Cardiac Magnetic Resonance Imaging in a Patient With Amniotic Fluid Embolism Associated With Severe Cardiopulmonary Complications

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Summary

Amniotic fluid embolism (AFE) is a rare but devastating complication of pregnancy. Acute circulatory failure and obstetric disseminated intravascular coagulopathy are often associated with AFE and lead to poor prognosis of this syndrome. Although many reports of AFE and its cardiopulmonary complications exist, their etiology remains unknown. Classically, it was believed that the fatal cardiopulmonary complication in AFE is due to acute and severe pulmonary hypertension caused by critical obstruction of the pulmonary vessels by embolized amniotic fluid. However, recent hypotheses are suggesting that anaphylactic reaction or a cytokine effect induced by amniotic fluid is the main pathophysiological mechanism. We report a case in which cardiac magnetic resonance imaging was performed at the chronic stage of AFE. Late gadolinium enhancement (LGE) was detected at the mid-wall of the left ventricle with no evidence of pulmonary hypertension. This finding suggests that the pathophysiological mechanism of severe cardiac complications in AFE may include direct left ventricular myocardial injury through an immune reaction or cytokine release, rather than pulmonary embolism. (Int Heart J 2013; 54: 119-122)

Key words: Late gadolinium enhancement

Since amniotic fluid embolism (AFE) was first reported by Meyer in 1926, it has become acknowledged as an obstetric complication associated with a high mortality rate. The main causes of poor prognosis are disseminated intravascular coagulation (DIC) and acute circulatory failure. Despite early recognition and intensive treatment, the mortality rate remains high. The pathophysiology of AFE has not been completely clarified to date, and a recent hypothesis that considers this syndrome as either a hormonal or immunological response is increasingly favored, as opposed to the conventional theory of physical obstruction of the pulmonary circulation by amniotic fluid. We present a case of AFE with severe cardiopulmonary complications. Cardiac magnetic resonance imaging (MRI) in the chronic stage showed interesting and valuable findings with regard to further investigation of the pathophysiological mechanism.

Case Report

A healthy 40-year-old woman in the 36th week of her first pregnancy visited the emergency room complaining of back pain and bloody vaginal discharge. Her blood pressure and oxygenation level were normal on arrival and her gynecological examination was unremarkable. Soon after, she lost consciousness and her blood pressure dropped, and she entered a pulseless state. Although rapid infusion of saline was initiated immediately, systolic blood pressure could not be increased to > 60 mmHg and fetal bradycardia was detected. Electrocardiography showed a normal sinus rhythm with slight ST elevation at the V1 and V2 leads, ie, no evidence of cardiogenic shock (Figure 1A). Uterine rupture or AFE was strongly suspected and an emergency cesarean section was performed under mechanical ventilation and catecholamine support. Parturition was accomplished with difficulty, and there was no evidence of uterine rupture. Nevertheless, uterine bleeding became uncontrollable after delivery. Her vital signs deteriorated due to hemodynamic collapse, accompanied by continuing ventricular tachycardia and cardiopulmonary arrest. Spontaneous recovery of circulation was not achieved for 1 hour despite cardiopulmonary resuscitation, electrical cardioversion, the administration of large amounts of catecholamine, and blood
transfusion. Moreover, adequate oxygenation could not be achieved even with mechanical ventilatory support, because of severe pulmonary edema. Percutaneous cardiopulmonary support (PCPS) and intra-aortic balloon pumping (IABP) were therefore instituted via a bilateral femoral approach. The circulation recovered spontaneously under the support of these mechanical devices, while severe hemorrhage from the abdominal cavity and lungs continued owing to the accompanying DIC. After transfer to the intensive care unit, massive blood transfusion and multifaceted treatment for DIC were continuously required.

Electrocardiography immediately after the surgery showed QT prolongation and poor R progression at the precordial leads (Figure 1B), while echocardiography showed systolic function of the left ventricle to be severely impaired (ejection fraction 15%). Serum cardiac enzyme was mildly elevated (93 U/L of Creatine kinase-Mb) and smoothly normalized in one day, while C-reactive protein rose to 20.78 mg/dL and gradually normalized in 4 weeks.

Cardiac systolic function rapidly recovered to within normal limits within a few days, and cardiopulmonary instability was reversed in the presence of the severe, ongoing bleeding tendency. The patient was weaned off PCPS and IABP on the 8th day after admission, and the ventilator support was removed on the 13th day. Several weeks were required for full recovery from DIC and abdominal edema, and the patient was discharged on the 33rd day. From the clinical progression observed, a retrospective diagnosis of amniotic fluid embolism was made.

