# Probable Breastmilk Transmission of HIV to an Infant

P. Hari V. Kalra I.C.Verma R. Ahuja

HIV disease in adults is increasing in our country, but in infancy it is still rare. Children are infected with HIV either perinatally or by transfusion of contaminated blood products or rarely by sexual abuse and exposure to infected breast milk(1,2). Owing to inadequate screening of blood, transfusion related HIV infection in children is perhaps more common in our country as compared to the West where it accounts for 18% of all pediatric infections. The frequency of perinatal HIV infection is expected to rise as the number of infected young women increases.

The understanding of the mechanism of perinatal transmission is not very clear. Transmission may occur *in utero* resulting in first and second trimester, abortions, during the third trimester, or in the process of birth where the infant is exposed to great quantity of infected fluid. Ingestion of breast milk is an important route from mothers with high viremic load(3,4).

From the Department of Pediatrics, All India Institute of Medical Sciences, New Delhi 110 029.

Reprint requests: Dr. Veena Kalra, Additional Professor, Department of Pediatrics, All India Institute of Medical Sciences, New Delhi 110 029.

Received for publication: August 12, 1993; Accepted: February 23, 1994 Most children infected in infancy will be symptomatic by 3 years of age. The HIV disease manifests by deficient cell mediated immunity, opportunistic infections and progressive dysfunction of multiple organ systems.

We herein report a case of acquired immune deficiency syndrome (AIDS) in an infant who acquired HIV from his mother. Transmission via breast milk seems the most likely mode of infection in this case.

## Case Report

The patient was the first child of a healthy 26-year-old woman, who had no risk factors for HIV infection. The child was born at term and the mother was transfused two units of blood for accidental hemorrhage. One unit of blood was obtained from a known relative, and the other from a commercial blood bank. The neonatal period was uneventful. Breastfeeding was established and continued for 12 weeks.

The child remained well till the age of 8 months when he developed moderate grade fever, failure to thrive and recurrent diarrhea. He did not respond adequately to therapy.

At 9 months age he was referred to us for further evaluation. He had mild pallor, active chorioretinitis in one eye, soft hepatomegaly and splenomegaly each measuring 4 cm and 3 cm below the costal margin, respectively. BCG scar was absent although he was vaccinated at the appropriate age. He weighed 6.5 kg (<5th centile) and his length was 65 cm (<5th centile). His ESR was 48 mm, in first hour. The peripheral smear had 60% monocytes, and no abnormal cells were seen. Liver and renal function tests were within normal limits.

Blood culture was sterile and Widal was negative (<1:40). Bone marrow revealed a myeloid predominance. *Geotrichum candidum* an uncommon fungus was grown on fungal culture.

Salmonella typhi and Candida albicans were isolated from stool cultures on two different occasions. Chest X-ray showed small parenchymal lesions in the right midzone. Serum tested positive for tubercular antigen on PCR-amplification. Though, tuberculin test and family survey for tuberculosis were negative, the child "was discharged on anti-tubercular therapy.

At 11 months of age he was readmitted for persistence of fever. His condition worsened in the hospital and he developed jaundice, increase in hepatomegaly and necrotizing skin lesions. *Pseudotnonas aeruginosa* was isolated from bone marrow, skin scrapings and urine culture showed a significant growth of *E. coli*. Serum transaminases were raised but the hepatitis B antigen was negative. Abdominal ultrasonography was unremarkable except for hepatomegaly. Cranial ultrasound showed mild hydrocephalus. The septicemia responded to antibiotics including aminoglycosides and ceftazidime.

In view of persistent nonspecific symptoms and infection by unusual organisms, an immunological workup was done. IgA level was low (25 mg/dl), IgG and IgM were normal. HIV antibodies by ELISA and Western blot were positive. There was cutaneous anergy, though a CD<sub>4</sub>: CD<sub>8</sub> ratio was not performed. The mother was also HIV seropositive but the father was uninfected.

