Fırat Tıp Dergisi 2010;15(1): 51-53

Case Report

www.firattipdergisi.com

Amniotic Fluid Embolism: A Case Report

Ozan KAHVECİ[2], Ahmet DEMİRCAN[2], Ayfer KELEŞ[2], Gülbin AYGENCEL[a1], Fikret BİLDİK[2], Elif ÇALIDAĞ[2], Tülin KAHVECİ[3]

[1]*Gazi Üniversitesi Tıp Fakültesi, İç Hastalıkları AD Yoğun Bakım BD, ANKARA, Türkiye*

[2]*Gazi Üniversitesi Tıp Fakültesi, Acil Tıp AD, ANKARA, Türkiye*

[3]*Ankara Üniversitesi Tıp Fakültesi, Kadın Hastalıkları ve Doğum AD, ANKARA, Türkiye*

ABSTRACT

Amniotic fluid embolism (AFE) is a rare occurrence, which accounts for about 10% of all maternal deaths. There is no single pathognomonic clinical or laboratory finding for this syndrome. Diagnosis is based on clinical presentation and supportive laboratory values. A case of acute embolic episode occurred during term labour in 32-year-old female patient with spontaneous rupture of membrane was presented. This case showed typical clinical findings of this syndrome. Caesarean section was performed immediately, but the baby and the mother died. In conclusion**,** AFE continues to be one of the most feared and devastating complications of pregnancy. It can be neither predicted nor prevented. The management of AFE is largely supportive.

Key words: *Amniotic fluid embolism, pregnancy*

ÖZET

Amniyon Sıvı Embolisi: Olgu Sunumu

Amniyon sıvı embolisi (ASE) nadir görülen ve tüm anne ölümlerinin %10'undan sorumlu olan bir sendromdur. Klinik ve laboratuar olarak bu sendromu düşündüren patognomonik bir bulgu yoktur. Tanı klinik ve destekleyici laboratuar bulgularına dayanılarak konur. Burada termde, spontan membrane rüptürü ve doğum eylemi ile acile gelen 32 yaşında bir bayan hastada gelişen akut embolik epizod sunulmuştur. Olgu ASE'nin tipik klinik bulgularını göstermektedir. Sonuçta olgu acil sezaryan operasyonuna alınmış; fakat anne ve bebek hayatını kaybetmiştir. Sonuç olarak ASE'si gebelikte çok korkulan ve zarar veren komplikasyonlardan biridir. Ne önceden bilinebilmektedir ne de önlenebilmektedir. Tedavisi ise sadece destekleyici tedavidir.

Anahtar Sözcükler: *Amniyon sıvı embolisi, gebelik*

Amniotic fluid embolism (AFE) is a rare life-threatening complication unique to pregnancy. The incidence ranges between 1 in 8,000 to 1 in 80,000 pregnancies (1). AFE has a mortality rate of 61 to 86% and accounts for approximately 10% of all maternal deaths in the United States (2). AFE has a variable presentation, ranging from mild degrees of organ dysfunction to cardiovascular collapse, coagulopathy and death. AFE usually presents at term during labour. In this report, a case of acute embolic episode occurred during labour in a 32-year-old patient with spontaneous rupture of membrane was presented. Caesarean section was performed immediately; the baby died and the mother died because of disseminated intravascular coagulation (DIC) and cardiorespiratory arrest.

CASE REPORT

A 32-year-old nulliparous woman at 40 weeks' of gestation, was admitted to the our emergency service with complaints of vaginal bleeding and pain. Her antepartum obstetric history was unremarkable, but her past medical history was significant for a diagnosis of asthma and some drug allergies. Vaginal examination revealed a cervix dilated 3-4 cm, effaciated 60%, and the fetal head stated at minus 2. Amniotic fluid loss was seen. She was accepted in early active labour. She was semi-orientated, tachycardic, tachypnoeic, dyspnoeic, hypotensive and cyanotic. Arterial blood gas analysis showed a severe hypoxaemia. The chest x-ray revealed central bilateral pulmonary oedema. The electrocardiogram showed only a sinus tachycardia. Endotracheal intubation was immediately performed, and the patient was mechanically ventilated. She was transferred to intensive care unit. A central venous catheter was inserted in the right internal jugular vein and central venous pressure was 8 mmHg. Laboratory values revealed mild coagulopathy. Cardiotocography revealed signs of foetal distress and ultrasound showed oligohydramnios of the foetus . Supportive measures, including fluid resuscitation, inotropic support and plasma, were administered. Caesarean section was performed immediately, but the male baby died. During operation, the patient developed severe haemorrhage and shock necessitating massive blood transfusion. Then, maternal bradycardia and asistoli was developed and cardiopulmonary resuscitation (CPR) was performed. The patient was declared dead 40 minutes after the onset of resuscitative