Follow-up echocardiography was almost normal immediately before discharge. QT prolongation and poor R progression had disappeared, and were replaced by a negative T wave at all chest leads (Figure 1C). However, the level of brain natriuretic peptide (BNP) remained high (119.5 pg/mL).

No chest symptoms were noted at follow-up examination 6 months after discharge. BNP level and electrocardiography were fully normalized (Figure 1D), and echocardiography showed no abnormal findings. However, cardiac MRI (1.5T system of Achieva, Phillips) showed linear delayed enhancement at the mid-wall of the left ventricle, indicating residual myocardial injury, which suggested the need for further follow-up examination (Figure 2).

**Discussion**

Amniotic fluid embolism is known as a perinatal complication with a high mortality rate. This syndrome presents with the following clinical features: sudden cardiopulmonary failure, coma, and obstetric DIC. The diversity of symptoms makes the clinical diagnosis of AFE difficult, and histological detection of amniotic fluid elements in the maternal pulmonary vascular bed is essential for confirmation of diagnosis.

Cardiopulmonary complications, including severe hypoxia and systemic circulation collapse, are the chief causes of poor prognosis in AFE. The patient presented in this report also showed severe cardiopulmonary complications and only narrowly survived. Cardiac MRI for evaluation of residual myocardial damage performed 6 months after discharge showed LGE in the mid wall of the left ventricle. This lesion of myocardial injury as indicated by LGE was limited to the left ventricle, and the right ventricle showed no abnormality. Cardiac function was normal, and there were no other abnormal findings even by T2-weighted enhancement or stress perfusion.
It was believed until recently that the major hemodynamic change in AFE is acute and severe pulmonary hypertension due to critical obstruction of the pulmonary vessels by embolized amniotic fluid. However, the cardiovascular events in certain AFE cases cannot be explained by “classical” embolism of the amniotic fluid in the pulmonary vasculature. For example, the occurrence of AFE during first-trimester abortions suggests that maternal exposure to even very small amounts of amniotic fluid can initiate the syndrome.

A more recent hypothesis is that amniotic fluid contains a direct myocardial depressant and that leakage of amniotic fluid into the maternal circulation can lead to hemodynamic instability through the deterioration of left ventricular myocardial function. Endothelin, which has a powerful vasoconstrictor effect on the coronary and pulmonary arteries, has been implicated as a possible etiological factor. Meanwhile, various authors have suggested that other humoral factors including proteolytic enzymes, histamine, serotonin, prostaglandins, and leukotrienes may also contribute to the hemodynamic changes and consumptive coagulopathy associated with amniotic fluid. These mediators have been implicated in other shock states, in particular, anaphylaxis and sepsis. In these shock states, a foreign substance within the circulation releases various mediators, which induce DIC and profound myocardial depression, leading to hemodynamic crisis, which has a similar clinical appearance to that described for AFE. Indeed, there was a histopathological study in patients with LV dysfunction and septic shock, and it revealed a contraction band necrosis in the left ventricular wall. These similarities suggest that AFE and other shock states may share common pathophysiological mechanisms.

Meanwhile, Sinicina, et al demonstrated an increase in the number of pulmonary mast cells and elevation of serum mast cell tryptase in cases of AFE. These findings also indicate an anaphylactic reaction to fetal antigens and could explain how a minimal amount of amniotic fluid can lead to fatal hemodynamics with no accompanying pulmonary vascular obstruction.

The cardiac MRI findings in the present case seem compatible with such speculation, ie, left ventricular myocardial injury not caused through acute pulmonary hypertension or right ventricular overload.

MRI was useful in the detection of irreversible myocardial injury. At present, LGE is thought to enable the visualization of myocardial fibrosis associated with various cardiomyopathies, whereas T2-weighted sequences are used in the detection of edema and inflammation.

The finding of a scar on the mid-wall of the left ventricle shows a marked similarity with those observed in peripartum cardiomyopathy (PPCM). The etiology of PPCM is not yet clear, but it is considered to arise from several pathophysiological mechanisms, including abnormal immune response, cytokine-mediated inflammation, and abnormal hormonal function. These mechanistic theories concerning PPCM bear some resemblance to those of AFE. From these findings, there appear to be many characteristics in common between AFE and PPCM, such as perinatal onset and MRI findings of left ventricular injury, both of which suggest that there may be a common pathophysiological mechanism, eg, anaphylactic reaction.

This is the first report on the use of cardiac MRI in AFE, although its diagnostic value remains to be evaluated and will require further examination. It would seem logical in the quest for more concise clarification of the mechanism of cardiac dysfunction in AFE to follow cases carefully over a number of years, with appropriate re-examination by MRI in addition to electrocardiography and echocardiography.

Figure 2. Cardiac MRI performed 6 months after discharge showing delayed linear enhancement at the mid-wall of the left ventricle.
REFERENCES