

REMAINING ALIVE AFTER AMNIOTIC FLUID EMBOLISM: A CASE REPORT

AMNİYON SIVI EMBOLİSİ SONRASI SAĞ KALIM : OLGU SUNUMU

Reyyan ÖZCAN TEMİZKAN, Şevki ÇELEN, Ayhan SUCAK, Sevtap HAMDEMİR

Zekai Tahir Burak Woman's Health Education and Research Hospital -ANKARA

Geliş Tarihi:10.05.2010

Kabul Tarihi: 09.10.2011

Abstract:

Amnion fluid embolism (AFE) is a rare but catastrophic complication of pregnancy. Its incidence varies from 1:8000 to 1:80.000 and the mortality rate is about 61-86%. Neonatal survival reaches 40-70% Our case was a healthy, 36 year old multigravida at 39 weeks gestation hospitalized for induction of labor, which developed AFE in the active phase of labor. Initial symptom was acute and severe hypotension, followed by collapse of peripheral circulation, cyanosis and acute respiratory failure. The patient underwent an urgent cesarean section with the survival of neurologically intact fetus but complicated with severe hemorrhage and DIC. Despite of severe coagulopathy, the mother was discharged from the hospital at the end of one week. Amniotic fluid embolism is a very complicated problem, difficult to manage. Complete management of AFE requires qualified, experienced and multidisciplinary physicians.

Keywords: Amnion fluid embolism, anaphylactoid reactions, coagulopathy.

Özet

Amniyon sıvı embolisi (AFE) gebelikte nadir görülen fakat çok ciddi seyirli bir komplikasyondur. İnsidansı 1:8000 ile 1:80000 arasında değişmektedir. Mortalite oranı %61-86 dır. Neonatal sağ kalım % 40-70' dir. Vakamız sağlıklı 36 yaşında multigravid eylemin indüksiyonu için yatırılmış 39 haftalık gebeydi. Eylemin aktif fazında AFE gelişti. Başlangıç semptomu akut ve ciddi hipotansiyon, takiben periferel dolaşım kollapsı, siyanoz ve akut solunum yetmezliğidir. Hasta acil olarak sezeryana alındı. Nörolojik olarak fetus sağlam olarak çıkarıldı sonrasında annede ciddi hemoraji ve DIC gelişti. Ciddi koagülopatiyeye rağmen hasta bir hafta içinde hastaneden taburcu edilebildi.

Amniyon sıvı embolisi yönetimi çok zor olan nadir görülen fakat çok ciddi seyirli bir komplikasyondur. Bu sebeple AFE yönetimi deneyimli ve nitelikli hekimlerin bulunduğu multidisipliner hastanelerde yapılmalıdır.

Anahtar Kelimeler: Amniyon sıvı embolisi, anafilaktik reaksiyon, koagülopati.

Amniotic fluid embolism (AFE) is a rare, unpredictable, life threatening complication unique to pregnancy (1). This new concept, anaphylactoid reactions in AFE became more important.

The incidence of AFE has been reported in the range of 1/8000 to 1/80000 deliveries. Two recent, AFE mortality rate changes between %61 to %86 in different studies. It is estimated that 10% of all maternal deaths are caused by

AFE (2,5) fetal outcome is poor when AFE occurs before delivery. We presented a case whose labor was complicated by AFE

Case Report

A healthy 36 year old multigravida (G4P1Y1D/C1EXU1) at 39 weeks gestation was admitted to Zekai Tahir Burak Woman's Health Education and Research Hospital. The patient had a 39 weeks of fetus who had a vertex

presentation with a cervical dilatation of 4 cm, ruptured uterine membranes, blood pressure 120/70mmh and fetal heart rate of 144 bpm. Two hours later cervical dilatation reached 5 cm, the patient suddenly showed manifestations of acute respiratory failure, cyanosis and hypotension 70/30 mmHg, followed by cardiovascular collapse. Severe decelerations were shown in cardiotocograph.

