Phenotype and function of monocyte-derived dendritic cells in neonates born to Hepatitis B virus-positive mothers

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Abstract

Hepatitis B Virus (HBV) infection in infancy or early childhood leads to high rate of persistent infection (25-90%). The immunological basis of high rate of viral persistence in vertically acquired HBV infections is not completely understood. Dendritic cells (DCs) are one of the most potent antigen-presenting cells (APC) and play pivotal roles in the enhancement or regulation of antiviral immune reactions. Aim of the present study was to investigate whether an HBV-infected maternal environment might influence the infants' DC phenotype and function. Monocyte-derived DC (MoDC) of neonates born to HBsAg-positive mothers were studied phenotypically by Flow Cytometry (FCM) and functionally by mixed lymphocyte reaction (MLR) and enzyme-linked immunosorbent assay (ELISA). An electron microscope was used to analyze the morphological changes of MoDC. MoDC from neonates whose maternal HBV DNA was>5×107 copies/ml showed a reduced surface expression of CD80, CD86, and HLA-DR as compared to that in neonates whose maternal HBV DNA was negative (CD80: t=3.238, P=0.002; CD86: t=3.543, P=0.001; HLA-DR: t=2.785, P=0.008). T-cell proliferation assays also showed an impaired allostimulatory capacity in comparison to that in neonates whose maternal HBV DNA was negative, especially in the cultures at a DC: T cell ratios of 1:5 and 1:10 (t=-5.442, P<0.001; t=-2.195, P=0.042). Therefore, it can be speculated that the presence of high level of HBVDNA in the maternal environment might lead to minor phenotypic and functional alterations of MoDC from neonates and subsequent deficits in T-lymphocyte activation may contribute to viral persistence.

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