Cardiol Young 2003; 13: 209–211

ISSN 1047-9511

Brief Report

Ischemia due to peripartum cardiomyopathy threatening loss of a leg

Paul J. Gagne,[1,2] John B. Newman,[1] Bart E. Muhs[2]

[1]*Departments of General Surgery and Vascular Surgery, Naval Medical Center, Portsmouth, Virginia;*
[2]*Division of Vascular Surgery, New York University Medical Center, New York, USA*

Abstract Ischemia of the leg in a peripartum female is an uncommon condition. Paradoxical arterial embolisation, and arterial dissection, are rarely encountered but recognized causes of this clinical condition. Peripartum cardiomyopathy is a rare life-threatening cardiac condition that can foster intracardiac thrombosis and produce peripheral vascular complications through embolization. We present here the case of a young, healthy, postpartum female who developed acute ischemia of the left leg, and asymptomatic arterial insufficiency of the right lower leg, as the presenting symptoms of peripartum cardiomyopathy, highlighting the fact that ischemia threatening the loss of a limb can be the initial manifestation of peripartum cardiomyopathy.

Keywords: Embolism; arterial ischemia; thrombus

Acute, limb-threatening lower extremity ischemia is uncommon in young adults with no prior symptoms of arterial insufficiency or risk factors for atherosclerotic occlusive disease. Peripartum cardiomyopathy is a rare affliction of healthy, young, females characterized by congestive heart failure, intracardiac thrombus, and frequently death. We present a case of a 38-year-old female who presented with acute ischemia of one leg, highlighting that this finding can be the initial manifestation of peripartum cardiomyopathy.

Case report

A 38-year-old white female presented to the emergency department with a four-hour history of a paralyzed and insensate left foot. She had no significant medical or surgical history. She denied any history of claudication, tobacco use, hypercholesterolemia, coagulopathy, congestive heart failure, cardiac arrhythmias, or diabetes mellitus. She had given birth to her fifth child seven days previously. Labour had been induced because of pre-eclampsia, and an uneventful vaginal delivery followed. She was discharged on the second post-partum day.

Four hours prior to presentation, she had noted the sudden onset of a heavy feeling in her left foot. Over the next 3 h, she developed severe pain, paresthesia, and paralysis from her left mid-calf to her toes. When seen in the emergency department she was afebrile, her heart rate was ranging between 100 and 127 beats per min, and her blood pressure was 146/100 mmHg. Her saturation of oxygen whilst breathing room air was 98%. Her lungs had scant basilar rhonchi. Her cardiac exam revealed a tachycardia, but no murmurs were audible. The electrocardiogram confirmed the presence of sinus tachycardia. Her left foot was cool, pallorous, insensate, and paralyzed distal to the left mid-calf. Her right lower leg was normal, and her femoral and popliteal pulses were palpable bilaterally. No foot or posterior tibial pulses, nor femoral and popliteal pulses were palpable bilaterally. Neither her dorsalis pedis nor posterior tibial pulses could be felt in the left leg, nor could doppler signals could be detected over these arteries. The right dorsalis pedis was also absent, and the right posterior tibial pulse was diminished. She was anticoagulated with heparin

The opinions or assertions expressed herein are those of the authors and are not to be construed as official or as reflecting the view of the Department of the Navy, the Department of Defense, or the Naval Medical Center Portsmouth. Financial support was not provided to the authors from any source other than the U.S. Navy.

Correspondence to: Paul J. Gagne MD, University Vascular Associates, 530 First Avenue, Suite 6F, New York, NY 10016, USA. Tel: (212) 263-7311; Fax: (212) 263-7722; E-mail: paul.gagne@med.nyu.edu

