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RESEARCH ARTICLE

AMNIOTIC FLUID EMBOLISM: ABOUT A CASE

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Abstract

Amniotic fluid embolism is a rare and unpredictable complication of childbirth.with about fifteen cases per year in France, but very serious with maternal mortality that can vary from 20 to 60% , associating severe cardiovascular collapse, acute respiratory distress syndrome and hemorrhage with disseminated intravascular coagulation (DIC). As soon as the diagnosis is mentioned, management must be multidisciplinary and intensive. Amniotic embolism is responsible for such significant neonatal mortality. Over the past ten years, the maternal-fetal prognosis seems to have improved thanks to progress in standardized multidisciplinary care at the delivery site. We report a case of amniotic fluid embolism which occurred during fetal extraction during caesarean section.

Introduction:-

Amniotic embolism is the unpredictable passage of amniotic fluid into the maternal circulation. In most cases, it occurs at the time of labor during thechildbirth. The feto-placental elements pass into the maternal blood via the placental veins or during tears of the uterus or the cervix. According to estimates, amniotic embolism is the third cause of maternal mortality (12% of deaths), ie less than ten cases each year in France.

This passage of amniotic fluid into the maternal blood isresponsible for a picture combining respiratory, circulatory, neurological and hematological signs.

In the absence of a simple test, the diagnosis is based on a range of clinical arguments and on the elimination of differential diagnoses.

Diagnosing amniotic embolism is not easy. The autopsy, which reveals the presence of feto-placental elements in the lungs, is the only definitive diagnosis. The use of an autopsy is not systematic, in this case or when the patient is resuscitated, it may be useful to take maternal samples for diagnostic purposes. When the outcome has not been fatal, the diagnosis of orientation or exclusion calls upon the biochemical analysis of the blood and the cytological analysis of the bronchial secretions.

Patient And Observations:-

Mrs. EJ is a 30-year-old parturient, third act, third pare and two live children by caesarean section, with no notable medical or surgical history. child of CHU Mohamed VI in labor. The patient was admitted to the operating room in emergency because of the bi-scarred uterus. she was anesthetized under spinal anesthesia.

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The patient then presented with agitation with chest pain, generalized tonic-clonic convulsions and cardiopulmonary collapse with hypotension at 06/03 mmHg and respiratory distress at the time of the fetal extraction, the patient was intubated with the placement of vasoconstrictors in a peripheral vein for the time to take a central venous catheter, but his condition was complicated by the occurrence of cardiorespiratory arrest, resuscitation measures were carried out with immediate recovery. The occurrence of a 2nd cardiorespiratory arrest after 2 min of recovery was done and effective cardiac activity was regained after cardiac massage alone. the evolution was marked by the occurrence of a lightning haemorrhage with uterine inertia favored by a CIVD. the patient was transfused by 10 CG, 15 PFC,4CP and she benefited from an infusion of 3g of fibrinogen. A hysterectomy for haemostasis was performed after failure of conservative treatment but given the persistence of bleeding with diffuse haemorrhages both in the operating field and at the points of puncture, packing was done. Then the patient was admitted to the maternal intensive care unit intubated ventilated and sedated for additional care.

The evolution was marked by the extubation of the patient 24 hours after her admission with a recovery of consciousness without neurological sequelae and normalization of the biological assessment

The search for elements of the amniotic fluid was not carried out to confirm the presence of amniotic cells due to the lack of reagents at the hospital and due to the lack of means of the patient's family.

Discussion:-

Amniotic embolism is a rare obstetric emergency and is estimated to occur in approximately 2-6/100,000 pregnancies. 2nd trimester.

Although mortality estimates vary widely (from about 20 to 90%), the syndrome clearly establishes a significant risk, and amniotic fluid embolism is one of the most likely causes of sudden death during labor (1,2). Survival depends on early diagnosis and initiation of immediate treatment.

