FETAL MATURITY IN PREECLAMPTIC PREGNANCIES

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About 10 – 15% of pregnancies are presented with hypertensive disorders worldwide, while preeclampsia as a more dangerous obstetric syndrome complicates approximately 4 – 10% of pregnancies. This rapidly progressive condition is a common global cause of both maternal and perinatal morbidity and mortality. By conservative estimates, hypertensive disorders are responsible for 50.000 – 76.000 deaths each year. According to ACOG, women who receive no prenatal care are more than seven times as likely to die from complications of preeclampsia and eclampsia as women who receive any prenatal care. The immediate and long-term annual costs could be estimated as billions dolars (in the US over 7 Billion dolars per year, approximately).

Preeclampsia is freequently associated with serious and unsolved obstetric problems like prematurity, cerebral pulsy, chronic uteroplacental insufficiency that leads to intrauterine growth retardation (IUGR), placental abruption, etc. In the absence of fetal illness (malformations, chromosomopathies, sepsis, metabolic diseases, ...), immaturity is perhaps the most important prognostic factor of neonatal outcome. It has been calculated that about 70 – 75% of neonatal mortality, morbidity and costs is related to premature deliveries.

Prematurity as a result of both spontaneous and especially iatrogenic premature deliveries, is one of the most freequent and most important consequence of severe preeclampsia. Although fetal maturity is predominantly determined by gestational age, it seems that some pathological conditions could influence on it. Namely, conditions of chronic fetal stress are thought to result in an accelerated lung maturation due to an increase in fetal cortisol production. Chronic uteroplacental insufficiency and fetal growth retardation have been well known such factors. Moreover, chronic hypertension, pregnancy induced hypertension, and preeclampsia are potential chronic stressors that are considered by many authors to accelerate fetal maturity. Some reports, 20 to 25 years ago, have described a decreased incidence of respiratory distress syndrome (RDS) and an earlier lung maturation in infants who are born to preeclamptic mothers. More recently, others failed to confirm these findings reporting no protective effect of preeclampsia in the development of hyaline membrane disease. It could be said that the influence of preeclampsia on fetal lung maturation is quite controversial.

Because published studies relating to the risk of neonatal RDS in preeclamptic patients demonstrate a range of results, we decided to investigate prospectively whether acute RDS is decreased or increased in newborn infants who are born to women with preeclampsia compared with controls.

Material and methods

A potential affection of lung maturity in pregnancies complicated by hypertension and preeclampsia was examined by use of amniotic fluid (AF) lamellar bodies counts (LBC). Amniotic fluid samples were obtained by amniocentesis 1 – 3 days before birth or during cesarean sections. All infants were evaluated by laboratory testing and clinically by attending neonatologists. Radiographic examinations have been performed only if there was a real indication.

Results

During the prospective study we have performed 117 invasive procedures in 72 patients with singleton pregnancies from 29 to 37 weeks of gestation. Actually, the pregnant women were divided into four groups: A (study group: patients with gestosis / preeclampsia; n = 15), B (real control group: patients without preeclampsia / IUGR; n = 15), C (control group: patients with preeclampsia + IUGR; n = 27), and D (control group: patients with IUGR and no preeclampsia; n = 15). There were no significant differences relating median values of gestational age between the groups A (32.5 weeks), B (33 weeks), and C (33 weeks), but the median value of gestational weeks in the group D (35.5 weeks) was significantly greater than in other three groups.

Amniotic fluid LB concentrations in preeclamptic pregnancies have shown a mild linear increase from weeks 29 (3.000/μL) to 37 (16.000 /μL). The measured concentrations of LB in amniotic fluid were very similar in both the study group A and control group B until 34 weeks of gestation. However, significantly higher values of LBs were found in control group B from weeks 35 to 37.

The values of amniotic fluid LBs were significantly increased in the group of preeclamptic pregnancies complicated with IUGR (group C) in relation to both the preeclamptic and the control group B, but after 35 weeks of gestation there were no differences in LB counts between the group C and the control group B. In other words, fetal lung maturation in the preeclamptic pregnancies was of a less degree than in the control group and the group of pregnancies with preeclampsia combined with IUGR.

Chronic stress in IUGR fetuses (group D) could explain an accelerated and pronounced lung maturity after 32 gestational weeks in contrast to the fetuses from pregnancies complicated by preeclampsia alone. Moreover, the only three cases (all of them ≤32 weeks) of severe neonatal RDS were registered in the study group with preeclamptic pregnancies.

Conclusions

We have failed to demonstrate any protective effect of preeclampsia in the development of acute neonatal RDS. According to our preliminary results which were based on amniotic fluid LBC and postnatal clinical findings, we should conclude that fetal lung maturity is commonly delayed in preeclamptic pregnancies. That is in agreement with several recent studies, in which a clear beneficial effect of betamethason administration with idea of improving lung maturity has been shown. However, if preeclampsia was combined with chronic uteroplacental insufficiency and real IUGR, newborn infants showed, with rare exceptions, a reduced risk of hyaline membrane disease as a consequence of an accelerated lung maturation.

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