CASE RECORDS of the MASSACHUSETTS GENERAL HOSPITAL

Case 22-2014: A 40-Year-Old Woman with Postpartum Dyspnea and Hypoxemia

Zoltan P. Arany, M.D., Christopher M. Walker, M.D., and Lin Wang, M.D.

PRESENTATION OF CASE

Dr. Daniel S. Ong (Cardiology): A 40-year-old woman was admitted to this hospital 10 days post partum, because of dyspnea and hypoxemia.

Ten days before admission, the patient went into labor at 39 weeks of gestation. A low transverse cesarean section and tubal ligation were performed electively at another hospital with the patient under spinal anesthesia, without complications. A healthy infant was delivered; the 1-minute and 5-minute Apgar scores were 8 and 9, respectively. A tuberculin skin test was negative, and a tetanus–diphtheria–acellular pertussis vaccine was administered to the patient. The patient returned home on the third postpartum day. She remained well until approximately 5 days before admission (5 days after the cesarean section), when gradually increasing orthopnea and dyspnea on exertion developed, associated with mild leg edema. Two days before admission, intermittent substernal chest tightness developed, associated with nausea, vomiting, and a cough productive of blood-tinged sputum but without fever or chills. The night before admission, she went to the emergency department of another hospital because of worsening dyspnea.

On examination, the blood pressure was 140/74 mm Hg, the pulse 113 beats per minute, the respiratory rate 16 breaths per minute, and the oxygen saturation 99% while the patient was breathing ambient air; the temperature was normal. The lungs were clear. An electrocardiogram (ECG) showed sinus rhythm at 130 beats per minute and possible left atrial enlargement, with no ST-segment elevation or depression. Blood levels of total bilirubin, total protein, albumin, calcium, alanine aminotransferase, and aspartate aminotransferase were normal, as were tests of renal function; other test results are shown in Table 1. Computed tomographic (CT) angiography, performed according to the pulmonary embolism protocol with the administration of iopamidol, reportedly revealed an enlarged heart; small, bilateral pleural effusions; patchy air-space disease, with areas of patchy consolidation and thickening of the interlobular septa bilaterally; and no evidence of pulmonary embolism, aortic aneurysm, or pericardial effusion.

Two hours after the patient's arrival at the other hospital, marked anxiety and increased dyspnea developed. Lorazepam was administered, followed by heparin. The blood pressure rose to 201/176 mm Hg and the pulse to 173 beats per minute, the respiratory rate was 21 to 30 breaths per minute, and the oxygen saturation

From the Department of Medicine, Beth Israel Deaconess Medical Center (Z.P.A.), the Departments of Radiology (C.M.W.) and Cardiology (L.W.), Massachusetts General Hospital, and the Departments of Medicine (Z.P.A.), Radiology (C.M.W.), and Cardiology (L.W.), Harvard Medical School — all in Boston.

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fell to 64%. Etomidate and propofol were administered, the trachea was intubated, and manual ventilation was begun. Approximately 200 ml of frothy, pink secretions were suctioned. Ten minutes later, the blood pressure fell transiently to 76/43 mg Hg; the pulse was 111 beats per minute, the respiratory rate 31 breaths per minute, and the oxygen saturation 81%. During the next hour, succinylcholine, furosemide (40 mg), and midazolam were administered intravenously. One liter of clear urine was collected by catheter. The oxygen saturation was 90% with manual ventilation