Accepted for publication 27 November 2002

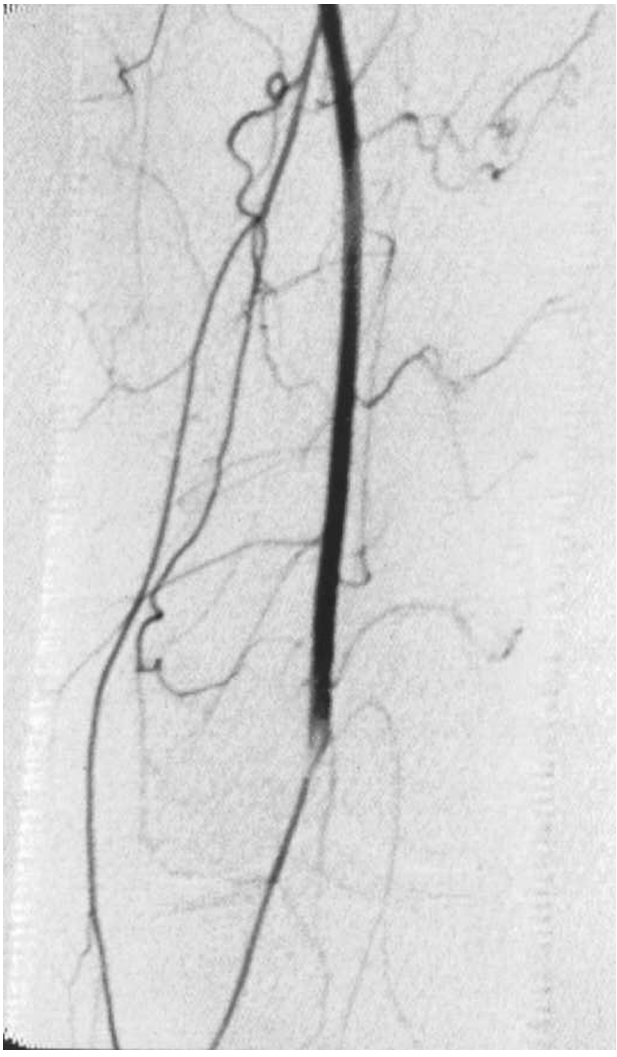

Figure 1.
Angiogram demonstrating occlusion of the left popliteal artery.

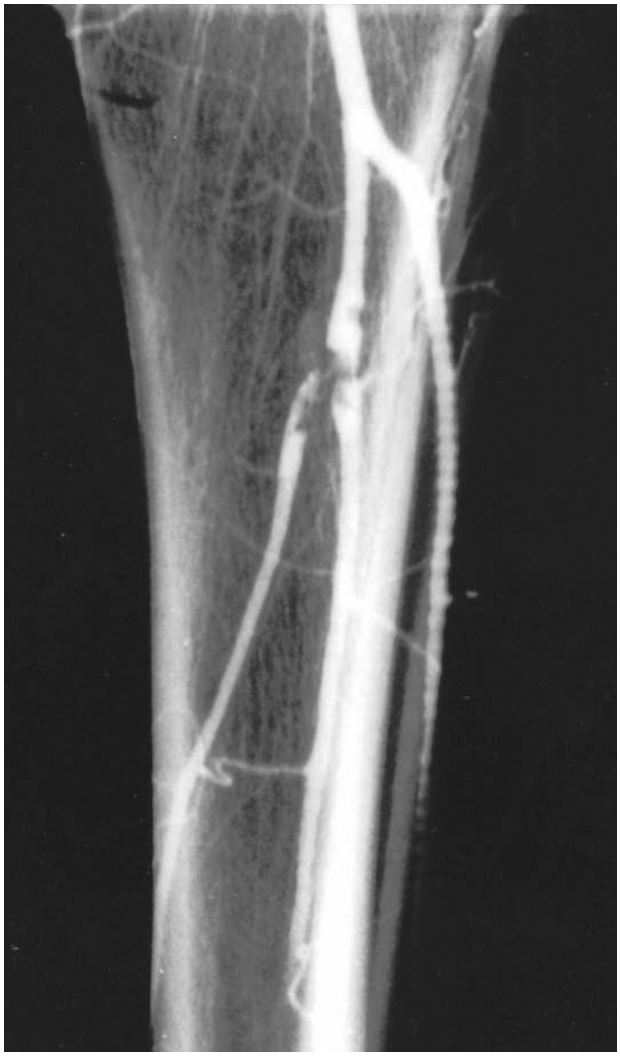

Figure 2.
Angiogram demonstrating occlusion of the right tibeo-peroneal trunk.

and, 20 min later, her left foot had improved, with resolution of the paralysis and paresthesias, though no pulse was palpable at the left ankle. The ankle brachial index was 0.49 on the left, and 0.70 on the right. Prior to emergent arteriography, a chest radiograph showed evidence of bilateral pleural effusions and mild cardiomegaly. Though the patient denied any shortness of breath or chest discomfort, her heart rate had increased to 130 beats per min, and her saturation of oxygen had decreased, requiring supplemental oxygen.

An aorto-femoral runoff showed bilateral arterial embolization, with occlusion of the left popliteal artery (Fig. 1), and the right tibio-peroneal trunk (Fig. 2). Thrombolysis was considered, but by then the symptoms of limb ischemia had resolved, albeit that her respiratory status had progressively deteriorated. A new arterial blood gas showed a mild respiratory alkalosis and profound hypoxemia. This was a marked change from 2 h earlier, when her saturation of oxygen had been 98%. Concerned that the patient may be manifesting pulmonary embolization, and paradoxical arterial embolization, we performed venous duplex interrogation of the legs, along with a pulmonary angiogram. No deep venous thrombosis or pulmonary embolization were identified. Five hours after admission to the emergency department, the patient was transferred to the intensive care unit and intubated for worsening hypoxemia. The patient then became hypotensive, requiring insertion of a Swan-Ganz catheter and vasopressor support. A transthoracic echocardiogram revealed an ejection fraction of 30%, global hypokinesis, and no intracardiac thrombus. A diagnosis of peripartum cardiomyopathy was made based on these findings coupled with her post-partum state. A transesophageal echo performed 12 h later confirmed a cardiomyopathy without intracardiac thrombus or septal defects.

https://doi.org/10.1017/S1047951103000416 Published online by Cambridge University Press

Over the next 24 h, the patient became hemodynamically stable. She was extubated 72 h after admission. No attempt was made to address the embolization of her legs because of her acute cardiac decompensation. Her legs were asymptomatic and viable. She was maintained on a therapeutic infusion of heparin and, 5 days later, the foot and posterior tibial pulses were palpable at both ankles. Duplex ultrasonography revealed resolution of the embolus in the right tibioperoneal trunk, and recanalization of the occluded left popliteal artery. The patient was discharged on the eighth hospital day on warfarin, digitalis, and captopril. Evaluation for a hypercoagulative state was negative. Eight months after discharge, she is free of symptoms from arterial insufficiency in the legs. Vascular examination of the legs is normal, and she has no symptoms of congestive heart failure.

