Hepatitis B in jaundiced neonates admitted to a special care nursery

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Summary

Sixteen of the 448 neonates admitted with unconjugated hyperbilirubinemia had hepatitis. Four of these babies were HbsAg positive at the time of admission. About 418 of the mothers of jaundiced neonates admitted to the nursery were screened for HbsAg; 47/418 of them were HbsAg positive. The percentages of affected mothers were the same among the Malays, Chinese and Indians. This study suggests that the staff in our nursery and the neonates admitted were at risk of contracting hepatitis B from the high percentage of HbsAg positive mothers of jaundiced neonates.

Introduction

Various studies have shown that hepatitis B can be transmitted from the mothers to their neonates in utero, during and after delivery.^{1,2,3,4} Studies have also shown that by giving hepatitis B immunoglobulin and hepatitis B vaccine to the neonates shortly after birth, the condition can be prevented in most of the children born to hepatitis antigen B positive mothers.^{5,6,7,8} In Malaysia, there are no data available yet on the exact incidence of hepatitis B in the general population.⁹

In the General Hospital, Kuala Lumpur, over a period of 12 months, between 1st of November 1984 to 31st of October 1985, 618 neonates were admitted to the University Unit in the Special Care Nursery and of these 448 (72%) were admitted because of jaundice. At the time of admission, specimens of blood were usually taken from the mothers of each of these jaundiced babies for blood grouping and crossmatching as the babies might require exchange transfusion for severe jaundice. There was, thus, a lot of potential exposure of the medical and nursing staff to the blood specimens of these mothers.

The objectives of our study were to find out during this 12 month period: a) how many of the jaundiced neonates admitted to ward 4, General Hospital, Kuala Lumpur, under the care of the University Unit, were due to hepatitis, b) what were the common causes of the neonatal hepatitis in these babies, and c) how many of the mothers of these jaundiced babies were hepatitis B surface antigen (HbsAg) positive at the time when these babies were admitted.

Materials and method

All neonates admitted with jaundice to the University Unit were included in this study. After taking the history and a physical examination, specimens of blood were collected from each baby by the doctors for the following investigations: full blood picture, total serum billirubin and fractionation, glucose-6-phosphate dehydrogenase screening, blood grouping and crossmatching, serum alanine aminotransferase (SGPT) and serum aspartate aminotransferase (SGOT). Neonates were diagnosed to have hepatitis when they had raised levels of SGPT (>55 U/L) and SGOT (>70 U/L) for at least two consecutive days. From babies with raised blood levels of liver enzymes, blood was also screened for HbsAg and TORCHES antibodies. Toxoplasma antibodies were measured by immunoflluorescence test (Wellcome), Rubella antibodies by haemaglutination inhibition test using W.H.O. method, cytomegalovirus and herpes simplex antibodies by complement fixation test, and VDRL test using rapid plasma reagin card from Brewer Diagnostic kits. Full septice work-up was carried out on any neonates with clinical features suggestive of septicemia.

From the mothers of all the jaundied babies, specimens of blood were collected either by the doctors or the nurses for blood grouping and crossmatching and for screening of hepatitis B surface antigen (HbsAg). When any of the mothers' blood were found to be positive for HbsAg, specimens of blood were also collected from their babies for testing of HbsAg irrespective of their serum enzyme levels.

Testing for hepatitis B surface antigen

Hepatitis B surface antigen was tested by enzyme immunoassays (EIA) with kits from Abbott Laboratories, North Chicago (Auszyme).

Results

The 448 neonates were admitted for treatment for jaundice. About 418 of the mothers of these jaundiced babies were screened for HbsAg at the time when their newborns were admitted; 30 of the mothers were not tested for HbsAg because they could not come to the hospital.

Forty-seven of the mothers tested were HbsAg positive. The racial distribution of the babies and their mothers and the results of hepatitis screening are shown in Table 1. These figures did not take into account the 30 mothers who were not screened.

Sixteen of the jaundiced babies had raised liver enzymes and 4 of these babies were HbsAg positive. All of them had predominantly unconjugated hyperbilirubinemia with conjugated bilirubin not exceeding 25 umol/L. One of these HbsAg positive babies with raised liver enzymes had indirect serum bilirubin level of 340 umol/L, and total serum bilirubin of 352 umol/L. No other cause of severe jaundice was found in this patient. The other three babies had only mild to moderately severe hyperbilirubinemia. Of the 4 HbsAg positive babies, only two mothers were proven to be HbsAg positive as the other two mothers did not come for HbsAg screening. The causes of hepatitis in the remainder of 12 patients could not be identified with our tests. None of the other 45 babies whose mothers were HbsAg positive had evidence of being infected by hepatitis B virus yet at the time of the study.

Discussion

The present study showed that hepatitis can cause neonatal unconjugated hyperbilirubinemia and unless test the serum liver enzyme levels in the jaundiced neonates are measured the diagnosis may be missed.

Table 1

Race	a Total no. of neonates admitted	b No. of babies admitted with jaundice (b/a x 100)	c No. of jaundiced neonates with HbsAg positive mothers (c/b x 100)	d No. of HbsAg positive neonates
Chinese	165	148 (90)	16 (10.8)	1
Indian	58	30 (52)	3 (10.0)	1
Others	24	14 (58)	0 (0.0)	0
Total	618	448 (72)	47	4

Racial distribution of the neonates admitted to the University unit, ward 4, General Hospital, Kuala Lumpur, between 1st November 1984 and 31st of October 1985.

(Figures in paranthesis indicate %)

Although the study was small, it revealed that hepatitis B was not common among neonates admitted to our nursery. The screening tests also showed that there was a high carrier rate of hepatitis B among mothers of the three main racial groups of Malaysia. The immeidate significance of this study was that it indicated that the nursing and medical staff working in the Ward 4 Nursery, General Hospital, Kuala Lumpur were among the high risk hospital personnel who required protection via vaccination against hepatitis B infection. This situation could be the same in the other neonatal nurseries in Malaysia, although further studies are required to confirm this suspicion.

The presence of four hepatitis B positive babies in our small study suggests that there is an urgent need to protect the neonates born in Kuala Lumpur and possibly in the whole of Malaysia by routinely screening their mothers before delivery. Once pregnant mothers have been identified to be HbsAg positive, they should preferably be tested for the presence of HbeAg in their blood. This is because 80 to 90 percent of the HbeAg positive mothers will transmit the virus to their neonates and young infants, and 85 percent of these infants will become chronic carriers. 50 percent of the male chronic carriers will develop cirrhosis of the liver and hepatocellular carcinoma.^{10,11} Although mothers who are positive for both HbeAg and the antibody to hepatitis B e antigen are relatively noninfectious, fatal cases of neonatal hepatitis B virus infection have been confirmed to be HbsAg positive, hepatitis B immunoglobulin and vaccine should be given to their neonates within 12 hours after birth. This should be followed by two further doses of vaccine at monthly intervals subsequently. Only then can this group of high risk neonates be prevented from the possibility of becoming chronic carriers of hepatitis B virus and its sequelae.

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