AIDS and Antibodies to Human Immunodeficiency Virus (HIV) in Children and Their Families: Clinical Experience at Yale–New Haven Hospital

GEORGE MILLER, M.D., KELSEY MARTIN, B.A., BEN Z. KATZ, M.D., AND WARREN A. ANDIMAN, M.D.

Departments of Pediatrics, Epidemiology and Public Health, and Molecular Biophysics and Biochemistry, Yale University School of Medicine, New Haven, Connecticut

Received March 16, 1987

As of December 1986, we have identified 23 symptomatic children with human immunodeficiency virus (HIV) infection in New Haven. Twelve developed AIDS as manifested by lymphocytic interstitial pneumonitis, Pneumocystis carinii pneumonia (PCP), and/or disseminated mycobacterial infections; seven of them have died. The remainder have milder clinical syndromes, which include failure to thrive, diffuse lymphadenopathy, and parotid swelling. When compared to adults with AIDS, children often have hypergammaglobulinemia and normal numbers of T4 lymphocytes. Intravenous drug abuse by the mother or mother's consort is the risk factor in 87 percent of these children. Two families have now been identified with more than one symptomatic child, but in no family is there evidence of spread from symptomatic children to uninfected siblings. A prospective study was begun to attempt to assess the risk of developing symptomatic HIV infection when a child is born to a mother with antibodies to HIV.

INTRODUCTION

One percent of reported cases of AIDS in the United States occur in children less than 13 years of age [1]. The number of childhood AIDS cases is likely to rise in the future as the epidemic spreads and involves larger numbers of heterosexual women. Children are important subjects for analysis of the etiologic, pathogenic, and epidemiologic factors involved in AIDS. Since they have been infected with and harbor fewer dormant infectious agents than adults, they should help decipher one of the principal unanswered questions—why only some individuals with human immunodeficiency virus (HIV) infection become symptomatic; that is, certain co-factors, which are important in the development of AIDS, might be more readily discernible in children. Furthermore, some children who are HIV-infected may remain healthy until their childbearing years. These children could be a reservoir of HIV infection for future generations.

The etiologic agent of AIDS, a retrovirus of the lentivirus group, was identified in 1983 and 1984 [2,3]. This identification permitted the development of the serologic

527

Abbreviations: ARC: AIDS-related complex  EBNA: Epstein-Barr nuclear antigen  EBV: Epstein-Barr virus  HIV: human immunodeficiency virus  LIP: lymphocytic interstitial pneumonitis  PCP: Pneumocystis carinii pneumonia

This work was partly supported by National Institutes of Health Grant No. AI21186 and a grant from the American Foundation for AIDS Research (No. 000192).

Address reprint requests to: George Miller, M.D., Dept. of Pediatrics, Yale University School of Medicine, 333 Cedar Street, New Haven, CT 06510

Copyright © 1987 by The Yale Journal of Biology and Medicine, Inc. All rights of reproduction in any form reserved.
tests needed to make a specific diagnosis of HIV infection and to define, in precise terms, transmission of the disease. In August 1984, we began studies of HIV infection of New Haven children and their families, using Western immunoblotting, which is the most specific technique available for measuring antibody to HIV [4]. The results of these studies through December 1986 form the basis of this report.

The First Cases

The earliest recognized childhood case in New Haven was a boy born in 1979. His mother's principal consort was an intravenous drug abuser. At 16 months of age, the child was admitted to the hospital with fever, weight loss, and lymphadenopathy. He had hypergammaglobulinemia; pulmonary infiltrates and hilar adenopathy were seen on chest X-ray. Periodic episodes of respiratory decompensation and one episode of pneumococcal sepsis occurred between three and five years of age, by which time he developed clubbing. On three occasions, lung biopsies revealed histologic changes consistent with lymphocytic interstitial pneumonitis (LIP) and were found to contain Epstein-Barr virus (EBV) DNA [5]. When the patient first presented, pediatric AIDS was not well-recognized and various other diagnoses were entertained, including pulmonary hemosiderosis and the "X-linked lymphoproliferative syndrome" [6]. We subsequently found that the child and his mother had HIV antibodies. This patient is now seven years old and doing well. He is being treated with small doses of steroids.

