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Atypical Presentation of Amniotic Fluid Embolism: A case report

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Abstract

Amniotic fluid embolism (AFE) is an acute, and unpredictable complication of pregnancy, with vital distress for the mother and fetus, that requires prompt recognition and supportive treatment .

We report a case of AFE after cesarean section ,at the moment of the cord clamping, in a 28-year-old woman ;resulting in respiratory distress; hemodynamic collapse; seizure and without coagulopathy. Diagnosis was made after excluding differential diagnoses.

Management included 100 % oxygen delivering with facial mask; hypovolemia correction is based on fluid resuscitation and low vasoconstrictors doses. A full recovery without sequelae was achieved in few minutes.

Some times; AFE leads to serious complications and death while in others it may leads to mild organ dysfunction, and quick recovery. However, mild forms should be recognized early to avoid complications. Multidisplinary experimented team combining anesthesiologist; obstetrician and neonatologist is essential to preserve maternal and fetal prognosis.

Keywords: Amniotic fluid embolism, diagnosis, management, Complications.

Introduction

Amniotic fluid embolism (AFE) is an acute, and unpredictable complication of pregnancy, with vital distress for the mother and fetus, occurring most often during labor or in immediate postpartum. **[1–3]**.

Case Presentation

A 28-year-old woman, with no pathological history, first gesture first par, was admitted for cesarean section on a 39-week term of gestation because of the presence of a grade 4 anterior placenta previa.

At the arrival in the operative room, vital signs were normal

After 500 cc of crystalloids given, she had spinal anesthesia. The cold sense check was performed to verify the sensory block level to the fourth thoracic spinal segment. A hysterectomy was allowed, and a 3370 g newborn was delivered with Apgar scores at 1 and 5 min at 7 and 9 respectively.

At the moment of the cord clamping, the patient felt vomiting and complained of dyspnea; O2 saturation decreased gradually, BP decreased to 45-65/20-30 mmHg, with an irregular pulse at 32 beat/min and parturient lost consciousness and convulsed. Her airway was managed with oxygen by facemask, and seizure activity stopped within 10– 15s without drug administration; at the same time a second peripheral intravenous access was obtained, ephedrine 12 mg and atropine 0.5 mg were injected. Then; the patient recovered to the sinus rhythm at 90b/min and BP at 124/75 mmHg and spontaneous breathing with SaO2 at 98 %. She was moved to the intensive care unit (UCI) where blood tests were performed **(Table 1)**; Electrocardiogram, bedside chest, cardiac ultrasound, chest angiography, brain scan were all normal. The woman continued to recover with no neurological deficit. She was discharged home on the 5thday.

Table 1: Laboratory data of the blood sample obtained 2 h after delivery.

Test	value	Normal values
WBC	17,9 10^9/L	(3.5 10^9–9.5 10^9/L)
RBC	3,25 10^{12}/ L	(3.8 10^{12}–5.1 10^{12}/L)
Hb	10,5 g/dL	(11,5–15g/dL)
Hct	30,7 %	(37 – 46 %)

Plt	110 10^9/L	(125 10^9–350 10^9/L)
PT	62 %	(75 %-100 %)
FIB	1,3 g/L	(2-4 g/L)
D-dimer	12.68 mg/L	(0.01–0.5 mg/L)
Troponin	734	(8-25)
Proteinuria	0g/24h	< 0.3g/24h

WBC: white blood; RBC: red blood cell counts; Hb: hemoglobin; Hct: hematocrit Plt: platelet counts; PT: prothrombin time; FIB: fibrinogen

Discussion

Physiopathology:

There are two theories explaining the pathogenesis of AFE:

A breach of the barrier between maternal blood and amniotic fluid allows the entry of amniotic fluid into the systemic circulation and results in mechanical obstruction of the pulmonary circulation.

