

case report

Unusual manifestations of peripartal cardiac disease

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Because peripartal cardiac disease occurs infrequently, its manifestations may be unfamiliar to most physicians. We report two unusual cases of postpartum cardiac disease: one patient presented with cerebral and peripheral arterial embolization, and the second patient developed late eclamptic seizures with subsequent myocardial infarction. Both patients recovered. Nonobstetric physicians should be aware of these pregnancy-associated medical complications to allow prompt diagnosis and aggressive therapy.

Peripartal cardiac disease is, fortunately, uncommon. Its presentation may include complications of pre-existent valvular dysfunction, acute myocardial infarction during labor and delivery, postpartum myocardial infarction, and postpartum congestive cardiomyopathy. The incidence of postpartum congestive cardiomyopathy has been estimated at one per 1300 live births or one per 4000 confinements (1). Postpartum myocardial infarction is even more rare, with only 11 cases reported in the medical literature. We report two cases of peripartal cardiac disease with unusual manifestations.

CASE REPORTS

Patient 1

A 26-yr-old female, gravida 5 para 3 abortus 2, came to the outpatient obstetrics clinic 2 wk after delivering a healthy child by an uncomplicated spontaneous vaginal delivery. Her chief complaint was garbled speech and difficulty walking, and she was admitted to the internal medicine service.

Medical history and review of systems were negative for chest pain, exercise limitation, recent viral syndrome, alcohol or tobacco use, hyperlipidemia, family history of cardiopulmonary disease, hypertension, heart murmur, fat diets, thyroid disease, migraine headaches, and congenital cardiac disease. Physical examination on admission showed normal fundoscopy, left hemiparesis, left facial nerve paresis, mild expressive aphasia, and left Babinski's sign. Cardiac examination revealed a +S3, +S4 gallop and a grade 2 holosystolic murmur, best

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heard at the apex, that radiated to the axilla. The pulses were within normal limits, with the exception of decreased left dorsalis pedis and posterior tibialis pulses. The lungs were clear, and the abdominal examination was normal.

Doppler examination of the peripheral arterial system confirmed decreased flow to the left foot. An ECG revealed evidence of right ventricular hypertrophy and left atrial enlargement with ST-segment changes compatible with a right ventricular strain pattern. A cerebral computed tomographic scan was within normal limits. Chest x-ray revealed biventricular cardiac enlargement with evidence of pulmonary venous hypertension. Lumbar puncture results were normal.

The initial assessment was that the patient had suffered a right parietal cerebrovascular accident and peripheral arterial embolization. The cardiac examination, chest x-ray, and ECG abnormalities suggested the heart as a likely source for emboli. The patient was begun on heparin therapy.

Approximately 4 wk after admission, all residua of the embolic stroke had completely cleared, and peripheral pulses were equal and strong. Combined M-mode and two-dimensional echocardiograms demonstrated left and right ventricular hypertrophy, left atrial enlargement, and apical densities suggestive of a mural thrombus. Cardiac catheterization revealed normal coronary arteries, 2+ mitral regurgitation, left atrial enlargement, anterior and posterior basal hypokinesis with an akinetic left ventricular apex, and a filling defect compatible with a mural thrombus. The pulmonary angiogram was normal. The left ventricular ejection fraction was 26% (normal 67% predicted). The pulmonary capillary wedge pressure was elevated at 25 mm Hg, and the pulmonary arterial pressure was markedly elevated at 94/42 mm Hg. The following laboratory tests were negative or within the normal range: thyroid functions, antinuclear antibody, rheumatoid factor, complement levels, and VDRL. Viral cultures and acute and convalescent viral serologies were nondiagnostic.

The clinical course and the physical and laboratory findings were compatible with a diagnosis of postpartum congestive cardiomyopathy. The cerebral and peripheral arterial embolization was probably caused by a left ventricular thrombus. Three years after hospital discharge, the patient remains physically active, limited only during extreme exertion.

Patient 2

A 22-yr-old female, gravida 2 para 0 abortus 1, came to the outpatient obstetrics clinic during the eighth month of her pregnancy, complaining of 3 days of epigastric pain. The patient's prenatal course had thus far been uncomplicated, and she had gained 18 pounds. Her medical history was remarkable for a cholecystectomy 4 yr earlier and a spontaneous abortion at 10 weeks gestation in an earlier pregnancy. She had no history of peptic ulcer disease or coronary vascular disease, she did not use alcohol or tobacco, and her only medications were vitamins. Admitting BP was 170/110 mm Hg, and the examination was noteworthy for a grade 2 systolic flow murmur and trace pedal edema. The intrauterine pregnancy was consistent with her dates. Laboratory tests were remarkable for 4+ proteinuria.

The patient was treated with magnesium sulfate and underwent an uncomplicated low transverse cesarean section the next day. Her postpartum course was complicated by low-grade temperatures and persistent hypertension (140/110 mm Hg) treated with clonidine and hydralazine. The patient also had several recurrences of epigastric discomfort associated with nausea and loss of appetite. The discom-

fort was unresponsive to trials of antacids and glycopyrrolate. On the fourth postpartum day, the patient experienced three generalized tonic-clonic seizures. Evaluation included normal serum chemistries, lumbar puncture, and a computed tomographic scan of the head. Seizures were attributed to late eclamptic seizures and were without recurrence. On postpartum day 12, an ECG obtained to evaluate a persistent tachycardia demonstrated 1-mm ST-segment depression in leads 2, 3, AVF, and V₁ to V₃. The next day, the patient complained of shortness of breath. Her temperature was 38.2°C, and respiratory rate was 30 breath/min.

