PRESENTATION OF AMNIOTIC FLUID EMBOLISM AS ACUTE HYPOXIA DURING ELECTIVE CESAREAN SECTION

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A case of a pregnant woman undergoing an elective cesarean section (CS) who developed an intra-operative hypoxemia that was refractory to treatment with oxygen and ventilation is presented.

Keywords: amniotic fluid embolism, acute hypoxia.

CASE REPORT

A 39-ye-old, 120 kg woman with a 38-week gestation presented to hospital for an elective CS. She gave a history of a previous CS under general anesthesia since 2 yrs. She suffered from mild bronchial asthma, had not needed her salbutamol inhaler for over 6 months, but was otherwise previously healthy. Preoperative cardiovascular and chest examination was normal. Hematological and biochemical blood results appeared within normal limits.

After preoxygenation and monitors application, anesthesia was induced with thiopental (5mg.kg\(^{-1}\)) preceeded by lidocaine (1.5 mg.kg\(^{-1}\)), and succinylcholine (1.5 mg.kg\(^{-1}\)). Tracheal intubation was performed and pancuronium (0.1 mg.kg\(^{-1}\)) administered in order to maintain an acceptable level of muscle relaxation (V\(_{t}\) 10 ml.kg\(^{-1}\), 10 cycles/ min, I:E 1:3). Then a dramatic clinical deterioration occurred; tachycardia (up to 160 beats /min), hypoxemia (with a pulse oximeter saturation of 60%). Systolic arterial blood pressure fell to a minimum value of 40 mmHg. Neither epinephrine (in repeated doses up to 1mg in total) nor additional fluid administration (lactated ringer’s solution) was effective in elevating it. Coarse inspiratory rhonchi were heard bilaterally on auscultation with increased peak inspiratory pressure. A healthy fetus was delivered (Apgar scoring: 8) but the mother became increasingly hypoxic and cardiac arrest followed profound hypoxia. Cardiopulmonary resuscitation started on the spot (external chest compression, epinephrine, cardioversion) but all the measures failed and death followed.

DISCUSSION

Amniotic fluid embolism (AFE) is a rare (1:20,000 deliveries) but potentially lethal complication (86% mortality rate) that can occur during normal delivery or cesarean section.\(^{(1)}\) Even with ideal care, AFE remain a disease with an extremely poor outcome.\(^{(2)}\) Entry of amniotic fluid into the maternal circulation can occur through any break in the uteroplacental membranes. In addition to desquamated fetal debris, amniotic fluid contain various prostaglandin and leukotriens which appear to play an important role in the genesis of this syndrome.\(^{(3)}\)

Presentation is usually sudden tachypnea, hypoxemia, shock and generalized bleeding. Pulmonary edema may develop which has both cardiogenic and non-cardiogenic components.\(^{(4,5)}\) Acute left ventricular dysfunction appears to be a common feature. The diagnosis of AFE is one mostly of exclusion. It can be firmly established only by demonstrating fetal elements in maternal circulation (autopsy). Amniotic fluid embolism should always be suggested by acute circulatory collapse, respiratory distress or coagulopathy.\(^{(6,7)}\)

Treatment consists of aggressive cardiopulmonary resuscitation and supportive care. Epinephrine may be the first-line agent of choice. Corticosteroids may be helpful. Therapeutic heparinization to minimize consumption coagulopathy remains controversial.\(^{(6)}\)

In summary, we have presented a mortality case due to intraoperative amniotic fluid embolism suggested by acute hypoxemia and circulatory collapse, who didn’t respond to resuscitative measures and diagnosis was established by demonstrating fetal elements in maternal circulation at autopsy.
REFERENCES


