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PROGNOSTIC IMPLICATIONS OF HBeAg IN INFANTS BORN FROM ASYMPTOMATIC CARRIER MOTHERS

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Transmission of HBV vertically from mother to baby is one of the major routes of HBV transmission through the world.

In high prevalence areas of Asia and Africa up to 40—50 % of HBsAg carrier mothers transmit the virus to their newborn babies (1, 5, 6, 14), in contrast to some European countries and USA where vertical transmission (VT) occurs in relatively small rates (9, 10, 12).

Women with acute hepatitis in the last trimester of the pregnancy fre-

quently transmit HBV (7); this transmission has also been demonstrated from asymptomatic carrier mothers, especially from those with serological evidence of HBeAg (1, 6).

Babies infected with HBV in the neonatal period rarely have clinical hepatitis. However, 60—80 % of infected babies become chronic HBsAg carriers. More importantly, studies in Asia and Africa now indicate that VT plays a role in the development of hepatocellular carcinoma and cirrhosis (15).

We have extended our previous examinations (8) to 11 babies born from asymptomatic carrier mothers, babies who developed HBs antigenemia after delivery, and have tried to correlate the presence of HBeAg in maternal sera with the transmission of this marker to infants and with progression to chronicity.

Materials and methods

Eleven babies born from 153 asymptomatic carrier mothers, who became HBsAg positive 3—6 months after delivery, were followed up with repeated clinical and biochemical investigations, including the determination of HBsAg and „e system" for a two years' period. HBsAg was tested by Hepanosticon technique and HBeAg/anti-HBe by Rheophoresis (2).

Starting from the premise that any detectable component of virus should be passed on with the infection, we have studied the transmission of HBsAg and HBeAg from mothers to their infants.

The presence and persistence in infants of both markers of HBV have been correlated with the progression to chronic liver disease or chronic HBsAg carrier state.

Results and discussion

One of the most important factor influencing the risk of infection in the baby is HBeAg positivity of the mother's serum (1, 6, 11, 13, 16).

Differences in the prevalence of HBeAg among HBsAg carrier women in various parts of the world probably account for the differences in the frequency of VT.

In our study among 153 pregnant women found to be asymptomatic carriers of HBsAg, 8,4 % were positive for HBeAg and 11,7 % positive for anti-HBe (Table I).

Table I

Prevalence of „e system" in investigated groups

Group investigated	nr.	HBeAg positive		anti-HBe positive		cronic liver disease	
		nr.	%	nr.	%	nr.	%
Mothers HBsAg positive	153	13	8,4	18	11,7	0	0
Children HBsAg positive	11	3	27,2	0	0	2	18,1
Vertical transmission	7,1%						

It should be noted that not all babies born to HBeAg positive women became HBsAg positive, and not all HBsAg positive babies had HBeAg positive mothers. Eighty two per cent of the babies whose mothers were HBeAg positive became HBsAg positive, while only 1,4 % of the babies became carriers when mothers were HBeAg negative ($p<0,01$).

Although it is a strong correlation between maternal „e" antigenemia and VT, in this study HBeAg was not a perfect predicator of the development of HBs antigenemia in the babies.

Five out of 11 children infected with HBV developed persistent HBs antigenemia. 3 with slight average of transaminases without other modifications and 2 with signs of chronic hepatitis. The remaining six, were transiently infected and eliminated the virus before the age of one year.

HBeAg was not consistently transmitted from mothers to their newborns. Thus, only one out of 3 children with HBe antigenemia was born from mother who carried this marker, too.

Yanagida et al. (19) have reported the failure of materno-fetal transmission of HBeAg. That can be the cause of our small percentage of HBeAg (33,3 %) transmitted from mothers to the babies.

Studying the correlation between transmission of HBeAg and HBsAg in primary and secundary cases of acute hepatitis B occuring in 15 families, Villarejos et al. (17) showed that HBeAg was not consistently transmitted along with HBV in the familial environment, these data being in agreement with our results, too.

They concluded that HBeAg is probably not an integral component of the virus, and represents rather a specific host response by the individual. Such a response, when prolonged, is associated with persistence of HBsAg, especially in young children (17).

This is in accord with the Ig G nature of HBeAg proposed by *Neurath* and *Strick* (4) and supported by *Visoná* et al. (18).

The disagreement between different studies concerning the prognostic value of HBeAg, besides the use of relative insensitive methods to determine „e system", reflects variations in time and frequency in sampling.

Iwarson et al. (3) showed that patients with HBV infection may or may not circulate HBeAg depending upon what point in the natural course of infection has been reached.

From our data, we can conclude that the presence of HBeAg is a perfect predicator neither for the development of HBs antigenemia in babies, nor in inducing chronic liver disease.

On the other hand, our results show that the progression to chronic liver disease was linked to the persitence of HBeAg in the childrens' serum.

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