

# Acute Respiratory Insufficiency Due to Peripartum Cardiomyopathy After Caesarean Section in a Term Pregnancy With Twins

## *İkiz Term Gebelikte Sezaryen Sonrası Peripartum Kardiyomiyopatiye Bağlı Akut Solunum Yetmezliği*

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### Abstract

We report a case of acute respiratory insufficiency due to peripartum cardiomyopathy after Caesarean section in a term pregnancy with twins. The patient was a 30-year-old woman with a spontaneous twin pregnancy at 32 weeks of gestation who was admitted to our obstetrics department with preterm premature rupture of membranes. After 48 hours, the tocolysis was stopped and an uneventful Caesarean was performed under general anesthesia. As the patient was waking up, her SPO<sub>2</sub> decreased to 32%, and she became cyanotic and tachypneic. Auscultation revealed rales in her lower lung lobes bilaterally. Her oxygen saturation did not increase in the hours that followed, and her cyanosis persisted, so we decided to admit her to the Intensive Care Unit. She was mechanically ventilated. Her chest X-ray showed an enlarged cardiac silhouette and pulmonary infiltrates in the lower lobes. On the second postoperative day, transthoracic echocardiography was performed and revealed an EF of 45%, mild left ventricular systolic dysfunction and moderate mitral valve failure. Lisinopryl and furosemide were started. On postoperative day four, her symptoms and radiological signs had resolved. She was weaned from mechanical ventilation and discharged from the obstetric ward on postoperative day seven.

**Key Words:** Peripartum cardiomyopathy, Pulmonary edema

### Özet

İkiz gebeliği olan ve postoperatif dönemde kardiyomiyopatiye bağlı akut solunum yetmezliği gelişen bir olgu sunulmuştur. Bizim olgumuzda, 30 yaşında 32 haftalık ikiz gebeliği olan kadın hasta preterm membran rüptürü nedeniyle Kadın Hastalıkları ve Doğum kliniğine yatırıldı. Tokolizden 48 saat sonra genel anestezi altında hasta operasyona alındı. Uyandırma döneminde SpO<sub>2</sub> %32 saptanan hastada taşikardi ve takipne gelişti. Oskültasyonda her iki akciğer alt zonda krepitan raller tespit edildi. Hasta pozitif basınçlı ventilasyona rağmen oksijen saturasyon yüzdesinin düşük seyretmesi ve siyanozunun devam etmesi nedeniyle, Yoğun Bakım Ünitesi'ne entübe halde alındı. Çekilen akciğer grafisinde, her iki akciğer alt loblarında pulmoner infiltratlar ve kalp gölgesinin genişlemiş olduğu tespit edildi. Ekokardiyografik incelemesinde ejeksiyon fraksiyonu %45, sol ventrikül sistolik disfonksiyonu, sol ventrikül boyutlarında artma, orta mitral kapak yetmezliği ve hafif triküspit kapak yetmezliği saptanan hastada, bu tabloyu açıklayacak anemi dışında bulgu saptanmadı. Peripartum kalp yetmezliği tanısı konan hastaya lisinopril ve furosemid başlandı. Postoperatif 4. günde hasta mekanik ventilatörden ayrıldı, postoperatif 7. günde taburcu edildi.

**Anahtar Kelimeler:** Akciğer ödemi, Peripartum kardiyomiyopati

### Case Report

A 30-year-old gravida 2 para 1 pregnant woman with a spontaneous twin pregnancy at 32 weeks of gestation was admitted to our obstetrics department with preterm premature rupture of membranes. Her history revealed postpartum hemiparesis following a vaginal birth nine years ago that

resolved spontaneously after two months. At presentation she had contractions. Tocolysis was started with nifedipine, and betamethasone therapy was initiated. After 48 hours, the tocolysis was stopped and the patient was prepared for Caesarean delivery. A Caesarean was performed under general anesthesia because the patient did not want regional anesthesia. A healthy 1590-gram girl with a first-minute APGAR

