

Summary

Women in childbearing age are often affected by autoimmune diseases (AD) associated with the presence of antiphospholipid antibodies (aPL) that may influence further development of their children. The primary objective of our prospective study was to determine the presence of the following aPL: anti β 2 glycoprotein I (anti- β 2GPI), anticardiolipin (aCL), antiphosphatidylserine, antiphosphatidylinositol, antiphosphatidylethanolamine, antiphosphatidylglycerol, antiphosphatidic acid, antiannexin V in mothers with defined AD and their children after birth, at 6 and 12 months of life, and to compare the incidence of aPL with a control group. A secondary objective of the study was a 2-year follow-up of children born to aPL negative and aPL positive mothers with AD in order to detect the possible impact of maternal AD on the health of the offspring. In children, we analysed anthropometric data, blood cell count, cerebral and abdominal ultrasound examination, transient evoked otoacoustic emission test (TEOAE), electrocardiograph (ECG), the presence and kinetics of aPL. At the age of 2 years the Bayley Scales of Infant Development (BSID-II) were used for children's assessment of motor, language and cognitive development. 31 mothers from the total examined 82 aPL positive women with AD delivered 34 neonates that were also aPL positive and were included in the study. The second group consisted of 24 aPL negative newborns born to 23 aPL negative mothers with AD. The control group comprised of 30 mothers without AD and 30 children born to women without AD. Home-made and commercial ELISA kits were used for aPL detection. The most frequently detected aPL in mothers with AD were antiphosphatidylserine, aCL and anti β 2GPI IgG. The quantitative determination of aPL IgG differed significantly ($p < 0.01$) between mothers with AD and mothers without AD except antiphosphatidylglycerol. The level of aCL IgM and anti- β 2GPI IgA was significantly higher in mothers with AD ($p < 0.05$, $p < 0.01$, respectively). The aPL incidence in mothers without AD was 3.3 %. The transplacental transfer of aPL IgG was detected at birth in 41.4 % of newborns. The antiphosphatidylserine IgG was the most frequently found aPL in newborns. In neonates born to healthy mothers, aPL presence was detected in 3.3 % of cases. At 6 months of age the aPL persisted in 38.2 % of children born to aPL positive mothers, the identical aPL were present in 5.8 % of those children even at 12 months of age. The de novo aPL production at 6 and 12 months of age was highest in the group of children of aPL positive mothers. Neither symptoms of neonatal lupus nor thrombotic event were found in any of children during follow-up. Evaluation of ECG and TEOAE did not reveal any pathology. Higher incidence of transient thrombopenia and mild CNS abnormalities were observed in children of aPL positive mothers. Mental development index (MDI) of BSID-II was significantly lower in these children ($p < 0.01$). The prevalence of the moderate damage differed significantly between the two groups ($p=0.03$). MDI in children of aPL positive mothers negatively correlated with anti β 2GPI IgG at birth ($p < 0.01$). We believe that maternal AD with presence of aPL may negatively influence the mental development of their offspring. Therefore a long term follow-up of the children with BSID testing is required in order to detect early abnormalities of their neuropsychological development and to enable timely correction.