Table 1. Laboratory Data.*

Variable	Reference Range, Adults†	Other Hospital	On Admission, This Hospital
Hematocrit (%)	36.0–46.0	31.5	40.1
Hemoglobin (g/dl)	12.0–16.0	11.0	13.6
White-cell count (per mm^3)	4500–11,000	8700	20,900
Differential count (%)			
Neutrophils	40–70	67.6	88
Lymphocytes	22–44	24.1	9
Monocytes	4–11	5.4	2
Eosinophils	0–8	2.1	1
Basophils	0–3	0.8	0
Platelet count (per mm^3)	150,000–400,000	292,000	608,000
Sodium (mmol/liter)	135–145	143	142
Potassium (mmol/liter)	3.4–4.8	3.3	3.1
Chloride (mmol/liter)	100–108	112	107
Carbon dioxide (mmol/liter)	23.0–31.9	26	17.5
Anion gap (mmol/liter)	3–15	5	18
Glucose (mg/dl)	70–110	100	174
Alkaline phosphatase (U/liter)	30–100	109	162
Creatine kinase (U/liter)	40–150		187
Troponin (ng/ml)			
I (quantitative)	0.00–0.04	0.03	
I (screening)	Negative		Positive
T	<0.03		0.12
Lactic acid (mmol/liter)	0.5–2.2		3.3
N-terminal pro–B-type natriuretic peptide (pg/ml)	0–450 (age <50 yr)		1090
Arterial blood gases			
Fraction of inspired oxygen			1.00
pH	7.35–7.45		7.29
Partial pressure of carbon dioxide (mm Hg)	35–42		45
Partial pressure of oxygen (mm Hg)	80–100		268
Bicarbonate (mmol/liter)	24–30		21
Base excess (mmol/liter)			–5.3
Mixed venous oxygen saturation (%)‡			83 (7 hr after arrival)

* To convert the values for glucose to millimoles per liter, multiply by 0.05551. To convert the values for lactic acid to milligrams per deciliter, divide by 0.1110.
† Reference values are affected by many variables, including the patient population and the laboratory methods used. The ranges used at Massachusetts General Hospital are for adults who are not pregnant and do not have medical conditions that could affect the results. They may therefore not be appropriate for all patients.
‡ The oxygen saturation was measured through a pulmonary-artery catheter.

(the fraction of inspired oxygen was not recorded); desaturation to 56 to 80% occurred when the patient was mechanically ventilated. Approximately 3.5 hours after arrival, she was transferred by ambulance to this hospital for admission. During transfer, she was manually ventilated; the blood pressure was 88/61 mm Hg, and the pulse was 136 beats per minute, weak, and regular.

Seven years earlier, the patient's first child was delivered at 40 weeks' gestation by cesarean section because of failure to progress and fetal distress. The patient had had two spontaneous abortions, 16 months and 13 months before this admission. She had a history of Graves' disease, carpal tunnel syndrome, anemia, and urinary tract infections. Medications before admission included ibuprofen, docusate, and methimazole, and she had taken micronized progesterone and propylthiouracil during the first trimester. She had no known allergies. She was born in South America and had moved to the United States 5 years earlier. She lived with her husband and children and worked as a housecleaner. She did not drink, smoke, or use illicit drugs. Her father had died at 60 years of age from myocardial infarction, her maternal grandmother had died of a stroke, her father and maternal grandmother had had hypertension and hypercholesterolemia, as did her mother, and her sister had nephrolithiasis and asthma.

On examination, the patient was sedated and intubated. She was breathing spontaneously and showed movement when examined. The temperature was 36.3°C, the blood pressure 126/87 mm Hg, the pulse 164 beats per minute, the respiratory rate 24 breaths per minute, and the oxygen saturation 94% while she was breathing 100% oxygen. The skin was diaphoretic. Jugular venous distention could not be assessed. There were crackles at both lung bases, without wheezing. The heart sounds were rapid and regular. The abdomen was soft and nondistended, the surgical incision appeared clean, and the extremities were cool. Blood levels of total protein, albumin, globulin, total and direct bilirubin, alanine aminotransferase, aspartate aminotransferase, calcium, phosphorus, magnesium, lipase, amylase, and thyrotropin were normal, as were results of tests of coagulation and renal function; testing for antinuclear antibodies (ANA) and anti–double-stranded DNA (dsDNA) antibodies was negative. Other test results are shown in Table 1. Urinalysis revealed 1+ occult blood and trace albumin, with mucin in the sediment, and was otherwise normal; testing for urinary legionella antigen and human chorionic gonadotropic hormone was negative.

