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Chickenpox

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Introduction

Background

The varicella-zoster virus (VZV) is the etiologic agent of the clinical syndrome of chickenpox (varicella). Zoster, a different clinical entity, is caused by reactivation of VZV after primary infection. VZV is a double-stranded DNA virus included in the Alphaherpesvirinae subfamily. Chickenpox is largely a childhood disease, with more than 90% of cases occurring in children younger than 10 years. The disease is benign in the healthy child, whereas increased morbidity is seen in adults and in patients who are immunocompromised.

Pathophysiology

Chickenpox is usually acquired by the inhalation of airborne respiratory droplets from an infected host. The highly contagious nature of VZV explains the epidemics of chickenpox that spread through schools as one child who is infected quickly spreads the virus to many classmates. High viral titers are found in the characteristic vesicles of chickenpox; thus, viral transmission may also occur through direct contact with these vesicles, although the risk is lower.

After initial inhalation of contaminated respiratory droplets, the virus infects the conjunctivae or the mucosae of the upper respiratory tract. Viral proliferation occurs in regional lymph nodes of the upper respiratory tract 2-4 days after initial infection and is followed by primary viremia on postinfection days 4-6. A second round of viral replication occurs in the body's internal organs, most notably the liver and the spleen, followed by a secondary viremia 14-16 days postinfection. This secondary viremia is characterized by diffuse viral invasion of capillary endothelial cells and the epidermis. VZV infection of cells of the malpighian layer produces both intercellular edema and intracellular edema, resulting in the characteristic vesicle.

Exposure to VZV in a healthy child initiates the production of host immunoglobulin G (IgG), immunoglobulin M (IgM), and immunoglobulin A (IgA) antibodies; IgG antibodies persist for life and confer immunity. Cell-mediated immune responses are also important in limiting the scope and the duration of primary varicella infection. After primary infection, VZV is hypothesized to spread from mucosal and epidermal lesions to local sensory nerves. VZV then remains latent in the dorsal ganglion cells of the sensory nerves. Reactivation of VZV results in the clinically distinct syndrome of herpes zoster (shingles).

Frequency

United States

Chickenpox is a common disease, with most cases occurring in the pediatric population. Since the

introduction of widespread pediatric immunization in the United States in 1995, the incidence of varicella has declined significantly, approaching up to 90% in one study. Mortality from varicella has also similarly decreased since the initiation of the US vaccination program, with the mortality rate decreasing by approximately 66% in one study.^[1]

International

Countries with tropical and semitropical climates have a higher incidence of adult chickenpox compared with countries with a temperate climate (eg, United States, Europe).

Mortality/Morbidity

Chickenpox affecting a healthy child is usually a self-limited disease. Secondary bacterial infection of skin lesions, manifesting as impetigo, cellulitis, or erysipelas, is the most common complication in this population. Staphylococci and streptococci are the most commonly implicated bacterial pathogens. Bacterial superinfection may predispose to scarring. Localized bacterial superinfection may rarely result in septicemia, culminating in a secondary bacterial pneumonia, otitis media, or necrotizing fasciitis (see Complications).

Congenital infection with the VZV is also a concern. Maternal chickenpox during pregnancy may produce latency of the VZV in the dorsal root ganglia of the fetus. These children may remain asymptomatic, or they may develop herpes zoster at a young age without previous history of primary chickenpox infection. Primary maternal chickenpox infection during the first 20 weeks of gestation may also rarely produce the congenital varicella syndrome, which is characterized by limb hypoplasia, muscular atrophy, skin scarring, cortical atrophy, microcephaly, cataract formation, and rudimentary digits (see Special Concerns).^[2, 3]

Race

Varicella has no racial predilection.

Sex

Varicella has no sexual predilection.

Age

Chickenpox is predominantly a pediatric disease.

