in 1973(3). Since then chloramphenicol resistance has been reported from many countries(1). The MIC values of the isolates in our cases were consistent with chromosomal resistance to chloramphenicol and ampicillin. Similar patterns of resistance have been reported for *S. typhi* from patients in the Middle East(4). Plasmid mediated resistance results in MIC values for chloramphenicol of 125-250 mg/L and renders the drug ineffective in clinical treatment. However, chromosomal resistance might also affect the therapeutic value of this antibiotic(4). Chromosomally mediated chloramphenicol resistance can develop *in vivo* during treatment with antibiotics(4). Since the first patient was treated with ampicillin before admission it is likely that our cases were caused by different strains of *S. typhi.*

The failure of our patients to develop a positive widal reaction is not unusual since the percentage of patients who develop antibodies in outbreaks of typhoid fever may be as low as 24-60%(5). Patients treated with antibiotics early in the course of their disease may not develop a significant rise in antibody titres.

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Perinatally Acquired AIDS

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AIDS is spreading like an epidemic in our country. According to the Indian Council of Medical Research reports(1), 6683 individuals are positive for HIV infection in India, this clearly highlights the magnitude of this problem. In India, the first adult AIDS case was reported in May, 1986. The first seropositive pregnant woman was reported in September, 1986, and the first seropositive infant was reported in October, 1987(2). The first autopsy on full blown AIDS was carried out in 1988(3). Since then the seropositivity due to HIV infection and clinical AIDS in Pediatric age group is also increasing(4,5). Our patient is the first case of perinatally

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acquired AIDS in India. This child died at the age of 47 days.

Case Report

A 1-month-old infant, weighing 2 kg was brought from an orphanage to our Outpatient Department on 6.10.1990 with complaints of distension of abdomen and loose motions of unknown duration. Detailed history was not available.

The baby's general condition was poor. There were signs of moderate dehydration, mild pallor, an erythematous maculopapular rash over both the cheeks, oral and perianal candidiasis and diaper dermatitis on both buttocks. Scleromatous changes were present on both the lower extremities. The baby's activity was depressed, with poor neonatal reflexes. Examination of respiratory, cardiovascular, central nervous system and gastro-intestinal system did not reveal any abnormalities.

With this clinical presentation and findings, the diagnosis of neonatal sepsis with acute gastroenteritis with oral and perianal candidiasis with diaper dermatitis was made and a possibility of HIV infection was kept in mind.

The investigations revealed a hemoglobin of 10.8 g/dl. The total and differential WBC counts were 14000/mm^3 with 76% neutrophils and 24% lymphocytes, the platelets were reduced on peripheral smear. Serum electrolytes showed hypokalemia. Blood urea nitrogen was normal and VDRL test was negative. The ELISA and Western blot tests for HIV were positive.

The baby was treated with broadspectrum antibiotics, antifungals and correction of dehydration and electrolyte disturbance. The baby had a fluctuating course in the ward and ultimately on the 17th day of admission, succumed to the illness.

A clinical autopsy was performed to know the cause of death. All reticuloendothelial organs were studied. The histopathology revealed lymphocytic depletion in the thymus, spleen and Peyer's patches of the gastrointestinal tract. In addition there was widespread cytomegalovirus infection in the lungs (*Fig.*), adrenals, kidneys and large intestine. Lung also showed fungal colonies of candida. Both showed pyogenic meningitis with abscess in right temporal lobe.

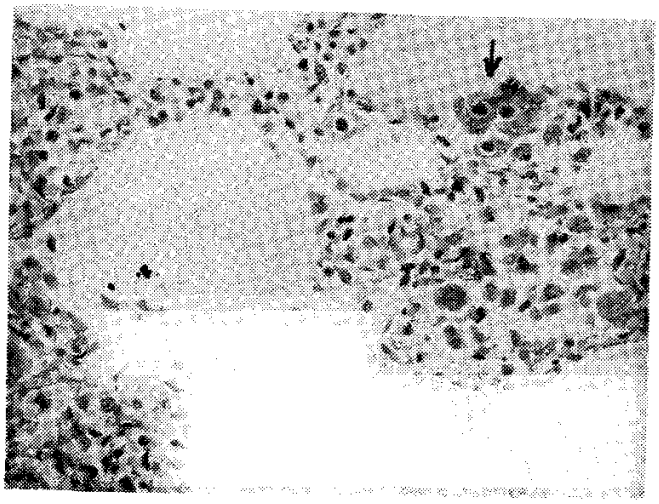

Fig. Lung histopathology-(H & E ×800) Arrow shows large alveolar lining cells containing intranuclear basophilic inclusions characteristic of cytomegalovirus.

Discussion

The clinical course of perinatal HIV infection is very variable with regards to its latency period, age of onset, signs and symptoms at presentation and the degree of neurological manifestations. The European Collaborative Study(6) of 600 children born to HIV infected mothers have shown antibody positivity in 64 children of whom 19 children (30%) presented with AIDS within 6 months of age. The youngest child in this study who presented with

AIDS was 2 months of age. Our patient was just one month old at the time of presentation and the presence of HIV antibodies at one month age is not diagnostic, in view of the possibility of passive transfer of maternal antibodies.

The presence of extensive oropharyngeal and perianal candidiasis, chronic diarrhea, failure to thrive, recurrent bacterial infections in an orphan baby with laboratory evidence of lymphocytopenia and the clinical course of the baby in the ward made us think in terms of AIDS related complex in this baby.

The histologic evidence of lymphocytic depletion in thymus, spleen and Peyer's patches of gastrointestinal tract is suggestive of the diagnosis of AIDS(3,7). Presence of indicator disease of AIDS like cytomegalovirus and candidiasis in a HIV positive infant suggest active HIV infection and AIDS in the infant(8,9). Other causes of primary immunodeficiency syndrome like Digeorge syndrome and Nezelof's syndrome were ruled out on clinical grounds as well as at postmortem study.

Thus, in clinical practice, higher suspicion of AIDS in children with multiple opportunistic infection and unusual course of the disease, we may be able to diagnose many more cases of AIDS in our country. We now understand AIDS as a chronic, preventable and manageable disease. Since it is rapidy spreading in our country, prevention remains the main tool in halting its progress.

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