Our second pediatric AIDS patient was born in 1982 to a prostitute who abused heroin. At the time of hospitalization at seven months of age for meningococcemia, the baby was noted to have persistent fever, hepatosplenomegaly, and thrush. Lymphocytic interstitial pneumonitis was demonstrated by lung biopsy. Laboratory findings included hypergammaglobulinemia and low numbers of T4 lymphocytes. At age 21 months, he developed a B-cell lymphoma of the central nervous system associated with EBV DNA [7]. At 31 months of age, he died of disseminated Mycobacterium avium-intracellulare infection. HIV serology was positive in the child and the mother.

The third patient, a girl born in 1982, was healthy until six months of age. She developed Pneumocystis carinii pneumonia (PCP) and rapidly died of respiratory failure. This fulminant course was mysterious since she had no identifiable underlying congenital immune deficiency. Both parents were subsequently found to be HIV-seropositive, and both developed AIDS and died. The father was a secretive intravenous drug abuser.

Clinical Characteristics of Twenty-Three Children with HIV Infection

Thus far we have identified 23 children with symptomatic HIV infection. Twelve of the 23 children with HIV antibodies had severe disease and met the case definition of pediatric AIDS; seven have died. Children with AIDS had well-recognized major complications such as PCP (five patients), lymphocytic interstitial pneumonia (LIP) (six patients), progressive encephalopathy (two patients), and/or disseminated mycobacterial infection, of which the latter was always fatal (four patients) [8,9,10]. One of the children with PCP also had received multiple blood transfusions and a bone marrow transplant for leukemia. Therefore, in this case, it is impossible to be certain whether PCP was related to the child's underlying disease and therapy or to the HIV infection.

The remaining 11 children have milder illnesses, manifesting as failure to thrive, developmental delay, persistent candidiasis, adenopathy, parotitis, or bacterial sepsis.
As noted by others, many children with HIV infection are first seen because of an episode of sepsis due to one of the common encapsulated bacterial pathogens, such as pneumococcus or meningococcus. The problem of classifying the milder disease has been emphasized, and there is at present no agreed-upon definition of "AIDS-related complex" (ARC) in childhood. The presence of antibody to HIV and the identification of a known epidemiologic risk factor, such as parental drug abuse or a history of multiple blood transfusions, help in the differential diagnosis. Only a few children in the New Haven group have had facies reminiscent of the dysmorphisms which have been described by others [11].

Some children with lymphocytic interstitial pneumonitis and no other opportunistic infection seem to do quite well. Episodes of respiratory failure have responded acutely to aggressive respiratory support and large doses of corticosteroids. After clinical recovery, most are maintained on very small every-other-day doses of prednisone.

CLINICAL LABORATORY FINDINGS

Several laboratory findings are distinctive in children with HIV infection. One is marked polyclonal hypergammaglobulinemia, a finding seen in ten of 12 children in our original series, as well as by others [4,12,13]. Only two of our patients did not manifest this abnormality; both were infants who died before the age of six months. It is not known whether these immunoglobulins have antigen specificity; however, a variety of autoantibodies have been observed in children with AIDS [8]. It is possible that these antibodies are one manifestation of EB virus-mediated B-cell stimulation.

A second unusual feature of childhood AIDS is that children usually do not have lymphopenia and often have normal numbers of T4 lymphocytes. In our earlier series, only two of ten children had low numbers of T4 cells; the remainder had normal or, occasionally, elevated numbers of T4 lymphocytes [4]. Thus, the hypothesis that the opportunistic infections associated with AIDS are due solely to virus-induced cytolysis of T4 lymphocytes does not adequately explain the pathogenesis of childhood AIDS.

Children with HIV infection do have reversals of the ratio of T4 to T8 cells; however, in most instances, the reversed ratio is due to a relative increase in the numbers of T8 cells. This increased number of T8 cells may be due to concurrent viral infections with agents such as Epstein-Barr virus (EBV) and cytomegalovirus, which are known to cause an increase in the number of cytotoxic and suppressor cells [14,15]. Perhaps the T8 cells exert an ameliorating effect on the course of the disease, as has been suggested by in vitro experiments [16].

