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Case report / Olgu sunumu

CARDIORESPIRATORY ARREST DURING CAESAREAN SECTION; AMNIOTIC FLUID EMBOLISM; CASE REPORT.

Amniotik sıvı embolisine bağlı olarak sezaryen sırasında gelişen kardiyopulmoner arrest olgusu; Olgu sunumu.

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ABSTRACT

Amniotic fluid embolism (AFE) occurs rarely. It is one of the major causes of maternal mortality in caesarean section. A 25-year-old primigravida was admitted for delivery at 39 weeks and 3 days gestation after a normal pregnancy. A live female infant weighing 2920 g was delivered. Then bradycardia and kardiac arrest occured.medical and obstetric management was started immediately. It was stopped inhalated agent and continued mechanical ventilation with 100% O_2 concentration. Atropine 2 gr and adrenaline 2 gr were given. After resuscitation performed the patient's blood pressure was 110/80 mmHg. At the end of surgery, after one hour of monitoring in the recovery room, the patient was transferred to the intensive care unit. The presentation of this syndrome was hypotension, massive bleeding and sudden cardiac arrest. Early diagnose and management are the key points for the favourable outcome. The final diagnosis was AFE leading to profuse post-partum haemorrhage sudden cardiac arrest.

Key words: Amniotic fluid embolism; caesarean section; cardiopulmonary arrest.

ÖZET

Amniyotik sıvı embolisi nadiren görülür. Sezaryen anne ölümlerinin en önemli nedenlerinden biridir. 25 yaşında, 39 hafta 3 gün ve ilk gebeliği olan hasta sezaryen operasyonuna alındı. 2920 gr ağırlığında canlı bir kız bebek doğdu. Doğumu takiben ani bradikardi ve kardiyak arrest gelişen anneye hemen medikal ve obstetrik düzenlemeye başlandı. İnhalasyon ajanı durduruldu ve %100 O_2 konsantrasyonu ile mekanik ventilasyona devam edildi. Toplam olarak atropin 2 gr ve adrenalin 2 gr intravenöz olarak verildi. Resüsitasyona cevap alınan hastanın kan basıncı 110/80 mmHg idi. Operasyon sonrası uyanma odasında bir saat izlenen hasta yoğun bakım ünitesine transfer edildi. Bu sendromda hipotansiyon, massif kanama ve ani kardiyak arrest gelişmesi tipiktir. Kesin tanıda amniyotik mayi embolisinin yol açtığı doğum sonrası kanama ve ani kardiyak arrest olması yolgösterici olmalıdır. Tedavide erken teşhis ve tedavi en önemli anahtar noktalardır.

Anahtar kelimeler: Amniotik sıvı embolisi, sezaryen, kardiyopulmoner arrest.

INTRODUCTION

Amniotic fluid embolism occurs rarely. It is one of the major causes of maternal mortality in caesarean section (1). It was first described in 1926 and in 1941. It became an established clinical entity (2). The classical description is sudden onset of dyspnoea,

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cyanosis and hypotension out of proportion to the blood loss, Cardiorespiratory arrest may follow these clinic symptoms. In addition to haemodynamic collapse and pulmonary injury, 40% of patients surviving the initial haemodynamic insult may develop a disorder of coagulation in a wide clinic spectrum like minor disturbances in platelet count or disseminated intravascular coagulation (DIC) (3–10). Observation of amniotic cells in the central venous blood as well as in the broncho-alveolar fluid supports AFE diagnose. Because initial characteristic signs may be mistaken and therefore make diagnosis difficult. The early management is the key point for a favourable outcome so the clinician have to be more quick for diagnose. We report on a non-fatal case of AFE occurring during a caesarean section.