Discussion

Peripartum cardiomyopathy is a rare form of congestive heart failure, which can afflict women one month prior to, and up to six months after, childbirth. Suggestions have been made that the cardiomyopathy is infectious or immunologic, but currently the cause remains unknown.[1] Symptomatic left ventricular dysfunction is the usual presentation. Coronary arterial, valvular, and pericardial disease are notably absent.[1,2] The reported prognosis has ranged from excellent, with most women surviving, to very poor, with up to half the patients dying in heart failure in the first three months after delivery unless they undergo cardiac transplantation.[1,3] Chronic refractory cardiomyopathy can occur, or recurrent heart failure with subsequent pregnancies.[4,5] Up to half of those affected will recover, with good long term prognosis.[4,5]

The differential diagnosis for acute ischaemia of the leg in women after delivery includes an arterio-arterio embolus, thrombosis of a stenotic artery, paradoxical embolization, primary arterial thrombosis due to a hypercoagulable state, and arterial dissection.[6] The postpartum respiratory decompensation in our patient could have been the result of pulmonary embolism, amniotic fluid embolism, or the exacerbation of sub-clinical cardiac insufficiency due to an acquired or congenital cardiac lesion such as mitral stenosis, aortic stenosis, or idiopathic hypertrophic subaortic stenosis.[7,8] The medical history and physical examination were helpful in excluding some of these diagnosis. Angiography was instrumental in identifying the peripheral arterial embolus, and excluding the diagnosis of pulmonary embolization. Venous duplex interrogation of the legs, along with echocardiography, identified no deep venous thrombosis nor right-to-left intracardiac shunting, thereby ruling out paradoxical embolization. The diagnosis of the cardiomyopathy itself was elusive until the echocardiogram revealed diffuse cardiac hypokinesis.

Ischaemia threatening a limb is extraordinarily rare as the initial presentation of peripartum cardiomyopathy. This is surprising, given the frequency with which ventricular thrombus has been identified in such women at autopsy. Cerebral embolism and stroke have been reported as the initial manifestation.[9] Previous reports of acute ischemia of the leg in this setting described women with several months of known ventricular dysfunction prior to a peripheral embolic event.[10] We believe that intracardiac thrombus formed in our patient as a result of her acute global cardiac dysfunction, and embolized to her legs.

Treatment of severe thrombo-embolic events should follow standard clinical techniques in these patients. Lytic therapy may be less morbid than open surgical techniques in these circumstances if the severity of ischemia requires intervention and no intracardiac thrombus is evident. Though we had planned thrombolysis to treat our patient's arterial insufficiency, her rapidly deteriorating cardio-pulmonary condition took priority once her legs were no longer threatened. Delayed thrombolysis was not performed, since she regained her pulses apparently due to spontaneous recanalization.

Peripartum cardiomyopathy, therefore, is a disease process that can present as ischemia of the leg due to cardiac embolization. Its etiology remains unknown. A high index of suspicion, and recognition of this entity in the peripartum female, will facilitate the diagnosis and lead to timely and appropriate management.

References

1. Homans DC. Current Concepts – Peripartum Cardiomyopathy. New England J Med 1985; 312: 1432–1437.
2. Veille JC. Peripartum cardiomyopathies: a review. Am J Obstet Gynecol 1984; 148: 805–818.
3. Felker GM, Thompson RE, Hare JM, et al. New England J Med 2000; 342: 1077–1084.
4. Demakis JG, Rahimtoola SH, Sutton GC, et al. Natural course of peripartum cardiomyopathy. Circulation 1971; 44: 1053–1061.
5. Carvalho A, Brandao A, Martinez E, et al. Prognosis in peripartum cardiomopathy. Am J Cardiol 1989; 64: 540–542.
6. Finley BE. Acute coagulopathy in pregnancy. Med Clin North Am 1989; 73: 723–743.
7. Le Jemtel TH, Demopoulos L, Tavel ME. A problem of pulmonary edema developing postpartum. Chest 1994; 106: 1883–1884.
8. Demakis JG and Rahimtoola SH. Peripartum cardiomyopathy. Circulation 1971; 44: 964–968.
9. Hodgman MT, Pessin MS, Homans DC, et al. Cerebral embolism as the initial manifestation of peripartum cardiomyopathy. Neurology 1982; 32: 668–671.
10. Bassaw B, Ariyanayagam DC, Roopnarinesingh S. Peripartum Cardiomyopathy and arterial embolism. West Indian Med J 1992; 41: 79.