The third unusual feature of childhood AIDS is lymphocytic interstitial pneumonitis (LIP). Most children with this disease have extremely high antibody titers to the replicative antigens (capsid and early antigens) of EB virus, and their lung tissue contains EBV DNA. On the other hand, children with AIDS and PCP usually have low or absent antibodies to the EBV replicative antigens and no EBV DNA in the lung [5]. Finally, most children with AIDS and LIP have low antibody titers to the Epstein-Barr nuclear antigen (EBNA) and often lack antibody to that component of EBNA (designated EBNA 1) encoded by the BamHI K region of the genome [17]. Thus elevated antibody to EBV capsid and early antigens and lack of antibody to EBNA 1 can serve as an adjunct to the diagnosis of LIP.

SERODIAGNOSIS OF HIV INFECTION IN CHILDREN

We have used the Western blot technique to measure antibodies to HIV [4]. Every serum was simultaneously tested against HTLV III B virus purified on sucrose
gradients, HTLV III B-infected H9 cells, and uninfected H9 cells. H9 cells are a clone of the HT T-cell leukemia line which support HIV replication [3]. Our purpose in using this panel of antigens is to distinguish HIV-specific reactions from those that are due to anticellular reactivity. In most instances, the sera from children have also been examined for antibodies using commercially available ELISA kits.

Several features of the serologic response to HIV in children merit comment. In general, the reactivity of sera from children is weaker than that from infected adults, particularly their parents. Children who are HIV-infected, as demonstrated by virus isolation or by the presence of antibody to HIV detectable by Western blotting, are sometimes seronegative by ELISA [4]. In some children, the presence of antibodies by Western blotting can only be shown after the use of bridging anti-immunoglobulins. Children recognize fewer polypeptides on Western blots than do their parents [4]. Most of the mothers and fathers in our series recognized nearly all of the nine polypeptide clusters which we see on immunoblots. Among our original group of 12 sera from 13 children, sera from six recognized four or fewer polypeptides. Three of these six children reacted with only one or two proteins, polypeptides of 62 KDa and 25 KDa. The response of the other six children was comparable to that seen in adults; sera from this group reacted with seven, eight, or all nine polypeptide clusters. The weak reactors were younger (mean age, 1.3 years) than those with an adult type of serologic response (mean age, 2.6 years).

In children less than six months of age, the presence of antibodies to HIV could reflect transfer of maternal IgG anti-HIV antibodies across the placenta. Thus, antibodies per se are not necessarily indicative of infection. At present there are two ways to determine whether the young infant with HIV antibodies is actually infected. The first is to detect virus—by isolation in culture, antigen detection, or by in situ hybridization. The second is to show that the infant's antibody titers fail to wane, increase with time, or that reactivities to specific proteins, initially absent in the neonatal period, begin to appear later in infancy [18,19]. The measurement of specific IgM antibodies, a technique which has been so useful in rubella and other congenital infections, has not yet been found to be useful for HIV.

**INTRAVENTOUS DRUG ABUSE IS THE MAIN RISK FACTOR FOR CHILDHOOD HIV INFECTION IN NEW HAVEN**

The epidemiology of AIDS in New Haven differs significantly from the epidemiology of the disease in the U.S. at large (Table 1). These differences help explain why childhood AIDS and HIV infection are so prevalent in New Haven. The epidemiology of AIDS in New Haven is characterized by a relatively large fraction of patients who are intravenous drug abusers or heterosexual partners of those who use intravenous drugs, whereas the majority of AIDS cases in the U.S. are male homosexuals. Therefore, locally, there are relatively more reported cases in women and, accordingly, in children.

Of pediatric cases reported from Yale–New Haven Hospital, intravenous drug use by a parent was the risk factor in 20 of 23 symptomatic children (87 percent) (Table 2). In contrast, one child's father was a bisexual who had spent time in prison, and two children apparently acquired the infection through blood transfusions. Of these latter two, one was the leukemic child discussed previously, and the other was a child who received an exchange transfusion while being treated for fulminant meningococcal meningitis.
AIDS IN CHILDREN

TABLE 1
Comparison of Reported AIDS Cases in the City of New Haven and the Country as a Whole (Cumulative through December 1986)

| Risk Groups* | USA | City of New Haven |
|--------------|-----|-------------------|
| Homosexual/Bisexual male | 66% | 31% |
| Intravenous drug abuser (IVDA) | 17% | 43% |
| Homosexual male and IVDA | 8% | 4% |
| Heterosexual sex partner | 4% | 11% |
| Hemophilia | 1% | 0 |
| Transfusion recipient | 2% | 3% |
| Children | 1% | 6% |
| Undetermined | 2% | 3% |
| Ethnic Group | | |
| White | 60% | 23% |
| Black | 24% | 61% |
| Hispanic | 14% | 14% |
| Other/Unknown | 2% | 2% |
| Gender | | |
| Male | 92% | 71% |
| Female | 8% | 29% |
| Cases/Million | 92 | 634 |

*Percentages do not add up to 100 percent because of rounding error.

