

5th World Congress on

Diabetes & Metabolism

November 03-05, 2014 Embassy Suites Las Vegas, USA

Does childhood type diabetes mellities start “in utero”

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Background and Aim: Findings of different seasonality of month of birth in children with T1D compared with the general population suggested possible initiation of childhood T1D in utero, triggered by viral infections. Thus we determined anti-virus and/or anti-islet cell antibodies in healthy pregnant mothers without diabetes background and their offspring at birth in two winter viral seasons.

Methods: Maternal and cord blood sera from 107 healthy pregnant women were tested for islet cell GAD65 autoantibodies by radioligand binding assays and anti-rotavirus and anti-CoxB3 virus antibodies by ELISA.

Results: GAD65 autoantibodies and rotavirus antibodies present in both maternal and cord blood sera correlated with an OR of 6.89 (95% CI: 1.01 to 46.78). For 5, 22, and 17 pregnancies antibodies to GAD65, rotavirus and CoxB3, respectively, were detected in cord blood only and not in the corresponding maternal serum. In 10 pregnancies rotavirus antibody titers in the cord blood exceeded those in the corresponding maternal serum by 2.5 to 5-fold. Increased antibody titers in the 20th week of gestation suggested CoxB3 infection in one and rotavirus infection in another of the 20 pregnancies from which serum was available.

Conclusion: The concurrent presence of GAD65 antibodies in cord blood and their mothers may indicate autoimmune damage to islet cells during gestation caused by cross-placental transmission of viral infections and/or anti-virus antibodies. Cord blood antibody titers exceeding those of the corresponding maternal sample by >2.5-fold or antibody positive cord blood samples with antibody-negative maternal samples may infer active in utero immune response by the fetus.

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