An alternative management for growth retarded fetus with absent end-diastolic velocity in umbilical artery and normal cardiotocography

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Abstract

OBJECTIVES: Intrauterine growth retardation (IUGR) is associated with fetal adverse conditions. The most important cause of growth restriction and poor perinatal outcome is chronic fetal hypoxemia (CFH). Adaptation to CFH can be studied by Doppler velocity waveform on umbilical and fetal arteries and cardiotocography (CTG).

METHODS: Preterm delivery, as an elimination of CFH, has to be confronted with the risks of prematurity. A special situation may occur when CTG is normal at the absence of end-diastolic velocity (AEDV). AEDV in the umbilical artery precedes the onset of abnormal CTG, whose duration differs considerably among the fetuses. The time after the onset of AEDV in pregnancy may be utilized for performing exact diagnosis by fetal blood analysis.

CASE: Primigravida at 30 gestational weeks was referred because of IUGR. IUGR, AEDV, oligohydramnion, and normal fetal anatomy were revealed. CTG was normal. Indication for cordocentesis was to perform cord blood gases analysis and to obtain fetal caryotype. Cordocentesis revealed normal caryotype, values of pH, and fetal blood gases were considered satisfactory. Continuation of pregnancy was decided in spite of persistent AEDV. At 33 gestational weeks pathological CTG was an indication for induction of labor. Labor, delivery, umbilical blood gases, postpartal and neonatal outcome were normal.

CONCLUSION: In the case of fetal monitoring controversy assessment of umbilical blood analysis may be crucial. This examination is significant and independent of the interval between cordocentesis and the onset of CTG pathology. This interval may be utilized for intrauterine treatment and for optimizing obstetric management.
INTRODUCTION

Decision for preterm delivery is very difficult in the cases of expected prematurity of the fetus with the diagnosis of chronic fetal malnutrition. The goal is to prevent intrauterine fetal hypoxemia and acidosis and to overcome the factors causing harm to the fetus and the newborn. Fetuses with intrauterine growth retardation (IUGR) are typical example of the described situation. Fetal biometrical measurement cannot distinguish the cause of the growth retardation, nor because of limited haemodynamic situation neither because of other reasons like chromosomal aberrations (Wienerroither et al., 2001). Pathological blood flow in fetoplacental unit is easily observed using Doppler technology by analysing the characteristics of the Doppler velocity waveform (DVWF). By studying DVWF on umbilical and fetal arteries it is possible to assess the characteristics of the blood flow from mother to the fetus and to monitor the aspects of the fetal adaptation if CFH is present. If present, it is possible to monitor the evolution obtaining information of clinical validity in order to optimize the management and the timing of the delivery, if necessary. This pathological situation can be caused for example by haemodynamic placental insufficiency (Šuška, 1995; Foltinova et al., 2007).

However, Doppler flowmetry is not the only way to monitor fetal condition. Therefore, some situations can bring controversy when Doppler flowmetry results are in controversy to the results of cardiotocography (CTG). It is therefore necessary, to evaluate objectively the fetal condition and to set the optimal management, especially in cases of premature fetus (Schwarze et al., 2005; Urdzik et al., 2007).

Cordocentesis can be performed in the case of fetus with serious IUGR in order to get fast caryotype and to analyze blood gases and pH level in the umbilical blood (Arduini et al, 1996, Ferianec et al., 2000; Figueras et al., 2005). Its efficacy is evident in cases of unclear or opposite results from different fetal monitoring modalities. Cordocentesis offers information that cannot be gained by any other diagnostic method. It can be helpful to set the best management for both, the monitored fetus and indirectly for the mother, with minimal risk taken.

The aim of this article is to present, in specific clinical case, possible diagnostic and therapeutic guideline in this controversial situation.

CASE

33-year-old woman, first time pregnant, was sent to our clinic in the 31th week of pregnancy with the suspicion of oligohydramnion. Ultrasound examination proved the result: oligohydramnion, fetal hypotrophy with severe flowmetry defect – AEDV (absent end diastolic velocity) (Figure 1). The patient was admitted to the hospital, taken blood samples for blood examination and was set for continual CTG monitoring. The CTG showed normal, reactive curve during the whole monitoring time without contractions of the uterus (Figure 2). Because the Doppler flowmetry remained pathological throughout 24 hours – AEDV persisted, we performed invasive prenatal diagnostics – cordocentesis, despite physiological CTG in order to get fast caryotype, umbilical blood pH, and fetal blood gases. At the same time we performed amniocentesis to evaluate the maturity of the fetus.

