

# HOSPITAL PHYSICIAN®

## PEDIATRIC MEDICINE BOARD REVIEW MANUAL

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## Pediatric HIV Infection

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# Pediatric HIV Infection

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## INTRODUCTION

Acquired immunodeficiency syndrome (AIDS) is caused by the human immunodeficiency virus (HIV). The pathologic hallmark of AIDS is severe immunosuppression; HIV-infected infants and children suffer considerable morbidity and mortality from opportunistic infections such as *Pneumocystis jiroveci* pneumonia (PCP), tuberculosis, and disseminated *Mycobacterium avium intracellulare* (MAI) infections. In addition to these catastrophic medical consequences, HIV-infected infants and children—along with their families—experience tremendous psychosocial upheaval owing to this chronic, often devastating illness.

Because of the complexity and vast clinical spectrum of HIV infection, this case-based article is limited to a general review of epidemiology, diagnosis, and management of pediatric HIV infection. The prevention of perinatal HIV transmission also is discussed.

## EPIDEMIOLOGY

Today, children acquire HIV infection primarily via perinatal transmission. The first AIDS cases in children were reported in 1982; by the year 2002, the cumulative number of AIDS cases in US children younger than 13 years was 9074.<sup>1</sup> At the end of 2001, 2499 children were reported to be living with AIDS in the United States and its territories. An additional 2909 children were known to be infected with HIV. Because not all states report to the Centers for Disease Control and Prevention (CDC), these numbers are considered underestimates. In 2002, the World Health Organization estimated that 3.2 million children younger than 15 years were living with HIV/AIDS.<sup>2</sup>

## IMMUNOPATHOLOGY AND TRANSMISSION OF HIV INFECTION

HIV virus types 1 and 2 (HIV-1 and HIV-2) are members of the *Lentivirus* genus of the Retroviridae family, which are RNA viruses that require the generation of viral DNA within the host.<sup>3</sup> High levels of viral replication characterize HIV-1 infection. Studies reveal that in infancy and early childhood, there is a large and renewable pool of permissive host cells that may contribute to

persistently high plasma HIV-1 RNA levels.<sup>4</sup> In vertically infected infants, the level of plasma HIV-1 RNA increases rapidly during the first few weeks of life, reaching levels as high as  $10^5$  to  $10^7$  copies/mL.<sup>5,6</sup> Plasma HIV-1 RNA levels then decrease over the first 1 to 2 years of life, but mean plasma levels in these children do not drop below  $10^5$  copies/mL until at least the third year of life. Studies in infants have revealed that high viral loads (ie, levels of 100,000 copies/mL) have correlated with high risk for disease progression and mortality, especially if CD4+ T cell percentage is less than 15%.<sup>7</sup>

The life cycle of the HIV virus is complex. Cells that express the CD4 molecule on their cell surfaces (CD4+ T lymphocytes and cells of monocyte or macrophage lineage) are the primary targets for the virus (specifically, the external glycoprotein 120 [gp120]-containing binding sites for the CD4+ cells and coreceptors). Viral attachment and entry are vital for the perpetuation of the virus, leading to the destruction of cells bearing CD4+ markers. Cell surface chemokine receptors serve as coreceptors for HIV-1.<sup>8</sup> All HIV-1 strains use CCR5 or CXCR4 (also known as LESTR or fusion) coreceptors, and CCR5 appears to be important in the initial establishment of infection.

Vertical (mother to child) transmission of HIV-1 may occur during the following periods: (1) in utero, (2) intrapartum, and (3) postpartum (through breastfeeding). Transmission of HIV has not been demonstrated to occur in schools or child care settings. In the absence of documented parenteral, mucous membrane, or skin contact with blood or blood-containing body fluids, transmission of HIV has rarely been demonstrated to occur in families or households or with routine care in hospitals and clinics.

## INCUBATION PERIOD AND PROGNOSTIC FACTORS

The onset of clinical symptoms occurs earlier among children with perinatal infection as compared with those who have transfusion-acquired infection.<sup>9</sup> The observed period to development of AIDS is estimated to be 12 months for perinatal HIV infection compared with 41 months for children with transfusion-acquired HIV infection. In recent years, however, an increasing number of children with perinatally acquired HIV