

Varicella-Zoster Virus Disease

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Panel's Recommendations
<p>Vaccination and Post-exposure Prophylaxis</p> <ul style="list-style-type: none"> Children aged ≥ 12 months and adolescents with HIV stage 1 or 2 without evidence of immunity to varicella should receive two doses of single-antigen varicella vaccine (VAR; Varivax) according to routine or catch-up vaccination schedules (AI). VAR is not recommended for children with HIV stage 3 (AIII). Children and adolescents with HIV who are susceptible to varicella or who have severe (stage 3) immunosuppression and have had a significant exposure to varicella or herpes zoster (HZ) should receive varicella-zoster immune globulin (available as intramuscular VariZig) as soon as possible (ideally within 96 hours but up to 10 days) after exposure (AI). Varicella vaccine should be administered 5 months after receipt of VariZig in eligible children (AIII). Refer to the Varicella Vaccine section of Figure 1. Recommended Immunization Schedule for detailed recommendations. <p>Treatment of Varicella and Herpes Zoster</p> <ul style="list-style-type: none"> Intravenous (IV) acyclovir is the preferred drug for the initial treatment of varicella infection in children with HIV, especially those with severe immunosuppression (stage 3) or those who have high fever, abdominal pain, respiratory symptoms, or numerous or deep, necrotic, or hemorrhagic skin lesions (AI). IV acyclovir should be initiated as soon as possible after varicella lesions appear and shows the most benefit when initiated within 24 hours of rash onset. For children with HIV stage 1 or 2 who have mild varicella disease, varicella can be initially treated with oral valacyclovir (if ≥ 3 months of age) or oral acyclovir (BII). For children ≥ 3 months of age with HIV and uncomplicated HZ, oral valacyclovir is the treatment of choice (AI*). Initial IV acyclovir administration is recommended for children with HIV with severe immunosuppression (stage 3) and HZ, extensive multidermatomal HZ, trigeminal or sacral nerve involvement, disseminated infection, visceral involvement, or otherwise complicated HZ (AI*). Children in whom lesions continue to develop, fail to heal, or progress after 7 days of treatment may have acyclovir-resistant varicella-zoster virus. Resistance is rare and primarily occurs in immunocompromised individuals. Because resistance testing may be difficult to arrange and delays treatment, the decision to change therapy should be based on current clinical status, and foscarnet should be administered immediately if clinical status is deteriorating (AII*). <p>Rating of Recommendations: A = Strong; B = Moderate; C = Optional</p> <p>Rating of Evidence: I = One or more randomized trials in children[†] with clinical outcomes and/or validated endpoints; I* = One or more randomized trials in adults with clinical outcomes and/or validated laboratory endpoints with accompanying data in children[†] from one or more well-designed, nonrandomized trials or observational cohort studies with long-term clinical outcomes; II = One or more well-designed, nonrandomized trials or observational cohort studies in children[†] with long-term outcomes; II* = One or more well-designed, nonrandomized trials or observational studies in adults with long-term clinical outcomes with accompanying data in children[†] from one or more similar nonrandomized trials or cohort studies with clinical outcome data; III = Expert opinion</p> <p>[†]Studies that include children or children/adolescents, but not studies limited to postpubertal adolescents.</p>

Epidemiology

Varicella-zoster virus (VZV) is the infectious agent responsible for chickenpox (varicella) and shingles (herpes zoster [HZ]). Before the implementation of universal immunization in the United States in 1995, approximately 4 million cases of varicella occurred annually. After initiation of universal immunization, disease has since declined overall by >97%. The most impressive declines

(99%) were seen among persons aged <20 years who were born after vaccine introduction and in whom severe disease has mostly been eliminated.¹ Another unanticipated benefit of the vaccination program is the reduction in risk of HZ in both healthy and immunocompromised children. In healthy children, vaccination is associated with an approximately 80% lower risk compared with unvaccinated children).²

VZV is highly contagious and can be transmitted either via direct contact with VZV lesions (from varicella or HZ) or through airborne particles.³ Clinical infection develops in about 80% (range: 61–100%) of susceptible individuals exposed to VZV within a household, and health care–associated transmission is well documented.⁴ Notably, varicella is more contagious than HZ.⁵ The virus is extremely labile, unable to survive for long in the environment, and, therefore, is not transmitted through fomites.

