were crepitant rales at the lung bases on ausculta-

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tion. Blood pressure was 95/60 mm Hg. Pulses were palpable. Heart rate was 106 beats/min and saturation was 92%. She did not have any family history, any chronic disease that she knew before, and a history of chronic drug use. She said that the increasing shortness of breath in the last month of pregnancy was attributed to pregnancy by the clinic she applied to. She also stated that her shortness of breath increased significantly after delivery.

No ischemic finding was detected in the electrocardiogram (ECG). Transthoracic echocardiography revealed dilated left ventricular cavities and low ejection fraction (30%), as well as a large thrombus mass starting from the apex and plastered to the mid-ventricular region (Figure 1A-C). In addition, a hypermobile pedunculated thrombus was observed to extend towards the aortic valve (Figure 1D), a finding that required close follow-up of the patient in terms of increased thromboembolic risk. The main pulmonary arteries were open and a mild pulmonary, mitral, and tricuspid regurgitation jet was observed (Figure 2A-C).

Thereupon, the patient was considered to have PPCM and was transferred to the cardiology intensive care unit for anticoagulation treatment in terms of intracardiac thrombus. The patient was started on fractionated intravenous heparin, HF medication and bromocriptine to suppress lactation. The patient's vital signs stabilized and the patient described sudden onset chest pain 1 day later. The 12-lead ECG was found to be consistent with inferior myocardial infarction and the patient was taken to the catheterization laboratory (Figure 3). Coronary angiography showed normal left coronary system (Figure 4A) and coronary embolism distal to the RCA (Figure 4B).

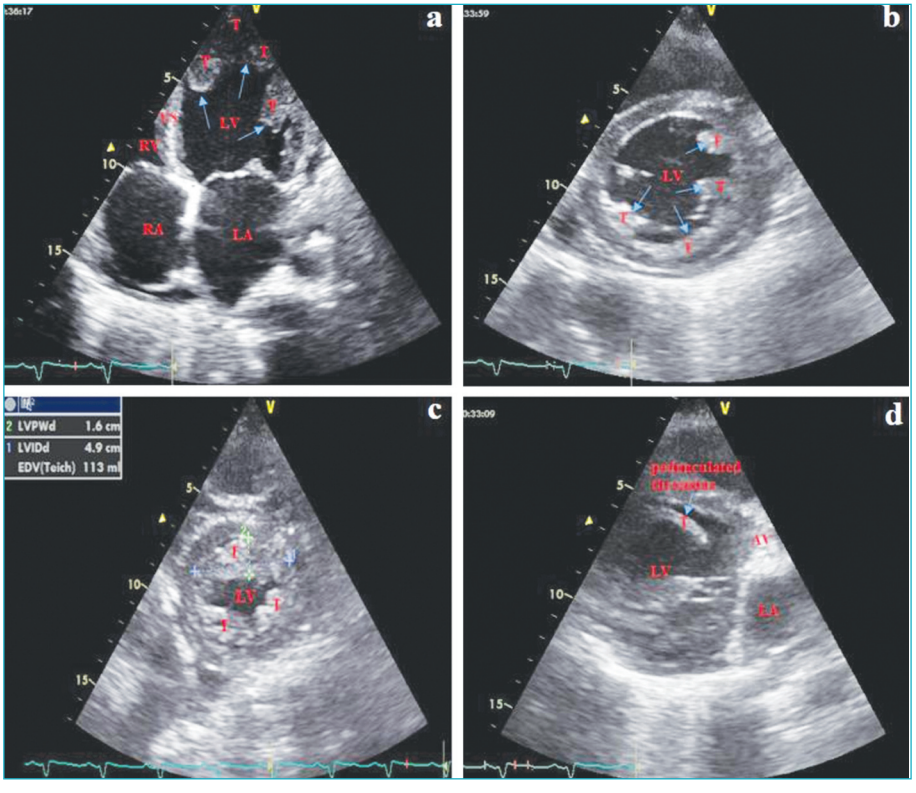

FIGURE 1: **a)** A hypoechoic thrombus mass originating from the apex and extending along the ventricular septum and lateral wall with enlarged left ventricular chambers in apical four-chamber view on transthoracic echocardiography; **b)** Thrombus mass with irregular contours, plastered to the left ventricular walls in mid-short axis view; **c)** Thrombus mass filling almost the entire left ventricular apex in apical short axis view; **d)** Parasternal long axis view shows a hypermobile pedunculated thrombus mass extending towards the AV.

