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Amniotic Fluid Embolism: How Can We Avoid The Worst? A Case Report

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Abstract **Case Report**

Amniotic fluid embolism (AFE) is a catastrophic syndrome typically occurring during labor and delivery or immediately postpartum. The pathophysiology of this entity is incompletely understood. The diagnosis of AFE is primarily based on the clinical observations even if not uniform. The classic triad of sudden hypoxia, hypotension, and coagulopathy forms the hallmark of AFE diagnosis. The primary management goal includes rapid maternal cardiopulmonary stabilization with treatment of hypoxia and maintenance of vascular perfusion. We describe the clinical case of a 36-year-old patient admitted for emergency caesarean section for acute fetal distress under spinal anaesthesia. She presents a few minutes after fetal extraction a cardiovascular collapse, sudden respiratory distress and disturbance of consciousness on a massive amniotic fluid embolism. We discuss also the modalities of management of this serious complication.

Keywords: Amniotic fluid embolism, pregnancy, cardiopulmonary arrest, resuscitation.

INTRODUCTION

The amniotic fluid embolism (AFE) remains one of the most enigmatic and devastating conditions in obstetrics practice. It is classically characterized by hypoxia, hemodynamic collapse, and coagulopathy.

AFE is a catastrophic syndrome typically occurring during labor and delivery or immediately postpartum. Although rare, it has a high case fatality rate and remains a leading cause of maternal mortality [1].

We describe here the clinical case of a 36-year-old patient admitted for emergency caesarean section for acute fetal distress performed under spinal anaesthesia, who presents a few minutes after fetal extraction a cardiovascular collapse, sudden respiratory distress and disturbed consciousness on a massive amniotic fluid embolism, and we discuss the modalities of management of this serious complication.

CLINICAL CASE

Mrs. F.N is a 36-year-old parturient, fourth gestation and three living children, with no notable medical or surgical history. The current pregnancy is estimated at 39 weeks, the patient was admitted to the operating room for emergency cesarean section for acute fetal distress (Fig 1).

The preoperative anesthetic evaluation found no abnormalities, after conditioning and monitoring, a spinal anesthesia was performed with 10 mg of Bupivacaine and 25 µg of Fentanyl at the L3-L4 level. The hemodynamic and respiratory constants were normal.

The cesarean section allowed the extraction of a male neonate APGAR 6/10 passed to 10/10 after 2 min with a birth weight of 3200 g.

A few minutes later, the patient presented a respiratory distress, loss of consciousness, and cardiovascular collapse with BP at 60/40 mmhg and severe bradycardia at 30 bpm unresponsive to repeated doses of ephedrine before she presented with cardiorespiratory arrest recovered after intubation, 2 minutes of cardiac massage, and two boluses of 1 mg of intravenous epinephrine.

Closure was completed after careful hemostasis and additional monitoring with a left radial arterial line and a right jugular central venous line were performed with hemodynamic stabilization achieved without vasoactive drug support.

The patient was then transferred to the intensive care unit where she presented a postpartum hemorrhage that improved after transfusion of two red blood cells units. The blood tests did not reveal any abnormalities other than thrombocytopenia, a low prothrombin and fibrinogen level.

The postoperative period was marked by hemodynamic and respiratory stabilization and by a complete awakening allowing the patient to be extubated three hours later. The clinical and biological assessment of the following day did not reveal any abnormalities, and the patient was discharged from the ICU.