Cordocentesis and amniocentesis were performed without complications. We have considered the results of the umbilical blood analysis to be satisfactory and therefore decided for expectant management. Thereafter we got the results of fetal maturity L/S – 1.5 and AF Krea – 157, that proved the expectant management as a good decision with regard to the fetal immaturity (Table 1).

Fast caryotypisation of the umbilical blood proved normal male caryotype. During the remaining time of the pregnancy the pathological Doppler waveform persisted to the mentioned degree and the CTG showed no pathology at all.

In the 33th week CTG showed some pathological patterns in the means of periodic early decelerations. After lung maturity induction we induced the delivery in the Table1. Summary results from invasive prenatal diagnostics

<table>
<thead>
<tr>
<th>Results</th>
<th>Amniocentesis + cordocentesis</th>
<th>Normal fetus</th>
</tr>
</thead>
<tbody>
<tr>
<td>L/S ratio</td>
<td>1.5</td>
<td>Over 2.0</td>
</tr>
<tr>
<td>AB</td>
<td>pH 7.39</td>
<td>7.38–7.42</td>
</tr>
<tr>
<td>pCO2</td>
<td>4.85 kPa</td>
<td>4.6–6.0</td>
</tr>
<tr>
<td>HCO3</td>
<td>21.5 mmol/l</td>
<td>21–29</td>
</tr>
<tr>
<td>BE</td>
<td>–2.8 mmol/l</td>
<td>–2 to +2</td>
</tr>
<tr>
<td>pO2</td>
<td>2.72 kPa</td>
<td>2.8–4.4</td>
</tr>
<tr>
<td>sO2</td>
<td>50.3%</td>
<td></td>
</tr>
<tr>
<td>Caryotype</td>
<td>46XY</td>
<td></td>
</tr>
</tbody>
</table>

Figure 1. AEDV at the time of admittance
33th week of pregnancy with the mature cervical status. The patient was set to continual CTG monitoring during I. and II. stage of the delivery. The delivery lasted 5 hours without severe CTG pathology (Figure 3). Newborn, male, with the Apgar scoring: 8, 9, 10. Postpartal newborn's blood gases and pH were in the physiological range. Morphological examinations of placenta and umbilical cord, which was twisted ones around newborn's neck, were normal. Following histopathological examination of the umbilical cord and placenta did not find any pathology. Postpartal course of the mother and the fetus were physiological.

DISCUSSION

Histopathological findings suggest that AEDV in umbilical arteries is associated with placental lesions characteristic for reduction in number of arterioles in tertiary villi (Giles et al., 1985). As a result, the exchange of oxygen and nutrition through placenta is reduced which causes alteration in acidobasic homeostasis in umbilical blood (Nicolaides et al., 1988). This state is associated with higher perinatal morbidity and mortality.

AEDV in umbilical artery usually precedes fetal heart rate alterations. However, the length of this preceding period cannot be exactly set (Arduini et al., 1993; Fairle et al., 1991; Ghidini et al., 1993). One study reports that AEDV precedes CTG alteration with a latency of 0 – 35 days (Chaoui et al., 1991).

Based on these facts, we suggest examining umbilical blood in fetus with IUGR in order to diagnose acidobasic status of the fetus and to specify the length of the period that precedes the onset of fetal distress (Figure 4).

It is obvious that cordocentesis is an invasive procedure with its relative risks especially for fetuses with IUGR (Ghidini et al., 1993). However, the final benefit, potentially gained by this process, highly exceeds its potential risks. The best-known fact is that up to 8 % of fetuses with AEDV in umbilical artery have abnormal caryotype, which is difficult to diagnose by sonographic and Doppler examination (Rizzo et al., 1994; Ferianec et al., 2000; Hecher et al., 2001; Mari et al., 2007). In these cases it is therefore indicated, in order to reduce unwanted surgical procedures (cesarean section), to perform fast examination of fetal caryotype. Simultaneous examination of pH in umbilical blood seems to be useful (Figure 5).
CONCLUSION

Up to 8% of fetuses with IUGR and AEDV may have an abnormal karyotype. Its rapid acquisition by fetal blood sampling is indicated to avoid unnecessary interventions. In the case of normal karyotype a simultaneous assessment of the fetal umbilical blood analysis is very useful for these fetuses. This kind of examination is very significant and independent to the interval between cordocentesis and the onset of cardiotocographic pathology. This interval may be utilized for intrauterine treatment, exact monitoring of the fetal wellbeing and to optimize obstetric management. The acidobasic state of the fetus is significant and does not necessarily rely on the time period between the onset of AEVD in umbilical artery and appearance of CTG pathology. The real significance of this information for everyday clinical management in these fetuses needs to be evaluated in future prospective trials.

REFERENCES