Dr. Christopher M. Walker: A chest radiograph (Fig. 1) shows an endotracheal tube with its distal end 2 cm above the carina. The heart is enlarged, and there is haziness over both costophrenic angles, indicating small bilateral pleural effusions. There are patchy opacities in the middle and lower lung zones, more in the right lung than in the left lung, findings that are most consistent with pulmonary edema.

Dr. Ong: An ECG showed sinus tachycardia with nonspecific ST-segment and T-wave abnormalities.

In the emergency department, a catheter was inserted into the radial artery. Furosemide (a 40-mg bolus), midazolam, and fentanyl were administered intravenously. Approximately 2 hours after arrival, the blood pressure decreased to 61/35 mm Hg, and norepinephrine bitartrate was infused, with improvement to 81/61 mm Hg.

Diagnostic procedures were performed.

DIFFERENTIAL DIAGNOSIS

Dr. Zoltan P. Arany: This is a dramatic presentation of a young, generally healthy woman with respiratory failure and hemodynamic collapse in the pu-

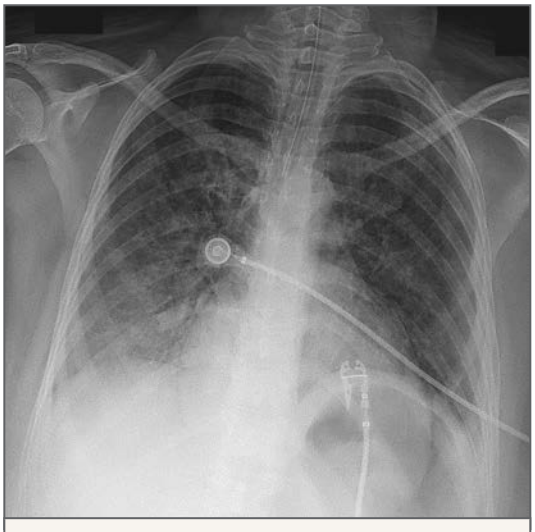

Figure 1. Chest Radiograph on Admission.
An anteroposterior chest radiograph shows an endotracheal tube with its distal end 2 cm from the carina. The heart is enlarged, and there is haziness over both costophrenic angles, indicating small bilateral pleural effusions. There are patchy opacities in the middle and lower lung zones, more in the right lung than in the left lung, findings that are most consistent with pulmonary edema.

erperium. Causes of dyspnea and hypoxemia in the peripartum period are predominantly of pulmonary or cardiovascular origin, the latter generally associated with pulmonary edema. The peripartum period predisposes women to a number of pathologic conditions, including pulmonary embolism, amniotic-fluid embolism, infection, aspiration, preeclampsia, and peripartum cardiomyopathy.

PULMONARY CAUSES OF DYSPNEA AND HYPOXEMIA POST PARTUM

This patient's tachycardia and bloody sputum are findings that are consistent with, but not diagnostic of, pulmonary embolism. Venous thromboembolism occurs in 1 or 2 in 1000 pregnancies[1] and is approximately 10 times as likely post partum as during pregnancy.[2] The peripartum period is a hypercoagulable state, most likely an evolutionary adaptation to minimize postpartum hemorrhaging. Pregnancy-associated venous stasis and endothelial damage further contribute to clotting risk. CT with the administration of appropriately timed arterial-phase contrast material renders a large pulmonary embolism highly unlikely in this patient. Emboli in the small branches of the pulmonary arteries cannot be ruled out, but it is unlikely that these would be manifested as hemodynamic collapse.