The pathophysiology of amniotic embolism is not clearly defined.The long-used term amniotic "embolism" implies a predominantly occlusive and mechanical disorder, as occurs in thromboembolism or gas embolism. However, since amniotic fluid is completely soluble in blood, it cannot cause obstruction. Additionally, the limited number of fetal cells and minimal amounts of tissue debris that may accompany amniotic fluid into the maternal circulation are too small to mechanically occlude the pulmonary vascular tree and cause the marked hemodynamic changes that occur in this syndrome. .

Instead, it is currently assumed that exposure to fetal antigens during childbirth activates proinflammatory mediators that trigger a catastrophic inflammatory cascade and release of vasoactive substances (eg, norepinephrine) similar to the syndrome systemic inflammatory response that occurs in thesepsis and septic shock.(2)

The inflammatory response causes damage to organs, particularly the lungs and heart, and triggers a clotting cascade, resulting indisseminated intravascular coagulation(DIC). The resulting maternal hypoxia and hypotension have profound negative effects on the fetus.

Since maternal exposure to fetal antigens is likely quite common during labor and delivery, it is not known why only a few women develop amniotic fluid embolism. It is thought that varying amounts of different fetal antigens likely interact with unknown maternal susceptibility factors.

The sudden clinical picture most often begins during labor or immediately after the birth of the child, vaginally or by cesarean section. Isolated cases are reported outside of any work. [4]. The main risk factors are intrauterine hypertension induced by polyhydramnios, amniocentesis, amnio-drainage, amnio-infusion, internal tocography, intrauterine fetal death and abdominal trauma. Cases of ELA have been described following medical or voluntary termination of pregnancy, and even during removal of cerclage wire. However, they are all discussed. The components of amniotic fluid have been implicated in the occurrence of an allergic reaction of the anaphylactic type responsible for pulmonary arterial hypertension [3]. The clinical manifestations usually described in ELA are of two types, hemorrhagic and cardiovascular. It is accepted that the first physiopathological consequence is severe pulmonary arterial hypertension (PAH) sometimes associated with damage to the left ventricle (LV). PAH would be due to two mechanisms, a pure mechanical phenomenon with more or less complete obstruction of the pulmonary

capillaries by amniotic fluid, associated with pulmonary vasoconstriction secondary to the presence in large quantities in the amniotic fluid of endothelin, a powerful vasoconstrictor, responsible of PAH [5–6]. The damage to the LV is indirect by compression by a dilated right ventricle or direct by toxicity of the amniotic fluid which would act on the contractility of the myocardium and would participate in the initial collapse. Circulatory arrest is frequent and early, even initial [7]. Postpartum hemorrhage favored by DIC due to the richness of the amniotic fluid in activating factor X released into the maternal circulation [3].

The brutality of onset of ELA imposes as a priority the immediate start of symptomatic treatments which temporarily exclude any paraclinical investigation. Biological examinations can guide resuscitation [4]. Biological diagnosis can and must be undertaken once effective resuscitation maneuvers have been initiated or, unfortunately, post-mortem. The search for LA elements (epithelial cells, vernix caseosa, lanugo, meconium) can be carried out on the peripheral maternal blood or on a central venous line, in the broncho-alveolar lavage fluid, and/or finally on the maternal lung tissues in the event of the patient's death. Reservations about diagnostic reliability affect each of these examinations. The presence of meconium in the LA is observed at term, as well as the lanugo and the vernix caseosa, but if the ELA occurs earlier, these elements are usually lacking. As for the desquamation cells observed in the maternal blood in case of ELA, their fetal origin would be questionable for certain authors [4].

The diagnosis of ELA should be considered in the face of any cardiovascular and hemorrhagic manifestation occurring in the peripartum. It will be confirmed by anatomopathologic analyses. We must also think of other differential diagnoses such as: myocardial infarction, pulmonary embolism, gas embolism, eclampsia, septic or anaphylactic shock [3].