The 20 children with symptomatic HIV infection which resulted from parental intravenous drug abuse were members of 17 families. In ten of the 17 families, the mother herself was known to use intravenous drugs; in seven families the mother denied intravenous drug abuse but had as her principal sexual consort an intravenous drug abuser. In these families the mother presumably became infected heterosexually and transmitted the infection to her infant. This conclusion must be tempered by the caveat that the histories from the mothers may not always be reliable.

Age

Our HIV-infected children developed symptoms between one month and six years of age (Table 3). The median age at onset of symptoms was 18 months. Therefore, the

TABLE 2
Summary of Symptomatic Childhood HIV Infection Diagnosed at Yale–New Haven Hospital, by Risk Factor, Through December 1986

| Risk Factor      | Number of Children with Symptoms | Number with AIDS |
|------------------|----------------------------------|-----------------|
| Parental Drug Use| 20                               | 10              |
| Father Bisexual  | 1                                | 1               |
| Transfusion      | 2                                | 1               |
| Total            | 23                               | 12              |
incubation period for clinically expressed pediatric AIDS is 18–27 months, depending on whether the infection is acquired at the time of birth or in utero.

**HIV Infection in Families of Symptomatic Children**

In our original group of 14 children, sera were available from 12 of the 14 mothers. All of the mothers’ sera contained antibodies to HIV. One mother died of AIDS, another of a drug overdose; the rest were not symptomatic. We could locate only five fathers; one of them, who was not living in the same household as the child and his mother, was HIV-seronegative. The other four fathers were seropositive, and one died of AIDS.

The situation in siblings was instructive in several respects. In our original study, only one of eight siblings of index cases was seropositive [4]. This finding indicated that HIV was not readily spread from infected parents or infected children to siblings, as others have also found [4,20,21]. Furthermore, parent surrogates who cared for two children during prolonged hospitalizations remained HIV-seronegative. Finally, we have studied one family in whom all members are seronegative, even after the parents dressed the open wounds of their infected child for two years prior to discovery of his serologic status.

In the expanded group presented in this report, we now have two families in which there is more than one infected symptomatic child: one family from our previous study now has a third symptomatic child and another newly recognized family has two. Thus three of 14 siblings are HIV-seropositive (Table 4). In the original family with two symptomatic children, the infected older sibling was presumably infected in utero, at birth, or in the neonatal period by the mother. After his younger brother presented to us, he was evaluated and found to have thrush and diffuse adenopathy. Although the two sibs have different fathers, both were intravenous drug users and the mother was seropositive at the time the younger child was first examined. Among the seronegative siblings of index cases was a fraternal twin and a younger sibling. These findings indicate that an infected mother can give birth both to infected and uninfected children.

Only prospective studies will define precisely the risk associated with being born to a carrier mother, although, in preliminary studies to date, the risk of being born with HIV infection to a mother who has previously given birth to a child with HIV infection is estimated to range from 33–92 percent [22,23].

**PREVENTION OF TRANSMISSION OF HIV INFECTION TO CHILDREN**

In virtually all instances, infected, but usually asymptomatic, mothers transmit HIV infection to their children. Occasionally, HIV infection has been transmitted to children through blood or blood products [24].

---

**TABLE 3**

Symptomatic HIV Infection in New Haven Children, by Age, Through December 1986

| Age at Onset | Number | %  | AIDS | Death |
|-------------|--------|----|------|-------|
| <12 months  | 7      | 30 | 3    | 2     |
| 12–24 months| 8      | 35 | 5    | 3     |
| 25–36 months| 3      | 13 | 1    | 0     |
| >36 months  | 5      | 22 | 3    | 2     |
| Total       | 23     | 100| 12   | 7     |
Interruption of transmission from mother to child requires a more detailed understanding of the pathogenesis of perinatal infection. There are several different means whereby transmission may occur. Virus may pass from the maternal blood across the placenta and infect the fetus in utero. Among the findings which would implicate in utero transmission are infection of the placenta, isolation of virus from the infant in the first days of life, and the presence of a distinct constellation of congenital malformations. The virus may be acquired during birth as the infant passes through the birth canal. If this is a significant mode of transmission, Cesarean section might help prevent infection. There are, however, cases of children with AIDS who have been born by Cesarean section [25]. Finally, the infant may be infected in the postnatal period by close personal contact, or breast feeding, as has been reported in a single case [26]. Since non-venereal personal contact does not appear to spread the infection to older children, one would need to postulate, under the latter circumstances, that the newborn infant is more susceptible to infection by personal contact than the older child.

