Acute ST-Elevation Myocardial Infarction During Labor Due to Amniotic Fluid Embolism

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**Financial support:** None declared

**Conflict of interest:** None declared

**Patient:** Female, 30-year-old

**Final Diagnosis:** Amniotic fluid embolism • disseminated intravascular coagulation • STEMI

**Symptoms:** Cardiorespiratory arrest • convulsions • hemorrhage

**Medication:** —

**Clinical Procedure:** —

**Specialty:** Anesthesiology • Cardiology • Obstetrics and Gynecology

**Objective:** Rare disease

**Background:** Amniotic fluid embolism (AFE) is an extremely rare, life-threatening complication of labor that leads to hyperacute induction of inflammation and disseminated intravascular coagulation (DIC). Usually, acute pulmonary hypertension results in acute right ventricular failure, while DIC manifests by hemorrhagic and ischemic complications, ultimately leading to multi-organ failure and death.

**Case Report:** A 30-year-old primigravida and primipara woman with no prior medical history was admitted for labor after intrauterine fetal death at 37 weeks of gestation. After medical birth induction, she had a convulsive seizure and cardiorespiratory arrest. Short mechanical resuscitation was performed before spontaneous circulation returned. Simultaneously occurring severe vaginal hemorrhage and an ST-elevation myocardial infarction (STEMI) triggered the diagnosis of AFE. Laboratory results fulfilled the criteria for DIC, and hemostatic resuscitation and mechanical hemostasis were performed. Transesophageal echocardiography revealed hypokinesia to akinesia of the inferior wall. Owing to the ongoing DIC, coronary angiography could not be performed. After the patient's transfer to the Intensive Care Unit, ST-segment elevations resolved and the myocardial infarct was managed medically. Cardiac magnetic resonance imaging performed 3 months later demonstrated myocardial scarring in 2 different areas. Referring to the coronary artery anatomy in a computed tomography scan of the chest, the infarcted areas correlated with 2 different coronary supply territories.

**Conclusions:** AFE should be considered in women with acute cardiorespiratory failure during labor. This is the first report of a STEMI triggered by an AFE. The 2 separate areas of infarction, corresponding to the 2 different coronary territories, suggest an AFE-related thrombotic/thromboembolic etiology.

**Keywords:** Acute Coronary Syndrome • Disseminated Intravascular Coagulation • Hemorrhage • Pregnancy • ST Elevation Myocardial Infarction

**Full-text PDF:** https://www.amjcaserep.com/abstract/index/idArt/936653
Background

The most common differential diagnoses for cardiac arrest during or shortly after delivery include acute pulmonary embolism, air embolism, eclampsia, and peripartum cardiomyopathy, as well as septic, anaphylactic, or anesthetic shock due to spinal anesthesia. Less common causes of cardiorespiratory failure are aortic dissection, tension pneumothorax, and acute coronary syndrome [1]. Even though amniotic fluid embolism (AFE) is very rare (2 to 8 in 100 000 deliveries), it has a high mortality rate of at least 20% and needs to be considered, particularly since survival can only be achieved by immediate and aggressive treatment [2]. Amniotic fluid, lanugo hair, fetal cells, and other fragments enter the mother's circulation and partially obstruct the pulmonary circulation. As some parts can pass the pulmonary capillaries and thus enter the systemic circulation, disseminated intravascular coagulation (DIC) is triggered, leading to the devastating clinical syndrome. AFE is often sudden and dramatic, sometimes preceded by chills, nausea, or agitation right before the onset. While seizures are a rare symptom [3], the classic presentation is a combination of cardiorespiratory failure with respiratory distress and thrombotic or hemorrhagic complications, due to DIC. Cardiorespiratory compromise can lead to cardiac arrest requiring cardiopulmonary resuscitation, intubation, ventilation, and sometimes extracorporeal membrane oxygenation. The hyperacute coagulopathy and hemorrhage can only be managed by urgent and extensive transfusion of blood products. If the patient is still pregnant at the onset of AFE, urgent delivery is indicated [4].

ST-elevation myocardial infarction (STEMI) is characterized by complete occlusion of a coronary artery, usually triggered by rupture of an atherosclerotic plaque and subsequent local thrombus formation. Rarer causes include coronary dissection, coronary spasm, and embolic events. Pregnancy-associated myocardial infarction accounts for 20% of maternal cardiac death. In contrast to the mechanisms for coronary artery disease in the general population, the most frequent mechanisms for coronary artery disease in pregnant women are nonatherosclerotic (43% spontaneous coronary artery dissection, 18% normal coronary arteries) [5]. In addition, Takotsubo cardiomyopathy can mimic a STEMI, even though it does not involve coronary occlusion or myocardial scarring [6].