Case

A 25-year-old primigravida was admitted for delivery at 39 weeks and 3 days gestation after a normal pregnancy. She was otherwise healthy, she had not been taking any medication and had no known allergy. Before anesthesia, biochemical parameters were normal in the patient. The labour was spontaneous with a blood pressure of 128/74 mmHg pulse; 88 /min on admission and no temperature elevation was noted. Her coagulation profile was also normal. Vaginal examination showed the cervix to be dilated 4 cm four hours later, vaginal examination showed the cervix to be only 5 cm dilated and a lower segment caesarean section was performed under general anaesthesia because of labour arrest and reduced fetal heart rate variability. During this period, maternal heart rate, blood pressure and pulse oximetry were within normal range. A live female infant weight 2920 gram was delivered. Apgar scores were 7 at 1 and 9 at 5 minutes later. The amniotic fluid was grossly stained and the umbilical artery pH was 7.24. Immediately following delivery of the placenta hypotension occured a profuse post-partum haemorrhage was observed with uterine atony. Then, bradycardia and cardiac arrest occured, medical and obstetric management was started immediately. The trachea was intubated and assisted mechanical ventilation maintaned with 100% O_2 concentration. Atropine 2 gr and adrenaline 2 gr were given. After resuscitation performed the patient's blood pressure was 110/80 mmHg. She was tachycardic and adrenaline infusion was arranged (0.5 lg/kg-1-min-1). Arterial blood gas values under assisted mechanical ventilation with a 100% inspired oxygen concentration were pH 7.23, $PaCO_2$ 38.5 mmHg, PaO_2 185 mmHg, $cHCO_3$ 15.9 mmol/L. Resuscitation included infusion of colloid Hydroxy Ethyl Starch (HES) 1L, Ringer's lactate 3 L. Intrauterine examination was performed. Two infusions of oxytocin (Syntocinon-10 units) were administered, one intravenously and one into the myometrium it was continued uterine massage. Uterine atony responded to oxytocin, intravenous infusion of prostaglandin was given. Five units of prostaglandin (Cytotec- 200 l gr) were given rectally with another prostaglandin intravenous infusion. At the end of surgery, after one hour of monitoring in the recovery room, the patient was transferred to the intensive care unit (ICU). Upon admission in the ICU her blood pressure was 120/80 mmHg. She remained tachycardic and auscultation of the chest was normal. Lactic acidosis was noted (lactic acid: 4 mmol/L; normal range 0.4 -2.2 mmol/L). Arterial blood gas values under assisted mechanical ventilation with a 50% inspired oxygen concentration were pH 7.29, $PaCO_2$ 33.5 mmHg, PaO_2 73 mmHg. Liver function tests and D-dimer level as0.27 mg/L were normal (normal range 0-0.3 mg/L). The chest X-ray was normal and the electrocardiogram showed only a sinus tachycardia.

Catecholamine use was continued and tracheal entubation was extubated 3 h after admission. Neurological examination was normal without sedation. She was discharged from the ICU on the second day postpartum. The final diagnosis was AFE leading to profuse post-partum haemorrhage.

DISCUSSION

Amniotic fluid embolism is a rare event, ranging from one in 8000 to one in 80.000 deliveries (4). The AFE syndrome was first described by Meyer in 1926. Steiner and Luschbaugh published a maternal mortality case series that included eight women who had squamous cells and mucin, presumably of fetal origin, within their pulmonary vasculature. After this publishment in 1941, AFE became a clinic entity. The classic descriptiton is sudden onset of dyspnoea, cyanosis and hypotension out of proportion to the blood loss, followed quickly by cardiorespiratory arrest (4). This initial episode is usually followed by disorders of coagulation. In our case report, a distinctive feature should be noted, coagulopathy disorder and massive bleeding. The clinical coagulation disorder was the first sign in the course of the caesarean section. Indeed, the biological signs of DIC occurring during AFE are difficult to ascribe to the AFE itself. The massive haemorrhage and the transfusion needed for management could induce the same coagulation factor deficit. Porter et al. isolated eight clinical cases where acute coagulopathy developed without antecedent hypotension, hypoxia or evidence of any other events or disease processes including placental abruption (5). Five patients were delivered by caesarean section. Uterine atony was noted in five patients. Acute haemorrhage occurred postpartum in seven patients and intrapartum in one patient. Six of eight patients exsanguinated despite appropriate medical management. Porter et al. concluded that isolated fatal DIC is a form fruste of AFE. It is mortality rate similar to more classic form. Recent studies confirmed the association of AFE and DIC (6–9). The pathophysiology of this coagulo-

pathic disorder remains controversial. As discussed in the review on AFE by Davies et al., the experimental data suggest that amniotic fluid has a direct factor X activating property and thromboplastin-like effect (7). A tissue factor found in the amniotic fluid could account for the activation of the coagulation pathway (11). Encephalopathy is not commonly seen in AFE. Indeed, if neurologic syndromes are present in the course of AFE, they usually develop after severe prolonged hypotension or even cardiac arrest. In all circumstances, the diagnosis of AFE is difficult to establish on the basis of clinical and laboratory findings. The usual clinical criteria are as follows; acute hypotension or cardiac arrest, acute hypoxia, coagulopathy absence of other explanations for the manifestation observed, onset during labour within 30 minutes of delivery or surgical abortion (12). The other diagnoses to consider in a patient presenting with the above signs are haemorrhagic shock, placental abruption, sepsis, pulmonary thrombo-embolism, aspiration of gastric contents and eclampsia. The finding of fetal squamous cells or other amniotic fluid material in the maternal pulmonary circulation is neither specific nor sensitive for the diagnosis of AFE. Finding fetal squamous cells in the broncho-alveolar lavage may support the diagnosis of AFE (13-14).

In conclusion, we report on a case of AFE in a 25-year-old woman. The presentation of this syndrome was hypotension, massive bleeding and sudden cardiac arrest. Early diagnose and management are the key points for the favourable outcome.

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