Immediate intubation was performed within 3 min with iv infusion of propofol 140 mg and 35 mg esmeron. The patient was transferred to the operating room with in 5 min. Emergency cesarean section was performed because of severe fetal bradycardia, resulting in the delivery of a viable female infant weighing 2870 gram with an APGAR score of 3 at 2 minutes, fetal hearth rate 70-80 bpm after successful cardiopulmonary resuscitation, an APGAR score of 7 at 5 min The newborn was extubated shortly.

After the delivery, twenty minutes later the patient developed severe coagulopathy. A rubber drain was placed to cul de sac. After the delivery, the woman was noted

to have sinus tachycardia, 165 bpm, and blood pressure 109/73 with vasopressor and sympathomimetic agents.

Uterus was not contracted in spite of continuous uterine massage, 0.2 mg methergine and oxytocin infusion. Estimated total blood loss was 2500 cc.

Two hours after the delivery, the hemoglobin levels decreased from 13.9 mg/dl to the level of 8.5 g/dl despite the replacement of two units of packed red blood cell transfusion.

Seven units of fresh frozen plasma, three units of platelet and four units of packed red blood cell was transfused shortly after the operation. The patient was consulted to cardiology Unit. Echocardiography showed minimally right atrial dilatation and sinus tachycardia.

Ten hours after the onset of first symptom, the patient became hemodinamically stable and was transferred to the intensive care unit (ICU).

Table-1: Laboratory findings during the patient's hospital surveillance.

| | Hb (g/dl) | Plt(10 ³ /µl) | PT | aPTT | INR | Fibrinogen (g/l) | D-Dimer |
|----------------------|------------------|--------------------------|--------------|--------------|-----------------|------------------|---------------|
| Preoperative | 13.9 | 169 | 11,3 | 28.6 | 0,83 | 240 | |
| Intraoperative | 11.2 | 79 | 33,1 | 113.6 | 3,25 | | |
| Postoperative 2 hrs | 8.5 | 120 | | | | | |
| Posttransfusion | 10,8 | 122 | | | | | |
| Postoperative 25 hrs | 7.3 | 72 | | | | 180 | 11,68 |
| Postoperative 48 hrs | 8.4 | 98 | | | | | 11 |
| Normal Range | 13,6-17.2 | 156-373 | 11-15 | 25-40 | 0,75-1.5 | 140-450 | 0-0,50 |

Discussion

70% of cases occurred during labor, 19% were recorded during cesarean section, and 11% of cases occurred immediately following vaginal delivery.(2) Our patient showed symptoms during active phase of labor. There is increased frequency of AFE in women who underwent cesarean delivery. Approximately 50% of cases were associated with fetal distress, suggesting that AFE and associated hypoxia proceed cesarean delivery (4,7) as in our case. The patient went cesarean section following severe fetal distress caused by AFE.

Mechanism of AFE is poorly understood. Normally intact fetal membranes do not permit the entrance of the amniotic fluid in the maternal circulation.(10) AFE occurs only when there is a breach in this barrier. Definitive diagnosis can be made by finding amniotic fluid components in maternal pulmonary vasculature in postmortem examinations. Amniotic fluid components composed to maternal

circulation through placental sites and endocervical vessels via electrochemical gradient or pressure. (8) AFE is a process that involves a chain reaction of many mediators of inflammation like prostaglandins, leukotriens, PAFs and complement activating factors which are elevated during labor. (9) This mechanism is similar to anaphylaxis and septic shock (2)

There is no specific diagnostic test and a high index of suspicion is necessary when a pregnant woman presents with any of the above signs and symptoms.