After initial infection or vaccination with VZV, VZV establishes latency in sensory (dorsal root, cranial nerve, and autonomic) ganglia, where its reactivation results in HZ. Once established, VZV latency persists for life, and reactivation causing HZ occurs in approximately 30% of people with varicella. Although HZ is less contagious than varicella, VZV can still spread from HZ lesions through direct contact or via airborne particles, causing varicella infection in unvaccinated people who have never had varicella. HZ occurs when VZV-specific cellular immunity, first stimulated by primary infection, declines with age. In addition, VZV-specific cellular immunity may be depressed by HIV infection, with evidence suggesting that children with HIV have difficulty maintaining anti-VZV immunoglobulin G (IgG) levels due to failure of reactivating memory responses.^{6–9} Although the incidence of HZ has declined significantly in children with HIV since the advent of antiretroviral therapy (ART), the incidence remains at 1 to 3 cases per 100 patient-years, approximately 10 to 25 times higher than in the general population.^{10–15} Risk factors for the development of HZ include low incident (i.e., coincidental with HZ reactivation) or nadir CD4 T lymphocyte (CD4) cell count/percentage, high HIV RNA plasma viral load (e.g., >10,000 copies/mL), and acquisition of varicella when the CD4 percentage is <15%.^{11,16,17} As in adults, the frequency of HZ recurrences in children correlates inversely with the CD4 count.^{11,18}

Immunocompromised people with primary or reactivated VZV infection are at increased risk of serious disease. Severe varicella and disseminated HZ more commonly develop in children with HIV [stage 3](#)—or congenital T lymphocyte defects—than in people with B lymphocyte abnormalities. Other groups at higher risk of severe or complicated varicella include infants, adolescents, children with chronic cutaneous or pulmonary disorders, and those receiving systemic corticosteroids, other immunosuppressive therapy, or long-term salicylate therapy.

Clinical Manifestations

Primary infection with VZV results in varicella, which is a generalized, pruritic, erythematous vesicular rash often accompanied by fever and other systemic symptoms. In unvaccinated individuals, the rash typically consists of hundreds of lesions in various stages of development (e.g., papules, vesicles). Crusted lesions are indicative of healing. Breakthrough cases may occur in vaccinated children, but the rash and other symptoms are usually milder. The incubation period for varicella ranges from 10 to 21 days (average of 14 days) in immunocompetent children. A brief prodrome of malaise and fever may occur, followed by the appearance of skin lesions, which occur more frequently on the face and trunk than on the extremities.

Varicella causes more morbidity in individuals with HIV than in the general population. Initial reports of varicella in children with untreated HIV described severe disease manifestations and chronic, atypical skin lesions, including nonhealing ulcers that become necrotic, crusted, and hyperkeratotic.^{19,20} Clinically important systemic involvement, especially in severely immunocompromised children, can include neurologic manifestations (e.g., encephalitis, cerebellar ataxia, transverse myelitis), hepatitis, rapidly progressive pneumonitis, and multiorgan failure with intravascular coagulation.²¹ Subsequent studies suggest less complicated varicella infections in children with HIV who are receiving ART or who have higher CD4 counts at the time of infection.^{16,22} However, active infection may last longer than normal, with new lesions and fever developing into the second week of illness. In addition, the rate of complications is higher in children with HIV than in otherwise healthy children with varicella.²³

Uncommonly, severely immunocompromised children with HIV can have persistent chronic varicella infection, with continued appearance of new VZV lesions for >1 month after onset of varicella.^{20,24} The lesions are characteristically varicelliform at onset but evolve into nonhealing ulcers or necrotic, crusted, and hyperkeratotic verrucous lesions. Chronic VZV was reported in 14% of children with HIV who had VZV in the pre-ART era, usually in children with low CD4 counts.¹⁸

Congenital varicella syndrome (CVS), which can occur when VZV crosses the placenta and is transmitted to the fetus, is characterized by multi-system anomalies. The estimated incidence of CVS is 0.59% and 0.84% for women infected with VZV during the entire pregnancy and for those infected during the first 20 weeks of pregnancy, respectively.²⁵ CVS only occurs in infants born to women with primary varicella infection and does not occur in infants born to women with HZ during pregnancy.

Perinatal VZV transmission can also occur in late gestation, resulting in neonatal varicella. In mothers who develop varicella 5 days before to 2 days after delivery, the attack rate for infants is approximately 20%, and mortality, before the availability of antiviral therapy, was approximately 30%.^{26,27} In comparison, in infants born to mothers who developed varicella ≥ 5 days before delivery, long enough to allow the transfer of VZV antibodies across the placenta, perinatal varicella can still occur but is typically less severe and with an extremely low mortality rate.

HZ classically presents as a painful or pruritic, vesicular, dermatomal rash. Typically, pain precedes the rash by 2 to 3 days. Less typical rashes—including rashes that extend beyond dermatomal boundaries, are bilaterally distributed, or that are generalized—can occur in children with HIV. Multiple recurrent episodes of HZ can also be seen.^{11,18} Disseminated HZ with multiorgan involvement can occur, with or without the typical rash. Encephalitis long after HZ, or without rash, has been reported in children with HIV.²⁸ Given similarities between the characteristic rash of HZ and herpes simplex virus (HSV), polymerase chain reaction (PCR) testing of vesicular fluid is necessary to rule out HSV for diagnosis and treatment.

