Amniotic fluid embolism and disseminated intravascular coagulation complicating hypertonic saline-induced abortion

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Summary

Amniotic fluid embolism (AFE) associated with disseminated intravascular clotting (DIC) is usually fatal. Such a combination generally occurs at the term of pregnancy or in the immediate postpartum period. A case of AFE with DIC following a hypertonic-saline-induced abortion, is reported. The patient was in the midtrimester with a live fetus. Prompt recognition and treatment of her condition ensured her complete recovery. Such a case has not, to our knowledge, been previously reported.

KEY WORDS: amniotic fluid, clotting, abortion, embolism.

Introduction

Intra-uterine instillation of hypertonic solutions to induce abortion was introduced as a simpler, safer alternative to hysterotomy, when uterine size makes interruption by curettage or vacuum aspiration, dangerous. Hypertonic solutions can be used from the 12th to the 28th weeks of pregnancy.

Many complications of this technique have been reported: fever and infection (Gillmer, Friend and Beard, 1971; Goodlin et al., 1969; Mackenzie, Roufa and Tovell, 1971; Stim, 1972), haemorrhage (Stim, 1972), coagulation defects (Beller et al., 1972; Brown, Davidson and Phillips, 1972; Goodlin, 1971; Halbert et al., 1972), hypernatraemia (Cameron and Dayan, 1966; Droegemuller and Greer, 1970; Kerenyi, 1969), water intoxication (Abdul-Karim and Assali, 1961; Goodlin, 1971; Whalley and Pritchard, 1963), transplacental haemorrhage (Jorgenson, 1969; Parmely, Montague and Miller, 1970; Voigt and Britt, 1969), retained placenta (Frisoletto and Pokoly, 1971; Futoran, Lowenstein and Peacock, 1969; Kerenyi, 1971) uterine lacerations (Swane, 1960; Wood, Booth and Pinkerton, 1962), and maternal death (Cameron and Dayan, 1966; Goldman and Eckerling, 1972; Pathak, 1968). Amniotic fluid embolism (AFE) is a further possible complication.

Case report

A 25-year-old woman, gravida I, para o, was admitted at 20 weeks' gestation, for induction of abortion following a first trimester rubella infection. The haemoglobin was 11-5 g/dl, the white cell count, platelet count coagulation tests, serum electrolytes, electrocardiogram (ECG) and chest X-ray were normal. At midday 200 ml of amniotic fluid were aspirated and replaced by 200 ml of 20% saline. Twenty-two hours later, routine coagulation tests and electrolytes were normal. Twenty-six hours after saline instillation, uterine contractions and a fetal heart were detectable. Thirty hours following instillation, intravenous oxytocin (52 mu./min) was given to increase irregular uterine contractions. The fetal heart was still detectable at this stage. Three hours before she delivered, the patient required analgesia for painful contractions, which were palpable, 4 in 10 min. At 33 hr the membranes ruptured and 45 min later, a dead fetus weighing 480 g, and complete placenta were delivered. The blood loss was about 100–150 ml and contained clots.

Acute respiratory distress appeared 5 min after completion of the abortion, followed by apnoea and cyanosis. The patient developed hypotension (systolic pressure 80 mmHg) and tachycardia (140/min). Brief generalized convulsions were followed by unconsciousness. The patient was intubated and ventilated with 100% oxygen. Vaginal bleeding had increased, but clotting was not now evident. A tentative diagnosis of amniotic fluid embolism and disseminated intravascular coagulation (DIC) was
later confirmed by laboratory studies and serial chest X-rays.

ECG shortly after the episode was suggestive of acute right heart strain. Chest X-ray 90 min after the respiratory distress showed pulmonary artery engorgement at the hilum and finely increased vascular marking in the right lower lung field (Fig. 1). Twelve hours later, a uniform opacity was seen in the right lower lobe in the chest X-ray. Hypoxaemia and hypocarboxaemia supported the diagnosis of acute pulmonary embolism.

Thirty minutes after the apnoeic episode, whole blood clotting time, prothrombin time and partial prothrombin time were all prolonged. Plasma fibrinogen concentration, previously normal, was less than 50 mg/dl. Blood electrolytes were normal at this stage. The patient was given 1000 ml of fresh whole blood to correct blood loss and coagulation defects. One gram of hydrocortisone was given intravenously, and this dose was repeated after 4 hr. Intravenous aminophylline 6 mg/kg was given in 15 min, and a continuous infusion of 0.5 mg/kg/hr was then maintained for 4 hr. The patient's hypoxaemia was treated with intermittent positive pressure respiration and 40% oxygen for 1 hr after the acute episode, when she was extubated. Respiratory support was continued with an oxygen mask for another 30 hr and blood gases were checked. The chest X-ray 2 days later showed dramatic clearing of the right lower lobe opacity. The subsequent course was uneventful.

Discussion

Amniotic fluid embolism is one of the causes of death during labour and in the immediate postpartum period. It is estimated that there is one death in 20,000–30,000 deliveries (Courtney, 1974; Resnik et al., 1976). Probably mild episodes of AFE are more frequent. AFE should be suspected in all cases of respiratory distress, maternal collapse, coagulation failure or uncontrolled haemorrhage during labour, delivery or postpartum (Courtney, 1974; Reis, Pierce and Behrendt, 1969).

We are not aware of any previous report of AFE.
after saline induced abortion involving a live midtrimester fetus and maternal recovery. Since AFE and DIC at term are the sequelae of uterine contractions, with intravasation of amniotic fluid (Sparr and Pritchard, 1958; Wagatsuma, 1965), it is logical that a similar event may occur during saline induced abortion.

Early diagnosis with appropriate and prompt treatment can ensure, as in the present case, pulmonary and cardiovascular resuscitation, with complete recovery.

References


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