Chicken pox

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Varicella-zoster virus (VZV) causes primary, latent, and recurrent infections. The primary infection is manifested as varicella (**chickenpox**) and results in establishment of a lifelong latent infection of sensory ganglion neurons. Reactivation of the latent infection causes herpes zoster (**shingles**).

Etiology. VZV is a neurotropic human herpesvirus with similarities to herpes simplex virus.

Epidemiology . Varicella is a more serious disease with higher rates of complications and deaths among infants, adults, and immunocompromised persons. Patients with varicella are contagious from 24 to 48 hr before the rash appears and until vesicles are crusted, usually 3–7 days after onset of rash. Susceptible children may also acquire varicella after close, direct contact with adults or children who have herpes zoster.

Pathogenesis . VZV is transmitted in respiratory secretions and in the fluid of skin lesions either by airborne spread or through direct contact. Primary infection (varicella) results from inoculation of the virus onto the mucosa of the upper respiratory tract and tonsillar lymphoid tissue. During the early part of the 10–21 day incubation period, virus replicates in the local lymphoid tissue followed by a brief subclinical viremia that spreads the virus to the reticuloendothelial system. Widespread cutaneous lesions occur during a **2nd viremic** phase that lasts 3–7 days. Peripheral blood mononuclear cells carry infectious virus, generating new crops of vesicles during this period of viremia. Host immune responses limit viral replication and facilitate recovery from infection. In the immunocompromised child, the failure of immune responses, especially cell-mediated immune responses, results in continued viral replication that may result in disseminated infection with resultant complications in the lungs, liver, brain, and other organs. Subsequent **reactivation** of latent virus causes **herpes zoster,** a vesicular rash that usually is dermatomal in distribution.. Suppression of cell-mediated immunity to VZV correlates with an increased risk for VZV reactivation as herpes zoster.

Clinical manifestation.

Varicella is an acute febrile rash illness that was common in children prior to the universal childhood vaccination program. It has variable severity but is usually self-limited. It may be associated with severe complications, including bacterial superinfection, pneumonia, encephalitis, bleeding disorders, congenital infection, and life-threatening perinatal infection. Herpes zoster, uncommon in children, causes localized cutaneous symptoms, but may disseminate in immunocompromised patients.The illness usually begins 14–16 days after exposure, although the incubation period can range from 10 to 21 days. Prodromal symptoms may be present, particularly in older children and adults. Fever, malaise, anorexia, headache, and occasionally mild abdominal pain may occur 24–48 hr before the rash appears. Temperature elevation is usually moderate, usually from 100 to 102°F but may be as high as 106°F;

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fever and other systemic symptoms persist during the 1st 2–4 days after the onset of the rash.Varicella lesions often appear first on the scalp, face, or trunk. The initial exanthem consists of intensely pruritic erythematous macules that evolve through the papular stage to form clear, fluid-filled vesicles. Clouding and umbilication of the lesions begin in 24–48 hr. While the initial lesions are crusting, new crops form on the trunk and then the extremities; the simultaneous presence of lesions in various stages of evolution is characteristic of varicella. The distribution of the rash is predominantly central or centripetal, in contrast to smallpox, where the rash is more prominent on the face and distal extremities. Ulcerative lesions involving the mucosa of oropharynx and vagina are also common; many children have vesicular lesions on the eyelids and conjunctivae, but corneal involvement and serious ocular disease is rare. The exanthem may be much more extensive in children with skin disorders, such as eczema or recent sunburn. Hypopigmentation or hyperpigmentation of lesion sites persists for days to weeks in some children, but severe scarring is unusual unless the lesions were secondarily infected.

The **differential diagnosis** of varicella includes vesicular rashes caused by other infectious agents, such as herpes simplex virus, enterovirus, monkey pox, rickettsial pox, or *S. aureus;* drug reactions; contact dermatitis; and insect bites. Severe varicella was the most common illness confused with smallpox before the eradication of smallpox.

Neonatal Chickenpox.