Retinitis, a potential complication of VZV infection in children and adolescents with HIV,^{29,30} may be confused with cytomegalovirus (CMV) retinitis.³¹ Acute retinal necrosis can be a complication of VZV infection in children with HIV regardless of CD4 count. Progressive outer retinal necrosis, often associated with HZ, typically occurs in individuals with CD4 counts <50 cells/mm³. A rapid decrease in visual acuity or occurrence of red eye or eye pain should prompt an immediate consultation with an ophthalmologist for diagnosis and specific therapy.

Diagnosis

Typical presentations of varicella and HZ are readily diagnosed clinically. Laboratory diagnostic methods are required for atypical presentations, prolonged course of disease, non-response to therapy, or to distinguish from HSV infection. VZV DNA PCR is the most sensitive and specific method for diagnosing VZV infection and can rapidly provide an etiologic diagnosis.³² In addition to lesion specimens (vesicular fluid or scabs), PCR can be applied to blood, cerebrospinal fluid, aqueous and vitreous humor, and pharyngeal and conjunctival swabs. Laboratories that can differentiate between wild-type and Oka (vaccine)-strain VZV using PCR include the Centers for Disease Control and Prevention (CDC) (404-639-0066), a safety research program sponsored by Merck & Co (1-800-672-6372), and four state public health laboratories (Wisconsin, Minnesota, California, and New York).³³ Prior methods of VZV detection, such as Tzanck smear, direct immunofluorescence for VZV antigen, cell culture, or shell vial culture, are less sensitive and specific than PCR and are no longer readily available.^{34,35} Serologic tests are of little value in diagnosing active VZV infection in children with or without HIV, but can be used to determine immunity to VZV.

Prevention Recommendations

Preventing Exposure

Children with HIV without evidence of immunity to varicella (no verified history of varicella or HZ and no evidence of appropriate vaccination or laboratory evidence of immunity) should avoid exposure to individuals with varicella or HZ. Commercially available VZV antibody assays can have false-negative and false-positive results, limiting the ability to determine varicella immunity with certainty.^{32,36} Non-immune people (e.g., no clear evidence of vaccination or previous VZV infection) who are exposed to VZV and have a household contact who is living with HIV should receive the varicella vaccine to reduce the chance of introducing wild-type VZV into the household.³⁷ For the same reason, household contacts aged 50 years and older should receive the HZ vaccine according to Advisory Committee on Immunization Practices (ACIP) recommendations.³⁸ No precautions are needed following immunization of healthy people who do not develop a rash. However, immunized people who develop a post-immunization rash should avoid direct contact with an immunocompromised host lacking evidence of immunity for the duration of the rash.

Preventing Disease

Active Immunization

Children with HIV [stage 3](#) may develop systemic disease from vaccine-strain VZV; therefore, single-antigen varicella vaccine (VAR; Varivax) **is not recommended** for children with HIV stage 3 (**AIII**).³⁹ The varicella vaccine can be safely administered to children in whom stable immune reconstitution (see [HIV Infection Stage table](#)) is achieved with ART for ≥ 3 months.^{40,41}

Children and adolescents with HIV stage 1 or 2 (see [HIV Infection Stage table](#)) without evidence of immunity to varicella should receive two doses of varicella vaccine according to [routine or catch-up vaccination schedules](#) (**AI**).³⁷ The response to varicella vaccination is optimal in children on effective ART for an extended period and in those with high CD4 counts and very low HIV RNA plasma viral loads.^{42,43} Limited data from a clinical trial in children 1 to 8 years of age with HIV (CD4 percentage $>15\%$) indicate that the vaccine was well tolerated and that $>80\%$ of the children had detectable VZV-specific immune responses (antibody or cell-mediated immune response or both) at 1 year after

vaccination.^{39,41} This finding has been validated by other investigators, including persistence of antibodies for 7 years or more post-vaccination.^{40,42,44,45} Immunogenicity of the varicella vaccine may decrease as children age, and data are limited on its use in older children and adolescents with HIV. In the absence of specific safety and immunogenicity data, the combination measles-mumps-rubella-varicella vaccine should not be administered to children with HIV in place of the single-antigen varicella vaccine **(AIII)**.