Clinical

History

Chickenpox is usually diagnosed clinically on the basis of the characteristic rash and successive crops of lesions. Lesions may be found in all stages of development and healing in affected sites. A history of exposure to an infected contact within the incubation period of 10-21 days is also an important clue in the diagnosis. Consider the following:

- Childhood chickenpox is usually not heralded by a prodrome, but rather it begins with the onset of an exanthem.
- Chickenpox in adults and adolescents may be preceded by a prodrome of nausea, myalgia, anorexia, and headache.
- The typical patient is infectious for 1-2 days prior to the development of rash and for 4-5 days

afterwards, which is usually the time at which the last crop of vesicles has crusted over.

- The triad of rash, malaise, and low-grade fever signals the onset of chickenpox.
- Small, erythematous macules appear on the scalp, the face, the trunk, and the proximal limbs, with rapid sequential progression over 12-14 hours to papules, clear vesicles, and pustules, with subsequent central umbilication and crust formation.
- New crops of lesions form, which subsequently progress to vesicles with crusting.
- Vesicles may appear on the palms and the soles and on the mucous membranes, with painful, shallow, oropharyngeal or urogenital ulcers.
- Intense pruritus commonly accompanies the vesicular stage of the rash.

Physical

The characteristic chickenpox vesicle, surrounded by an erythematous halo, is described as a dewdrop on a rose petal (see the images below).



Dewdrop on rose petal characteristic vesicle of chickenpox. Reprinted with permission from *Cutis* 65: 355, 2000.



Vesicular eruption on the trunk demonstrating papules, vesicles, and crusts. Reprinted with permission from *Cutis* 65: 355, 2000.

Chickenpox is clinically characterized by the presence of active and healing lesions, in all stages of development, within affected locations. Lesions characteristically heal without scarring, though excoriation or secondary bacterial superinfection predispose to scar formation.

Adults with chickenpox have a more complicated course than that occurring in children. Adults may experience a more widespread rash; prolonged fever; and an increased likelihood of complications, the most common being varicella pneumonia.

Clinical variants of chickenpox infection also occur. Hemorrhagic lesions (see Complications) are rare and are most commonly associated with patients who are immunocompromised or immunosuppressed. Bullous chickenpox is a rare variant in which bullae appear instead of the characteristic vesicles.^[4] The possibility of bullous impetigo from *Staphylococcus aureus* must be addressed, especially in a child with persistent fever or relapse after he or she appeared to be improving. Bullous chickenpox may affect both children and adults and must be differentiated from other bullous disorders (eg, bullous pemphigoid, pemphigus). The course of the disease is believed to be unchanged, although a delay in diagnosis and treatment of elderly patients and patients who are immunocompromised may lead to serious morbidity.

Chickenpox and other viral exanthems may appear concentrated in areas where intense sun exposure occurred during the incubation period. Patients with atopic dermatitis may show an atypical distribution of varicella, in which the characteristic eruption is primarily found on lichenified areas.^[5]

Causes

Chickenpox is usually acquired by the inhalation of airborne respiratory droplets from a VZV-infected host. High viral titers are found in the characteristic vesicles of chickenpox; thus, viral transmission may also occur through direct contact with these vesicles.

Differential Diagnoses

Bullous Pemphigoid	Herpes Simplex
Dermatitis Herpetiformis	Impetigo
Drug Eruptions	Insect Bites
Erythema Multiforme	Syphilis

Other Problems to Be Considered

Viral exanthems
Pityriasis lichenoides et varioliformis acuta
Disseminated herpes simplex virus (HSV) infection
Atypical herpes zoster
Rickettsial disease
Neonatal syphilis

Workup

Laboratory Studies

A Tzanck smear of vesicular fluid, which can be prepared in an office setting, demonstrates multinucleated giant cells and epithelial cells with eosinophilic intranuclear inclusion bodies.^[6]