AV: Aortic valve; LA: Left atrium; LV: Left ventricle; RA: Right atrium; RV: Right ventricle; VS: Ventricular septum; T: Thrombus; Blue arrows point to thrombus mass.

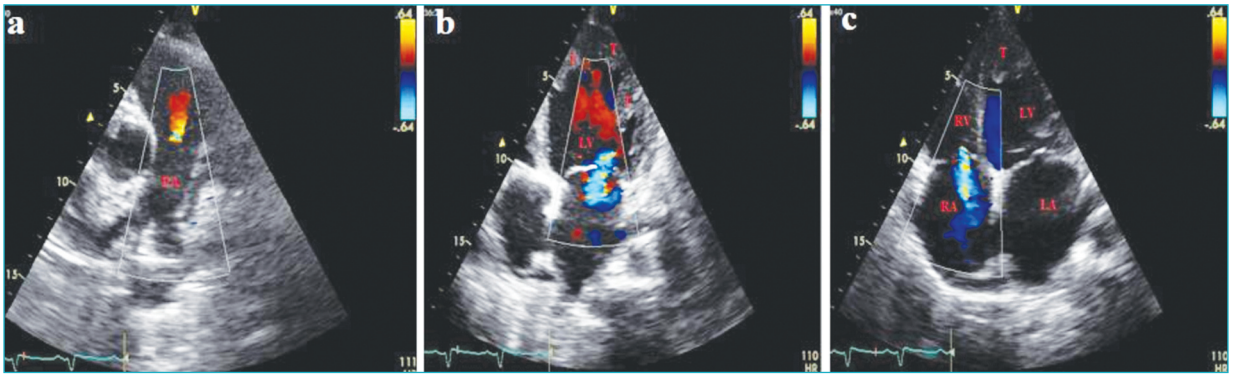

FIGURE 2: **a)** Pulmonary arteries were open and mild pulmonary regurgitation was present; **b)** Mild functional mitral regurgitation was present as expected in dilated heart failures; **c)** Mild tricuspid regurgitation was present and systolic pulmonary arterial pressure calculated over the tricuspid jet was 35 mmHg.
AV: Aortic valve; LA: Left atrium; LV: Left ventricle; RA: Right atrium; RV: Right ventricle; PA: Pulmonary artery; T: Thrombus.

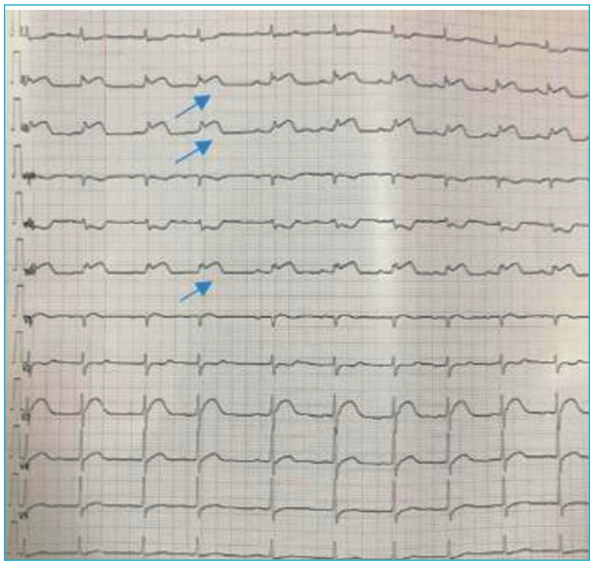

FIGURE 3: ST-segment elevation in inferior leads (blue arrows) on electrocardiogram taken after sudden onset of chest pain under intravenous heparin therapy.

Subsequently, a balloon followed by a drug-coated stent was inserted into the thrombused area restricting coronary flow (Figure 4C), and coronary blood flow was successfully achieved (Figure 4D).