The effectiveness of the varicella vaccine in children with HIV is suggested by long-term follow-up studies of vaccinated children at several institutions.^{10,46} In one study, the incidence of primary VZV infection and HZ in the 57 patients who received the VZV vaccine was 22.3 per 1,000 person-years and 4.5 per 1,000 person-years, respectively.¹⁰ In another study, vaccination (one or two doses) was 82% effective against varicella, and no cases of HZ were observed in previously vaccinated children.⁴⁶ This compares favorably with the efficacy of the vaccine in healthy children (after one dose) and in children with underlying leukemia (after two doses), where an efficacy of 80% to 85% was observed for prevention of clinical infection. In vaccinated children without HIV, most breakthrough varicella cases (i.e., varicella that occurs ≥ 42 days after receipt of varicella vaccine) are mild, with fewer lesions (commonly < 50), less fever, and a shorter duration of illness than with varicella in unvaccinated children.^{4,47} Comprehensive information on the severity of breakthrough varicella in children with HIV is lacking.

Although children with HIV stage 1 or 2 (see [HIV Infection Stage table](#)) tolerate the vaccine well, like healthy children, up to 5% of vaccine recipients may develop a rash 2 to 3 weeks after vaccination. Antiviral therapy is rarely required, and skin lesions usually clear in 3 to 5 days without treatment. Oka (vaccine)-strain VZV is susceptible to acyclovir, should antiviral treatment be necessary. Because low levels of wild-type VZV are still circulating, VZV rashes (especially when they are extensive) that develop shortly after vaccination require virologic investigation to distinguish vaccine-associated rashes from those caused by wild-type VZV. Oka strain VZV can also cause HZ in vaccinated healthy children, although this occurs less frequently than wild-type HZ among those with natural varicella infection.^{14,48} Data on the frequency of Oka strain HZ after vaccination in children with HIV are lacking.

At this time, the recombinant subunit HZ vaccine is licensed only for use in healthy people aged ≥ 50 years and for adults ≥ 19 years who are immunodeficient or immunosuppressed, including those with HIV.³⁸ It has not been studied in children with HIV and should not be given to those aged ≤ 18 years.

Post-exposure Prophylaxis With Passive Immunization

Children and adolescents with HIV stage 1 or 2 (see [HIV Infection Stage table](#)) who lack evidence of immunity to varicella (as defined by ACIP)³⁷ or who have severe (stage 3; see [HIV Infection Stage table](#)) immunosuppression (regardless of prior immunity to varicella) and have a non-transient, significant exposure (e.g., face-to-face exposure for ≥ 5 minutes) to a person with varicella or HZ should receive varicella-zoster immune globulin (VariZIG) prophylaxis as soon as possible, ideally within 96 hours, but up to 10 days after exposure **(AI)**.^{49,50} VariZIG, which is administered as a single intramuscular dose, is a purified human varicella-zoster immune globulin made from plasma containing high levels of anti-varicella antibodies (IgG) and is commercially available in the United States from a broad network of specialty distributors (<https://www.varizig.com>). A large observational study of immunocompromised children without HIV infection indicated that VariZIG given within 72 hours of VZV exposure reduced varicella severity compared to historical controls.⁵¹

Subsequent studies indicated that some protection occurred with passive immunization for up to 10 days after exposure.⁵² Thus, varicella- or HZ-exposed children with HIV are likely to benefit from passive immunization.

Receipt of VariZIG may prolong the incubation period for varicella from 10 to 21 days to up to 28 days, simultaneously extending the period of potential infectiousness. In some cases, VariZIG may attenuate but not prevent varicella.⁵¹ If varicella does not develop, subsequent active immunization with VAR, provided the vaccine is not contraindicated, should be administered 5 months after receipt of VariZIG **(AIII)**. If VariZIG is not available, one dose of intravenous immune globulin (IGIV) can be administered once as soon as possible (ideally within 96 hours after exposure). However, the titer of anti-VZV antibodies of any specific lot of IGIV is uncertain because this is not tested routinely. Children who receive IGIV monthly for other indications are likely to be protected from VZV infection if their last dose was received within 3 weeks of varicella or HZ exposure.

Neonates with peripartum exposure to varicella in the immediate peripartum period (onset 5 days before to 48 hours after delivery) may benefit from VariZIG.³³ There are no data to suggest that infants with HIV infection or HIV exposure are at higher risk of neonatal varicella or should be managed differently from infants who have not been exposed to HIV.

Post-exposure Prophylaxis With Antivirals

When passive immunization is not possible for severely immunocompromised individuals, some experts recommend oral acyclovir or valacyclovir for post-exposure prophylaxis (PEP) based on several smaller studies suggesting prevention or attenuation of varicella in healthy children,⁵³⁻⁵⁵ with additional evidence supporting some benefit in immunocompromised children.⁵⁶⁻⁵⁸ When used for PEP, oral acyclovir or valacyclovir should be initiated 7 days after exposure and continued for 7 days.³³ The use of acyclovir for prophylaxis in VZV-exposed children with HIV has not been studied. For that reason, while some experts recommend using antivirals for PEP, other experts consider it prudent to wait until rash appears to start treatment with acyclovir (or valacyclovir) in VZV-susceptible, VZV-exposed children with HIV who did not receive passive immunization.

