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Varicella in Children

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Abstract

Varicella (chickenpox) and herpes zoster are caused by the varicella-zoster virus (VZV) (shingles). Varicella, or chickenpox, is caused by a primary VZV infection, which causes a diffuse vesicular rash. The Varicella Zoster Virus (VZV) is always in circulation, and the illness burden rises throughout the spring season. Clinical diagnosis is difficult due to a wide range of clinical presentations ranging from vesicular rash to hemorrhage or neurological sequelae.

Keywords: Varicella; chickenpox; shingles; varicella zoster virus; herpes zoster.

INTRODUCTION

The Varicella Zoster Virus (VZV) is responsible for two highly contagious diseases: varicella (chickenpox) and herpes zoster (Shingles). VZV is a highly contagious virus that has only been found in humans until now. The virus's infectivity rate is greater than 85 to 90%, implying that VZV transmits easily among contacts, increasing the illness burden (Singh *et al.*, 2011). Nasopharyngeal secretions, cutaneous vesicular and pustular lesions are the main sources of viral transmission. VZV infection is characterized by viremia and viruria. During the months of January to May, there was a continual presence and circulation of VZV in the community, as well as an increase in the number of cases. In these, there are outbreaks of infection. Though Varicella is most commonly associated with children, it is shown that young adults in tropical countries such as India are more susceptible to infection. VZV seroprevalence is at 16 percent in children aged 1-4 years, 54 percent in children aged 5 to 14, and 72 percent in those aged 15-25 years (Lee, 1998).

In most cases, the incubation time for VZV infection is 1 to 3 weeks. From 3 days after the rash appears to scabbing, there is the most contagiosity (Heininger and Seward, 2006). Varicella presents a wide range of clinical signs, including high grade fever with vesicular rash, sore throat, cough, diarrhea, and, in severe cases, bleeding features. The disease is usually harmless, with less than 1% of cases

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resulting in death (Singh *et al.*, 2011). Patients with bleeding or respiratory issues have high mortality rate, and it's easy to mix it with other viral hemorrhagic fever or respiratory virus illnesses. If the expectant woman contracts VZV during the third trimester of pregnancy, there is always the danger of Congenital Varicella Syndrome (CVS). Congenital Varicella Syndrome is characterized by scarring lesions (cicatricle) and neurological and ocular deficits. CVS is characterized by limb shortening and hypotonia (Singh *et al.*, 2011; Gnann, 2002; Ojah et al., 2016; Lee, 1998).

EPIDEMIOLOGY

Secondary home attack rates of >90% in vulnerable persons make it highly contagious. Contact with aerosolized droplets from an infected person's nasopharyngeal secretions or direct cutaneous contact with vesicle fluid from skin lesions results in transmission. The incubation period is between 14 and 16 days (range 10 to 21 days). Infectivity lasts for 48 hours before the rash appears until the skin sores have entirely hardened. Primary infection in children is a relatively minor illness. Neonatal, teenagers, adults, pregnant women, and immuno compromised people are all susceptible to severe disease. Secondary cases are more serious than primary cases. Herpes zoster is caused by the reactivation of VZV, which is dormant in the sensory ganglia. Secondary cases in immunocompetent people are uncommon, although they do happen. Although it is less prevalent than measles, it is nonetheless highly contagious, with secondary infection rates of more than 85% in vulnerable household contacts.[8,9] However, immunologic evidence suggests that VZV subclinical reinfection is common.[9] Varicella has a distinct seasonal pattern, peaking in the winter and spring, or during the cool, dry season.[11,12] Outbreaks are most common in places where children assemble, such as daycare centers and schools, but they can also happen in other age groups and contexts, such as hospitals, institutionalized people's facilities, refugee camps, and military and correctional facilities.[12,13] In 1% of afflicted pregnancies, varicella contracted during the first two trimesters of pregnancy causes serious congenital abnormalities in the newborn.[14]

