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Amniyotik Sıvı Embolisi Olgusunun Başarılı Yönetimi

Successful Management of a Case with Amniotic Fluid Embolism

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ÖZ

Amniyotik sıvı embolisi (AFE), yüksek maternal ve fetal mortalite ve morbiditeye sahip obstetrik acil bir durumdur. Prognozu belirleyen en önemli faktör erken tanı ve tedavidir. Erken tanı ve zamanında müdahale ile başarıyla tedavi edilen bir amniyotik sıvı embolisi olgusunu sunuyoruz.

Fetal böbrek anomalisi nedeniyle 10 gün önce ürinom drenajı ve kordosentez yapılan 20 yaşındaki hastada doğum sırasında solunum durması gelişti ve ardından vajinal doğumdan yaklaşık 90 dakika sonra yoğun kanama meydana geldi. AFE tanımızla masif kan transfüzyonu, fibrinojen replasmanı ve intrauterin balon hızla uygulandı. AFE tanısı klinik olarak ve ayırıcı tanılar dışlanarak konulmuştur. Hastamız, komplikasyonsuz bir şekilde taburcu edildi.

Anahtar Kelimeler: amniyotik sıvı embolisi, yüksek riskli gebelik, maternal risk, doğum öncesi tanı.

ABSTRACT

Amniotic fluid embolism (AFE) is an obstetric emergency with high maternal and fetal mortality and morbidity. The most important factor in determining the prognosis is early diagnosis and treatment. We present a case of amniotic fluid embolism that was successfully treated with early diagnosis and intime management.

Respiratory arrest developed during labor in a 20-year-old patient who underwent urinoma drainage and cordocentesis for fetal renal anomaly 10 days ago and then profuse bleeding occurred approximately 90 minutes after vaginal delivery. With our definition of AFE, massive blood transfusion, fibrinogen replacement and intrauterine balloon were applied quickly. The diagnosis of AFE was made clinically and excluding differential diagnoses. She was discharged without complications.

Keywords: amniotic fluid embolism, high risk pregnancy, maternal risk, prenatal diagnosis.

BACKGROUND

Amniotic fluid embolism (AFE) is a rare obstetric emergency that can lead to sudden death. Amniotic fluid and fetal components that pass into the maternal circulation activate the maternal complement system, causing an anaphylactic reaction and as a result, sudden cardiopulmonary collapse and early-onset disseminated intravascular coagulation (DIC) develops(1, 2). Amniotic fluid embolism was first described by Meyer in 1926(3). The mortality rate is associated with early diagnosis and rapid resuscitation, and when more than 17 million births and 751 AFE cases in eight countries were analyzed, the overall maternal mortality rate was found to be 20.3%(4). Informed consent was obtained from the patient to present a case of amniotic

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fluid embolism, which was treated with an early diagnosis and multidisciplinary approach.

CASE PRESENTATION

The patient was 20 years old, gravida 2, parity 1 and had a history of COVID-19 infection at the 7th week of her pregnancy. During the fetal ultrasonographic scan, fetal left kidney dimensions and parenchymal echo together with a thin-walled, anechoic cyst of 46x32 mm significantly increased was detected. Therefore, an invasive prenatal diagnostic procedure was recommended to the patient, but the patient refused. In the control examination performed 1 week later, the cyst in the left kidney was measured as 54x39 mm. Thereupon, urinoma drainage and cordocentesis were performed after the betamethasone dose and there were no early complications due to the procedure. The patient was admitted to hospital with 4 cm cervical dilatation 10 days after the procedure. Her blood pressure was 114/66 mmHg, heart rate was 97/min, oxygen saturation (SpO2) was 96%, body temperature was 36.5°C, and fetal heart rate was 120-130/min. Antibiotic and neuroprotective magnesium treatments were applied to the patient with high C-Reactive Protein (CRP) and cervical dilation. The patient delivered 9 hours after her hospitalization. During the straining, her breathing became shallow and she lost consciousness. An Emergency Code was given for the patient who developed respiratory arrest at 13:50 and then an oropharyngeal airway was inserted. Oxygen was given at a rate of 12 L/min, she was monitored and blood analysis was requested. The first venous blood gas analysis result showed pH 7.27, base excess (BE) -9.4, lactate 4.06.