Dual antiplatelet therapy was added to her current treatment, as a stent was implanted in the patient. The patient was discharged with coumadin, dual antiplatelet, HF medication, and bromocriptine.

Written informed consent was obtained from the patient for the publication of her information and images.

DISCUSSION

PPCM is a rare form of cardiomyopathy specific to pregnant women or women who have just given birth, presenting in the last months of pregnancy or within 5 months of delivery and in which no other etiology of HF has been identified.[1] PPCM is characterized by impaired cardiac systolic function, decreased left ventricular ejection fraction, and dilated left ventricular diameter.[1] The epidemiology of PPCM differs between regions and races, and its incidence varies between 1/3,000 and 1/15,000.[2] Nonspecific symptoms such as shortness of breath, edema and chest pain due to physiological changes due to pregnancy are frequently encountered especially in the last stages of pregnancy and in the first months after pregnancy and do not require specific treatment. Since these symptoms are also the most prominent symptoms of HF, the diagnosis of these patients is often missed at the early stage, and these patients are therefore diagnosed late and may sometimes present with more mortal findings such as thromboembolism. Here, we will discuss a PPCM patient with coronary thromboembolism due to intracardiac thrombus and briefly review the literature.

Dilated cardiomyopathies are the most important risk factor for intracardiac thrombus due to intracavitary stasis of blood, and the risk of intracardiac thrombus in PPCM is higher than in other types of cardiomyopathy.[12] The main reason for this is thought

