Cytomegalovirus Infection Acquired Through Blood Transfusions

Swarna Rekha
M.K. Chandrasekhara
Malathi Yeshwanth

Cytomegalovirus (CMV) infection is found worldwide and is spread by close contact. The prevalence of congenital CMV infection varies from 0.2-2.4%(l). Neonates may be infected transplacentally or during birth through a CMV positive mother (congenital CMV infection) or may acquire infection in the neonatal period through blood transfusions and breastmilk of infected mothers(l-3). We report three infants with symptomatic CMV infection, presumably acquired through blood transfusions in the neonatal period.

Case Reports

Case 1: A premature baby (birth weight 1890 g and gestation 33 weeks) was admitted to the neonatal ward with respiratory distress syndrome. The neonate received assisted ventilation for six days and blood/plasma transfusions for a low hematocrit and shock. The patient was discharged at 15 days of age and followed in the neonatal follow up clinic. At 4 months of age the baby had jaundice, hepatosplenomegaly and thrombocytopenia. Investigations showed presence of TgM antibodies against CMV in the infant's blood. Maternal CMV IgM antibodies were absent. At the age of 2.5 years the growth and development was normal. There was no icterus or chorioretinitis but splenomegaly and hepatomegaly was present.

Case 2: This neonate was born to a primigravida mother at 34 weeks gestation and weighed 1720 g at birth. The baby had multiple problems in the neonatal period including unconjugated hyperbilirubinemia, apnea, sepsis, thrombocytopenia and anemia. The baby received multiple blood and platelet transfusions. The patient was discharged at 9 weeks of age and readmitted a week later with jaundice, hepatosplenomegaly, thrombocytopenia and central nervous system bleeds. The infant showed IgM antibodies to CMV and simultaneous maternal CMV IgM was negative. The baby was treated symptomatically and discharged after 10 days. On follow up at 1 year 3 months of age there was no icterus or choreoretinitis. Hepatosplenomegaly was present and the developmental age of the child was 1 year (not corrected for gestational age).

Case 3: This baby was born at 30 weeks gestation with a birth weight of 1160g. The baby had unconjugated hyperbilirubinemia requiring exchange transfusion on day 2. At 7 days of age the baby presented with features of sepsis, jaundice, hepatosplenomegaly

From the Department of Pediatrics, St. John's Medical College Hospital, Bangalore.

Reprint requests: Dr. Swarna Rekha, Department of Pediatrics, St. John's Medical College Hospital, Bangalore.

Received for publication: March 4, 1994; Accepted: November 1, 1994

and thrombocytopenia. IgM antibodies against CMV were positive in the neonate and negative in the mother. Maternal CMV IgG was also negative. On follow up at 6 months of age there was no jaundice, choreoretinitis or hepatosplenomegaly. The developmental age was 3 months (uncorrected).

All our cases had received blood transfusions and CMV TgM was negative in the mothers. We feel that these babies may have acquired it through blood. All the babies were preterm and were symptomatic.

Discussion

CMV infection is found worldwide and CMV is known to be endemic in India also. Pal *et al.(4)* from Chandigarh reported a prevalence of CMV antibody positivity in 90-100% of the population. CMV infection has been reported in India(5). One fifth of children with intrauterine infections have GMV antibody positivity. However, transfusion acquired CMV has not been reported.

CMV infection may be acquired perinatally through a CMV positive mother(6) or through breastmilk(7). One study has shown that 13.5% of infants transfused with CMV positive blood developed CMV infection of whom 50% were symptomatic(8). Majority of the babies who develop CMV infection are asymptomatic but may have sequelae, the most common being deafness(9,10). In contrast, preterm babies are usually symptomatic, symptoms include jaundice, hepatosplenomegaly and thrombocytopenia.

Transfusion acquired CMV can be prevented by donor screening; donor titres of less than 1:4 do not result in transfer of infection(3). Use of washed packed cells and deglycerolized blood has also been shown to reduce CMV infection(ll).