[a] Corresponding Adress: Dr. Gülbin AYGENCEL, Gazi Üniversitesi, Tıp Fakültesi, İç Hastalıkları AD Yoğun Bakım BD, ANKARA, Türkiye
Tel: +90 312 2024216 e-mail: aygencel@hotmail.com

efforts. Autopsy was not performed.

DISCUSSION

Amniotic fluid embolism (AFE), first described by Meyer in 1926, is a topic of great concern in obstetrics today because of its dramatic presentation and high rate of mortality (3). There is no one specific test that can confirm this syndrome. The diagnosis of AFE is based on its clinical presentation and supportive laboratory studies. The diagnosis is therefore made by exclusion of other causes. Any condition that presents as acute cardiorespiratory collapse or massive hemorrhage in the peripartum period must be systematically evaluated. The differential diagnosis includes air or thrombotic pulmonary emboli, septic shock, aspiration pneumonia, acute myocardial infarction, placental abruption, eclampsia, complication of tocolytic therapy with β-sympathomimetics, transfusion reaction and local anaesthetic toxicity (4). AFE should be suspected in any pregnant patient, specifically those with ruptured membranes, who develop sudden onset dyspnea with hypoxia, acute hypotension and/or cardiac arrest followed by a profound coagulopathy. Both the United States and the United Kingdom have a national registry for suspected AFE, and entry criteria consist of the presence of the four following factors: 1) acute hypotension or cardiac arrest; 2) acute hypoxia; 3) coagulopathy or severe clinical hemorrhage in the absence of other explanations; 4) all of these occurring during labour, Cesarean delivery, or dilatation and evacuation (D & E) or within 30 min postpartum with no other explanation for the findings (5). The patient described in this case report had all of these factors.

Amniotic fluid embolism (AFE) was thought to more likely occur in the elderly multiparous patient who had an unusually strong or rapid labour or who had just delivered following such a labour. The use of uterine stimulants, meconium staining of the amniotic fluid or the presence of a large or dead fetus were also felt to increased the risk (6). Subsequent experiences have shown that there are a number of exceptions to this classical description. Our case was also one of these exceptions.

Although authors noted that patients were most likely to present during labour or shortly thereafter, there are several case reports of AFE occurring during Caesarean sections and therapeutic abortions as well as occasional cases in the late postpartum period, or very rarely in a non-labouring patient. Other cases have been associated with abdominal trauma, ruptured uterus or intrapartum amnioinfusion (7).

Amniotic fluid embolism (AFE) can occur only when there is a breech in the barrier between the amniotic fluid and maternal circulation. If even a small volume of amniotic fluid enters the maternal circulation, the initial haemodynamic response consists of acute pulmonary hypertension and vasospasm complicated by severe hypoxaemia and right-sided heart failure, followed by a second phase of more sustained left ventricular failure. The first phase might be due to the introduction of a potent vasoconstrictor arising from the amniotic fluid, and the second phase is thought to be due to a direct myocardial depressant also from the amniotic fluid. The responsible substance might be endothelin, which has been found in high concentrations in the amniotic fluid (8).

New theories suggest that AFE is actually a type 1 hypersensitivity reaction with mechanisms similar to anaphylaxis and septic shock, and that the variation in the nature and severity of the clinical syndrome appears to depend on the variation of the antigenic exposure and the individual response. Clark suggests that AFE should be renamed “Anaphylactoid Syndrome of Pregnancy” (2). Forty-one (41%) of the patients analyzed had a history of atopy or known drug allergies. Clark showed that 67% of those analyzed had been pregnant with a male fetus (2). Interestingly, the patient in this case report had a male fetus and she had a diagnosis of asthma with several drug allergies.