Isolation of the varicella-zoster virus (VZV) through culture of vesicular fluid provides a definitive diagnosis, although culturing of the VZV is technically difficult and positive less than 40% of the time. Direct immunofluorescence study offers excellent sensitivity and is more rapid than tissue culture. Polymerase chain reaction (PCR)-based techniques are very sensitive in identifying VZV, although they are not readily available.^[7]

Serologic evidence of immunity (native IgG formation) can be achieved through a number of different assays (eg, enzyme immunoassay [EIA], indirect fluorescent antibody [IFA], complement fixation, fluorescent antibody to membrane assay [FAMA], latex agglutination test). EIA, FAMA, and indirect fluorescence are not widely available, and complement fixation is not highly sensitive for VZV. The latex agglutination test is the most popular serologic assay for determining exposure and immunity to VZV.

Imaging Studies

Chest radiography is indicated for pulmonary symptoms in adults.

Histologic Findings

Histologic examination of skin lesions does not differentiate VZV from herpes simplex virus (HSV) infection. Intranuclear eosinophilic inclusion bodies are seen in epithelial cells in both infections. Leukocytoclastic vasculitis and hemorrhage are more common in VZV lesions than herpes simplex.

Treatment

Medical Care

Healthy children

Primary varicella infection in the healthy child is a rather benign disease that requires symptomatic therapy only. Pruritus can be treated with calamine lotion or pramoxine gel, powdered oatmeal baths, or oral antihistamines.

The nucleoside analogue acyclovir given orally (20 mg/kg PO qid for 5 d) to children, though shown to decrease the duration and the symptoms of primary varicella infection when administered within 24 hours of onset of symptoms, is not commonly prescribed in healthy children.^[8]

Children with defects in cell-mediated immunity, chronic atopic dermatitis or asthma, iatrogenic immunosuppression or long-term systemic steroid use, splenic dysfunction, or nephrotic syndrome should be treated because of the high risk of varicella-related complications.

Immunocompetent adult population

Consider oral acyclovir for healthy persons at increased risk of severe varicella infections, most notably people older than 12 years. Oral acyclovir therapy in this population (800 mg 5 times/d for 7 d), begun within 24 hours of onset of symptoms, has been shown to decrease the duration of lesions and pyrexia and to diminish symptoms and duration of disease.

Valacyclovir, the L-valyl ester of acyclovir, is a prodrug that has higher oral bioavailability than acyclovir. Valacyclovir is used in the treatment of herpes zoster, but, currently, no large-based clinical trials have demonstrated its efficacy in primary varicella infection of healthy, immunocompetent individuals.

Famciclovir is a prodrug of penciclovir, which is a nucleoside analogue similar to acyclovir. Like valacyclovir, famciclovir has demonstrated efficacy in the treatment of herpes zoster, but it has not been extensively studied for use in primary varicella infection of healthy populations.

A few case reports also have found sorivudine, a nucleoside analogue that is a potent in vivo inhibitor of varicella-zoster virus (VZV) replication, to be effective in the treatment of primary varicella in healthy adults. Larger scale clinical trials are needed to demonstrate the efficacy of this medication.

Immunocompromised/immunosuppressed population^[9]

Intravenous acyclovir therapy is recommended for this population because of the life-threatening complications of primary varicella infection that may develop in immunocompromised hosts. Severe disseminated disease, with the development of varicella pneumonia, encephalitis, hepatitis, and hemorrhagic complications (see Mortality/Morbidity), is much more common in this population than in other populations. Secondary complications (eg, bacterial pneumonia, meningitis) caused by bacterial superinfection of cutaneous lesions with subsequent septicemia, are also more common and dangerous in the immunocompromised population.

