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Rescue of an intrauterine dead fetus with IUGR: a resurrection

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Correspondence to Huriye Ayse Parlakgumus, ayseparlakgumus@yahoo.de**Summary**

Intrauterine growth restriction is associated with increased perinatal morbidity and mortality, as well as certain metabolic diseases such as coronary heart disease, stroke, diabetes and hypertension in adult life. In this report, the authors describe a case of successful neonatal resuscitation after caesarean delivery in a severe growth-restricted fetus with absent cardiac activity following prolonged intrauterine bradycardia. The neonatal outcome was favourable, with the infant showing only unilateral hearing loss at 13 months' follow-up.

BACKGROUND

Even the fetuses with absent cardiac activity, exposed to chronic hypoxemia in the intrauterine life, can respond to resuscitation.

CASE PRESENTATION

A 30-year-old, gravida 5 para 2 pregnant woman was admitted to our emergency department at 33 weeks 4 days of gestation; she complained of pelvic pain and vaginal bleeding and had a history of amniotic fluid leakage for 2 days. She had no remarkable history; she did not smoke. She did not attend any antenatal follow-up until 33 weeks of gestation.

INVESTIGATIONS

The patient was referred to the Baskent University Department of Obstetrics and Gynecology with a diagnosis of severe intrauterine growth restriction (IUGR),¹ reverse flow in the umbilical artery as well as preterm premature rupture of the membranes. On admission, the patient's blood pressure was 110/70 mm Hg, heart beat was 80/min and there were no clinical signs of chorioamnionitis. Vaginal examination revealed a posterior, 1 cm dilated cervix, but amniotic fluid leakage was not detected. Ultrasound examination revealed a severely growth-restricted fetus with an estimated fetal body weight of 1150 g (below 3rd percentile), as well as anhydramnios and severe persistent fetal bradycardia (40 beats/min) with reversed flow in the umbilical artery.

TREATMENT

The patient was turned on her left supine position, nasal oxygen was administered and intravenous fluids were commenced. The bradycardia did not improve despite these measures. Therefore, a decision was taken to conduct an emergency caesarean delivery, and the patient was immediately transferred to the operating theatre. Since the condition was an obstetric emergency, venous doppler studies were not performed. Preparation of the surgical team occurred within 20 min. Before placement of sterile surgical drapes, sonographic re-evaluation revealed an absence of fetal cardiac activity. Verbal consent was

obtained from the patient to proceed with the caesarean delivery. A 1200 g hypotonic female infant without cardiac activity was delivered within 3 min after making the decision to proceed. The infant had a first-minute Apgar score of 0/10 and an umbilical artery pH of 6.81. Resuscitation of the neonate was commenced immediately after delivery and continued for 30 min. After regaining bradycardia, the neonate was transferred to a neonatal intensive care unit with an absence of deep tendon reflexes and spontaneous movements. A chest x-ray revealed pneumothorax, and a chest tube was inserted.

OUTCOME AND FOLLOW-UP

The postnatal period was further complicated by neonatal seizures. Electroencephalogram revealed baseline electrical activity distortion; a combination of antiepileptic agents was administered. Cranial MRI revealed subacute haemorrhage in supra- and infratentorial areas, intraventricular haemorrhage and bilateral ischaemia at the level of the cerebral cruri. Fifty-seven days later, the infant was discharged from the hospital with cranial ultrasonography findings consistent with bilateral ventriculomegaly and fronto-parietal encephalomalacia. The mother had an uneventful postoperative period and was discharged 36 h after caesarean delivery. Pathological examination of the placenta revealed focal infarction areas and no signs of intrauterine infection.

The infant is now 13 months old and she walks, and exhibits appropriate mental and motor development for her age except for unilateral hearing loss.

DISCUSSION

Although there is no reported case like this in the literature, successful neonatal resuscitation after emergency caesarean delivery of a fetus with absent cardiac activity is possible. The presence of severe IUGR and persistent fetal bradycardia followed by cardiac asystole indicates sustained fetal hypoxia. Severe fetal acidaemia detected at birth also supports this. Hypoxia is the main cause of ischaemia-derived neural damage of the neonates.² It has been established that acute asphyxia of the fetus in utero may result in a spectrum of effects on the fetus, including death, or

survival with permanent neurologic damage, or apparent complete recovery. The severity of damage depends on the degree and duration of asphyxia and a number of sensitising factors, including prior metabolic and cardiovascular status of the fetus, differential sensitivity of the heart and brain to asphyxia and gestational age. The fetus has a number of compensatory mechanisms that allow it to survive periods of oxygen limitation without permanent damage to the brain. The fetus can increase cerebral blood flow to increase oxygen delivery to the brain and can decrease its metabolism by electrophysiological and behavioural state changes.³ Cerebral ischaemia and reduced metabolism to <50% of control is probably necessary for permanent brain damage to occur. In human pregnancy, factors consistent with intrapartum asphyxia lasting until delivery as a cause of fetal neurologic damage include absent fetal heart rate variability, umbilical cord arterial pH <6.8, base excess ≤20 mEqL⁻¹, severe and prolonged newborn depression with Apgar score of ≤3 at 10 min, seizure activity in the first day of life and damage to the non-cerebral organs and regions.⁴ However, these factors are neither independently nor collectively predictive of asphyxial brain damage. It is concluded that permanent neurologic damage or death can occur in the fetus due to single or repetitive episodes of hypoxia or asphyxia, but it is not yet possible to predict the occurrence or extent of such damage in an individual fetus.⁵ There are many factors that influence the fetal response to an asphyxial insult. The effect upon the fetus may be influenced by whether the asphyxial event is the first or the last of a series of asphyxial episodes and the duration of the asphyxial episode. The characteristics

Learning points

- The interval between the onset of cardiac asystole and delivery is not established in this case.

of the fetus, that is, maturity (preterm vs term), and fetal growth small for gestational age or appropriate for gestational age may influence the fetal response to an asphyxial insult.⁶ Less severe but prolonged asphyxia is associated with severe brain injury than when an insult is brief but severe.⁷

Competing interests None.

Patient consent Obtained.

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