Pelvic examination demonstrated a cord-like, right adnexal tenderness suggesting ovarian venous thrombophlebitis, and the patient was begun on heparin, clindamycin, penicillin, and gentamicin. However, lung ventilation and perfusion scan were of low probability for pulmonary embolus, and her continued respiratory deterioration required transfer to the medical ICU. In the ICU, arterial blood gas analysis revealed a PaCO_2 of 31 torr, a PaO_2 of 23 torr, and a pH of 7.48 on an oxygen flow of 6 L/min through nasal prongs. An endotracheal tube was placed, and the patient was mechanically ventilated. Serial ECGs showed sinus tachycardia with first degree arterial-venous block, arterial-venous dissociation with left bundle-branch block, and Mobitz I block. There was a loss of R-waves in leads V₁ to V₃ compared to prior ECGs. Creatine phosphokinase was elevated to 666 IU with 11% MB fraction, and the lactate dehydrogenase isozymes revealed a pattern consistent with myocardial infarction. Pulmonary arterial catheterization demonstrated a high pulmonary capillary wedge pressure and low cardiac index compatible with Forrester IV hemodynamics. A temporary pacemaker was prophylactically placed for 48 h. M-Mode and two-dimensional echocardiography showed a small pericardial effusion, septal paradoxical movement, and diffuse left ventricular hypokinesis. The patient became afebrile and was extubated on postpartum day 18. Repeat cardiac echocardiography revealed persistent left ventricular apical and septal hypokinesis. The patient underwent cardiac catheterization, which revealed normal coronary arteries, posterior-basal akinesis, anterior hypokinesis, and an ejection fraction decreased at 35%.

DISCUSSION

These cases illustrate two unusual presentations of peripartal cardiac disease. Patient 1 had peripartal congestive cardiomyopathy with cerebral and systemic arterial embolization, reminiscent of the case reported by Hodgman et al (2). Like their patient, ours had experienced a puerperal stroke that led to the diagnosis of a previously unrecognized cardiomyopathy. However, our patient differed from theirs in that a left ventricular mural thrombus was identified as the source of the cerebral and peripheral arterial emboli (3).

Peripartal cardiomyopathy is a rare disease, occurring in one of every 3000 to 4000 pregnancies (4). Contributing factors may include hypertension, familial cardiomyopathy, tropical climate, black race, poor nutrition, and twin pregnancies. The mortality appears to be around 40% to 60% for mothers and 10% for infants. The typical symptoms of congestive heart failure with x-ray evidence of cardiomegaly are most obvious 2 months postpartum. As in patient 1, peripartal cardiomyopathy may cause cerebral embolism from a left ventricular mural thrombus (2, 3, 5). In fact, up to 53% may present with cerebral or peripheral embolism from a cardiac source. Autopsy studies show enlarged flabby hearts with cardiac fiber degeneration and mural thrombi in a majority of cases (3).

Treatment of patients with postpartum cardiomy-

opathy consists of rest, salt restriction, careful digitalis administration to avoid toxicity, anticoagulants for possible mural thrombi or grossly dilated hearts, and preload/afterload reduction as for congestive heart failure. Prognosis for patients with this unusual disease is related to cardiac size 6 months after diagnosis; those with normal heart size have a mortality of 11% to 14%, and those with cardiomegaly have a mortality of 40% to 85% during subsequent pregnancies (4).

The second case typifies the rare occurrence of postpartum acute myocardial infarction. Patient 2 was in her twenties, with a pregnancy complicated by eclampsia but no history of cardiopulmonary disease or risk factors for ischemic cardiac disease. Her myocardial infarction occurred 14 days postpartum and, like eight of 11 patients reviewed by Beary et al. (6), her infarct was located in the anterior wall of the left ventricle.

Mortality from peripartal myocardial infarction reportedly ranges from 18% to 45% (6). Our patient developed cardiogenic shock, which is associated with an inhospital mortality of over 60%. Her excellent outcome was attributed to early diagnosis, aggressive medical management, and prior good health.

Because most postpartum myocardial infarctions occur in women whose pregnancies were complicated by the eclampsia-preeclampsia syndrome, it is tempting to invoke coronary arterial vasospasm as the precipitating factor (7). The uterus, like the kidney, secretes renin. Although patients with eclampsia/preeclampsia have normal circulating serum renin levels, they seem to have an increased sensitivity to circulating angiotensin (8). Angiotensin is a powerful vasoconstrictor, approximately 10 times as potent as norepinephrine, and in the face of extracellular fluid expansion and increased angiotensin sensitivity, a normal renin value may cause hypertension. Maier et al. (9) reported that angiotensin constricts the coronary vessels in isolated perfused rabbit hearts, and Fowler and Holmes (10) showed that angiotensin increases coronary resistance in man. Postpartum myocardial infarction in young healthy women may be caused by coronary arterial vasospasm mediated by an increased sensitivity to circulating angiotensin. Equally possible, but difficult to prove, is that coronary arterial embolization, precipitated by the hypercoagulable state of pregnancy, is the inciting event in postpartum myocardial infarction (6).

Although rare, patients with eclampsia/preeclampsia appear to be at risk for acute myocardial infarction in the first 2 to 3 wk postpartum. Complaints of chest pain during this period should be thoroughly evaluated by chest x-ray and ECG. Nitrate therapy or calcium channel blockade may be advisable to treat coronary arterial vasospasm. The increased sensitivity of the renin-angiotensin axis in conjunction with diffuse vasospasm may also indicate therapy with angiotensin-converting enzyme-inhibitors. This would help normalize

BP and might decrease coronary artery resistance and improve myocardial oxygen delivery. Symptoms of unexplained exercise limitation, shortness of breath, weakness, or headache should prompt the physician to consider the diagnosis of a postpartum myocardialopathic syndrome.

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