Although all three modes of transmission may occur, some evidence favors the pathway of intrauterine infection, i.e., the craniofacial abnormalities which have been described, the occasional isolation of virus from placenta and fetus, and the rapid onset of disease during the first months of life in some infants [8]. Perhaps sampling of fetal blood or other tissues for virus will help confirm the incidence of intrauterine infection.

If public health policy is to be directed at the prevention of HIV infection of children, we need to learn more about the risks associated with being born to a seropositive mother. Our study of families, albeit small, permits some tentative conclusions. Not all infected mothers give birth to infected children. We and others have described twins, one of whom escaped infection [27]. Occasionally younger siblings of children with AIDS remain uninfected [23]. Nonetheless, some families appear to be at greater risk. Two mothers in our series have borne successive children with symptomatic HIV infection.

The only way to assess the risk to infants of seropositive mothers is to carry out a prospective study. Such a study was started in New Haven in December 1985 (Table 5). The idea was to identify infants who were seropositive at or around the time of birth and then follow them. Twenty-two infants born to twenty mothers were identified in a year. These infants' mothers were enrolled in a methadone program, were known intravenous drug addicts who did not participate in a treatment program, or were consorts of intravenous drug addicts (Table 5). The infants will be followed serologically and for the development of symptomatic HIV infection. Thus far, three of them
appear to be symptomatic: one who presented with pneumococcal sepsis and shock, one who presented with pneumococcal bacteremia and subsequently developed PCP, and one who presented with Salmonella sepsis and was found to have diffuse cortical atrophy. Several infants who were antibody-positive at birth have lost their antibody, which was presumably of maternal origin, and remain well.

PUBLIC POLICY QUESTIONS

Questions are frequently asked about the potential infectivity of children with AIDS or pre-AIDS in school situations and at home. Our opinion that these children represent no great hazard to caregivers and classmates is based on the observation that individuals who provide close care to infected infants and children, such as nursing staff, grandmothers, foster mothers, and mother surrogates are not seropositive, although there is one very unusual case report to the contrary [28]. Furthermore, older siblings and even twins in the same household, exposed both to infected parents and to infected younger brothers and sisters, remain seronegative in our studies and in others. When more than one seropositive sibling is found, it has been assumed that a carrier mother has passed the infection to more than one child in utero or during the neonatal period. For these reasons, we believe that infected children can be placed in their natural homes, foster care, and in school with very little risk.

REFERENCES

1. Centers for Disease Control: Immunization of children infected with human T-lymphotropic virus type III lymphadenopathy-associated virus., JAMA 256:2477–2483, 1986
2. Barre-Sinoussi F, Cermann JC, Rety F, et al: Isolation of a T-lymphotropic retrovirus from a patient at risk for acquired immune deficiency syndrome (AIDS). Science 220:868–871, 1983
3. Gallo RC, Salahuddin SZ, Popovic M, et al: Frequent detection and isolation of cytopathic retroviruses (HTLV-III) from patients with AIDS and at risk for AIDS. Science 224:500–503, 1984
4. Martin K, Katz BZ, Miller G: AIDS and antibodies to Human Immunodeficiency Virus (HIV) in children and their families. J Infect Dis 155:54–63, 1987
5. Andiman WA, Eastman R, Martin K, et al: Opportunistic lymphoproliferations associated with Epstein-Barr viral DNA in infants and children with AIDS. Lancet ii:1390–1393, 1985
6. Sullivan JL: Epstein-Barr virus and the X-linked lymphoproliferative syndrome. Adv Pediatr 30:356–399, 1983
7. Katz BZ, Andiman WA, Eastman R, et al: Infection with two genotypes of Epstein-Barr virus in an infant with AIDS and lymphoma of the central nervous system. J Infect Dis 153:601–604, 1986
8. Scott GB, Buck BE, Leterman JG, et al: Acquired immunodeficiency syndrome in infants. N Engl J Med 310:76–81, 1984
9. Rubinstein A, Morecki R, Silverman B, et al: Pulmonary disease in children with acquired immune deficiency syndrome and AIDS-related complex. J Pediatr 108:498–503, 1986