Post-exposure Prophylaxis With Varicella Vaccine

Varicella vaccine has been shown to be effective for PEP in children and adults without HIV.⁵⁹ However, this preventive approach is predicated on a prompt and robust immune response, which is why it has not been studied or recommended in children with HIV.

Treatment Recommendations

Treating Disease

Based on controlled trials in children with malignancies^{60,61} and response to therapy in children with untreated HIV and severe varicella,¹⁹ intravenous (IV) acyclovir is the preferred drug for treating varicella infections in children with HIV, especially those with severe immunosuppression (stage 3—see [HIV Infection Stage table](#)) or those who have high fever; abdominal pain; respiratory symptoms; or numerous or deep, necrotic, or hemorrhagic skin lesions **(AI)**. IV acyclovir should be initiated as soon as possible after varicella lesions appear, with the greatest benefit demonstrated when initiated within 24 hours of rash onset. In the setting of a national shortage of IV acyclovir, IV ganciclovir or

foscarnet can be substituted. IV antiviral therapy is typically continued for 7 to 14 days, provided that at least 48 hours have elapsed since the appearance of new lesions.

When a child with severe varicella is clinically improved and no new lesions appear, oral therapy can be considered for the remainder of the treatment cycle. Valacyclovir is a prodrug of acyclovir with two- to threefold higher bioavailability when administered orally and is rapidly converted to acyclovir after absorption. Oral valacyclovir is recommended over oral acyclovir for treatment of severe varicella due to its improved bioavailability. For children with HIV stage 1 or 2 (see [HIV Infection Stage table](#)) who have mild varicella disease, varicella can be initially treated with oral valacyclovir or oral acyclovir **(BII)**. Safety and efficacy data support valacyclovir dosing as young as 3 months of age.⁶²⁻⁶⁵ While no pediatric formulation is available, crushed valacyclovir caplets can be used to make an extemporaneous suspension with good bioavailability.⁶⁴⁻⁶⁶

For children ≥ 3 months of age with HIV and uncomplicated HZ, a 7- to 14-day course of oral valacyclovir is recommended **(AI*)**. For children < 3 months of age or if oral valacyclovir is unavailable, oral acyclovir can be used. Oral treatment (as opposed to IV) is considered safe for HZ because the risk of disseminated, life-threatening disease is lower with HZ than with varicella. However, initial IV acyclovir administration is recommended for children with HIV stage 3 (see [HIV Infection Stage table](#)) and HZ, or with extensive multidermatomal HZ, disseminated infection, visceral involvement, or otherwise complicated HZ **(AI*)**. IV acyclovir may also be considered for trigeminal nerve or sacral dermatomal involvement. IV acyclovir was demonstrably efficacious in a controlled trial of HZ in immunocompromised adults, including those with disseminated HZ.⁶⁷ If treatment is initiated intravenously, it should be continued until cutaneous lesions and visceral disease are clearly resolving, after which oral administration can be considered to complete the 10- to 14-day course of therapy.

Famciclovir is an oral prodrug of penciclovir that is U.S. Food and Drug Administration approved for the treatment of HZ in adults aged ≥ 18 years. Famciclovir has been evaluated for pediatric use in a single clinical trial of children with varicella (none with HZ), and a sprinkle formulation is available; however, safety and efficacy data are insufficient to support the use of famciclovir in children with HIV < 18 years of age who have varicella or HZ.^{65,68}

Varicella-Zoster Virus Ocular Disease

Progressive outer retinal necrosis evolves rapidly, and despite aggressive therapy, the prognosis for visual preservation is poor. Involvement of an ophthalmologist with experience in managing VZV ocular disease and its complications in children is strongly recommended when ocular involvement is suspected (e.g., eye pain, conjunctival injection, photophobia, blurred vision). Optimal therapy for VZV-related retinopathy has not been defined. Regardless of VZV-specific antiviral therapy, optimization of ART is recommended and monitoring for the emergence of immune reconstitution inflammatory syndrome (IRIS) is warranted, particularly among ART-naïve children. Most experts recommend IV anti-VZV therapy that includes combinations of systemic antivirals (e.g., acyclovir or ganciclovir plus foscarnet), frequently given in conjunction with twice-weekly intravitreal injections of ganciclovir and/or foscarnet.⁶⁹⁻⁷¹ Adjunctive retinal surgery is sometimes recommended, along with corticosteroids and/or low-dose aspirin for associated occlusive vasculopathy and optic neuropathy. In contrast to progressive outer retinal necrosis, acute retinal necrosis appears more responsive to high-dose IV acyclovir for 10 to 14 days, followed by prolonged (i.e., 4 to 6 weeks) oral treatment with valacyclovir or acyclovir.^{72,73}