Case reports have described vidarabine, a purine nucleoside analogue, and interferon-alpha to be effective in the treatment of primary varicella infection of immunocompromised hosts. Acyclovir-resistant strains of VZV have been reported in patients with AIDS. Foscarnet, an inorganic pyrophosphate analogue that acts as a selective inhibitor of viral DNA polymerases and reverse transcriptases, is a potentially efficacious drug in patients with acyclovir-resistant VZV strains. Optimal dosage, duration of therapy, and efficacy in primary varicella infection need further investigation. Treatment of primary varicella

in these populations is difficult and needs an integrated team approach.^[10]

Continuing research into new antiviral agents and ongoing clinical trials are constantly adding new information to the pharmacotherapy against VZV infections. Proper consultation with specialists who keep abreast of the most recent pharmacotherapeutic advances is highly advised before treating these patients.

Passive immunization

Varicella-zoster immune globulin [VZIG], a human immunoglobulin preparation, is indicated for use in highly susceptible, VZV-exposed immunocompromised or immunosuppressed populations (eg, patients who have undergone bone marrow transplantation, those with leukemia, patients with congenital or acquired immunodeficiency syndromes, patients undergoing immunosuppressive therapy for transplant procedures, infants born to mothers who experience onset of chickenpox 5 d prior to delivery or within 2 d after delivery). VZIG, given within 96 hours of exposure, can modify the course of disease but does not prevent it. Maximal effectiveness is seen with administration as soon as possible after exposure.

VZV vaccine^[11,12,13,14,15]

A live attenuated varicella vaccine (Oka strain) was approved by the US Food and Drug Administration in 1995 for prophylactic use in healthy children and adults. Vaccination recommendations consist of 1 dose for healthy children aged 12-18 months and 2 doses, in a 4- to 8-week interval, in persons older than 13 years who are susceptible. Studies in Japan point to high seroconversion rates and long-term immunity in children after vaccination.^[16] The need for revaccination, or a booster immunization, will be addressed after more long-term studies have been completed.

The effectiveness of the vaccine wanes over time, as has been shown in several studies. The effectiveness of vaccination ranges from 97% in the first year after vaccination to 84% 8 years postvaccination.

Breakthrough varicella, which is seen in previously immunized persons, is a well-known clinical entity. The disease course is much milder than conventional primary varicella, and an atypical clinical presentation, in which only a few papules or papulovesicles are present, is usually seen. Transmission of VZV to other individuals may occur, although at lower rates than in nonimmunized people with primary varicella.

Adverse effects to vaccination include pain and erythema at the site of injection, allergic reactions to gelatin, and the development of a localized chickenpox. Vaccine-induced herpes zoster infection in immunocompetent and immunocompromised populations has also been reported, albeit, it is a rare phenomenon. Rarer still, with only a small number of reported cases, is the transmission of vaccine-associated virus from vaccinated individuals to susceptible contacts.

Clinical guideline summaries

Centers for Disease Control and Prevention - A new product (VariZIG) for postexposure prophylaxis of varicella available under an investigational new drug application expanded access protocol^[17]

American Academy of Pediatrics Committee on Infectious Diseases -Prevention of varicella: recommendations for use of varicella vaccines in children, including a recommendation for a routine 2-dose varicella immunization schedule^[18]

Centers for Disease Control and Prevention, Advisory Committee on Immunization Practices - 1)
Prevention of varicella: recommendations of the Advisory Committee on Immunization Practices (ACIP). 2)
Update: recommendations from the Advisory Committee on Immunization Practices (ACIP) regarding
administration of combination MMRV vaccine^[19]

American Academy of Pediatrics Committee on Infectious Diseases -Recommended immunization
schedules for persons aged 0 through 18 years: United States, 2009^[20]

Medication

The goals of pharmacotherapy are to reduce morbidity and to prevent complications, especially in individuals who are immunocompromised/immunosuppressed.

Immune globulins

For passive immunization, use VZIG, a human immunoglobulin preparation. This agent is indicated for use in highly susceptible, VZV-exposed immunocompromised or immunosuppressed populations.

Varicella-zoster immune globulin, human (VZIG)

When given within 96 h of exposure, can modify the course of disease but does not prevent it. Maximal effectiveness is seen with administration as soon as possible after exposure. Administer by deep IM injection in gluteal muscle or in another large muscle mass.