Pneumonia needs to be considered. Pregnant women have immune tolerance, which is most likely an evolutionary adaptation to preclude maternal rejection of the fetus. Immune tolerance, anatomical changes during pregnancy, and the risk of aspiration during labor or cesarean delivery place women in the puerperium at risk for infection, including common causes of community-acquired pneumonia. Influenza pneumonia and varicella pneumonia can be virulent, and the acute respiratory distress syndrome is a more common complication among pregnant women than among nonpregnant women.[3] The absence of both fever and an elevated leukocyte count on presentation, in concert with the findings on chest radiography and CT, makes infection unlikely in this patient. The immune-tolerant state of pregnancy can also provide a "holiday" from vasculitides, which can rebound post partum. However, the absence of inflammatory signs and negative tests for ANA and dsDNA make a diagnosis of vasculitis unlikely. Results of tests for antineutrophil cytoplasmic antibodies and human immunodeficiency virus would be helpful.

Advanced maternal age and delivery by cesarean section place this patient at risk for amniotic-fluid embolism, a rare but catastrophic complication of pregnancy or labor.[4,5] Patients with amniotic-fluid embolism usually present with cardiorespiratory collapse, which this patient had; this is typically accompanied by disseminated intravascular coagulation and systemic inflammatory responses, which this patient did not have. Delayed manifestation of amniotic-fluid embolism beyond 48 hours after delivery is extremely rare, and thus this diagnosis is unlikely in this case.

Other pulmonary causes of peripartum dyspnea and hypoxemia are listed in Table 2. There are few indications of a pulmonary cause for this patient's presentation. However, the patient's orthopnea, copious pink and frothy discharge, bilateral pleural effusions, leg edema, elevated serum B-type natriuretic peptide and troponin levels, tachycardia, cool extremities, and hypotension, although not pathognomonic individually, together suggest pulmonary venous congestion and a cardiovascular cause.

CARDIOVASCULAR CAUSES OF DYSPNEA AND HYPOXEMIA POST PARTUM

Acute pulmonary edema complicates approximately 1 in 1000 pregnancies and could explain this patient's symptoms. Peripartum acute pulmonary edema is most commonly due to iatrogenic causes, preeclampsia, or cardiac disease.[6]

Fluid Overload

Iatrogenic fluid overload, which compounds ongoing reabsorption of extracellular fluids, is the leading cause of acute pulmonary edema post partum. Women with this diagnosis usually have had a positive fluid balance of 3 to 9 liters for the preceding 48 hours. Tocolysis (the prevention of uterine contractions) predisposes to pulmonary edema by unclear mechanisms; pulmonary edema usually occurs within hours after the administration of a tocolytic agent and occurs most frequently when multiple tocolytic agents are used simultaneously. This patient was not reported to have received tocolytic agents or excess fluid administration.

Preeclampsia

Pulmonary edema is a frequent complication of preeclampsia, a systemic vascular disease that affects 3 to 5% of all pregnancies.[7] The hallmarks of preeclampsia are hypertension and proteinuria, which are most likely elicited by the secre-

tion of antivascular factors, including soluble fms-like tyrosine kinase 1 (sFlt-1, also known as soluble vascular endothelial growth factor receptor 1), from the placenta in late gestation.[8,9] Preeclampsia-associated pulmonary edema often becomes evident post partum and is thus a consideration in this case. Pulmonary edema in cases of preeclampsia is multifactorial: endothelial damage confers a predisposition to vascular leak, diastolic dysfunction[9,10] and acutely elevated blood pressure confer a predisposition to pulmonary venous congestion, and loss of serum protein lowers intravascular oncotic pressure. In this case, neither elevated blood pressure nor proteinuria was noted during or after pregnancy, including at the time of the patient's initial presentation at the other hospital (before acute decompensation). Postpartum preeclampsia cannot be ruled out, but it is not a likely diagnosis. Testing of antepartum blood, if available, for the levels of sFlt-1 and placental growth factor may be useful to rule out preeclampsia.[8]

Myocardial Infarction

Could this patient have an acute myocardial infarction? The risk of acute myocardial infarction among pregnant or recently pregnant women is three times as high as that among nonpregnant women.[11] The absolute incidence is less than 1 in 10,000 but has climbed as maternal age at the beginning of pregnancy increases. Coronary dissection causes one third of acute myocardial infarctions in this population, most of which occur peripartum.[12] In this patient, the absence of ECG changes, the normal serum creatine kinase level, and the only slightly elevated serum troponin level, make this diagnosis unlikely, although it cannot be ruled out. Myocardial infarction could have occurred days before the presentation. Echocardiography would be useful, since segmental wall-motion abnormalities or papillary muscle rupture and mitral regurgitation would suggest a diagnosis of recent myocardial infarction.