Monitoring of Response to Therapy and Adverse Events (Including IRIS)

Primary toxicities are similar between acyclovir, valacyclovir, and famciclovir and include phlebitis (for IV acyclovir), renal toxicity, nausea, vomiting, and rash. Infants who received high-dose acyclovir for neonatal HSV disease experienced neutropenia (defined as absolute neutrophil count $<1,000$ cells/mm³) as a major side effect.⁶⁴ However, monitoring for neutropenia is not necessarily indicated for children receiving short courses of acyclovir for varicella or HZ. Among severely ill children without HIV receiving high-dose IV acyclovir, renal injury or failure was observed in $>10\%$ of patients.⁷⁴ Renal function should be assessed upon initiation of acyclovir treatment and at least once weekly during treatment, especially in children with underlying renal dysfunction who are receiving prolonged therapy. If possible, avoid concomitant administration of other nephrotoxic drugs. Maintaining adequate hydration (via IV fluids if necessary) can help to avoid crystallization of the drug in the renal tubules. IV acyclovir must be adequately diluted and administered slowly over 1 to 2 hours. Dose adjustment based on creatinine clearance is needed in children with renal insufficiency or renal failure ([see package insert](#)).

HZ has been considered an IRIS event in numerous reports in which the incidence of HZ was increased transiently after the institution of ART.⁷⁵ However, an analysis that compared the incidence of HZ in children in the 3 months before ART initiation to that in the 3 months after ART initiation indicated no difference in incidence rates.¹¹ This suggests that the high incidence of HZ occurring in the 3 months after ART is initiated represents persistence of the inability to develop a robust VZV-specific cell-mediated immune response in this early post-ART initiation period. As immune reconstitution proceeds beyond this time, the incidence of HZ declines. This relationship has been demonstrated with numerous opportunistic infections⁷⁶ and confirmed for HZ.¹⁰

Managing Treatment Failure

Children in whom lesions continue to develop, fail to heal, or progress after 7 days of treatment may have acyclovir-resistant VZV.⁷⁷ Resistance is rare and primarily occurs in immunocompromised children with inadequate VZV-specific cell-mediated immunity to rapidly clear the VZV infection. If possible, viral isolation in culture or viral sequencing should be attempted to perform susceptibility testing and confirm drug resistance. Because drug susceptibility testing may be difficult to arrange and may involve significant delay, the decision to change therapy should be based clinically. All acyclovir-resistant VZV strains are also resistant to valacyclovir, famciclovir, and ganciclovir and should be treated with IV foscarnet immediately, particularly if clinical status is deteriorating (**AII***).^{72,78} Drugs with a mechanism of action different from viral DNA polymerase inhibition have shown promise in the treatment of acyclovir-resistant VZV, but they are not currently licensed in the United States.⁷⁹

The most common toxicities associated with foscarnet are renal impairment, electrolyte disturbances, and seizures. Infusing foscarnet with saline fluid loading can minimize renal toxicity, and infusion through a central venous catheter can prevent thrombophlebitis. Doses should be modified in children with renal insufficiency and can be extrapolated from dose adjustments used for management of CMV ([see package insert](#)).⁸⁰ Complete blood counts and comprehensive metabolic panels should be monitored in those receiving foscarnet at least two to three times per week during induction therapy and once weekly thereafter.

Preventing Recurrence

No measures are available to prevent HZ in children and adolescents with HIV. However, varicella vaccination reduces the incidence (and perhaps severity) of HZ such that the risk of HZ is lower in vaccinated children with HIV than in those who had wild-type varicella infection.^{10,46,81} The recombinant subunit HZ vaccine can be administered to adults with HIV who are ≥ 19 years of age. In addition, the likelihood of initial or recurrent HZ attacks is reduced with effective ART.¹¹

Secondary Prophylaxis

There is no known benefit of secondary prophylaxis (chronic maintenance therapy) for children with HIV after an episode of varicella or HZ.

