AMNIOTIC FLUID EMBOLISM AND DEFIBRINATION

(A Case Report)

by

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Introduction

Among the hazards of oxytocic agents, amniotic fluid embolism is a grave complication which has a poor prognosis for the patient. Ever since Steiner and Lushbaugh in 1941 reported a fatal case of amniotic fluid embolism, a number of similar cases have been reported. Reid *et al* (1953) explained that clinically a syndrome of cardiorespiratory failure together with intense shock is always associated and haemorrhagic syndrome is often associated. It is a very serious condition when it occurs in the last stage of labour or during delivery. Usually the patient dies of shock, if she survives shock, she may get uncontrolable postpartum haemorrhage due to disseminated intravascular coagulation and the fibrinolysis, especially the latter which can lead to severe life endangering situations. The case recorded here documents an association between oxytocic drug administration and amniotic fluid embolism which subsequently produced defibrination and uncontrolable haemorrhage.

CASE REPORT

A, 19-year-old primigravida was admitted on February 18, 1978 at 02.00 hours as an emergency with history of term pregnancy and labour pains of 24 hours duration. Prior to admission the patient had been attended by a doctor who had administered oxytocin as a drip for 16 hours in her nursing home. When the delivery was found difficult, the case was referred to hospital. At 03.20 hours, a live female baby was delivered by outlet forceps. The placenta and membranes were delivered spontaneously and completely. Prophylactic ergometrin 0.25 mg was administered intravenously. At 04.00 hours there was profuse bleeding per vaginum. Exploration revealed no tear in the cervix. The uterus was intermittently contracting and relaxing. Ergometrine 0.25 mg was administered intravenously and 40 units of syntocinon in 500 ml of 5% dextrose were infused as a drip. Blood was withdrawn for clot observation and blood grouping and cross matching. The blood pressure had fallen from original 130/80 mm hg. to 80 mm Hg systolic. Three bottles of fresh blood were infused and 8 mg of dexamethasone was administered intravenously. General condition of the patient had deteriorated. She had developed cardiorespiratory distress and shock. Bimanual compression of the uterus was attempted for 2 hours. Bleeding persisted after the compression was removed. It was observed that the blood had failed to clot. A tentative diagnosis of defribrination was made. The patient was transfused 8 bottles of fresh blood, 2 bottles of dried plasma and 6 bottles of Haemacoele. She was administered calcium chloride 5 ml intravenously, soda bicarb 60 ml intravenously and dexamethasone 40 mg intravenously. Inspite of vigorous treatment, the general condition of the patient did not improve although clot was formed at that stage. At 11.00 hours the uterine cavity was packed with ribbon gauze. The patient developed pulmonary oedema. She was given diuretics and digitalized. She was kept in Fowler's position and administered oxygen continuously. At 15.00 hours the patient started gasping. Her blood pressure and pulse were unrecordable. At 17.00 hours the patient expired. Due to lack of laboratory investigation facilities, we could not plan either Heparin or Trasylol therapy.

Discussion

Amniotic fluid embolism has been reported to occur as a complication of oxy-

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tocic drug administration. The case recorded in this report has demonstrated an association between oxytocic drug infusion and amniotic fluid embolism which subsequently produced afibrinopoenia and uncontrolable post-partum haemorrhage. We record this case to emphasize that defibrination is not an uncommon entity associated with pregnancy as it has been reported in the literature. Coagulation profiles, and qualitative and quantitative assessment of fibrinolytic activity during labour, delivery and the early puerperium in normal pregnancies has suggested that a minor degree of physiological defibrination develops during normal labour i.e. qualitatively similar to, but of much lesser magnitude than, the pathological defibrination syndrome commonly associated with abruptio placentae or prolonged intrauterine death (Kleiner and Greston 1976).

Some degree of defibrination which occurs in pathological pregnancies may not usually reach clinical significant levels to cause hypofibrinoganemic haemorrhage. It has been observed that only 26 per cent of the cases with proved diffuse intravascular coagulation may have clinically significant haemorrhage.

The author can recall two other cases of amniotic fluid embolism which had occured following syntocinon drip. One patient had caesarean section for foetal distress. Half an hour after the operation, she had severe post-partum haemorrhage and bleeding from the abdominal wound which was treated effectively with dried plasma and fresh blood. The case expired 13 days after the operation due to acute renal failure. The other patient had caesarean section for foetal distress. During the operation, she developed shock and cardiorespiratory distress and expired in the theatre. The liquor amnii is thought to reach the maternal circulation through the placental site or through lacerated endo-cervical veins depending upon the rupture of the membrane in the upper or the lower segment respectively. The original embolic theory of Steiner and Lushbaugh (1941) has been rejected by Reid *et al* (1953) who believe that the liquor amnii and its anticoagulant activity is lethal and amniotic fluid perfusion is a better term than amniotic fluid embolism.

Theoretically, pathologic defibrination can develop due to diffuse intravascular clotting. As fibrin is being deposited throughout the vascular tree, fibrinogen as well as other coagulation moieties are removed from the circulation and bleeding occurs because the blood is depleted of the proteins necessary for normal hemostasis. The basic mechanism in this coagulation disorder, the factory which initiates diffuse I.V. clotting is not known with certainty. Among obstetricians and gynaecologists it is generally assumed that thromboplastic substances from foetal tissues are released into the maternal circulation during or immediately prior to delivery. There are convincing animal experiments and clinical observations to support this contention.

Acknowledgement

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References

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