

Fetal growth impairment in a cohort of pregnancies complicated by spontaneous preterm labor

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Objective

Placental insufficiency is a known pathological process implicated in the preterm parturition syndrome. Spontaneous preterm labor (sPTL), preterm prelabor rupture of membranes (PPROM) and fetal growth restriction share this common placental etiology. The aim of this study was to explore the differences in fetal growth in a cohort of pregnancies complicated by sPTL or PPROM as compared to uncomplicated pregnancies.

Methods

A prospective cohort study including 212 singleton pregnancies from 24 to 34 weeks of gestation, divided into three groups: sPTL (n=83), PPROM (n=63), and 66 uncomplicated pregnancies. Fetal ultrasound was performed at hospital admission for fetal biometry and feto-maternal Doppler study. Small for gestational age (SGA) was defined as estimated fetal weight (EFW) < 10th centile and as birthweight < 10th centile. Intrauterine-growth restriction (IUGR) was defined as EFW <3rd centile or abnormal feto-maternal Doppler study at first ultrasound (middle cerebral artery pulsatility index (PI) <5th centile and/or umbilical artery PI > 95th centile and/or cerebro-placental ratio <5th centile) and as birthweight < 3rd centile.

Results

There were no differences in gestational age at first ultrasound among groups (uncomplicated median (p25;p75) 29.1 (26.7;32.1), sPTL 29.7 (27.6;31.6), PPROM 31.0 (27.6;32.6) weeks; p=0.2). As expected, cases complicated with sPTL or PPROM showed lower gestational age at delivery (uncomplicated 39.9 (38.9;40.6), sPTL 35.9 (32.3;39.9), PPROM 32.1 (30.4;34.0) weeks; p=0.0001), as compared to uncomplicated pregnancies. Both sPTL and PPROM presented a higher proportion of SGA and IUGR cases at recruitment ultrasound (SGA: uncomplicated 9,1%, sPTL 13,4%, PPROM 34,9%; IUGR: uncomplicated 0,0%, sPTL 7,2%, PPROM 14,3%), and at birth (SGA: uncomplicated 10,1%, sPTL 25,0%, PPROM 42,9%; IUGR: uncomplicated 1,5%; sPTL 10,8%, PPROM 25,4%). There was a linear trend across the study groups with worse fetal growth in PPROM as compared to sPTL (p<0,0001), both at first ultrasound and at birth. This linear trend was also detected when comparing head circumference, height, cephalization index and BMI at birth (p<0,0001).

Conclusion

Fetal growth impairment was more pronounced in PPROM cases, but remarkable differences were also observed in the sPTL group. This linear trend across the groups suggests a biological gradient of severity of the placental insufficiency that may be under the origin of preterm parturition, being milder in sPTL cases and more severe in PPROM pregnancies. Further research is guaranteed by the placental pathological exam and the analysis of the expression of placental biochemical markers related to vascular damage, infection and inflammation and placental aging, now in progress.