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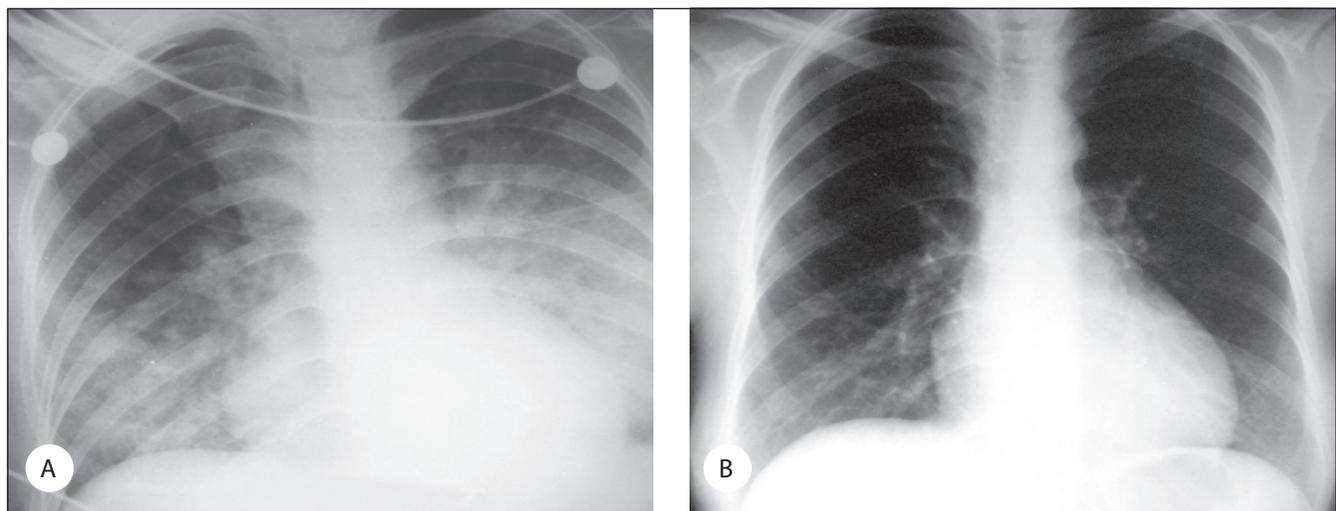
score of 6 and a 1670 gram boy with a first-minute APGAR score of 8 were delivered. When the patient was waking up, her SpO<sub>2</sub> decreased to 32%, and she became cyanotic. She also became tachypneic, and her heart rate reached about 112 bpm. She became slightly hypotensive (with a blood pressure of 90/50 mmHg). Auscultation revealed bilateral rales in the lower lobes of her lungs. The patient's oxygen saturation did not increase in the hours that followed, and cyanosis persisted despite the administration of oxygen at 100% O<sub>2</sub> (The patient could not be extubated due to her continued hypoxia). Her ETCO<sub>2</sub> was 45 cmH<sub>2</sub>O. Blood gas results were pH: 7.25, PaCO<sub>2</sub>: 57 mmHg, pO<sub>2</sub>: 45 mmHg, HCO<sub>3</sub>: 27, BE: -4 and SpO<sub>2</sub>: 70%. We decided to admit her to the Intensive Care Unit (ICU) due to Acute Respiratory Insufficiency. She was mechanically ventilated with PEEP 10 cmH<sub>2</sub>O, pressure level 14 cmH<sub>2</sub>O, 60% O<sub>2</sub> concentration and CPAP-PSV mode. A blood gas was taken after half an hour and results were pH: 7.30, pO<sub>2</sub>: 75, pCO<sub>2</sub>: 44, HCO<sub>3</sub>: 24, BE:-3 and SpO<sub>2</sub>: 94%. Her chest X-Ray showed an enlarged cardiac silhouette and pulmonary infiltrates in the lower lobes (Figure 1). An ECG and laboratory work-up, including C-reactive protein and sedimentation, did not identify any pathologies other than anemia (Hb: 8.7 gr/dl). The patient was empirically treated with antibiotics, low-molecular-weight heparin, acetylcysteine and bronchodilators. Despite the therapy, no improvement occurred. Pulmonologists were consulted, and their differential diagnosis included cardiac valvular disease, pulmonary thrombo-embolism and amniotic fluid embolism. Doppler examination of the patient's lower extremities showed no evidence of thrombosis, but low-molecular heparin (enoxaparine 4000 IU/ day) was started anyway. On the second postoperative day, transthoracic echocardiography was performed and revealed an EF of 45%, mild left ventricular systolic dysfunction and moderate mitral valve failure. Lisinopryl

and furosemide were added to the treatments. On the fourth postoperative day, her symptoms and radiological signs had resolved. She was weaned from mechanical ventilation, and was discharged from the obstetrics ward on the seventh postoperative day. Another pregnancy was discouraged, and follow-up with a cardiologist was arranged.