DIAGNOSIS

Clinical: Within fifteen days of exposure, clinical signs include a prodrome of fever, malaise, or pharyngitis, and loss of appetite[1], followed by a widespread vesicular rash that appears within 24 hours. Vesicular rash is typically pruritic and manifests itself over many days in successive crops. Varicella patients usually have lesions on their face, trunk, and extremities in various phases of development. In normal hosts, new vesicle development stops after four days, and lesions crust completely by day six. Within one to two weeks, the crusts break off, leaving transient hypopigmentation. The immunosuppressed can develop crops of vesicles over weeks, massive and hemorrhagic skin lesions, pneumonia, or extensive disease with disseminated intravascular coagulation (underlying malignancy, steroid use or immunosuppressive medication, HIV infection, or solid organ transplantation). Zoster without rash (also known as zoster sine herpete, sometimes either with facial palsy[15], meningitis, stroke, myelitis, and enteric (gastrointestinal) infections are all examples of VZV infection excluding rash

LAB FINDINGS

Chickenpox is diagnosed mostly through signs and symptoms, with typical early symptoms followed by a distinctive rash. Examining the fluid within the vesicles of the rash or testing blood for indications of an acute immune response can help confirm the diagnosis.

A Tzanck smear or a direct fluorescent antibody test can be used to assess vesicular fluid. The fluid can also be "cultured," which involves attempting to develop the virus from a sample of the fluid. Blood tests can be used to determine if a person's immune system is responding to an acute infection (IgM) or to a past infection and subsequent immunity (IgG).

Ultrasound can be used to diagnose fetal varicella infection during pregnancy, although it's best to wait 5 weeks after the main maternal infection. A PCR (DNA) test of the mother's amniotic fluid is also possible, albeit the risk of spontaneous abortion from the amniocentesis technique is higher than the risk of fetal varicella syndrome developing in the baby.

The following techniques are now most useful for laboratory diagnosis of VZV infection: If neurological symptoms or signs are present, PCR on material from skin vesicles (provided as swabs, fluid, or scabs, saliva, and cerebrospinal fluid.

VZV antigens can also be detected by direct immunofluorescence from vesicles; however, it is less sensitive than PCR110. In symptomatic individuals with or without rash, viral DNA can be found in saliva during varicella and zoster, and this approach is diagnostically useful and specific, VZV

resistance to acyclovir can be determined using PCR, restriction enzyme digestion, and sequencing of particular regions of the viral genome.

COMPLICATIONS

Immunocompromised people are more susceptible. Bacterial super infection of the skin or soft tissues, usually caused by streptococci or staphylococci. Encephalitis, Reye syndrome, transitory focal deficits, aseptic meningitis, transverse myelitis, vasculitis, and hemiplegia are all neurological consequences.

Encephalitis: occurs near the end of the first week of exanthema, however central nervous system involvement may occur before the rash. Acute cerebellar ataxia is the most common kind, affecting roughly 1 in 4000 varicella infections in children under the age of 15. It has a short duration and is usually followed by complete recovery.

Diffuse encephalitis is more common in young children. Delirium, convulsions, and localized neurologic abnormalities are some of the symptoms. According to 10% of survivors, up to 15% of survivors sustained long term neurologic effects.

Pneumonia develops one to six days after the rash appears.

Hemoptysis, tachypnea, dyspnea, and a persistent dry cough Growing hypoxia obstructs gas exchange. Chest radiographs show diffuse bilateral infiltrates that are nodular in character (early stage) but can calcify later.

Hepatitis is characterized by a high fever as well as significant stomach or back pain. DIC and gastrointestinal bleeding are prevalent in fulminant liver failure.

DIFFERENTIAL DIAGNOSIS

Herpes simplex, entero-viral infections, insect bites and drug reactions.

TREATMENT

Antihistamines are used to treat pruritis as a symptom. While acetaminophen is used to treat fever, aspirin can trigger Reye syndrome when used during a viral infection. Secondary infections require antibacterials. Antiviral treatment. Children over the age of 12, those with chronic cutaneous or pulmonary problems, or those on steroid or salicylate therapy, as well as secondary contacts, should be treated.

Dosage: For uncomplicated varicella, oral acyclovir 20 mg/kg PO (up to 800 mg each dose) four times daily for five days lowers the duration of the rash by one day and the lesions by 25%.

For immunosuppressed children with difficult or uncomplicated varicella or those with disseminated illness, such as pneumonia or encephalitis, intravenous acyclovir (10 mg/kg every eight hours) for 14 to 21 days.

PREVENTION

Live attenuated varicella vaccine and VZIG or IVIG with oral acyclovir for post-exposure prophylaxis.

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