Laboratory data may be supportive, but they alone can never diagnose or exclude AFE. Laboratory investigations which may be useful include complete blood count, coagulation parameters, arterial blood gases, maternal serum tryptase and plasma zinc coproporhyrin levels, chest x-ray, VQ scan, ECG and echocardiogram. The finding of squamous cells in the maternal pulmonary circulation in autopsy or alive, once considered pathognomonic, is neither specific nor sensitive for the diagnosis of AFE. The identification of mucin, however, seems to be a more sensitive indicator of AFE (9, 10).

In this case, autopsy was not performed; but diagnosis of amniotic fluid embolism was highly suspected and diagnosis was made on clinical grounds. We believe this case represents one of the typical presentations of AFE in which severe hypoxaemia and haemodynamic disturbances were the presenting symptoms followed by consumptive coagulopathy.

The management of AFE remains largely supportive with emphasis on maintaining left ventricular function and output. If hypoxia refractory to O2 occurs or the patient becomes unconscious, the patient should be rapidly intubated and placed on 100% O2 and mechanical ventilation. Maternal circulation should be supported with several large bore intravenous (iv) catheters and central access if possible. Initially, the goals are to rapidly increase the circulating volume and cardiac output with infusions of crystalloids, dopamine and other vasopressors as needed. Pulmonary artery and intraarterial catheters may be beneficial in guiding further therapy. If the patient is pregnant at the time of the AFE, plans should be made for immediate delivery after initial resuscitative efforts. Rapid delivery might not only improve fetal outcome, but may facilitate resuscitation particularly in term pregnancies. Because of the high maternal mortality rate, with more than half dying in the first hour, the surgeon should be prepared to perform a postmortem Caesarean section (5). Coagulopathy also occurs in approximately 83% of cases, and is treated with component therapy including platelets, fresh frozen plasma, cryoprecipitate, and red blood cells (2).

Since the pathophysiological mechanisms of AFE and anaphylaxis are similar, therapy directed at the management of AFE is analogous to the treatment of anaphylaxis. Primary treatment of anaphylaxis includes; maintain an adequate airway and give 100% O2, rapid iv volume resuscitation, and epinephrine for cardiovascular support. Secondary treatment includes the administration of; antihistamines (H1/H2 blockers), bronchodilators, corticosteroids and the maintenance of catecholamine infusions (epinephrine, norepinephrine, isoproterenol) to maintain hemodynamic stability. Indiscriminate prophylactic treatment to prevent anaphylaxis is not recommended and thus would not be indicated for the pre-

vention of AFE. However primary and secondary treatment is indicated when necessary (5).

In summary, AFE continues to be one of the most feared and devastating complications of pregnancy. It can be neither predicted nor prevented. AFE should be considered as a differential diagnosis in pregnant patients or immediate postpartum patients with acute profound haemodynamic, pulmonary or haematological disturbances. The therapy for AFE is nonspecific and directed towards ventilatory and circulatory support and correction of the coagulopathy.

REFERENCES

1. Morgan M. Amniotic fluid embolism. Anaesthesia 1979; 34: 20-32.
2. Clark SL, Hankins GD, Dudley DA, Dildy GA, Porter TF. Amniotic fluid embolism: analysis of the national registry. Am J Obstet Gynecol 1995; 172: 1158-1169.
3. Meyer JR. Embolis pulmonar amnio-caseosa. Bras Med 1926; 2: 301-303.
4. Sprung J, Cheng EY, Patel S, Kampine JP. Understanding and management of amniotic fluid embolis. J Clin Anesth 1992; 4: 235-240.
5. Ray BK, Vallejo MC, Creinin MD, Shannon KT, Mandell GL, et al. Amniotic fluid embolism with second trimester pregnancy termination: a case report. Can J Anaesth 2004; 51: 139-144.
6. Steiner PE, Lushbaugh CC. Maternal pulmonary embolism by amniotic fluid as a cause of obstetric shock and unexpected death in obstetrics. JAMA 1941; 117: 1245-1254.
7. Mato J. Suspected amniotic fluid embolism following amniotomy: a case report. AANA J 2008; 76: 53-59.
8. Perrozzi KJ, Englert NC. Amniotic fluid embolism: an obstetric emergency. Crit Care Nurse 2004; 24: 54-61.
9. Davies S. Amniotic fluid embolus: a review of the literature. Can J Anaesth 2001; 48: 88-98.
10. Fletcher SJ, Parr MJ. Amniotic fluid embolism: a case report and review. Resuscitation 2000; 43: 141-146.

Kabul Tarihi: 18.10.2009