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A Case of Amniotic Fluid Embolism : Detection by Postmortem Autopsy

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Amniotic fluid embolism(AFE) is perhaps the most devastating condition known in Obstetrics, with a mortality as high as 80 %, Despite widespread recognition and numorous attempts to develops an animal model, the etiology of AFE remains enigmatic and therapeutic recommendation are often confusingly contradictory.

Recently we experienced a case of AFE, confirmed by postmortem autopy at the patient with postpartum hemorrhage and disseminated intravascular coagulation. So we present this case with a brief review of literature concerned.

Key Words : AFE, postmortem autopsy, postpartum hemorrhage.

Introduction

AFE is a highly lethal and rare disorder that first reported by Meyer [1] in 1926. Despite its rarity it remains the most common cause of death in labor, during pregnancy, and the immediate postpartum period. The breakdown of the maternal-fetal barrier with the release of fetal materials into the maternal circulation is responsible for the clinical manifestations of this disorder, which in clude severe coagulopathy, cardiovascular collapse, and variable signs and symptoms.Because there has been no specific therapy for this disorder, theatment has been supportive and involve the maintenance of blood volume, pharmacologic support of the coagulopathy with blood components.

Despite this effort, the morbidity and mortality of AFE is over 80%[2] and the cause of the death involve cardiorespiratory neurologic, and hematologic mechanisms.
This report describes a case of amniotic fluid embolism with no premonitor signs and symptoms, even though She ultimately compromised and died.

Case Report

A 34-year-old Korean woman, gravida 2 para 0 was transfered to Dong San Medical Center, Tae gu, Korea on Aug. 3.1993, for the treatment of postpartum hemorrhage and postpartum shock.
Earlier that day, the patient had uncomplicated induction of labor at 40-week gestation

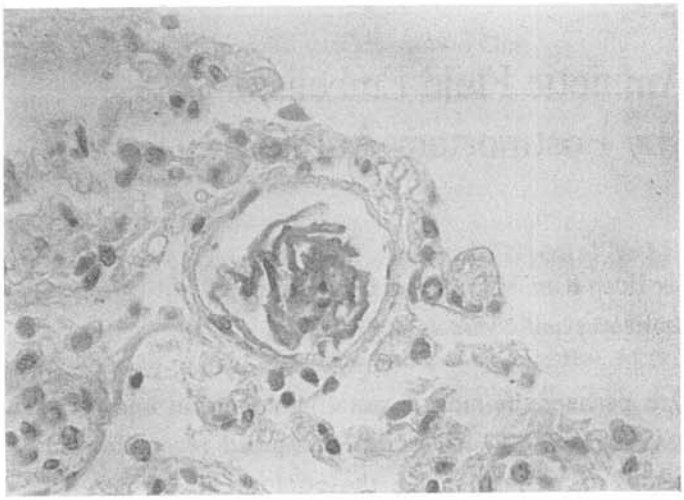

Fig. 1. Fetal squames packed into a small pulmonary artery from a fatal case of amniotic fluid embolism(H&E ×250).

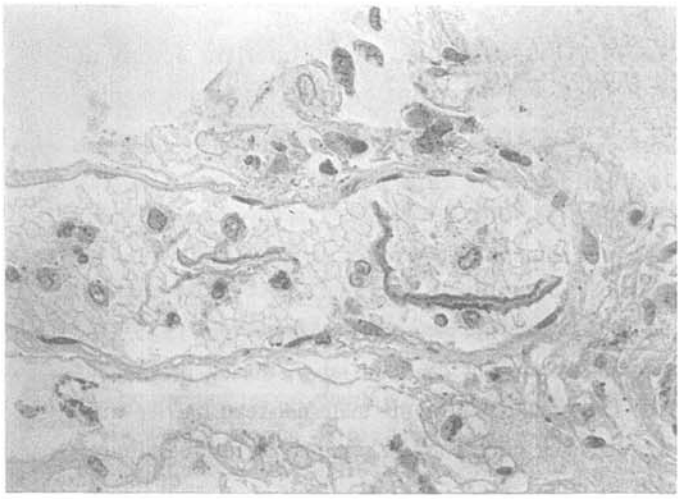

Fig. 2. Most of the empty spaces within the vessel were demonstrated by appropriate staining for lipid to have been filled with vernix caseosa(H&E ×250).

with the delivery of a normal male infant.

One hour postpartum, the baby became cyanotic and showed respiratory difficulty, then the baby was transfered to pediatric ICU under the endotracheal intubation with O_2 supply, but baby was expired at arrival of ICU. Two hour postpartum, the patient sustained a sudden postpartum hemorrhage and then she became irritable and semi-comatous. She was resuscitated with closed cardiac massage and assisted ventilation. Marginal blood pressure was maintained with a large dose of inotropic drugs and a large volus of fluids. The diagnosis of postpartum hemorrhage with uterine atony was suspected, and the patient was transfered to Dong San Medical center for possible medical and surgical treatment.