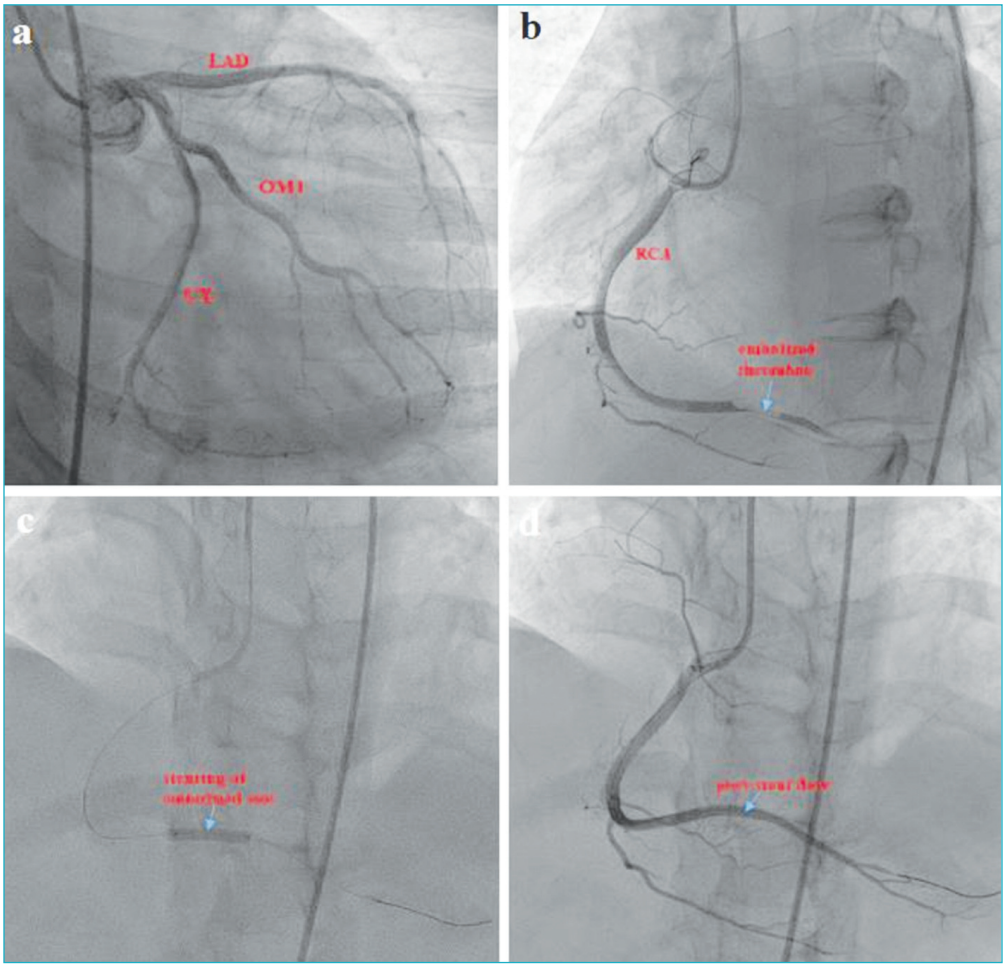

FIGURE 4: **a)** Coronary angiography shows open LAD artery, CX artery, and OM1 artery; **b)** Coronary angiography shows embolized thrombus material distal to the RCA (blue arrow); **c)** Post-balloon drug-eluting stent placement in the embolized thrombus region that restricts coronary flow and causes inferior myocardial infarction (blue arrow); **d)** Ensuring TIMI-3 coronary blood flow in the embolized area after the stent (blue arrow).
LAD: Left anterior descending; CX: Circumflex; OM1: Obtus marginalis 1; RCA: Right coronary artery.

to be the increased tendency of the peripartum period to hypercoagulation. In order to maintain the normal function of the utero-placental unit and to minimize the risk of bleeding during childbirth, a large number of clotting factors (including factors VIII, X, and XII) and fibrinogen are increased in the blood. In addition, the amount of natural anticoagulants (including tissue pathway factor inhibitor, protein C and protein S), platelet count, and fibrinolysis are reduced.[13] Therefore, the peripartum period creates a predisposition to thrombogenicity. In the case of dilated cardiomyopathy, systolic dysfunction, decreased myocardial compliance and dilatation of the ventricles impair intracardiac hemodynamics, leading to intracavitary stasis. In addition, chronic oxidative stress, endothelial damage, increased blood viscosity, states of relative hypoxia, and proinflammatory changes also contribute to the increased hypercoagulable state in dilated cardiomyopathy.[14] Therefore, the procoagulant environment created by both the peripartum period and dilated cardiomyopathy presents with an increased risk of intracardiac thrombus in PPCM compared to other dilated cardiomyopathies.

A number of PPCM cases presenting with thromboembolism have been reported in the literature.[15] Although most of them are central, pulmonary and peripheral, coronary thromboembolism has been

reported in only 4 cases and is one of the most risky and challenging in differential diagnosis among all thromboembolic complications.[8-11] No clinical studies are available to guide treatment for cases of coronary embolism without underlying coronary disease, but initiation of reperfusion therapy is indicated (standard for myocardial infarction). Unlike the previous 4 cases, in our case, the thrombus material was embolized to the RCA, not to the left coronary arteries (Figure 4). As seen in previous cases, an intracardiac thrombus material embolizing towards the aortic root is more prone to embolism to the left coronary system, probably because the ostium of the left main coronary artery is larger than that of the RCA. Interestingly, in our case, the thrombus material was embolized into the RCA. As seen in Figure 1D, a pedunculated thrombus extending towards the aortic valve may predict a high risk of systemic and coronary embolism.

The incidence of intracardiac thrombus and associated coronary embolism in PPCM is not known clearly. Non-specific symptoms such as shortness of breath and chest pain of a PPCM patient may overlap with such non-specific symptoms of a normal healthy peripartum woman. This may lead to PPCM being overlooked or misdiagnosed at an early stage. This situation may delay the intervention of mortal complications such as thromboembolic complications in the early stage if intracardiac thrombus has developed in PPCM. Therefore, gynecologists should be alert for this entity and should approach such patients multidisciplinary.

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Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

Idea/Concept: *Kenan Toprak, Asuman Biçer;* ***Design:*** *Kenan Toprak;* ***Control/Supervision:*** *Kenan Toprak, Asuman Biçer;* ***Data Collection and/or Processing:*** *Mustafa Beğenç Taşcanov;* ***Analysis and/or Interpretation:*** *İbrahim Halil Altıparmak;* ***Literature Review:*** *Kenan Toprak;* ***Writing the Article:*** *Kenan Toprak;* ***Critical Review:*** *Asuman Biçer;* ***Materials:*** *Kenan Toprak, Asuman Biçer, Mustafa Beğenç Taşcanov, İbrahim Halil Altiparmak.*

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