**TABLE 5**

Prospective Study of 22 Infants Who Were Born HIV-Seropositive, December 1985–December 1986

| Maternal Risk Factor        | Number of Mothers* |
|-----------------------------|--------------------|
| Mother in Methadone program | 8                  |
| Mother drug-addicted         | 6                  |
| Consort drug-addicted        | 4                  |
| Unknown                      | 2                  |
| Total                        | 20                 |

*Each of two mothers gave birth to a pair of seropositive twins.
10. Epstein LG, Shaver LR, Oleske JM, et al: Neurologic manifestations of Human Immunodeficiency Virus infection in children. Pediatr 78:678–687, 1986
11. Marion RW, Wiznia AA, Hutcheon G, Rubinstein A: Human T-cell lymphotropic virus type III (HTLV III) embryopathy. Am J Dis Child 140:638–640, 1986
12. Oleske J, Minnefor A, Cooper R, et al: Immune deficiency syndrome in children. JAMA 249:2345–2349, 1983
13. Rubinstein A, Sicklick M, Gupta A, et al: Acquired immunodeficiency with reversed T4/T8 ratios in infants born to promiscuous and drug-addicted mothers. JAMA 249:2350–2356, 1983
14. Carney MP, Rubin RH, Hoffman RA, et al: Analysis of T-lymphocyte subsets in cytomegalovirus mononucleosis. J Immunol 126:2114–2116, 1981
15. Haynes BF, Schooley RT, Payling-Wright C, et al: Emergence of suppressor cells of immunoglobulin synthesis during acute Epstein-Barr virus-induced infectious mononucleosis. J Immunol 123:2095–2101, 1979
16. Walker CM, Moody DJ, Stites DP, Levy JA: CD8+ lymphocytes can control HIV infection in vitro by suppressing virus replication. Science 234:1563–1566, 1987
17. Miller G, Grogan E, Fischer DK, et al: Antibody responses to two Epstein-Barr virus nuclear antigens defined by gene transfer. N Engl J Med 312:750–755, 1985
18. Harnisch DG, Hammerberg O, Walker IR, et al: Early detection of HIV infection in a newborn (letter). N Engl J Med 316:272–273, 1987
19. Johnson JP, Nair P, Alexander S: Early diagnosis of HIV infection in the neonate (letter). N Engl J Med 316:273–274, 1987
20. Kaplan JE, Oleske JM, Getchell JP, et al: Evidence against transmission of human T-lymphotropic virus lymphadenopathy-associated virus (HTLV-III/LAV) in families of children with the acquired immunodeficiency syndrome. Pediatr Infect Dis 4:468–471, 1985
21. Friedland GH, Saltzman BR, Rogers MF, et al: Lack of transmission of HTLV III/LAV infection to household contacts of patients with AIDS or AIDS-related complex with oral candidiasis. N Engl J Med 314:344-349, 1986
22. Luzi G, Ensoli B, Turbassi G, et al: Transmission of HTLV III infection by heterosexual contact (letter). Lancet ii:1018, 1985
23. Scott GB, Fischl MA, Klimas N, et al: Mothers of infants with the acquired immunodeficiency syndrome. JAMA 253:363–366, 1985
24. Amman AJ, Cowan MJ, Wara DW, et al: Acquired immunodeficiency in an infant: possible transmission by means of blood products. Lancet i:956–958, 1983
25. Cowan MJ, Hellman D, Chudwin D, et al: Maternal transmission of acquired immunity deficiency syndrome. Pediatr 73:382–386, 1984
26. Ziegler JB, Cooper DA, Johnson RO, et al: Postnatal transmission of AIDS-associated retrovirus from mother to infant. Lancet i:896–897, 1985
27. Merez-Bautista R, Fikrig SM, Pahwa S, et al: Monozygotic twins discordant for the acquired immunodeficiency syndrome. Am J Dis Child 140:678–679, 1986
28. Centers for Disease Control: Apparent transmission of Human T-lymphotropic virus type III/lymphadenopathy-associated virus from child to mother providing health care. JAMA 255:1005–1010, 1986