REFERENCES

1. Baley JE, Goldforb J. Viral infections. *In:* Neonatal—Perinatal Medicine: Dis ease of the Fetus and Infant, 5th edn. Eds. Fanaroff AA, Martin RJ, Chicago Mosby Year Book, 1992, pp 666-667.
2. Cuerine NG. Viral infections in the newborn. *In:* Manual of Neonatal Care, 3rd edn. Eds. Cloherty TP, Stark A. Boston Little Brown, 1992.
3. Kim HC. Blood component therapy in the neonate. *In:* Developmental and Neonatal Hematology. Eds. Stockman JA, Pochedly C. New York Raven Press, 1988, pp 169-193.
4. Pal SR, Chitkara NL, Krech V. Seroepidemiology of cytorn egalovirus infection in and around Chandigarh. Indian J Med Res 1972, 60: 973-978.
5. Broor S, Kapil A, Kishore J, Seth P. Prevalance of rubella virus and cytomegalovirus infection in suspected cases of congenital infection. Indian J Pediatr 1991, 58: 75-76.
6. Yeager AS, Palumbo PE, Malchowski N, Aragno RL, Stevenson DK. Sequelae *of* maternally derived cytomegalovirus infection in premature infants. J Pediatr 1983,102: 918-922.
7. Dworsky M, Yow M, Stagno S, Pass RF, Alford C. Cytomegalovirus infection of breastmilk and transmission in infancy. Pediatrics 1983, 72: 295-299.
8. Yeager AS, Grumet C, Hafleigh EB, Arvin AM, Bradley JS, Prober *CG.* Prevention of transfusion acquired cytomegalovirus infection in newborn infants. J Pediatr 1981, 98: 281-287.

9. Saigal S, Zunyk O, Larke B, Chernesky M. The outcome of children with congenital cytomegalovirus infection—A long term follow up study. Am J Dis Child 1982,136: 896-901.

10. Deka RC. Management of hearing impaired children. Indian Pediatr 1993, 30: 977-979.

11. Brady MT, Milian JD, Anderson DC, *et al.* Use of deglycerolized RBC to prevent post-transfusion infection with cytomegalovirus in neonates. J Inf Dis 1984,150: 334-339.

Sarcoidosis

S.K. Kabra
A. Bagga
Madhulika
A. Chatterjee
V. Seth

Sarcoidosis is a chronic multisystem disease of unkmbwn etiology, usually affecting adults. Only **a** few reports describing the clinical features and course of sarcoidosis in children have been published. Only one case in a child has previously been reported from this country(1). The rarity of the condition prompts us to report the clinical features in two such patients.

From the Department of Pediatrics, All India Institute of Medical Sciences, New Delhi U0 029.

Reprint requests: Dr. S.K. Kabra, Department of Pediatrics, All India Institute of Medical Sciences, Ansari Nagar, New Delhi 110 029.

Received for publication: May 6, 1994; Accepted: October 4, 1994

Case Reports

Case I: Two and a half year old girl presented with fever, weight loss and cough of 5 months duration. The weight and height were below the fifth percentile for the age. Examination showed marked pallor. The liver and spleen were palpable 5 cm and 3 cm, respec--lively below the costal margin. Rest of the systemic examination was normal. Investigations showed a hemoglobin level of 7 g/dl, total leucocyte count of 7800/cu mm with 60% polymorphonuclear leucocytes, 30% lymphocytes and 10% eosinophils. The blood levels of transaminases, alkaline phosphatase, serum proteins, creatinine, calcium and phosphate were normal. The liver biopsy showed non-caseating granulomatous lesions. An X-ray film of the chest showed bilateral enlarged hilar lymph nodes. The Mantoux test (using 1 TU injected intradermally) and VDRL test were negative.

A diagnosis of disseminated tuberculosis was made and the patient treated with isoniazid (5 mg/kg), rifampicin (10 mg/kg) and pyrazinamide (30 mg/kg) daily for 2 months. Subsequently pyrazinamide was stopped and therapy continued with rifampicin and isoniazid.