This report is of a 30-year-old primigravida woman with intrauterine fetal death at 37 weeks of gestation, induction of labor, amniotic fluid embolism, and acute STEMI.

Case Report

A 30-year-old primigravida woman at 37 weeks’ gestation with no prior medical history was admitted for labor induction after intraventricular fetal death due to true umbilical cord knot. Birth was induced by the administration of mifepristone 600 mg and misoprostol 125 mcg. To provide anesthesia, an epidural catheter was placed. The patient's blood pressure, oxygenation level, and body temperature were normal. During the second stage of labor, the patient had dizziness followed by emesis and a convulsive seizure. Based on a differential diagnosis of eclampsia, magnesium was administrated intravenously (MgSO4 4 g over 15 min). At the onset of cardiorespiratory arrest, immediate cardiopulmonary resuscitation was performed. After 3 episodes of pulseless electrical activity, bridged in each case with 5 cycles of chest compressions and invasive ventilation, return of spontaneous circulation was achieved. In anticipation of a possible postpartum hemorrhage, blood products for mass transfusion protocol were ordered.

An instantaneously recorded 12-lead electrocardiogram showed marked ST-elevation in II, III, aVF, and V6 consistent with an inferolateral STEMI (Figure 1). At the same time, the baby was delivered by forceps extraction, and severe postpartum hemorrhage led to the suspected diagnosis of AFE. Since transfer to the catheter laboratory at this point was impossible, echocardiography was ordered. Due to poor transthoracic image quality (Video 1), transesophageal echocardiography was performed and revealed a globally hyperkinetic left ventricle (LV) with hypokinesia to akinesia of the inferior wall (Video 2). The right ventricle was of normal size and normal systolic function. No signs of aortic dissection or valvular heart disease were detected. This suspicion of AFE was confirmed by coagulation and fibrinolysis markers: thromboelastometry and coagulation essays showed completely abolished clotting and fulfilled the criteria for DIC (platelets 97×10^9/L [150-400×10^9/L], D-dimers 4154 ng/L [<250 ng/L], prothrombin time [11-12.5 s] not quantifiable, and fibrinogen level 0.35 g/L [2.0-4.0 g/L]) [7], and the diagnostic criteria for AFE according to the Society for Maternal-Fetal Medicine were fulfilled [4]. No AFE-specific laboratory tests were performed. During the following hours and under close monitoring with thromboelastometry and repeated coagulation assays, correction of the plasmatic coagulation was achieved by infusion of high doses of fibrinogen (12 g), human coagulation factor XIII (1250 E), and protrombin concentrate complex (4500 IE), as well as with antifibrinolytic therapy, with a total of 3 g tranexamic acid. Additionally, 1 unit of thrombocytes and 4 units of packed red blood cells were transfused to avoid a hemoglobin level below 70 g/L under persistent bleeding.

An amount of 25 IE of oxytocin was slowly administered intravenously, and the uterus contracted adequately. As a local hemostatic measure, a Bakri balloon was installed in uterus. A full-body computed tomography scan was performed to rule out occult bleeding complications and pulmonary embolism.
Because of DIC in combination with severe vaginal hemorrhage, coronary angiography could not be performed. Within the first hours after the patient was transferred to the Intensive Care Unit, ST-segment elevations resolved. The cardiac biomarkers initial high-sensitivity cardiac troponin T (hs-cTnT) 135 ng/L [0-14 ng/L] and creatine kinase-myoglobin binding (CK-MB) 3.6 μg/L [0-9 μg/L] peaked after 11.5 h (peak hs-cTnT 6535 ng/L, peak CK-MB 49.7 μg/L). Low-dose aspirin (100 mg once daily), an angiotensin converting enzyme inhibitor (lisinopril 5 mg once daily), and prophylactic heparin therapy were administered. The patient was extubated on the same day, with
subsequent normal oxygenation. The patient was initially dis-oriented but recovered within hours and exhibited no residual neurological impairment. Vasoactive agents were tapered off, as the patient stabilized under volume substitution.

Transthoracic echocardiography 2 days after cardiac arrest revealed a normal LV chamber dimension and wall thickness with a mildly reduced biplane ejection fraction (LVEF) of 51% (54-74%), with akinesia of the inferior wall and hypokinesia in the basal and midventricular inferolateral segments. The right ventricle was still normal in size and systolic function. There were no signs of pulmonary hypertension.

