CMV IN PREGNANCY CASE REPORT AND REVIEW OF LITERATURE

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Man is the only reservoir of cytomegalovirus.[1] 13% of the general population are carriers of this virus with a high incidence of intra uterine infection. A wide variety of abnormalities has been observed, including hearing loss, mental retardation, cerebral palsy, seizures, blindness, defects in tooth structure and poor school performance[2]. Congenital CMV infection is usually due to transplacental spread of the virus[3], however, the mother is usually asymptomatic and may have either a primary or reactivated latent infection[4].

CASE REPORT

This 32 years old lady was in her second pregnancy. She had blood group 0 rhesus positive. Her TPHA was negative and she was rubella immime at the time of her booking in the ante-natal clinic. Her first pregnancy was normal and she was delivered of a normal male babywho weighed 4.080 kg. She was first seen in the ante-natal clinic at 10 weeks gestation. She attended the ante-natal clinic regularly until 39 weeks. Her diastolic blood pressure was slightly raised in the last trimester. Other parameters of ante-natal care were normal in each visit. She hada normal delivery ofa live male infant after a labour of 4 hours and 40 minutes. The baby responded to suction and 02. The baby was small for dates and transferred to the speeial care baby unit after paediatric consultation. The clinical features of bilateral cataracts, microcephaly and facial dysmorphism were noted. The baby weighed 2.010 kgs and died 2 days after developing marked jaundice despite exchange transfusions. Serological testing revealed a CMV titre of 128 i.u. and Igm positive, but the mother showed no significant amount of antibodies.

POST MORTEM FINDING

The baby had facial dysmorphism with hypertelorism, microcephaly associated with premature fusion of sutures, partial collapse of liver parenchyma associated with bile staining and a normal bihary tract, a wide spread petechial rash and massive splenomegaly. Histological examination of liver, spleen, kidney and lungs revealed the presence of CMV inclusion bodies. The overall features were characteristic of generalized cytomegalic inclusion disease.

DISCUSSION

CMV infection is endemic throughout the world with marked geographical variation in the incidence of infection. InAfrica andAsia over 90% of the population become infected and most of them acquire antibodies in childhood. In contrast in western countries more than 60% acquire infection later in life[5], particularly in higher socioeconomic groups in whom the major exposure occurs in adolescence and early adult life. In the recent study of Griffiths and Baboonian (1984), it was shown that one woman in every 133 experienced primary CMV infection duringpregnancy, which is twice as frequent as rubella infection during pregnancy[6]. It is now evident that the two viruses present quite different problems and only by prospective studies can the risk be assessed[7]. It is shown that 1st trimester infection will

definitely cause congenital abnormalities[8], and the infection early in pregnancy is associated with a sevenfold excess of fetal loss[6]. In one study it is noticed that fetal immune responses, both humoral and cell mediated are more vigorous in late gestation than early in pregnancy while maternal immune response is diminished in all stages of pregnancy and the most striking decrease occurred in the third trimester[9]. In the same study it was found that pregnant women with CMV infection are at in-creased risk of giving birth to congenitally infected infants particularly if active infection occurs late in pregnancy. These findings of infant damage due to third trimester infection are confirmed by Griffiths and Baboonian in humans and by Kumar in guinea pigs[9,6,10]. Neonatal CMV infection is multisystem disease and new born babies may either be normal or show manifestations of disease as in the above case, or be still born[4]. 1-2% of all live births have C.M.V. and 10-20% of these infected neonates have some degree of handicap attributable to CMV[2,6,11]. Perinatal and neonatal management is still not satisfactory. Two attenuated strains of live virus (AD-169 and TOWNE 1) are available but vaccination is still controversial. Therapy with interferon is under trial at the moment[12]. Recently a case is reported in which a3 month old boywith life threatening CMV infection was successfully treated with high titre anti CMV plasma[13].

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