Dosing

Adult

One vial (1.25 mL/125 U)/10 kg IM; not to exceed 625 U

Pediatric

Administer as in adults; minimum dose is 125 U IM

Interactions

Increases toxicity of live virus vaccine (MMR); do not administer within 3 mo of vaccine

Contraindications

Documented hypersensitivity; IgA deficiency; anti-IgE/IgG antibodies

Precautions

Pregnancy

C - Fetal risk revealed in studies in animals but not established or not studied in humans; may use if benefits outweigh risk to fetus

Precautions

Do not inject IV

Antiviral agents

Nucleoside analogs are initially phosphorylated by viral thymidine kinase to eventually form a nucleoside triphosphate. These molecules inhibit herpes virus' polymerase 30-50 times more than the human host cells' alpha-DNA polymerase.

Acyclovir (Zovirax)

Inhibits activity of both HSV-1 and HSV-2. Has affinity for viral thymidine kinase and, once phosphorylated, causes DNA chain termination when acted on by DNA polymerase. Patients experience less pain and faster resolution of cutaneous lesions when used within 48 h from rash onset. May prevent recurrent outbreaks. Early initiation of therapy is imperative.

Dosing

Adult

Immunocompetent population: 800 mg PO 5 times/d for 7 d

Immunocompromised/immunosuppressed population: 10 mg/kg IV, infused at a constant rate over 1 h, q8h for 7 d

Patients who are obese: Base dose on ideal body weight

Pediatric

Immunocompetent population: 20 mg/kg PO qid; not to exceed 800 mg/dose

Immunocompromised/immunosuppressed population:

<12 years: 20 mg/kg IV, infused at a constant rate over 1 h, q8h for 7 d

>12 years: Administer as in adults

Interactions

Concomitant use of probenecid or zidovudine prolongs half-life and increases CNS toxicity

Contraindications

Documented hypersensitivity

Precautions

Pregnancy

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Caution in renal failure or when using nephrotoxic drugs (adjust dose); caution in pregnant and breastfeeding women (benefits must outweigh risks); in IV administration, must give adequate hydration and should infuse over 1 h to reduce risk of renal damage (precipitation of acyclovir crystals in renal tubules may occur, possibly leading to acute renal failure)

Famciclovir (Famvir)

Prodrug that, when biotransformed into active metabolite, penciclovir, may inhibit viral DNA synthesis/replication.

Dosing**Adult**

500 mg PO q8h for 7 d

Pediatric

Not established

Interactions

Coadministration of probenecid or cimetidine may increase toxicity; coadministration increases bioavailability of digoxin

Contraindications

Documented hypersensitivity

Precautions**Pregnancy**

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Caution in renal failure or coadministration of nephrotoxic drugs

Valacyclovir (Valtrex)

Prodrug rapidly converted to the active drug acyclovir. More expensive but has a more convenient dosing regimen than acyclovir.

Dosing**Adult**

1000 mg PO q8h for 7 d

Pediatric

Not established

Interactions

Probenecid, zidovudine, or cimetidine coadministration prolongs half-life and increases CNS toxicity

Contraindications

Documented hypersensitivity

Precautions**Pregnancy**

B - Fetal risk not confirmed in studies in humans but has been shown in some studies in animals

Precautions

Caution in renal failure and coadministration of nephrotoxic drugs; associated with onset of hemolytic uremic syndrome

Follow-up

Complications

Secondary bacterial infection of skin lesions, manifesting as impetigo, cellulitis, and erysipelas, is the most common complication in healthy children.^[21] Staphylococci and streptococci are the most commonly implicated bacterial pathogens. Bacterial superinfection may predispose to scarring. Localized bacterial superinfection rarely may manifest in septicemia, culminating in secondary bacterial pneumonia, otitis media, or necrotizing fasciitis.

