Breakthrough Hepatitis B Virus (HBV) Infection From Mother-to-Infant Transmission Is the Key Problem Hindering HBV Eradication

TO THE EDITOR—We greatly appreciate the editorial commentary by Schwarz [1], which provided a comprehensive overview of the impact of our study on the universal hepatitis B virus (HBV) vaccination program in Taiwan [2]. It was pointed out that the 8 infants who were born to mothers positive for HBV e antigen (HBeAg) and some of the infants born to HBeAg-negative mothers developed acute HBV infection in the first year of life. We would like to clarify the details about these infants who developed acute hepatitis B in infancy. As shown in Supplementary Table 2 in our article [2], 13 infants with acute hepatitis B, also known as perinatal HBV infection, included 5 who were born to HBeAg-positive mothers and received HBV immunoglobulin (HBIG); 3 who were born to HBV-carrying mothers with an unknown HBeAg status, of whom 1 received HBIG; and 5 who were born to HBV-carrying, HBeAg-negative mothers and did not receive HBIG. According to Taiwan’s HBV immunization policy, HBIG was given to infants of HBeAg-positive mothers within 24 hours after birth, in addition to 3 doses of HBV vaccine at 0, 1, and 6 months. Among mothers who were HBeAg negative but HBV surface antigen (HBsAg) positive or who did not adhere to instructions to undergo screening for serum HBsAg and HBeAg, HBIG was not given to their infants, and only 3 doses of HBV vaccine were given.

The need to change current Taiwan HBIG policy to prevent breakthrough perinatal HBV infection remains under debate. Chen et al found that, among children who were born to HBeAg-negative mothers and received HBIG versus those who were born to HBeAg-negative mothers and did not receive HBIG, the rate of chronic HBV infection did not differ (0.14% vs 0.29%; \( P = .65 \)) [3]. Similar rates of antibodies against HBV core protein (0.99% vs 1.88%; \( P = .19 \)) were noted among children who were born to HBeAg-negative mothers and did or did not receive HBIG [3]. This suggests that adding HBIG cannot significantly reduce the total infection (ie, acute and chronic infection) rate. On the other hand, a high rate (9.26%) among children born to HBeAg-negative mothers who received full coverage by HBIG and HBV vaccine were HBsAg positive. The data indicated that HBIG plus HBV vaccination could not completely prevent mother-to-infant HBV transmission. It is also known that infants born to HBeAg-negative, HBsAg-positive mothers were prone to develop fulminant hepatitis, although overall infection rates were low [3, 4]. The studies mentioned above may also pertain to the infants with perinatal HBV infection in our study [2], who might become chronically infected with HBV even though HBIG was given to them regardless of their mothers’ HBeAg status, although the chance of chronic infection is very small. The cost-effectiveness of giving HBIG to HBeAg-negative, HBsAg-positive mothers requires further confirmation. More clinical information about and follow-up of vaccinated subjects born to high-risk mothers are needed to delineate this important issue and to develop strategies to eradicate HBV infection.

Note

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References


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