Apart from the classic symptomatic treatment, few new therapies are proposed. The initial symptomatic management of collapse has no specificity, combining vascular filling and catecholamines. The resuscitation of a cardiac arrest in a pregnant woman has some particularities and early fetal extraction has two objectives: to save the child and to improve maternal resuscitation [4–8].

The other aspects of obstetrical care during labor for these patients, including surgical techniques or interventional radiology, are not specific, except that they most often end with hysterectomy for maternal salvage hemostasis [4]. In recent years, the prognosis of ELA seems moderately better thanks to better initial multidisciplinary management. Cardiorespiratory arrest is the main cause of poor vital prognosis and maternal neurological sequelae. The presence of meconium amniotic fluid and fetal death in utero are also factors of poor prognosis. In the cases of favorable evolution, the evolution does not present any specific characteristic. Finally, the occurrence of ELA does not contraindicate a subsequent pregnancy [4–9].

The vital and neurological prognosis of the fetus is correlated with the extraction time in the most serious patients presenting with cardiopulmonary arrest [4].

Conclusion:-

Amniotic embolism represents one of the most formidable complications in obstetrics by its unpredictability, its brutality and its fatality since it represents the second cause of maternal death in France.

The diagnosis of amniotic embolism remains a diagnosis of exclusion and must be considered in the face of any neurological, cardiovascular and hemorrhagic manifestation occurring in the peripartum, but it remains difficult because of its rarity.

The prognosis can be improved by rapid diagnostic and therapeutic management, early suspicion of the diagnosis, aggressive treatment of disseminated intravascular coagulation, rapid elimination of other causes of postpartum hemorrhage, and effective resuscitation with a transfer to an appropriate service.

The improvement of the resuscitation techniques implemented has certainly contributed to the improvement in recent years of the maternal prognosis, even if it remains with the still severe fetal prognosis.

References:-

1. Clark SL: Amniotic fluid embolism. Obstet Gynecol 123:337-348, 2014. doi: 10.1097/AOG.0000000000000107
2. Society for Maternal-Fetal Medicine (SMFM), Pacheco LD, Clark SL, Klassen M, Hankins GD: Amniotic fluid embolism: Principles of early clinical management. Am J Obstet Gynecol 222 (1):48–52, 2020. 222(1):48-52. doi: 10.1016/j.ajog.2019.07.036
3. Rault S, Pirrello O, Langer B, Nisand I. Amniotic fluid embolism: about two cases. obstetrics gynecology and fertility. 2006;34:127–130.
4. Tramoni G, Boisson C, Gamerre L, Clement HJ, Bon C, Rudigoz RC, Viale JP. Amniotic fluid embolism: review. Ann Fr Anesth Reanim. 2006 Jun;25(6):599–604.
5. Dorne R, Pommier C, Boisson C. About amniotic embolism. Ann Fr Anesth Reanim. 2003 Mar;22(3):246–7.
6. Stanten RD, Iverson LI, Daugharty TM, Lovett SM, Terry C, Blum E. Amniotic fluid embolism causing catastrophic pulmonary vasoconstriction: diagnostic by transesophageal echography and treatment by cardiopulmonary bypass. Obstet Gynecol. 2003;102:496–498.
7. Perdrix C, Gioanni G, Delest A, Constantopoulos P. Rapidly fatal amniotic fluid embolism Extraction by caesarean section of a child living without neurological sequelae. Ann Fr Anesth Reanim. 2004 Sep;23(9):912–6.
8. Compain M, Tourtier J, Benhamou D. Treatment of cardiorespiratory arrest in pregnant women An analysis from the French register of maternal deaths. Ann Fr Anesth Reanim. 2003;22(Suppl 2):303.
9. Gilbert WM, Danielsen B. Amniotic fluid embolism: decreased mortality in a population-based study. Obstet Gynecol. 1999;93:973–977.