On arrival at emergency room, the systolic pressure was 60 mmHg with patient receving 30 ug/min/kg dopamine infusion.

She was immediately transfered to ICU where an EKG showing no specific finding within normal limits. Pulmonary artery catheterization was performed for CVP monitoring, hyperalimentation, and massive transfusion with packed cell, FFP, whole blood. Laboratory finding showed severe anemia(Hb 6.4), prolonged PT(over 80), no checkable fibrinogen. In addition, there was external evidence of severe ongoing coagulopathy with bleeding from all intravenous puncture sites, the vagina, and the CVP site. Because the patient was too unstable for further workup, she was taken directly to the operating room for total hysterectomy. The uterus was exposed through a median incision.

The most striking finding was a severe couvalaire uterus, then total hysterctomy was performed.

The patient was taken to ICU after operation, but there was no clinical and laboratory improvement.

Twenty minute post-operation and six hour post-delivery, she had no response to medical treatment, then became asystolic. The patient was pronounced dead after two hours of resuscitation maneuvers including endotracheal intubation, ventilation and pharmacologic intervention Postmortem examination revealed widerspread evidence of a massive AFE, including multiple pulmonary infarcts with microscopic evidence of foreign materials (Fig. 1, 2), but no evidence of chronic pulmonary hyperthension or hemodynamically important change in heart.

Discussion

The incidence of recognized AFE is between 1:8,000 and 1:80,000 pregnancies[2]. Clinical features are variable, but AFE is usually heralded by the acute onset of cardiorespiratory collapse associated with subsequent coagulopathy and neurologic compromise. Minor symptoms of fevers, chills, and hyper-reflexia has also been reported. AFE accounts for 4~5% of maternal death in some major centers[3].

The entry of amniotic fluid into the maternal circulation may be more common than previously thought. Clark et al.[4] found that squamae found in the pulmonary circulation of pregnant and even nonpregnant women is common, thus making the diagnosis of AFE difficult.

Malinou et al.[5], using precardial Doppler showed that greater than 40% of patients undergoing cesarean section had evidence of air embolism on incision of the uterus. Perhaps amniotic fluid could enter the circulation as easily.

It is conceivable that, in many deliveries, some amniotic fluid escapes into the maternal circulation without consequence. In the healthy parturient with normal cardiorespiratory reserve, minor episodes of AFE will be clinically undectected, while compromised patients may be less able to withstand the insults.

While the most common presentation of AFE is sudden cardiorespiratory collapse, these patients usually have not been intensively monitored prior to their collapse.

Ultimately even if a patient survives the initial insult, the patient die of multisystem

failure.

The actual mechanism of cardiac failure caused by AFE is belived to be both-mechanical(cloging) and biochemical(prostaglandin-induced global dysfunction). These mechanisms would fit the patient's clinical picture. Severg life-threatening hemorrhage is another characteristic of amniotic embolism that occurs in 40% of AFE[2]. The prominent feature appears to be disseminated intravascular fibrin deposition in response to the procoagulant effect of amniotic fluid. Traditionally the diagnosis of AFE has been made by postmortem examination of the pulmonary artery contents. Demonstration of fetal squamae from a fatal case of AFE is considered to be pathognomonic of this disorder. Antemortem diagnosis has been confined to the finding of fetal squamous cells in the maternal circulation retrieved either through a central venous, or pulmonary artery catheter; this method is imprecise because of high false-positive yield[4]. Therefore extraction of pulmonary artery thrombus during pulmonary thrombectoy[8] and subsequent pathologic examination represents the most accurate means of establishing the diagnosis during life.

Furthermore treatment of the coagulopathy by single replacement therapy without removal of the thrombogenic stimulus may aggravate the pulmonary vascular obstruction by causing continued thrombogenesis and resulting in progressive arterial desaturation and right heart failure.

In conclusion, on the basis of this case report, emergent diagnostic method for postpartum shock cauesd by AFE is needed.

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사후 병리조직학적 검사에 의해 확인된 양수색전증 1예

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양수색전증은 치사율이 80 %에 달하는 산과 영역에서 가장 치명적인 상태로 알려져있으며, 많은 연구에도 불구하고 양수색전증의 원인은 불분명하며, 또한 권하여지는 치료법도 일정하지 않다.

저자들은 34세의 경산부에서 유도분만에 의한 질식분만후 심한 산후 출혈 및 혈액응고 장애의 소견을 보여 본원에 전원되어 사후 병리학적 검사에 의하여 양수 색전증으로 확인된 1예를 경험하였기에 문헌 고찰과 함께 보고하는 바이다.

중심어휘 : 양수 색전증.