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Postnatal cytomegalovirus (CMV) transmission by breast milk to preterm infants: new findings and prevention strategy

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Objectves and hypothesis

The epidemiology of CMV is mainly influenced by breast feeding. During lactation CMV is reactivated in the breast of seropositive mothers. The reactivation rate equals the maternal CMV seroprevalence. Both, mothers of term and preterm infants reactivate CMV during lactation and transmit it via breast milk to their infants. In term infants, CMV transmission is associated in nearly all cases with an asymptomatic CMV infection. Preterm infants however, may acquire symptomatic CMV infections. In certain risk preterm infants, serious life-threathening disseminated CMV infections were observed.

Methods

We analysed CMV reactivation in breast milk of CMV IgG-positive mothers of preterm infants longitudinally for up to 2 months post partum under clinical observation by virus culture and PCR. Virolactia and DNAlactia were each quantitated. The urine of the newborns was screened also by PCR and virus culture. Virus inactivation of seropositive breast milk was performed using our new developed short-term heat-inactivation procedure. Maternal transmitters and non-transmitters were evaluated according to their virus reactivation dynamics.

Results: We found a very high rate of CMV reactivation in breastfeeding mothers of preterm infants. Like in HIV-mother-to infant-transmission, CMV-transmission occurs mainly cell free. The dynamics of CMV reactivation of maternal transmitters and non-transmitters can be described as an unimodal process. Transmission depends on onset of DNAlactia, peak levels of DNAlactia and on newborn parameters like low birthweight (<1000g) and gestational age below 30 weeks. Long-term outcome of postnatal CMV-

transmission of the preterm infants is principally different from that of congenital infected newborns.

Conclusions: CMV reactivation in breast milk during lactation is a unique example for the study of in vivo herpes virus reactivation. The role of factors involved in triggering virus excretion as well as the cellular and humoral immune mechanisms controlling the self-limited virus excretion in breast milk are mostly unknown.