POSSIBLE TERATOGENIC POTENTIAL OF CERTAIN VIRUS INFECTIONS AS RELATED TO PATIENTS WITH OROFACIAL CLEFT

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Viral infections seem more frequently responsible for lesions of the neonates as previously expected (1, 2, 3, 4, 5). We followed the antibody levels to rubeola, influenza, parotitis, hepatitis B, EB virus and cytomegalovirus (CMV) in mothers and their children with orofacial cleft aged 1 day till 10 months comparing them with a group of control sera from mothers with healthy neonates. The antibodies were measured by various tests such as ELISA, RIA, CF, HIT, and indirect immunofluorescence (IF).

Specific IgG antibodies to the capsid antigen of EB virus were examined in 208 paired sera from neonate children with cleft lip and palate and their mothers. We found 43.7% seropositive children and 72.2% seropositive mothers, however, when considering serum titres of 40 or higher only 7.6% children and 21.2% mothers revealed such high values. Out of 119 control mother and neonate serum paires the positive rates took 33.4% children and 42.8% mothers, respectively; titres of 40 or higher were found in 3.8% children and 10.2% of mothers, i. e. the positive rates were higher in children with orofacial cleft (p=0.05) as well as in their mothers (p=0.01). IgM type antibodies were tested in 52 mother/neonate serum paires (malformation group) and in 48 paired controls. The results were positive in 27.1% neonates and 17.2% mothers (titres 10 or higher) from the malformation group as compared to 10.4% children and 4.3% mothers in the control group (the differences were significant in children at p=0.01, in mothers at p=0.02).

CF antibody titres of 8 or higher to CMV (strain AD169) were found in 64.5% children with the malformation and in 84.2% of their mothers, while as the same titres were detected in 32.2% of healthy children and in 49.1% of their mothers (these results differed at p=0.001 in children as well as in their mothers). HI antibodies to rubeola virus were present in 13.3% children and 65.9% of their mothers as compared to 2.8% control children and to 48.6% of control mothers (titres of 80 or higher); the results were significant at p=0.001 in children and also in their mothers. HI antibodies to influenza A2/Bangkok/H2N2 in titres of 40 or higher were found in 72.5% of children with orofacial cleft and in 87.5% of their mothers as compared to 27.1% control children and 44.5% of their mothers (significance rate p=0.001). Finally, HI antibodies to parotitis virus in titres 40 or higher were found in 4.1% children with malformations and in 10.2% of their mothers (as compared to 3.6% in control children and to 9.4% in their mothers); results differed at p<0.05, i.e. the difference was not significant. No antibodies to HBs antigen were found in the sera tested.

Based on careful comparison of these results, i.e. of the antibody titres to abovementioned viruses including the analysis of individual paired sera repeatedly investigated for IgM antibodies we assume some association to foetal infection with influenza virus, human CMV, rubeola virus and possibly also with EB virus. No association was found to hepatitis B and parotitis viruses.

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