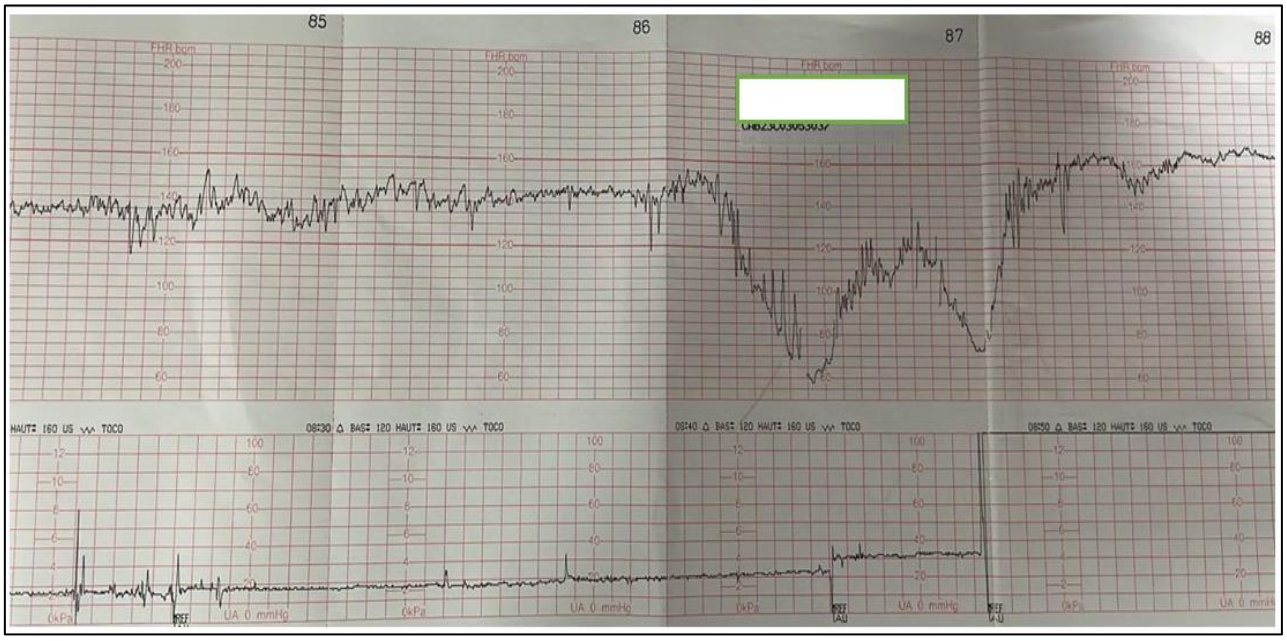

Fig 1: Fetal heart rate recording compatible with acute fetal distress

DISCUSSION

Amniotic fluid embolism (AFE) remains one of the most devastating conditions in obstetrics practice with reported mortality of 20% to 60%.

The pathophysiology of the amniotic fluid embolism is incompletely understood. Old studies assumed a simple, mechanical mechanism of injury, which results in obstruction of pulmonary arterial blood flow, such obstruction leads to hypoxia, right ventricular heart failure, and death. Actually, the mechanism seem to involve a complex sequence of reactions resulting from abnormal activation of pro-inflammatory mediators similar to the systemic inflammatory response syndrome (SIRS) [2].

Risk factors reported for AFE include situations in which the exchange of fluids between the maternal and fetal compartments is more likely, such as cesarean delivery, instrumental delivery, cervical trauma, placenta previa, and abruption. Association of induction of labor and AFE is inconsistently reported. Other reported risk factors include advanced maternal age and parity, male fetus, eclampsia, polyhydramnios, and multiple gestations [3, 4].

In our patient, the risk factors was cesarean delivery, advanced maternal age and male fetus.

Amniotic embolism remains a diagnosis of exclusion and should always be considered in any obstetrical emergency with initial cardiovascular collapse. Typically, AFE occurs during labor, delivery, or in the immediate postpartum [5].

The diagnosis of AFE is primarily based on the clinical observations even if not uniform. The classic triad of sudden onset of hypoxia, hypotension, and coagulopathy with onset during labor and delivery within 30 minutes postpartum forms the hallmark of AFE diagnosis [6].

Maternal death usually results from sudden cardiorespiratory arrest, massive hemorrhage, or secondary multi-visceral failure.

The first phase of shock is called obstructive, due to the embolism itself and which can be illustrated by an immediate cardio-circulatory arrest. In the initial stage, pulmonary hypertension and vasospasm lead to hypoxemia and right ventricular failure with mitral leak, increased pulmonary vascular resistance with normal

capillary pressures. The second phase is that of distributive shock, where a "septic shock like" appears with a major inflammatory response. At this stage, pulmonary hypertension usually disappears, to be replaced by left heart failure with increased capillary pressures and pulmonary edema leading to hypoxemia. Hypovolemia and secondary hemorrhage may aggravate this shock [7].

Like hypoxemia, neurological disorders are of multiple origin. Hypoxemia and low cerebral output secondary to cardiogenic shock explain the initial neurological disorders.

In our patient, the clinical presentation was dominated by cardiac collapse, respiratory distress, and postpartum hemorrhage.

Like the clinical symptoms, the results of the laboratory tests were non-specific. Leukocytosis may be observed. The diagnosis of DIC is based on a fall in PT, a fall in fibrinogen and/or the presence of thrombocytopenia. Hypoxemia is detected by arterial blood gas. Diffuse bilateral, heterogeneous or homogeneous nonspecific lung abnormalities may be seen on chest radiography. Transthoracic or ideally transesophageal echo (TEE) is useful for diagnostic.

Detection of squamous cells in the pulmonary artery bed in pregnant patients is not helpful for diagnosis of AFE. Such studies are not generally useful in either the diagnosis or exclusion of this condition [6].

Other substances in the peripheral blood have been suggested as markers of AFE: zinc-coproporphyrin complex, a component of meconium, Tn Syalin antigen, Tryptase and complement factors. However, not all of these tests are available in routine practice and their diagnostic value has yet to be confirmed.

In cases of maternal death, autopsy examination is essential to confirm the diagnosis of EA. Pathological examination of the lungs often shows pulmonary edema associated with atelectasis, as well as alveolar lesions with interstitial and endothelial edema and infiltration by alveolar macrophages. Elements of amniotic fluid-squamous cells, mucin, vernix caseum lipids, and lanugo can be seen separately or together in the small pulmonary arteries, but also in other organs [7].