Her blood pressure dropped to 62/43 mmHg and SpO2 to 73%. Within 10 seconds, at 13:50, a 950 gram male infant was delivered vaginally with APGAR scores of 3 and 7 at one and five minutes, respectively. Following delivery, the placenta and its appendages were completely separated. Postpartum 10 units of oxytocin intramuscularly and 20 units of oxytocin infusion were given intravenously at a rate of 125 ml/hour. Active vaginal bleeding was not observed and the uterus was contracted. At 13:58, the patient was intubated and taken to the intensive care unit (ICU). Post-intubation blood pressure was 48/35 mmHg, heart rate was 138/min, SpO2 was 80% and body temperature was 37.6°C. Fluid resuscitation was started, she was monitored and blood analysis was requested. The patient was administered 80 mg of methylprednisolone intravenously daily for 3 days. Misoprostol was given 2 tablets rectally. Considering the possibility of DIC due to the presence of hematuric urine, 2 g of fibrinogen was given intravenously. Following fluid resuscitation, systolic blood pressure values increased to 90, 110, 120 mmHg, respectively and SpO2 to 98%. The patient started to move and it was observed that she had limited range of motion in her right upper extremity. Upon the normalization of blood pressure and oxygen saturation values, emergency Cranial-Thorax-Abdominal Computed Tomography (CT) was performed on the patient. CT results were evaluated as normal. During the vaginal examination performed on the patient who came back to the ICU at 15:25, it was observed that the patient had excessive vaginal bleeding consistent with DIC. Abdominal ultrasound was performed, bleeding focus was not observed. 2 units of red blood concentrates (RBCs) and 4 units of freshly frozen plasma (FFP) were immediately transfused. Another 2 g of fibrinogen was given. An intrauterine balloon was applied to the patient and the balloon was inflated to 300 cc. Abdominal ultrasound was repeated and no bleeding was observed. In the meantime, it was observed that the patient's bleeding decreased. A left femoral catheter was inserted into the patient. COVID PCR was studied and the result was negative. Control blood tests were repeated and another 2 g of fibrinogen was given at 16:50 (fibrinogen 0.85 g/dL). Methylergonovine ampoule was administered intramuscularly. 40 units of oxytocin infusion were continued at a rate of 50 ml/hour. Broad-spectrum antibiotic treatment was started due to suspicion of antenatal subclinical chorioamnionitis. As a result of echocardiography at 18:30, the right atrium and ventricle were observed as slightly dilated. Elevated cardiac markers (troponin I 4213 ng/L, nt-pro BNP 506 ng/L, CK-MB mass 5.95 µg/L, myoglobulin 158 µg/L) and changes in electrocardiography (sinus tachycardia and anterior t negativity) was thought to be secondary to hypoxia that developed after DIC as a result of the patient's follow-up. According to results of the repeated blood test at 18:00, 20 units of cryoprecipitate, 3 units of FFP, 5 ampoules of calcium and 8 ampoules of potassium were given (fibrinogen 1.08 g/dL, potassium 2.9 mEq/L, calcium 7.7 mg/dL). 2 units of RBCs were inserted as a result of the blood tests performed at 20:00 (hemoglobin 7.5 g/dL).

The patient was extubated on the second day. Oxygen was given by mask at 6 L/min.

Diffusion magnetic resonance imaging was requested from the patient due to loss of consciousness during delivery, loss of strength in the right extremity and slowness of speech. As a result, acute infarction was detected in the left capsula externa

and posterior capsula interna. Therefore enoxaparin sodium was started subcutaneously.

On the third day, 1 unit of RBCs and 8 ampoules of potassium were given (hemoglobin 8.9 g/dL, potassium 3.1 mEq/L).

The time series results of blood sampling, vital signs and oxygen saturations are shown in Table 1.