Cardiac magnetic resonance imaging performed 3 months after the hospitalization showed a preserved LVEF of 55%, with inferior and inferolateral akinesia of the basal segments. Late gadolinium enhancement revealed mostly transmural ischemia-related myocardial scarring in 2 different areas: mid inferolateral (25×22 mm) as well as apical inferior and midventricular inferolateral (31×27 mm, Figure 2). Those 2 myocardial infarction areas were not connected, thus representing 2 supply territories: 1 from the left circumflex coronary artery and 1 from either the right coronary artery or the very distal left anterior descending artery. A benign variant with separated origins of the left anterior descending and left circumflex coronary artery was observed. Adenosine stress and rest perfusion imaging showed no inducible ischemia. Aspirin and lisinopril were continued and the patient underwent cardiac rehabilitation.

At follow-up 6 months after the event, the patient had taken up regular physical exercise and was free from any symptoms. Echocardiography showed improved systolic LV function (3D-LVEF 70%, GLS -24% [-18%]) with only a small mid inferolateral hypokinetic area (Video 3).

After thorough counseling, the patient decided to become pregnant again and had an uneventful pregnancy and delivery 18 months after the incident.

Discussion

AFE is a very rare, hyperacute, and severe birth complication with a high mortality rate. A disruption of the maternal/fetal interface with intrusion of amniotic debris into the maternal circulation induces a hyperacute activation of proinflammatory mediators and the coagulation cascade, usually triggering DIC. In the present case, we postulate that intracoronary thrombus formation due to DIC led to myocardial infarction with ST-segment elevation and regional LV wall motion abnormality.
This is a remarkable presentation, since the commonly suggested mechanism of circulatory failure is acute pulmonary hypertension and subsequent right ventricular failure because of mechanical and thrombotic obstruction due to components of the amniotic fluid as well as an increase of pulmonary vasoconstrictors and DIC [8,9]. Other reports describe acute left heart failure, supposedly mediated by hypoxic injury due to hypotension and the release of inflammatory mediators [1], resulting in elevated cardiac biomarkers [10]. In addition, DIC usually manifests by hemorrhagic complications as well as by ischemic organ dysfunction, ultimately leading to multi-organ failure.

There is insufficient data to reliably determine the risk in subsequent pregnancies. However, no cases of recurrent AFE have been published, while several cases of uneventful pregnancies in patients with prior AFE have been reported [11].

As present in this case, labor induction with prostaglandins and intrauterine fetal death are risk factors for AFE [12]. The patient exhibited the characteristic triad of sudden hypotension and hypoxia in combination with severe coagulopathy during labor. The sudden onset of a seizure in combination with cardiorespiratory failure shortly after labor induction was typical for AFE.

Other differential diagnoses for the patient’s presentation were considered. A chest computed tomography scan showed no pulmonary embolism. The patient had no fever or other clinical signs of infection or elevated inflammatory markers, thereby making septic shock very unlikely. An anaphylactic shock would not explain the myocardial infarction and the very urgent and extensive need for blood products. Classical (atherosclerotic) coronary artery disease was also unlikely considering the patient’s low cardiovascular risk profile and the absence of ischemia in a follow-up cardiac stress MRI. Furthermore, an isolated acute coronary syndrome of any nature would not explain the whole clinical picture with coagulopathy and DIC.

The most likely cause of the acute STEMI was coronary thrombus formation in the context of the DIC. A post-mortem study in patients with DIC of other causes demonstrated fresh myocardial infarction in 8.7%, which were predominantly caused by coronary thrombosis (7%) [13]. Coronary artery dissection or vasospasm cannot be excluded completely. However, the clinical context and the fact that there were 2 separated infarcted areas support the hypothesis that the thrombi formed directly in the coronary artery. Expert consensus on the treatment of STEMI in pregnancy recommends invasive management [14]. However, in our case coronary angiography was not an option owing to the ongoing DIC. Nevertheless, the favorable outcome of our patient and the normal left ventricular systolic function at follow-up suggests that conservative management, perhaps restricted to cases where nonobstructive coronary artery disease is assumed, can be considered in specific situations.

Another AFE case report described slight ST-elevation not fulfilling the diagnostic criteria for STEMI and linear, non-transmural late gadolinium enhancement in the left ventricular myocardium at follow-up. Even though the case has interesting similarities, the described findings do not suggest occlusion of a coronary artery. In fact, cases of similar linear late gadolinium enhancement of the left ventricle have been found in patients after septic shock or peripartum cardiomyopathy [15]. AFE-related left ventricular failure without ST-elevation has been described [16], suggesting that left ventricular impairment in AFE can be mediated through different mechanisms than were present in our case.

Conclusions

AFE is a rare and often fatal peripartal systemic complication and should be considered in women with acute cardiorespiratory failure during labor. Only immediate correction of coagulation, mechanical hemostasis, and cardiorespiratory support can allow survival. This is the first report of a STEMI in AFE, which was most likely caused by intracoronary thrombus formation. This case demonstrates that in acute coronary syndromes in the setting of AFE and DIC conservative management can result in an excellent outcome.

Declaration of Figures’ Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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