However, in some cases of AFE, one or more components of this triad may be minimal or absent. In such cases, the diagnosis is more difficult, and one should consider other possible differential diagnoses as pulmonary thromboembolism, hemorrhage, anaphylaxis, high spinal anesthesia, air embolism, myocardial infarction, eclampsia or a septic shock.

The primary management goal includes rapid maternal cardiopulmonary stabilization with treatment of hypoxia and maintenance of vascular perfusion. This management may require endotracheal intubation to maintain adequate oxygen saturation [8].

Administration of vasopressor and inotropic drugs is usually necessary to treat arterial hypotension refractory to filling and to manage cardiogenic failure. If cardiopulmonary arrest occurs, maternal resuscitation should be prolonged. When the AFE occurs in the antepartum period, fetal extraction should be considered without delay, to improve not only the fetal prognosis, but also the maternal prognosis by increasing venous return [9].

Administration of blood products (Red blood cells, fresh frozen plasma, and platelet concentrates) and fibrinogen is also a central component of treatment for the management of coagulopathy in case of associated secondary hemorrhage. Administration of recombinant activated factor 7 (rFVIIa) is proposed for massive postpartum hemorrhage but its use remains controversial [10].

In our clinical case, the rapid and early management of the cardiorespiratory arrest that occurred in the operating room and the correction of the coagulopathy improved the prognosis.

Other therapeutic techniques have been successfully reported as clinical cases in AFE such as inhaled nitric oxide for the management of pulmonary hypertension and right heart failure. Maternal survival without sequelae has been reported after placement of ECMO and intra-aortic balloon pump for cardiac arrest secondary to AFE [11].

CONCLUSION

AFE remains a rare but serious syndrome of pregnancy for which the risk factors are not very predictive. The pathophysiology still incompletely understood, but the hypothesis of an immunological mechanism would prevail over the obstructive mechanism historically considered.

The diagnosis of AFE, which is mainly a diagnosis of exclusion and a clinical diagnosis, must be evoked very quickly in order to set up a very early and aggressive management, essential to the prognosis of these patients. The use of new therapies such as ECMO must be considered in the most severe cases.

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REFERENCES

1. Stolk, K. H., Zwart, J. J., Schutte, J., & Van Roosmalen, J. (2012). Severe maternal morbidity and mortality from amniotic fluid embolism in the Netherlands. *Acta obstetricia et gynecologica Scandinavica*, *91*(8), 991-995.
2. Dildy, G. A., Belfort, M. A., & Clark, S. L. (2010). Anaphylactoid syndrome of pregnancy (amniotic fluid embolism). In: Belfort, M., Saade, G., Foley, M. editors. Critical Care Obstetrics. 5th edition. Oxford (UK): Whiley-Blackwell; p. 466-474.
3. Clark, S. L. (2014). Amniotic fluid embolism. *Obstetrics & Gynecology*, *123*(2 PART 1), 337-348.
4. Pacheco, L. D., Saade, G., Hankins, G. D., Clark, S. L., & Society for Maternal-Fetal Medicine (SMFM. (2016). Amniotic fluid embolism: diagnosis and management. *American Journal of Obstetrics and Gynecology*, *215*(2), B16-B24. http://dx.doi.org/10.1016/j.ajog.2016.03.012.
5. Benson, M. D. (2017). Amniotic fluid embolism mortality rate. *Journal of Obstetrics and Gynaecology Research*, *43*(11), 1714-1718. doi:10.1111/jog.13445.
6. Shamshirsaz, A. A., & Clark, S. L. (2016). Amniotic fluid embolism. *Obstetrics and Gynecology Clinics*, *43*(4), 779-790. doi:10.1016/j.ogc.2016.07.001. PMID: 27816160.
7. Marie-Pierre, B., Christelle, S., & Anne, C. (2022). EMBOLIE AMNIOTIQUE Le Congrès Conférence d'actualisation © 2012 Sfar.
8. Sultan, P., Seligman, K., & Carvalho, B. (2016). Amniotic fluid embolism: update and review. *Current Opinion in Anaesthesiology*, *29*(3), 288-296. doi:10.1097/ACO.0000000000000328
9. Tamura, N., Farhana, M., Oda, T., Itoh, H., & Kanayama, N. (2017). Amniotic fluid embolism: pathophysiology from the perspective of pathology. *Journal of Obstetrics and Gynaecology Research*, *43*(4), 627-632. doi:10.1111/jog.13284.
10. Coggins, A. S., Gomez, E., & Sheffield, J. S. (2022). Pulmonary Embolism and Amniotic Fluid Embolism. *Obstetrics and Gynecology Clinics*, *49*(3), 439-460. doi: 10.1016/j.ogc.2022.02.015
11. Hynek, H., Adéla, T. F., Martina, V., Gabriela, D., Eliška, H., & Radovan, P. (2022). Amniotic fluid embolism-review and multicentric case analysis. *Ceska Gynekologie*, *87*(4), 261-268. doi:10.48095/cccg2022261