Table 1: Laboratory Data, Vital Signs and Oxygen Saturation

	During hospitalization	Post-partum 1st minute	After 2nd hour	After 4th hour	After 6th hour	After 2nd day	After 3rd day	After 4th day
Laboratory Data								
Hemoglobin (g/dL) [12-15.6]	11.3	11.8	10.2	9.5	7.5	9.7	8.9	10.1
Hematocrit (%) [35.5-45.5]	34.5	32.9	29.1	28.5	21.7	27.2	27.7	31.5
WBC ($x10^6$/L) [3900-10200]	12200	23010	26070	20280	12060	13140	9570	6880
PLT ($x10^9$/L) [150-400]	234	142	188	158	142	155	152	160
Fibrinogen (g/L) [1.7-4.2]	5.22	N/A	0.85	1.08	2.65	3.36	3.79	3.50
D-dimer (mg/L) [<0,55]	N/A	1.49	N/A	>35.2	N/A	>35.2	12.48	N/A
Calcium (mg/dL) [8.7-10.4]	8.1	7.5	7.4	7.7	8.0	9.0	8.4	8.6
Magnesium (mg/dL) [1.3-2.7]	1.7	3.7	2.5	2.2	2.1	1.7	1.6	1.7
Potassium (mEq/L) [3.5-5.5]	3.7	3.6	2.5	2.9	3.1	4	3.1	3.6
pH [7.37-7.45]	N/A	7.28	7.29	7.33	7.40	7.42	7.45	7.43
BE (mmol/L) [(-2)-(+3)]	N/A	-9.4	-6.8	-3.6	-1.2	-2.8	-3	-1.4
Lactate (mmol/L) [4.5-20]	N/A	4.06	5.48	3.44	3.22	1.04	0.67	0.68
Vital Signs								
Blood Pressure (mmHg)	114/66	62/43	118/62	77/45	116/62	103/75	101/56	117/81
Pulse Rate (beats/min)	97	133	113	110	116	93	65	63
Body Temperature (°C)	36.5	37.6	36.8	36.6	37	36.5	36.4	36.2
Respiration Rate (breaths/min)	16	0	20	18	16	18	16	14
Oxygen Saturation								
SpO2 (%)	96	73	98	97	99	96	95	96

In total, 5 units of RBCs, 20 units of cryoprecipitate, 7 units of FFP and 6 g of fibrinogen were given to the patient. She was discharged from the hospital on the 14th postnatal day without any complications.

DISCUSSION

AFE is a complication of labor that usually results in death. The classic triad of AFE is sudden onset hypotension, hypoxia and coagulopathy. Body temperature should be normal and symptoms should have appeared during labor, placental delivery or up to 30 minutes later. The diagnosis of AFE is made clinically and excludes pulmonary embolism, peripartum cardiomyopathy, septic shock, myocardial infarction, venous air embolism, eclampsia, anaphylaxis, cephalad spread of spinal anesthetic(5).

The main risk factors for the development of AFE are advanced maternal age, placenta previa, placenta accreta, ablatio placenta, preeclampsia, eclampsia, gestational diabetes, multiple pregnancies, polyhydramnios, cerebrovascular, heart and kidney diseases, multiparity, male fetuses, trauma such as uterine rupture, cervical laseration, amniocentesis, cordocentesis, amnioinfusion, amniotomy, labor, labor induction, cesarean delivery, dilatation and curettage (5, 6). Regardless of the etiology, early diagnosis and treatment of AFE can be lifesaving.

In our case, the risk factors for AFE were male fetus, history of invasive prenatal diagnostic procedure, labor and multiparity. Our patient did not suffer from chronic hypoxia, as the patient who developed respiratory arrest during delivery was intubated in the early period. Bleeding was controlled with uterotonic treatments, intrauterine balloon, replacement of depleted blood products and electrolytes applied rapidly in the postpartum period. With this approach we could effectively manage two serious complications of AFE, hypoxia and DIC.

There is no specific diagnostic test or treatment for AFE. The main cause of mortality and morbidity associated with AFE is DIC and its complications. In the present case, early diagnosis, intensive and serial fibrinogen replacement are the main factors behind the patient's sequela-free survival.

DISCLOSURE

No potential conflict of interest was reported by the authors.

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