A 32-year-old second gravid woman without known cardiovascular risk factors underwent an elective cesarean delivery during the 33rd week of gestation because of intrauterine growth retardation secondary to placenta insufficiency. The latter was most likely related to a previously diagnosed symptomatic antiphospholipid syndrome. Twenty hours after an uncomplicated delivery, the patient reported a sudden onset of typical chest pain after a severe cough attack. Pulmonary embolism was ruled out by computed tomography. Chest pain had subsided 1 hour after onset following the initiation of medical therapy. The ECG showed transient, nonsignificant ST-segment changes (Figure 1) in the inferior leads, and cardiac enzymes were slightly elevated, reaching peak values 12 hours later. The diagnosis of subacute non–ST-elevation myocardial infarction (peak troponin I 2.240 μg/L [<0.010 μg/L], peak creatine kinase 1316 U/L [<170 U/L], and peak creatine kinase MB 87.9 μg/L [<4.0 μg/L]) was established, and the patient was transferred to the coronary care unit. Transthoracic echocardiography showed a modest inferolateral hypokinesia with preserved global systolic left ventricular function (Movie I in the online-only Data Supplement). The patient underwent coronary angiography 16 hours after the onset of chest pain with the intention to define the nature of the coronary event (atherothrombosis, thromboembolism, spontaneous dissection). The coronary angiogram revealed complete occlusion of a side branch of the first marginal branch with discrete signs of an embolic occlusion. The remaining coronary arteries were free of atherosclerosis (Figure 2A). The left ventricular angiography confirmed the echocardiographic findings (Movie IIA and IIB in the online-only Data Supplement). After crossing the occlusion with a guidewire, thrombus aspiration and balloon angioplasty of the side branch were performed without stent implantation. The aspirate consisted of a small piece of white solid tissue. The histological analysis of the sample revealed a solid necrotic tissue fragment (Figure 2B) in a small area with conserved mononuclear cells (Figure 2B, inset). The positive cytoplasmic immunohistochemical staining for human placental lactogen identified these cells as trophoblasts and the whole necrotic tissue fragment as a placental embolus (Figure 2C). Transesophageal echocardiography was performed to verify the expected intracardiac shunt. Because of inadequate Val-salva maneuver, it was not possible to exclude a patent foramen ovale); however, intracardiac shunts of other origin were excluded. Following a positive transcranial Doppler test, a right heart catheterization was performed without any evidence of either a patent foramen ovale by catheter exploration or a pulmonary arteriovenous malformation by pulmonary angiography.

Because the patient refused further investigations, she was discharged on oral anticoagulant therapy for the symptomatic
antiphospholipid syndrome without aspirin, because atherosclerosis was not present in the coronary arteries.

Acute myocardial infarction (AMI) in women of childbearing age is rare. Pregnancy, however, increases the risk of AMI 3- to 4-fold.1 The incidence of pregnancy-related AMI ranges between 1:16,000 and 35,000, and a mortality rate of 5% to 11% has been reported.2 The presence of antiphospholipid syndrome further increases the propensity for venous and arterial thromboses.3 Because the harvested embolus was mainly composed of necrotic placental tissue, the antiphospholipid syndrome may have played a minor role in the formation of the embolic occlusion of the coronary artery. Multiple ischemic infarctions of the placenta were caused by the antiphospholipid syndrome and resulted in placenta insufficiency. It is unknown whether these ischemic alterations of placental tissue facilitate embolic events during the peripartal period.

The most frequent causes of AMI during pregnancy are (in descending order) atherothrombosis, spontaneous coronary dissections, and thromboembolic events.1 Only a few case reports have indicated paradoxical embolism or spasm as the cause of AMI. Amniotic fluid is the most common source of embolism and reflects a catastrophic event that accounts for 5% to 10% of maternal peripartal deaths.4 In addition, trophoblastic cells and tissue have been identified in autopsies to be responsible for peripartal pulmonary embolisms. Sources of trophoblastic tissue other than the placenta can only be observed in the presence of a metastatic gestational trophoblastic disease such as choriocarcinomas, which is an extremely rare finding in advanced pregnancies and was not observed in this patient, including the histological investigation of the placenta.5

To the best of our knowledge, this is the first published case of acute peripartal myocardial infarction caused by a histologically documented placental embolus. A pathway of this paradoxical embolus through a patent foramen ovale, atrial septum defect, or pulmonary fistula was not found and the responsible right to left shunt site remained undefined.

In conclusion, coronary angiography with coronary aspiration may be indicated to define the etiology of a peripartal myocardial infarction.

Disclosures

None.

References

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Circulation. 2011;124:e26-e27
doi: 10.1161/CIRCULATIONAHA.110.015735
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2011 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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