## Discussion

Peripartum cardiomyopathy (PPCM) is a rare and potentially fatal disease that causes peripartum heart failure in previously healthy women [1, 2]. A brief review of the literature is provided with emphasis on recent data indicating that an antiangiogenic cleavage product of prolactin contributes to the molecular mechanisms underlying peripartum cardiomyopathy and that blocking the release of prolactin with bromocriptine can ameliorate the condition [3]. Although PPCM is an uncommon form of heart failure with a relatively sudden onset in previously healthy women, the lethal and recurrent nature of this disease following pregnancies is eye-catching. The reported mean incidence is about 1/3000-1/4000 and constitutes approximately 4% of maternal mortality [4].

Mortality ranges between 20% and 85% and commonly results from thromboembolic complications and serious arrhythmias due to the heart failure. Severe left heart failure shortly after delivery in a previously asymptomatic young woman can be caused by a peripartum cardiomyopathy, an exacerbation of valvular heart disease due to both congenital and acquired etiologies, a sustained chronic tachyarrhythmia (tachycardiomyopathy), a fulminant pulmonary embolism or an infective endocarditis. The etiology remains to be elucidated, but risk factors have been identified including old maternal age, multiparity, multifetal pregnancy, preeclampsia, long term tocolytic therapy and malnutrition. Also, infectious processes



**Figure 1. A)** Postoperative acute pulmonary edema **B)** After medical therapy.

and anemia are common coexisting conditions. Myocarditis, either autoimmune or virally-induced, has been proposed as an etiological factor, but an exact consensus has not been reached [5]. Many of the cited factors were present in our case. Mann proposed a role for increased inflammatory cytokine activity in PPCM, which supports the role of cytokines in heart failure [6]. The possibly increased levels of cytokines due to the preterm premature rupture of membranes, the negative inotropic effect of nifedipine, the multifetal pregnancy and general anesthesia could have stimulated the PPCM in our case.

Patients often present with classic symptomatology of cardiac failure: relatively sudden onset of dyspnea, cough, tachypnea, tachycardia and cyanosis. Priority should be given to more common diseases and complications of the peripartum period such as pulmonary thromboembolism, pneumonia, amniotic fluid embolism and asthma. Diagnosing PPCM may not be easy because the symptoms and signs are common for many peripartum pathologies. The clinical and echocardiographic diagnostic criteria have been set for PPCM as cardiac failure encountered in the last month of pregnancy or in the five months postpartum, absence of an identifiable cause for the cardiac failure, absence of cardiac disease prior to the last month of pregnancy and echocardiographic evidence of systolic dysfunction with a left ventricular ejection fraction of <45% and / or decreased fractional shortening <30% and/or an end diastolic dimension of 2.7 cm/ m<sup>2</sup> [7, 8].

A specific therapy for peripartum cardiomyopathy does not exist. Supportive treatments such as sodium and water restriction, afterload-reducing medication and inotropic and diuretic agents are used, and patients almost always need respiratory support in the Intensive Care Unit. For patients with progressive deteriorating cardiac function, heart transplantation may be an option. A recommendation for a future pregnancy is questionable. Another pregnancy could be dangerous, especially if ventricular function has not returned to normal echocardiographically in the six months after the illness [1, 9]. Safety of a future pregnancy is still not guaranteed even if ventricular function has returned to normal echocar-

diographically because ventricular systolic function has been shown to still be suboptimal in such cases [10].

In conclusion, peripartum cardiomyopathy should be part of the differential diagnosis for an acute onset of respiratory and cardiac pathology during the peripartum period. This is true especially in cases unresponsive to empiric therapeutic maneuvers. Standardized diagnostic criteria could make the diagnosis easier, and supportive therapy could be life-saving in puerperal women.

**Conflict of interest statement:** The authors declare that they have no conflict of interest to the publication of this article.

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