The presence of preexisting myocardial or valvular heart disease must also be considered. The patient is from South America, which places her at risk for Chagas' cardiomyopathy, rheumatic heart disease, or both. Pregnancy generates profound hemodynamic changes, including increases in total blood volume, cardiac output, and cardiac work.[13,14] Underlying cardiac disease is thus frequently aggravated or unmasked by pregnancy. However, the hemodynamic changes of pregnancy primarily occur during the first two trimesters, and clinical presentation with heart failure typically occurs during this time, unlike the timing in this patient.

Table 2. Causes of Dyspnea and Hypoxemia Post Partum.

Pulmonary causes
Common
Pulmonary embolism
Pneumonia
Aspiration pneumonitis
Reactive airway disease
Rare
Acute respiratory distress syndrome
Amniotic-fluid embolism
Vasculitis
Pulmonary hypertension
Pneumothorax
Cardiovascular causes
Common
Preeclampsia
Iatrogenic fluid overload
Edema associated with tocolysis
Myocardial infarction
Preexisting cardiac disease
Peripartum cardiomyopathy
Rare
Septic cardiomyopathy
Pericardial process
Takotsubo cardiomyopathy
Toxic cardiomyopathy (e.g., from alcohol or cocaine)
Thyrotoxicosis
Aortic dissection
Pericardial process
Mitral-valve chordal rupture (usually with mitral-valve prolapse)

Peripartum Cardiomyopathy

The patient's hypotension and evidence of peripheral vasoconstriction suggest a state of low cardiac output, which in the absence of a more likely explanation, suggests a diagnosis of peripartum cardiomyopathy. Peripartum cardiomyopathy is characterized by congestive heart failure and left ventricular systolic dysfunction toward the end of pregnancy or in the months after delivery, in the absence of other identifiable causes of cardiac disease. The ejection fraction on echocardiography is nearly always less than 45%.[15,16] More than 90% of cases are manifested in the first weeks post partum,[17] as in this case. The incidence varies from 1 in 300 live births to 1 in 3000 live births; two "hot spots" are Haiti and Nigeria. Peripartum cardiomyopathy is most common in women of African descent but is seen throughout the world. The manifestation is similar to that of other cardiomy-

opathies, including such signs and symptoms of venous congestion as dyspnea, orthopnea, edema, and, in extreme cases (as in this patient), hypoxemia. B-type natriuretic peptide levels do not normally rise during pregnancy but are typically elevated in peripartum cardiomyopathy, as are troponin levels. Chest radiographs typically show cardiomegaly, as in this case, although peripartum cardiomyopathy can occur without cardiac dilatation.

Research suggests that peripartum cardiomyopathy is triggered by insults to the cardiac vasculature that occur during late pregnancy and the postpartum period,[9,18] including inappropriate cleavage in the heart of circulating prolactin into a potently antivascular fragment[18,19] and the secretion of sFlt-1 from the late gestational placenta.[9] Prolactin and sFlt-1 begin to circulate late in pregnancy, which may explain the timing of peripartum cardiomyopathy. The sFlt-1 level is also elevated in preeclampsia and in multiple gestation, a finding consistent with the epidemiologic association between peripartum cardiomyopathy and these conditions.[13,20]

Other cardiac causes of postpartum dyspnea and hypoxemia are listed in Table 2. Because of the normal results of thyrotropin tests in this patient, severe thyrotoxicosis (thyroid storm) from reactivation of Graves' disease is unlikely. There is low suspicion in this case for toxic cardiomyopathy from cocaine or another drug, although the postpartum period can predispose to depression, drug abuse, and suicidal ideation. Aortic dissection may occur at higher frequency in the peripartum period than at other times, and a high degree of suspicion must be maintained for this diagnosis, since it is often missed. CT angiography did not show a dissection in this case.