Dosing Recommendations for Preventing and Treating Varicella-Zoster Virus

Indication	First Choice	Alternative	Comments/Special Issues
Pre-exposure Prophylaxis	VAR (Varivax) vaccine 2-dose series	N/A	<p>VAR is indicated for children aged ≥ 12 months with HIV stage 1 or 2.</p> <p>VAR is not recommended for children with HIV stage 3.</p> <p>See the Varicella Vaccine section of Figure 1. Recommended Immunization Schedule for detailed vaccine recommendations information.</p>
Primary (Post-exposure) Prophylaxis	<p>Varicella-zoster immune globulin (human [VariZIG]) administered IM, ideally within 96 hours (up to 10 days) after exposure</p> <p>Dosing Based on Body Weight</p> <ul style="list-style-type: none"> • ≤ 2 kg: 62.5 international units • 2.1–10 kg: 125 international units • 10.1–20 kg: 250 international units • 20.1–30 kg: 375 international units • 30.1–40 kg: 500 international units • ≥ 40.1 kg: 625 international units 	<p>If VariZIG Is Not Available</p> <ul style="list-style-type: none"> • IGIV 400 mg/kg actual body weight administered ideally within 96 hours (up to 10 days) after exposure <p>If Passive Immunization Is Not Possible</p> <ul style="list-style-type: none"> • Consider acyclovir 20 mg/kg/dose (max: 800 mg/dose) PO four times daily OR valacyclovir 20 mg/kg/dose (max: 1,000 mg/dose) PO three times daily for 7 days, beginning 7 days after exposure. 	<p>Primary (Post-exposure) Prophylaxis Is Indicated for—</p> <ul style="list-style-type: none"> • Children with HIV stage 1 or 2 and substantial exposure to varicella or HZ with no verified history of prior infection, no evidence of vaccination, or who are seronegative for VZV on a sensitive and specific antibody assay; <i>or</i> • Children with HIV stage 3 and substantial exposure to varicella or HZ, regardless of immunity history to varicella <p>Note: VariZIG is commercially available in the United States from a broad network of specialty distributors.</p> <p>Varicella vaccine should be administered 5 months after receipt of VariZIG in eligible children.</p> <p>Some experts start acyclovir upon first appearance of rash in children with HIV, rather than providing acyclovir as prophylaxis.</p>
Secondary Prophylaxis	N/A	N/A	There is no indication for secondary prophylaxis.

Dosing Recommendations for Preventing and Treating Varicella-Zoster Virus

Indication	First Choice	Alternative	Comments/Special Issues
Treatment	<p>Varicella</p> <p><i>Children With Severe Immunosuppression (HIV Stage 3) or Severe Varicella Disease (see Treating Disease in text)</i></p> <ul style="list-style-type: none"> Acyclovir 10 mg/kg/dose or 500 mg/m²/dose IV every 8 hours for at least 7 days and until no new lesions appear for 48 hours <p><i>Children With No or Moderate Immunosuppression (HIV Stage 1 and 2) and Mild Varicella Disease</i></p> <ul style="list-style-type: none"> If age ≥3 months: <ul style="list-style-type: none"> Valacyclovir 20 mg/kg/dose (max: 1,000 mg/dose) PO three times daily for 7–14 days If age <3 months OR unable to obtain valacyclovir suspension: <ul style="list-style-type: none"> Acyclovir 20 mg/kg/dose (max: 800 mg/dose) PO four times daily for 7–14 days 	<p><i>Children Unresponsive to Acyclovir When Resistance Is Suspected</i></p> <ul style="list-style-type: none"> Foscarnet: 120–180 mg/kg/day IV divided every 8–12 hours for at least 7 days and until no new lesions appear for 48 hours <p><i>Children in Whom IV Acyclovir Is Indicated, in the Setting of a National Shortage of IV Acyclovir</i></p> <ul style="list-style-type: none"> Foscarnet: 120–180 mg/kg/day IV divided every 8–12 hours for at least 7 days and until no new lesions appear for 48 hours, or Ganciclovir 5 mg/kg/dose IV every 12 hours for at least 7 days and until no new lesions appear for 48 hours 	<p>For children aged <2 years, the dose of IV acyclovir is 10 mg/kg/dose every 8 hours. For children aged ≥2 years, some health care providers administer the same dose or base the dose on body surface area (500 mg/m²/dose IV every 8 hours).</p> <p>When children treated with IV acyclovir show signs of clinical improvement with no new lesions for 48 hours, completion of antiviral therapy with an oral agent may be considered.</p> <p>Renal dose adjustments listed below are only applicable for IV acyclovir:</p> <ul style="list-style-type: none"> CrCl >50 mL/minute/1.73 m²: No dose adjustment necessary CrCl 25 to 50 mL/minute/1.73 m²: Administer the usual recommended dose every 12 hours CrCl 10 to <25 mL/minute/1.73 m²: Administer the usual recommended dose every 24 hours CrCl <10 mL/minute/1.73 m²: Administer 50% of the usual recommended dose every 24 hours (e.g., if the usual recommended dose is 10 mg/kg/dose every 8 hours, then administer 5 mg/kg/dose every 24 hours) Intermittent hemodialysis: 5 mg/kg/dose IV every 24 hours; administer after hemodialysis on dialysis days Peritoneal dialysis: 5 mg/kg/dose IV every 24 hours; no supplemental dose needed Continuous renal replacement therapy: 10 mg/kg/dose IV every 12 hours

