Postpartum Acute Myocardial Infarction Induced by Ergonovine Administration

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Abstract

We report a primigravida woman with acute myocardial infarction caused by coronary artery spasm induced by intravenous administration of methyl ergometrine maleate just after delivery. Despite the frequent usage of ergot derivatives to promote uterine contractions, cardiac complications related to this drug are rare. Myocardial infarction may be overlooked in young women in the early postpartum period. Careful monitoring and prompt evaluation should be performed when this drug is administered for obstetrical purposes.

Key words: myocardial infarction, pregnancy, vasospasm, ergonovine

Introduction

Ergonovine is known to induce coronary artery spasm and is typically used in cardiac catheterization laboratories for provoking vasospasm. However, this agent may precipitate acute myocardial infarction in some patients by induction of prolonged spasms. While ergot derivatives are frequently administered after delivery to promote uterine contractions, serious ischemic cardiac events related to this drug have rarely been described. We report a case of acute myocardial infarction just after delivery caused by coronary artery spasm induced by methyl ergometrine maleate administered intravenously.

Case Report

A 25-year-old woman primigravida was admitted to our hospital with threatened premature labor at 35 weeks of gestation. She was healthy and had no coronary risk factors such as hypertension, diabetes mellitus, hyperlipidemia, obesity, coagulative disorder, or smoking, with the exception of her family history. Her mother had angina pectoris, but it was not confirmed by cardiac catheterization. She had a passive smoking history in her family and occupational environment. She had no history of migraine headache. After admission, tocolytic treatment with ritodrine hydrochloride was given intravenously for seven days. However, at 36 weeks, her cervix dilated, and a healthy boy was delivered vaginally. Due to excessive uterine bleeding, methyl ergometrine maleate (0.2 mg) was administered intravenously. Within minutes, the patient complained of chest oppression, palpitation, and nausea. Chest oppression and nausea persisted for 4 hours and were treated symptomatically. An internal medicine consultation was obtained 5 hours after the onset of chest oppression, and an electrocardiogram at that time showed deep ST segment depression in anterior precordial leads (Fig. 1). The ST segment depression was not improved by nitrate administration. Serum activity of creatine phosphokinase (CK) peaked (928 IU/l, CK-MB isoenzyme; 66 IU/l) 18 hours after the onset of symptoms. Echocardiography revealed akinesis of anteroseptum. She was diagnosed with anteroseptal myocardial infarction. Pulmonary congestion was not noted. She was treated with intravenous nitrate, but thrombolytic and anticoagulation therapies were withheld immediately after delivery. Her recovery was uncomplicated.

After the onset of acute myocardial infarction, we attempted to obtain her medical history in detail, and discovered that she had felt chest oppression at rest early in the morning, which quickly disappeared, three times a year since she was 23 years old. Whether her symptom was angina pectoris or not was unclear, and had not been documented by electrocardiography.

Three months after myocardial infarction, cardiac cathe-
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Figure 1. Time course of electrocardiogram. A. At 22 years old 3 years before pregnancy. B. 5 hours after the onset of acute myocardial infarction. C. 24 hours after the onset of acute myocardial infarction. ST segment depressed in precordial leads (B). Transient left axis deviation probably due to left anterior hemiblock (B) and T wave inversions (coronary T) were observed (B).

terization was performed, which revealed normal coronary arteries (Fig. 2). Left ventriculography showed hypokinesis of segments 2, 3 and 5 (AHA classification), and left ventricular ejection fraction was 58%. Although we performed a provocative test by intravenous administration of ergonovine (the total amount 0.2 mg, but not administered selectively into coronary artery), coronary artery spasm was not induced. However, in this case, these findings strongly suggested that postpartum acute myocardial infarction was caused by coronary artery spasm induced by methyl ergometrine maleate. We followed her up with diltiazem and angina attacks were controlled, however, she stopped coming to our hospital eight months after delivery.

After 4 years, at the age of 29 years-old, she was referred to our hospital again for her second pregnancy. At 15 weeks' gestation, she felt chest oppression at rest early in the morning. From then on she had the same symptom four times a month. Electrocardiography revealed ST segment depression in the precordial leads (Fig. 3). She was diagnosed with vasospastic angina for the first time, and oral nitrates (slow-release isosorbide dinitrate) were administered. Although increasing experience with the use of a calcium antagonist during gestation has shown the safety of this drug, we chose oral nitrates (slow-release isosorbide dinitrate) as the first medication, since no known adverse effects of this therapy during pregnancy have been reported (1). At 27 weeks of gestation, her attacks occurred more frequently. We added slow-release nifedipine (10–20 mg) twice daily to her medication, but her attacks were not completely controlled.