Echocardiography is diagnostic in peripartum cardiomyopathy. I assume that it was the diagnostic procedure in this case.

How should this patient be treated? As with other cardiomyopathies, management of peripartum cardiomyopathy should focus on reducing preload and afterload and interrupting the maladaptive neurohormonal response to systolic heart failure. Diuretic agents and nitrates are the treatments of choice for volume overload, although caution is required with the use of these agents before delivery. Angiotensin-converting–enzyme inhibitors and angiotensin II–receptor blockers could be administered in this patient but would be contraindicated if she were still pregnant. In light of the hypercoagulability of the puerperium, anticoagulation is advised for the prevention of systemic embolism, if her ejection fraction is less than 35%. Implantation of an automatic implantable cardioverter–defibrillator, to prevent death from arrhythmia, is relatively contraindicated, because systolic function frequently recovers. If the patient were pregnant, neither premature discontinuation of pregnancy nor delivery by cesarean section would be indicated.

The implication of prolactin in the cause of peripartum cardiomyopathy suggests that bromocriptine, which suppresses the secretion of prolactin from the pituitary, may have therapeutic use. A small, randomized, phase 1 trial showed promising results[21] but awaits confirmation from ongoing larger trials. By the same reasoning, discontinuation of breast-feeding has been advocated, although a retrospective Internet-based study showed that breast-feeding was associated with a better rather than a worse outcome. Neither bromocriptine nor discontinuation of lactation can thus be recommended for this patient.

What is the patient's prognosis? As many as 50% of women with peripartum cardiomyopathy eventually recover cardiac function, but 25% have progression to advanced heart failure, which often leads to cardiac transplantation or death. A lower ejection fraction at presentation predicts a worse outcome and delayed recovery.[22-24] Peripartum cardiomyopathy usually recurs in subsequent pregnancies,[25] so decisions about repeat pregnancies in a patient such as this one need to be considered on an individual basis.

Dr. Nancy Lee Harris (Pathology): Dr. Ong, can you tell us the thinking of the team that was caring for the patient?

Dr. Ong: Because of the patient's initial clinical presentation, peripartum cardiomyopathy was at the top of the list in our differential diagnosis. We also considered acute coronary syndrome, coronary-artery dissection, pulmonary embolism, pericardial tamponade, and thyroid storm. We had the great benefit of a preliminary workup at the outside facility, including CT imaging that did not identify pulmonary embolism or a large pericardial effusion. Thyroid-function tests and a transthoracic echocardiogram were obtained to confirm that the most likely diagnosis was peripartum cardiomyopathy.

CLINICAL DIAGNOSIS

Peripartum cardiomyopathy.

DR. ZOLTAN P. ARANY'S DIAGNOSIS

Peripartum cardiomyopathy.

DIAGNOSTIC TESTING

Dr. Lin Wang: On admission, transthoracic echocardiography (Fig. 2A and 2B; and Videos 1 and 2, available with the full text of this article at NEJM.org) revealed that the left ventricular cavity was dilated and the left ventricular systolic function was severely impaired, with an estimated left ventricular ejection fraction of 11%, as determined with the use of the Quinones method.[26] The left ventricular walls appeared diffusely hypokinetic and did not correspond to any specific coronary-artery territory. There was incomplete closure of the mitral-valve leaflets, a finding consistent with papillary muscle displacement, and there was reduced mitral leaflet excursion, a finding consistent with decreased cardiac output. There was mild-to-moderate mitral regurgitation on color and spectral Doppler imaging. The left atrium was dilated. The right ventricle was normal in size and systolic function. There was evidence of mild tricuspid insufficiency on color and spectral Doppler imaging. The right ventricular systolic pressure was estimated at 30 mm Hg from the regurgitant tricuspid velocity, which makes the diagnosis of pulmonary embolism unlikely. There was no evidence of pericardial effusion. The echocardiographic findings are consistent with peripartum cardiomyopathy.

