UMBILICAL BLOOD GASES, FETAL GLYCEMIA AND FETAL-PLACENTAL HAEMODYNAMICS IN PREGNANCIES COMPLICATED BY INSULIN-DEPENDENT DIABETES MELLITUS

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Objective: *Fetal acidaemia and hypoxemia may be the result of placental insufficiency as well as the metabolic effect of maternal diabetes mellitus.*
Methods: *Fetal blood (FB) was sampled from the umbilical vein by transabdominal cordocentesis in 24 women with pregnancy, complicated by insulin-dependent diabetes (IDD) and 22 normal pregnancies at 33-37 weeks gestation, which was performed immediately after Doppler examination. Blood velocity systolic-diastolic (S/D) ratio was calculated in fetal descending aorta, umbilical and middle cerebral arteries, and also cerebro-placental ratio. Blood gases were determined using blood gas analyzer "ABL-30" "Radiometer". Fetal blood samples were assayed for glucose levels by standard glucose-oxidase-perocxidase method. The statistics was made using Student t-test and correlation analyze.*
Results: *Some fetuses (n=7) in diabetic pregnancies were acidaemic, but in the absence of acidosis in the most of them umbilical venous hypoxemia (n=18) and hypercapnia (n=16) were present. The mean S/D ratio in umbilical artery, fetal descending aorta in diabetic pregnancies were statistically significantly higher than in normal pregnancies and cerebro-placental ratio – lower. Associations between S/D ratio in umbilical artery , cerebro-placental ratio and umbilical venous pH (r=-0,596, p<0,01 and r=0,404, p<0,05, respectively), pCO2 (r=0,613, p<0,01 and r=-0,506, p<0,05, respectively) were found. Fetal blood glucose concentrations in pregnancies, complicated by IDD were significantly associated with umbilical venous pH (r=-0,616, p<0,01), pO2 (r=-0,469, p<0,05) and pCO2 (r=0,404, p<0,05).*
Conclusions: *In pregnancies, complicated by IDD fetal-placental haemodynamic changes (increase of peripheral placental resistance and centralization of fetal circulation) and fetal hyperclycaemia have been associated with fetal acid base disturbance.*

FETAL ADRENAL FUNCTION IN NORMAL AND INSULIN-DEPENDENT DIABETIC PREGNANCIES

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Objective: *The functional role of fetal adrenal gland in processes of prenatal and postnatal adaptation is known. Fetal endocrine responses in normal pregnancy and pregnancy, complicated by maternal insulin-dependent diabetes mellitus (IDD) were examined in this study.*
Methods: *Fetal blood (FB) and amniotic fluid (AF) samples were obtained by transabdominal cordocentesis and amniocentesis in 24 women with IDD, at 34-37 weeks gestation and 11 women with normal pregnancies at 30-37 weeks gestation. Normal fetal cortisol synthesis dynamic was examined in 17 normal pregnancies at 20-30 weeks gestation. Maternal venous blood was collected immediately after cordocentesis. Cortisol plasma and AF concentrations were measured using immunoenzimatic assay kit. The statistics was made using Student t-test and correlation analyze.*
Results: *No significant difference in mean fetal cortisol concentrations between different gestation ages of normal pregnancy was found: 49,9±22 nmol/l at 20-25 weeks, 34,5±52,5 nmol/l at 25-30 weeks and 52,5±12 nmol/l at 25-30 weeks gestation. But cortisol levels in FB increased gradually in the III trimester of normal pregnancy from 22,4 nmol/l at 26 weeks to 69,4 nmol/l at 37 weeks gestation and was significantly associated with gestational age (r=0,766, p<0,01). Statistically significant fetal hypercortisolemia was observed in pregnancies, complicated by IDD: cortisol levels in FB were 95,9±41,1 nmol/l in compare with the same normal figures 52,5±12 nmol/l. Mean cortisol levels in AF in diabetic pregnancies were also higher (M=95,7±36,2 nmol/l) from normal (M=71,3±36,2 nmol/l). There was no difference in maternal cortisol concentrations in normal pregnancies (M=688,2±107,8 nmol/l) and diabetic pregnancies (M=788,9±196,07 nmol/l). Significant correlation between fetal and maternal cortisol concentrations was found both in normal (r=0,450, p<0,05) and diabetic pregnancies (r=0,433, p<0,05).*
Conclusions: *Dynamic of fetal cortisol levels in normal pregnancy showed increased activity of the fetal adrenal in late gestation age. This process considered to be one of the factors initiated parturition. Association between fetal and maternal cortisol concentrations acknowledged the active placenta role in fetal cortisol metabolism and transplacental transport of maternal cortisol to fetus. Fetal hypercortisolemia may be the fetal adrenal adaptation reaction to chronic stress associated fetal hyperinsulinemia in diabetic pregnancies.*