### **Discussion**

This infant required multiple, protracted hospitalizations for severe and repeated infections. This nonspecific clinical complex of fever, weight loss, hepatosplenomegaly, recurrent diarrhea and opportunistic infection was suggestive of AIDS(5).

Recurrent diarrhea may be a major symptom in HIV infected children as in our case. The etiology is commonly infectious or HIV associated disease. Gastrointestinal infections can be due to Cryptosporidium, Salmonella, Isospora belli or cytomegalovirus(6). This child had an active chorioretinitis which may be due to cytomegolovirus, or HIV infection per se.

Symptomatic liver disease is uncommon in HIV infected children(7) and commonly results from superimposed opportunistic infection as in this case report. HIV infection in this child could be classified as P-2, subclass D as per CDC classification<sup>^</sup>).

The infection in this child is unlikely to have been transmitted *in utero*, since the mother was probably infected after delivery through blood transfusion where she received unscreened blood from a private blood bank. Her husband was seronegative and no other risk factors were operative. Since HIV has been demonstrated in the breast milk(8), we presume that breast feeding represents the likely mode of transmission in this case.

We conclude that children with nonspecific protracted illnesses like chronic diarrhea, weight loss, fever and hepatosplenomegaly should be tested for HIV disease. A legislative measure should be undertaken to implement national blood screening, and till then transfusion of inadequately screened blood should be condemned.

#### REFERENCES

- Caldwell MB, Rogers MF. Epidemiology of pediatrics HIV infection. Pediatr Clin North Am 1991,38:1-16.
- Centers for Disease Control. HIV/AIDS Surveillance Report. April, 1990.
- 3. Jovaisas E, Koch M, Schafer A, *et al.* LAV, HTLV III in 20 week fetus. Lancet 1985, 2:1129.
- Zeigler JB, Cooper DA, Johnson RO, et al. Postnatal transmission of AIDSassociated retovirus from a mother to an infant. Lancet 1990,1: 896-897.

- 5. Barret D. The clinician's guide to pediatric AIDS. Contemp Pediatr 1980, 5: 24.
- Soave R, Johnson W. Cryptosporidium *Isospora belli* infection. J Infect Dis 1988, 157: 225-229.
- Schneiderman D. Hepatobiliary abnormalities of AIDS. Gastroenterol Clin North Am 1988,17: 615.
- 8. Center for Disease Control. Classification system for human immunodeficiency virus infection in children under 13 years of age. MMWR 1987, 36: 225-235.

# **Rett Syndrome**

V. Kalra D.T. Sud

Rett syndrome is a fairly newly described disorder which consists of a devastating lifelong disability producing severe degeneration of motor skills and intellectual function in girls. We have recently seen six patients of Rett syndrome. To our knowledge Rett syndrome is being reported for the first time from India.

From the Department of Pediatrics, All India Institute of Medical Sciences, New Delhi 110 029.

Reprint requests: Dr. Veena Kalra, Additional Professor, Department of Pediatrics, All India Institute of Medical Sciences, New Delhi 110 029.

Received for publication: June 22, 1993; Accepted: March 31, 1994

### **Case Reports**

In the year J.992 and 1993 at the Child Neurology Clinic of the All India Institute of Medical Sciences (AIIMS), New Delhi we encountered six patients who were referred with the diagnosis of cerebral palsy but actually had Rett syndrome. All the six were girls ranging from 2.5 to 6.5 years age, at initial contact. Their clinical profile is summarized in Table I. All patients were born after nonconsanguinous marriages with no identifiable antenatal or perinatal insults. The birth weight was normal in 3 and unrecorded in others and they were reported normal till 15 to 24 months age. The complaints included poor mental progress, loss of attained hand skills, and stereotypic hand movements which included hand wringing in 4, mouthing in 1 and inappropriate clapping in 1. In all patients, the movements disappeared in sleep. The older subjects (cases 3, 4, 6) could no longer hold a pencil or turn pages of a book skillfully.

Gait was clumsy and wide based in