Videos showing transthoracic echocardiography are available at NEJM.org

Dr. Harris: Dr. Sarswat, would you tell us how you treated the patient and how she is doing?

Dr. Nitasha Sarswat (Cardiology): The patient was taken directly from the emergency department to the cardiac catheterization laboratory,

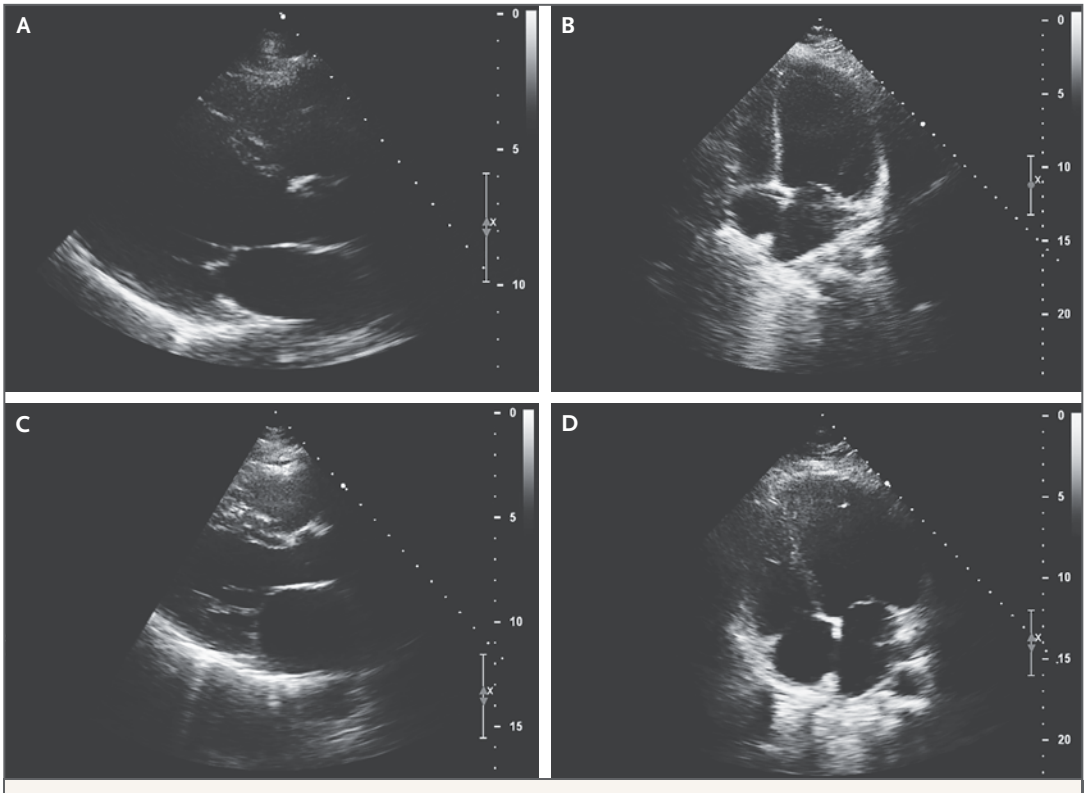

Figure 2. Transthoracic Echocardiographic Studies.