Disseminated primary varicella infection, usually seen in the immunocompromised or adult populations, may have high morbidity. Ninety percent of cases of varicella pneumonia occur in the adult population. Rarer complications of disseminated chickenpox also include myocarditis, gangrene, hepatitis, and glomerulonephritis.^[22]

Central nervous system complications of primary varicella-zoster virus (VZV) infection may occur, albeit very rarely. Reye syndrome, Guillain-Barré syndrome, acute cerebellar ataxia, and encephalitis have all been documented to occur after VZV infection.

Thrombocytopenia and purpura secondary to VZV infection have been described in more than 100 patients. Hemorrhagic complications are more common in the immunocompromised or immunosuppressed populations, although healthy children and adults have been affected. Five major clinical syndromes have been described: febrile purpura, malignant chickenpox with purpura, postinfectious purpura, purpura fulminans, and anaphylactoid purpura. These syndromes have variable courses, with febrile purpura being the most benign of the syndromes and having an uncomplicated outcome. In contrast, malignant chickenpox with purpura is a grave clinical condition that has a mortality rate of greater than 70%. The etiology of these hemorrhagic chickenpox syndromes is not known, although an autoimmune pathophysiologic mechanism has been implicated.

Prognosis

Chickenpox affecting a healthy child is usually a self-limited disease. Increased morbidity occurs in adult and immunocompromised populations.

Patient Education

Instruct parents to trim children's fingernails to minimize skin damage from scratching and the associated complications of bacterial superinfection. Advise parents not to use aspirin for fever control because the development of Reye syndrome is associated with salicylate administration in children with chickenpox.

For excellent patient education resources, visit eMedicine's Bacterial and Viral Infections Center. Also, see eMedicine's patient education articles Chickenpox and Skin Rashes in Children.

Miscellaneous

Medicolegal Pitfalls

Note the following medicolegal pitfalls for chickenpox:

- Failure to quickly diagnose and treat primary varicella in immunocompromised, immunosuppressed, or geriatric populations (The incidence of severe disease, with increasing morbidity and mortality, occurs in these populations.)
- Failure to include primary varicella in the differential diagnosis when considering the etiology of a vesicular or bullous eruption in immunocompromised, immunosuppressed, or geriatric populations
- Failure to quickly recognize primary varicella infection in patients who are hospitalized and to limit the spread of the virus to other patients and hospital workers
- Failure to diagnose and treat primary varicella in pregnant women who develop the characteristic varicella rash very close to delivery, and failure to immediately consult a neonatologist and a pediatric infectious disease specialist for the newborn's treatment

Special Concerns

In utero infection with VZV is a concern.^[2,3] Primary maternal chickenpox during pregnancy may produce latency of VZV in the dorsal root ganglia of the fetus. These children may remain asymptomatic, or they may develop zoster at a young age without previous history of primary chickenpox infection. Maternal chickenpox infection in early to mid-pregnancy is estimated to have a 1-2% risk of causing the congenital varicella syndrome, which is characterized by limb hypoplasia, muscular atrophy, skin scarring, cortical atrophy, microcephaly, cataract formation, and rudimentary digits.

Peripartum infection of the fetus before sufficient maternal antibody has crossed the placenta to confer transient passive immunity to the fetus (ie, when the mother experiences onset of chickenpox <5 d before delivery or within 2 d after delivery) often results in severe disseminated varicella in the newborn infant, which has a substantial mortality rate.

Prepartum infection with onset of chickenpox in the mother 5 or more days previous to delivery allows transplacental passage of sufficient maternal IgG antibody to protect the newborn from severe, disseminated varicella infection.

Multimedia



Media file 1: Dewdrop on rose petal characteristic vesicle of chickenpox. Reprinted with permission from *Cutis* 65: 355, 2000.



Media file 2: Vesicular eruption on the trunk demonstrating papules, vesicles, and crusts. Reprinted

with permission from *Cutis* 65: 355, 2000.

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