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Influence of intra-amniotic infection and inflammation on fetal cortical development

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Objective

To evaluate whether fetal cortical brain changes were present in fetuses of mothers admitted for preterm labor (PTL) or preterm prelabor rupture of membranes (PPROM) and if these changes were more pronounced in fetuses exposed to IAI.

Methods

In this prospective cohort study, fetal neurosonography and amniocentesis were performed at admission in singleton pregnant women with PTL and/or PPROM between 24.0-34.0 weeks. This group was further subdivided into two groups that were: 1) PTL/PPROM with intra-amniotic infection and/or inflammation (IAI group), and 2) PTL/PPROM without IAI (non-IAI group). The control group included outpatient pregnant women without PTL or PPROM matched for gestational age at study ultrasound. Fetal cortical development was evaluated by neurosonography and neuronspecific enolase concentrations were measured in amniotic fluidfrom women with PTL/PPROM and compared to 20 amniotic fluid Biobank samples for reasons other than PTL/PPROM or fetal brain pathology, matched for gestational age at amniocentesis. Fissures depth ratios were calculated by dividing each fissure depth (mm) by the biparietal diameter (mm). Data was adjusted for estimated fetal weight below the 10th centile and for PPROM at admission, and also for gestational age at amniocentesis when amniotic fluid biomarkers were compared.

Results

From 2018-2021, 143 fetuses were included: 95 fetuses were from mothers admitted with symptoms of PTL or PPROM: 41 (28.7%) were in the IAI group and 54 (37.8%) in the non-IAI group. 48 (33.6%) fetuses were included in the control group. There were no significant differences in maternal age, BMI, race, smoking habit, nulliparity, assisted reproductive techniques, gestational age at ultrasound or fetal sex. There was higher prevalence of fetuses below 10th percentile (26.8% in the IAI group, 13.0% in the non-IAI group and 8.3% in the control group) without fetal-placental Doppler significant differences. Fetuses in the IAI group had lower Sylvian fissure depth ratio compared to controls: (median (25;75 percentile) 0.14 (0.12;0.16) in the IAI group, 0.14 (0.13;0.16) in the non-IAI group and 0.16 (0.15;0.17) in the control group, p<0.001) and lower right parieto-occipital sulci depth ratio (0.09 (0.07;0.12) in the IAI group, 0.11 (0.09;0.14) in the non-IAI group and 0.11 (0.09;0.14) in the control group, p=0.012) after adjustment for confounding factors. Amniotic fluid concentrations of neuronspecific enolase were higher after adjustment for possible confounding factors: 11804.6pg/ml (6213.4;21098.8) in the IAI group, 8397.7pg/ml (3682.1-17398.3) in the non-IAI group and 2393.7pg/ml (1717.1;3209.3), p=0.001. The non-IAI group seems to be an intermediate group regarding Sylvian fissure depth and amniotic fluid biomarkers.

Conclusion

Fetuses with PTL / PPROM present smaller Sylvian fissure and parieto-occipital sulci, and higher amniotic fluid concentration of neuronspecific enolase, a grey matter damage biomarker. These findings support that the neurological damage observed in children born preterm have, at least in part, a prenatal origin.