Newborns have particularly high mortality in the circumstances of a susceptible mother contracting varicella around the time of delivery. Infants whose mothers develop varicella in the period from 5 days prior to delivery to 2 days afterward are at high risk for severe varicella. The infant acquires the infection transplacentally as a result of maternal viremia, which may occur up to 48 hr prior to the maternal rash.. Because perinatally acquired varicella may be life threatening, the infant should be treated with acyclovir (10 mg/kg every 8 hr IV) when lesions develop. Infants with community-acquired chickenpox who develop severe varicella, especially those who develop a complication such as pneumonia, hepatitis, or encephalitis, should also receive treatment with intravenous acyclovir (10 mg/kg every 8 hr IV).

Congenital Varicella Syndrome.

When pregnant women contract chickenpox early in pregnancy, experts estimate that as many as 25% of the fetuses may become infected. Fortunately, clinically apparent disease in the infant is uncommon: up to 2% of fetuses whose mothers had varicella in the 1st 20 wk of pregnancy may demonstrate a VZV embryopathy. Many infants with severe manifestations of congenital varicella syndrome (atrophy and scarring of a limb) have significant neurologic deficiencies, whereas those with only isolated stigmata, amenable to treatment, develop normally throughout childhood. Infants with neonatal chickenpox who receive prompt antiviral therapy have an excellent prognosis.

Diagnosis;

Laboratory evaluation has not been considered necessary for the diagnosis or management of healthy children with varicella or herpes zoster. Unusual or very severe varicella in otherwise immunocompetent individuals must be distinguished from smallpox, which may occur following deliberate release of smallpox virus A suspected case of smallpox should be reported immediately to the local and state health departments

Treatment;

Antiviral treatment modifies the course of both varicella and herpes zoster. Any patient who has signs of disseminated VZV, including pneumonia, severe hepatitis, thrombocytopenia, or encephalitis, should receive immediate treatment. Intravenous acyclovir (500 mg/m^2 every 8 hr IV) therapy initiated within 72 hr of development of initial symptoms decreases the likelihood of progressive varicella and visceral dissemination in high-risk patients. Treatment is continued for 7 days or until no new lesions have appeared for 48 hr.

Complications;

The complications of VZV infection occur with varicella, or with reactivation of infection, more commonly in immunocompromised patients. In the otherwise healthy child, **mild varicella hepatitis is relatively common** but rarely clinically symptomatic. **Mild thrombocytopenia** occurs in 1–2% of children with varicella and may be associated with **transient petechiae**. **Purpura, hemorrhagic vesicles, hematuria, and gastrointestinal bleeding** are rare complications that may have serious consequences. **Cerebellar ataxia** occurs in 1 in every 4,000 cases. Other complications of varicella, some of them rare, include **encephalitis, pneumonia, nephritis, nephrotic syndrome, hemolytic-uremic syndrome, arthritis, myocarditis, pericarditis, pancreatitis, and orchitis**.

Secondary bacterial infections of the skin, usually caused by group A streptococci and *S. aureus,* may occur in up to 5% of children with varicella. **Encephalitis** (1/50,000 cases of varicella) and acute cerebellar ataxia (¼,000 cases of varicella) are well-described neurologic complications of varicella; morbidity from CNS complications is highest among patients younger than 5 yr or older than 20 yr. **Reye syndrome of encephalopathy and hepatic dysfunction** associated with varicella has become rare since salicylates are no longer routinely used as antipyretics .

Varicella pneumonia is a severe complication in adults and other high-risk populations, but pneumonia may also complicate varicella in young children.

Prognosis;

The most common complications among people who died from varicella were **pneumonia, CNS complications, secondary infections, and hemorrhagic** conditions. **Neuritis with herpes zoster** should be managed with appropriate analgesics. Postherpetic neuralgia can be a severe problem in adults and may persist for months, requiring care by a specialist in pain management.

Prevention;

VZV transmission is difficult to prevent because the infection is contagious for 24–48 hr before the rash appears.. Varicella is a vaccine-preventable disease. Live virus varicella vaccine is available as a monovalent vaccine and is also available in combination with measles, mumps, and rubella (MMRV) vaccines.

Postexposure Prophylaxis.

Vaccine given to normal children within 3–5 days after exposure is effective in preventing or modifying varicella, especially in a household setting where exposure is very likely to result in infection. Varicella vaccine is now recommended for postexposure use, for outbreak control. Oral acyclovir administered late in the incubation period may modify subsequent varicella in the normal child; however, its use in this manner is not recommended until it can be further evaluated.