Entry of amniotic fluid into the maternal circulation activates inflammatory mediators, causing a humoral or immunologic response. Amniotic fluid contains vasoactive and procoagulant products including platelet-activating factors, cytokines, bradykinin, thromboxane, leukotrienes, and arachidonic acid. Maternal plasma endothelin concentrations are increased by the entry of amniotic fluid. Endothelin is not only a bronchoconstrictor but also a pulmonary and coronary vasoconstrictor, which may contribute to respiratory and cardiovascular collapse. **[2,4,5]**

Clinical presentation:

The classic description of AFE includes sudden symptoms which involve many organ systems. The first phase is characterized by, respiratory distress, dyspnea, hypoxia, tachypnea, peripheral oxygen desaturation, decrease in end-tidal carbon dioxide in intubated patients, cyanosis, tachypnea, bronchospasm, pulmonary edema frequently and cardiovascular collapse and arrhythmia. Hypotension is the most frequent sign which occurs in 100 % of AFE.

Management

There is no specific treatment protocol unique to AFE. Anesthesiologist, neonatologist, and obstetrician must decide on a rapid cesarean section to deliver the baby. **[3,5,7]**

Hypoxemia must be managed by 100 % oxygen supplementation and airway management with tracheal intubation and positive pressure ventilation if necessary. **[3,6,10,11]**

A circulatory support must be accomplished by aggressive fluid resuscitation and vasopressor administration when necessary. Two large-bore intravenous cannulas and central venous access are recommended. **[3,6]**

Fluid administration needs to be monitored:

-An arterial catheter to monitor the blood pressure aiming a mean arterial pressure of greater than 65 mmHg. **[6]**

-Transthoracic or transesophageal echocardiography to manage fluid resuscitation and evaluate left ventricular filling.

-A pulmonary artery catheter, central venous pressure, pulmonary capillary wedge pressure, and pulmonary artery pressure to monitor cardiac output.

Encephalopathy seems to be a consequence of prolonged hypoxemia. The mental state is frequently altered, 10–50% of patients with AFE present with seizures. **[2,3,5,6]**

The second phase involves coagulopathy and hemorrhage. Disseminated intravascular coagulation (DIC) does not occur in all cases of AFE, **[5,7]** but causes the death of the patient even if appropriate cardiopulmonary resuscitation and bleeding management are performed. **[6]**

The last phase of AFE is characterized by tissue injury and end-organ system failure. **[8]**

Diagnosis:

The diagnosis of AFE is now based on the clinical features: hypoxia, hypotension, and coagulopathy occurring during labor; or within 30 minutes of placental delivery, and absence of fever **[6]**.

Atypical manifestations of AFE include isolated coagulopathy with either a sudden or a gradual onset **[9]**. In some patients, DIC or uterine atony may be the only sign, **[9,10]** however DIC does not develop in all cases of AFE. **[5]** Our patient seems to be an atypical case of AFE. Blood tests, chest radiography, and echocardiogram help, but they are nonspecific.**[9]**

Those investigations were performed for our patient and were all normal.

Urinary catheter to monitor urinary out pout aiming at least 0.5 mL/kg/h. **[3,6,9]**

During the resuscitation, left uterine displacement must be performed to decompress the inferior vena cava and improve preload and stroke volume until the baby will be delivered. **[9,12]**

Coagulopathy should be anticipated by blood and blood products administration including fresh frozen plasma (FFP), platelets, and cryoprecipitate. The optimal Red Blood cells (RBC) to FFP ratio is not known but given 1: 1–1.5 ratios is preferable. Blood tests such as complete blood count, electrolytes, coagulation studies, international normalization ratio, and fibrinogen must be repeated to evaluate the coagulopathy therapy. **[2,3,6]** The aim is to maintain a platelet count of over 50000/mm, a normal partial thromboplastin time, and a normal international normalization ratio[6]. The use of recombinant

activated factor VIIa should be restricted to patients where hemorrhage cannot be managed by blood product administration **[9]** and its efficacy is controversial. **[6,9]**

Uterotonics should be administrated early. Insertion of Bakri catheter into the uterus to stop blood loss could be tried. Arterial embolization can be used. Hysterectomy is the ultimate solution to control bleeding. **[3,5,9].**

Key message

AFE seems to be a catastrophic complication unique to pregnancy. Classic clinical presentation is characterized by the tirade made by hypoxemia, hypovolemia, and coagulopathy, but atypical forms are not exceptional. The early recognition, prompt resuscitation and quick extraction of the baby improve both maternal and fetal prognosis.

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All authors declared that the manuscript has been read and approved by all the authors, that the requirements for authorship as stated earlier in this document have been met, and each the author believes that the manuscript represents honest work if that information is not provided in another form.

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