At gestational age of 38 weeks, cesarean delivery was performed under epidural anesthesia with bupivacaine hydrochloride. She delivered a normal boy weighing 2,958 g. Due to uterine bleeding, dilute synthetic oxytocin was administered. Neither ergonovine nor prostaglandins were administered. The puerperium was uneventful. In the postpartum period, her attacks were completely controlled by slow-release nifedipine (10–20 mg) and slow-release isosorbide
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Figure 3. Electrocardiogram on second pregnancy. A. At 13 weeks gestation. 1. At 15 weeks gestation during angina attack. The ST segments were depressed in precordial leads. Peaking of T waves and inverted U waves were apparent in precordial leads, suggesting severe ischemia of anterior septum.

dinitrate 20 mg twice daily. A provocative test of coronary artery spasm was not done after the second delivery.

Discussion

Ergot derivatives are widely used in cardiac catheterization laboratories to elicit coronary artery spasm, but this agent may precipitate prolonged spasms, resulting in acute myocardial infarction. Recently, because of its short duration of action, acetylcholine has been preferred as the agent for provocation of coronary artery spasm. While ergot derivatives are routinely administered after delivery or abortion in order to promote uterine contractions, cardiac complications related to this drug are rare (2–8). Including the present case, there were 9 reported cases of myocardial infarction after postpartum administration of ergot derivatives. In these reports, postulated risk factors of coronary artery spasm by ergot derivatives include age greater than 30, smoking, a family history of ischemic heart disease, and a history of migraines. The present patient had a family history of angina pectoris. Including our case, 7 of 9 cases were Asians. This observation is compatible with the higher incidence of variant angina among Asians than Caucasians (9, 10).

Except for coronary artery spasm, it was reported that the etiologies of acute myocardial infarction during pregnancy were atherosclerotic disease, thrombus, coronary artery dissection, Kawasaki disease and collagen vascular disease. Atherosclerotic disease was found in less than half of the patients in whom the coronary anatomy was investigated (1). In this case, there were no abnormalities in the coagulative system including protein C and protein S, but the hypercoagulable state of pregnancy might induce coronary artery thrombus under transient coronary artery spasm. Pregnancy-related coronary arterial dissections occurred postpartum in most cases (1). It has been suggested that angiographically normal coronary arteries seen in patients with peripartum acute myocardial infarction may represent a healed or spontaneously repaired coronary dissection (11).

Before the onset of acute myocardial infarction just after the first delivery, the present patient felt chest oppression, but whether this symptom was caused by vasospastic angina or not was not clear. During her second pregnancy, electrocardiograms documented her attacks as vasospastic angina for the first time. She might initially have had vasospastic angina. There is no report of myocardial infarction after postpartum administration of ergot derivatives and later diagnosed with vasospastic angina.

There are fewer than 30 cases of pregnancy after myocardial infarction reported in the literature (12, 13). Regarding the mode of delivery, vaginal delivery is not contraindicated, and cesarean delivery should be performed for obstetric indications, and in situations that are life-threatening to the mother and cannot be corrected immediately (1, 12). In our case, angina attacks were not completely controlled medically before her second delivery. In addition, we speculated that hyperventilation at the time of vaginal delivery might induce coronary artery spasm. Therefore, we performed cesarean delivery under epidural anesthesia.

There is also an important problem selecting which agent should be administered to promote uterine contraction in such cases. There are reports that not only ergonovine but also prostaglandins induce coronary artery spasms (14, 15). Dilute synthetic oxytocin may be recommended because it causes no adverse hemodynamic effect (16).

Recently, it was reported that estrogen has both rapid and long-term effects on the blood vessel wall. The short-term coronary vasodilatory effects of estrogen in humans are primarily mediated by the increased production of nitric oxide (17). Generally, in pregnancy, the serum level of estrogen is markedly increased. If estrogen can cause short-term vasodilation, vasospastic angina attack may be decreased during pregnancy. However, in this case, angina attacks were increased at 15 weeks and 27 weeks of gestation. Therefore, the relation between estrogen and coronary artery spasm is unclear.

Myocardial infarction may be overlooked in young women in the early postpartum period, because it is uncommon, and symptoms may be vague during this time. However, ergot derivatives, even in obstetrical patients, may induce coronary artery spasm resulting in acute myocardial infarction. We should carefully obtain a detailed medical history of patients. Careful monitoring and prompt evaluation should be performed when this drug is administered to pregnant women, particularly in those with a history of chest pain.

References