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A case of early onset intrahepatic cholestasis of pregnancy

Spina MR, Murgano D, Celentano C, Pavone G, Fanfani F, Liberati M
G. D'Annunzio" University, SS Annunziata Hospital, Chieti, Italy

Objective

Intrahepatic cholestasis of pregnancy (ICP) pregnancy complicates 0. 2–2% of pregnancies; the incidence is higher in multiple gestations and among certain ethnic populations and it has been correlated with adverse maternal and perinatal outcome. It characteristically involves maternal pruritus without a rash and elevated serum bile acid levels, typically during third trimester. The evaluation of maternal serum bile acids has been used to confirm the diagnosis of intrahepatic cholestasis of pregnancy and identify women at the highest risk (especially when bile acid concentration is >40 μ mol/l) for adverse perinatal outcomes. These include spontaneous preterm birth (via increased oxytocin receptor sensitivity and expression), meconium-stained amniotic fluid, and stillbirth (via sudden cardiac arrhythmia or placental vasospasm).

Methods

This is a case report.

Results

A 34 year-old gravida 2 para 1 patient with history of 1 previous caesarean section was referred to our clinic at 24 weeks of gestation because of intrahepatic cholestasis of pregnancy with worsening pruritus and biochemistry. She had been treated from 21 weeks. First admission was at 24 weeks of gestation with a hospitalization of 3 weeks; there was a second admission at 28 weeks for three days, then at 37 weeks of gestation to deliver via caesarean section. She had been screened for viral and autoimmune hepatitis; no bile duct stones were detected at abdominal ultrasound scan, other possible causes of impaired liver function were excluded. The maximum value of bile acids was of 469, 2 μmol/L at 28 weeks; the maximum value of alanine aminotransferase (ALT) was 217 U/L at 25 weeks; the maximum value of aspartate aminotransferase (AST) was 104 U/L at 24 weeks of gestation. All the values were double checked and confirmed. Fetal surveillance consisted of daily cardiotocography and biophysical profile weekly. Treatment with ursodeoxycholic acid at maximum dosage (450 mg x 4 times a day) improved biochemical markers, although they remained at the upper limits of normal. The patient delivered a healthy female neonate by caesarean section, with a prior corticosteroids course for induction of pulmonary maturity with intramuscular injections of Betamethasone (12 mg a day for two days). Amniotic fluid was mildly stained with meconium. Apgar score was 9 at 1 minute and 10 at 5 minutes. Neonatal pH was 7, 32 and the weight was 3140 gr. Neonatal and postoperative courses were uneventful.

Conclusion

Obstetric cholestasis is a maternal condition, but the fetal outcome is still unpredictable. No specific method of antenatal fetal monitoring for the prediction of fetal death can be firmly recommended. Ultrasound and cardiotocography are not reliable methods for preventing fetal death in obstetric cholestasis. Delivery decisions should not be based only on results of these tests. Risk of stillbirth should be carefully evaluated, balancing against the risk of iatrogenic prematurity.