Classic presenting symptoms of AFE include respiratory distress, altered mental status, profound hypotension, coagulopathy and death. (5) Other signs and symptoms include nausea, vomiting, fever, chills, and headache. Clark (2) from USA and Tuffnell (6) were worked systematically on this issue providing for suspected AFE. In our case, all the four criteria were met.DIC is a common feature of AFE. About 83% of patients demonstrated labora-



tory abnormalities or clinical findings consistent with DIC. Onset is variable. In 50% of cases, coagulopathy starts within 4 hours, often within 20 to 30 minutes of symptom onset. (2) In our case, clinical coagulopathy started intra-operative, about 20 minutes after the initial symptom and clinical coagulopathy is confirmed with abnormal coagulation profile.

There are no specific manners of management of AFE. The management of women with AFE must be multi-disciplinary and patient should be managed in an intensive care unit. Following the vital signs of the patient should be goal of the management. The basis of the management of AFE is support of airway, tissue oxygenation, breathing and circulation and correction of coagulopathy. (3, 13, 14) To maintain tissue oxygenation, 100% supplemental oxygen is necessary via intubation and positive pressure ventilation.

Intravascular volume must be preserved with crystalloids and colloids, if necessary inotropes and vasopressors may be administered. The aim is to improve circulation and maintain renal perfusion and urine output. (12)

In case of severe hemorrhage, blood and blood products such as platelet, fresh-frozen plasma and cryoprecipitate should be prepared and ordered rapidly. Uterine artery embolization and recombinant factor VII may be used in cases of severe coagulopathy resistant to conventional blood and blood product replacement. (11, 15)

In conclusion, treatment of patient who undergoes AFE resembles treatment of the anaphylactic shock. Thanks to early intervention and successful transfusion, patients can survive. Thus, labor management should be performed in a hospital that has blood center.

References

1. Meyer JR (1926) Embolia pulmonar amnio caseosa. J Bras Med 2:301–303
2. Clark SL, Hankins GD, Dudley DA et al. Amniotic fluid embolism: Analysis of the national registry. Am J Obstet Gynecol 1995; 172: 1159–1167.
3. Kramer MS, Rouleau J, Baskett TF, et al. Amniotic-fluid embolism and medical induction of labour: a retrospective, population-based cohort study. Lancet 2006;368(9545):1444–8.
4. Gilbert WM, Danielson B. Amniotic fluid embolism: decreased mortality in a populationbased study. Obstet Gynecol 1999;93(6):973–7.
5. Morgan M (1979) Amniotic fluid embolism. Anaesthesia 34:20–32
6. Tuffnell DJ. United Kingdom amniotic fluid embolism register. BJOG 2005;112(12):1625–9.
7. Lau G, Chui PP. Amniotic fluid embolism: a review of 10 fatal cases. Singapore Med J 1994; 35:180–3.
8. Resnik R, Swartz WH, Plummer MH, et al. Amniotic fluid embolism with survival. Obstet Gynecol 1976;47:295–8.
9. Karetzky M, Ramirez M (1998) Acute respiratory failure in pregnancy. An analysis of 19 cases. Medicine 77:41–49
10. Roche WD Jr, Norris HJ (1974) Detection and significance of maternal pulmonary amniotic embolism. Obstet Gynecol 43:729–731
11. Benson MD, Kobayashi H, Silver RK, et al. Immunologic studies in presumed amniotic fluid embolism. Obstet Gynecol 2001;97(4):510–4.
12. Aurangzeb I, George L & Raoof S. Amniotic fluid embolism. Crit Care Clin 2004; 20: 643–650.
13. Locksmith GJ. Amniotic fluid embolism. Obstet Gynecol Clin North Am 1999; 26: 435–444.
14. Kane SK. Historical perspective of amniotic fluid embolism. Int Anesthesiol Clin 2005; 43: 99–108.
15. Lim Y, Loo CC, Chia V, et al. Recombinant factor VIIa after amniotic fluid embolism and disseminated intravascular coagulopathy. Int J Obstet Gynecol 2004;87:178–9.

*Yazışma Adresi: Reyyan ÖZCAN TEMİZKAN
Zekai Tahir Burak Womans Health Education and Research
Hospital , ANKARA /TURKEY
Phone: +90 312 306 50 00
Email: reyyantdr@hotmail.com*