Parasternal long-axis (Panel A) and apical four-chamber (Panel B) views from a transthoracic echocardiographic study obtained on admission show a diffusely hypokinetic and dilated left ventricle and severely impaired left ventricular systolic function. The left ventricular ejection fraction was 11%, as determined with the use of the Quinones method.[26] Parasternal long-axis (Panel C) and apical four-chamber (Panel D) views from a transthoracic echocardiographic study obtained at the 7-month follow-up visit show that the left ventricular systolic function has improved and the ventricle is less dilated. The left ventricular ejection fraction was 46%, as determined with the use of the biplane Simpson's method.[27]

where a coronary angiogram showed patent coronary arteries. Right heart catheterization revealed a right atrial pressure of 7 mm Hg (reference range, 2 to 6), right ventricular pressure of 32/6 mm Hg (reference range, 15 to 28 systolic and 0 to 8 diastolic), pulmonary-artery pressure of 31/22 mm Hg (reference range, 15 to 28 systolic and 8 to 15 diastolic) with a mean pulmonary-artery pressure of 25 mm Hg (reference range, 10 to 20), and a pulmonary-capillary wedge pressure of 11 mm Hg (reference range, 6 to 12). Her cardiac output (as measured by thermodilution) was 6 liters per minute (reference range, 4 to 8), with a cardiac index of 3.3 liters per minute per square meter of body-surface area (reference range, 2.5 to 4.0). Systemic vascular resistance was calculated to be 1000 $dyn \cdot sec \cdot cm^{-5}$ (reference range, 800 to 1200). These hemodynamic results are not typical of cardiogenic shock, but they must be considered in the context of the patient's postpartum state, during which we expect cardiac output to be high and systemic vascular resistance to be low. An intraaortic balloon pump was placed.

On arrival in the coronary care unit, the patient was noted to have a fever, with a peak temperature of 39°C. An abdominal ultrasound examination showed no retained intrauterine products of conception, and specimens of blood and urine were cultured. She was given broad-spectrum antibiotics until the cultures were negative. We thought the fever could have been due to a component of distributive shock or may have been a marker of inflammation due to peripartum cardiomyopathy.

The patient required hemodynamic support with norepinephrine for 24 hours, as well as diuresis with small doses of intravenous furosemide, to which she had a good response. The trachea was extubated approximately 48 hours after her arrival at this hospital.

There was concern for thyrotoxicosis, and an endocrinology consultation was obtained. The patient had normal levels of free thyroxine (T_4), triiodothyronine (T_3), and thyrotropin. The total T_4 level was slightly elevated, although this was thought to be due to excess binding protein present in the peripartum state and therefore was consistent with appropriate thyroid function. Her preadmission dose of methimazole was continued. Other pertinent negative studies included an ANA test, an antibody titer for Lyme disease, a titer for *Trypanosoma cruzi*, and iron studies.

Shortly after the patient was weaned from the norepinephrine, hypertension developed, and she was treated with nitroprusside. Captopril and spironolactone were administered, and the balloon pump was removed. She remained in the coronary care unit for 4 days and was transferred to the cardiac step-down unit. The administration of captopril was discontinued, and treatment with lisinopril and carvedilol was begun. She was discharged home on the 10th day.

The patient has made regular visits to the outpatient clinic. She remains euvolemic and has not required further diuretic therapy, but she continues to take lisinopril and carvedilol. She has not had dose-limiting side effects from her medications and is happy at home with her baby.

Dr. Wang: The left ventricular systolic function improved, with an ejection fraction of 27% on day 1, 36% on day 6, and 39% at the 2-month follow-up visit. Transthoracic echocardiography performed at the 7-month follow-up visit revealed that the left ventricular systolic function was mildly impaired, with an ejection fraction of 46%, as determined with the use of the biplane Simpson's method[27] (Fig. 2C and 2D, and Videos 3 and 4). The left ventricular size and left atrial size had decreased, and there was reduced mitral regurgitation. The right ventricular size and function remained normal.

Dr. Harris: Dr. Arany, do you have any comments?

Dr. Arany: The patient recovered fairly quickly but not completely, which is fairly typical. Did she breast-feed the baby?

Dr. Sarswat: The patient was pumping breast milk for a short time, and then stopped.

ANATOMICAL DIAGNOSIS

Peripartum cardiomyopathy.

This case was presented at Obstetrics and Gynecology Grand Rounds.

Dr. Walker reports receiving royalties from Amirsys. No other potential conflict of interest relevant to this article was reported.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

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