Dosing Recommendations for Preventing and Treating Varicella-Zoster Virus

Indication	First Choice	Alternative	Comments/Special Issues
			<p>For patients with renal impairment on PO acyclovir, the Panel recommends consultation with a pediatric pharmacist and extrapolation of dose adjustments used for adults with renal impairment.</p> <p>In children receiving acyclovir, it is important to maintain adequate hydration to avoid crystallization of the drug in the renal tubules.</p> <p>Valacyclovir doses should be adjusted for renal impairment; however, no formal pediatric renal dosing guidelines exist for valacyclovir. The Panel recommends consultation with a pediatric pharmacist and extrapolation of dose adjustments used for adults with renal impairment (see package insert).</p> <p>There is no pediatric preparation of valacyclovir, although 500 mg caplets can be extemporaneously compounded to make a suspension.</p> <p>For patients with renal impairment on IV foscarnet, the Panel recommends consultation with a pediatric pharmacist and extrapolation of dose adjustments used for management of cytomegalovirus.</p>
	<p>Herpes Zoster</p> <p><i>Children With Severe Immunosuppression (HIV Stage 3), Trigeminal or Sacral Nerve Involvement, Visceral Involvement, Extensive Multidermatomal, or Disseminated HZ</i></p> <ul style="list-style-type: none"> Acyclovir 10 mg/kg/dose or 500 mg/m²/dose IV every 8 hours for 10–14 days 	<p><i>Children Unresponsive to Acyclovir When Resistance Is Suspected</i></p> <ul style="list-style-type: none"> Foscarnet IV: 120–180 mg/kg/day divided every 8–12 hours for at least 7 days and until no new lesions appear for 48 hours 	<p>For most cases, oral treatment (as opposed to IV) for HZ is considered safe because the risk of disseminated, life-threatening disease is lower with HZ than with varicella.</p> <p>For renal dose adjustment and other administration considerations with acyclovir, valacyclovir, and foscarnet, see Varicella above.</p>

Dosing Recommendations for Preventing and Treating Varicella-Zoster Virus

Indication	First Choice	Alternative	Comments/Special Issues
	<p><i>Children With No or Moderate Immunosuppression (HIV Stage 1 and 2) and Uncomplicated HZ</i></p> <ul style="list-style-type: none"> • If age ≥ 3 months: <ul style="list-style-type: none"> ◦ Valacyclovir 20 mg/kg/dose (max: 1,000 mg/dose) PO three times daily for 7–14 days • If age <3 months or unable to obtain valacyclovir suspension: <ul style="list-style-type: none"> ◦ Acyclovir 20 mg/kg/dose (max: 800 mg/dose) PO four times daily for 7–14 days 	<p>Famciclovir is approved for use in adults (age ≥ 18 years) with HZ at a dose of 500 mg/dose PO three times daily for 7–14 days; however, data are insufficient to support the use of famciclovir for HZ in children with HIV.</p>	
	<p><i>Children With Progressive Outer Retinal Necrosis</i></p> <ul style="list-style-type: none"> • Acyclovir 10 mg/kg/dose or 500 mg/m²/dose IV every 8 hours OR ganciclovir 5 mg/kg/dose IV every 12 hours, <i>plus</i> • Foscarnet 90 mg/kg/dose IV every 12 hours, <i>plus</i> • Ganciclovir 2 mg/0.05 mL intravitreal twice weekly AND/OR foscarnet 1.2 mg/0.05 mL intravitreal twice weekly <p><i>Children With Acute Retinal Necrosis</i></p> <ul style="list-style-type: none"> • Acyclovir 10 mg/kg/dose or 500 mg/m²/dose IV every 8 hours for 10–14 days, followed by valacyclovir 20 mg/kg/dose (max: 1,000 g/dose) PO three times daily for 4–6 weeks OR acyclovir 20 mg/kg/dose (max: 800 mg/dose) PO four times daily for 4–6 weeks 		<p>Involvement of an ophthalmologist with experience in managing HZ ophthalmicus and its complications in children is strongly recommended when ocular involvement is evident.</p> <p>Optimal management of progressive outer retinal necrosis has not been defined.</p>

Key: CrCl = creatinine clearance; HZ = herpes zoster; IGIV = intravenous immune globulin; IM = intramuscular; IV = intravenous; the Panel = Panel on Opportunistic Infections in Children With and Exposed to HIV; PO = oral; VAR = varicella; VZV = varicella-zoster virus

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