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Varicella-Zoster Virus (Chickenpox): Nursing, Laboratory, and Epidemiological Perspectives

Abbas Mohammed Alnami [1], Ahmed Ibrahim Hussein Alnajie [1], Mohammed Ahmed AMohammed Okayri [2], Layla Yahya Mansour Matary [1], Kamlah Mortada Ahmad Alhazm [1], Fatimah Ahmad Abdo Suraihi [3], Nouf Ali Ahmed Nahari [4], Hussain Ebrahim Tawashi [3], Rajaa Ibrahim Ahmed Alakrash [3], Muteeb Mohamed Alasiri [5], Abdulelah Omar Mousa Sharwani [6], Maram Khalid Alatwan [7]

(1) Al-Alia Primary Health Care Center, Ministry of Health, Saudi Arabia,
(2) Manshaba Health Center, Ministry of Health, Saudi Arabia,
(3) Al-Alia Health Center, Ministry of Health, Saudi Arabia,
(4) fsuraihy@moh.gov.sa, Saudi Arabia,
(5) king salman hospital, Ministry of Health, Saudi Arabia,
(6) Ministry of Health, Saudi Arabia,
(7) Alhasa, Ministry of Health, Saudi Arabia

Abstract

Background: Varicella (chickenpox) is a highly contagious viral disease caused by the varicella-zoster virus (VZV), characterized by a generalized vesicular rash and systemic symptoms. Despite widespread vaccination, varicella continues to pose clinical and public health challenges, particularly among high-risk populations such as adults, pregnant women, neonates, and immunocompromised individuals.

Aim: This article aims to comprehensively review varicella from nursing, laboratory, and epidemiological perspectives, emphasizing etiology, transmission, clinical features, diagnosis, management, prevention, and public health implications.

Methods: A descriptive narrative review approach was used, synthesizing established clinical, epidemiological, and laboratory data on VZV infection. Key aspects reviewed include pathophysiology, histopathology, clinical presentation, diagnostic modalities, treatment strategies, complications, and prevention through vaccination.

Results: Varicella is primarily transmitted via airborne droplets and direct contact with vesicular fluid. While generally self-limiting in children, severe disease and complications such as pneumonia, encephalitis, and secondary bacterial infections are more common in adults and immunocompromised hosts. Polymerase chain reaction testing is the most sensitive diagnostic method. Antiviral therapy reduces disease severity in high-risk patients, and vaccination has significantly decreased incidence, hospitalizations, and mortality.

Conclusion: Varicella remains a clinically relevant disease requiring ongoing surveillance, vaccination advocacy, early diagnosis, and interprofessional collaboration to minimize complications and transmission.

Keywords: Varicella, Chickenpox, Varicella-Zoster Virus, Epidemiology, Vaccination, Nursing Care

Introduction

Chickenpox, clinically termed varicella, is a highly contagious viral illness caused by the varicella-zoster virus, a member of the Herpesviridae family. Varicella-zoster virus produces two distinct clinical syndromes during the human lifespan. Primary infection manifests chickenpox, whereas reactivation of latent virus within sensory nerve ganglia results in herpes zoster, commonly known as shingles. This dual disease pattern reflects the virus's neurotropic nature and its ability to establish lifelong latency following initial exposure. Varicella remains a disease of major clinical, nursing, laboratory, and public health relevance despite the availability of effective vaccination programs. The differentiation of chickenpox from other vesicular illnesses developed gradually through historical observation. In 1767, Heberden clearly distinguished chickenpox from smallpox, a crucial advancement in infectious disease classification. The etymology of the term "chickenpox" has been debated, with proposed origins including the French word *chiche-pois*, referencing the chickpea-like size of the vesicles, and the Old English term *gigan*, meaning "to itch," which reflects the intense pruritus associated with the rash. A pivotal milestone in understanding varicella occurred in 1888, when von Bokay observed the development of chickenpox in children following exposure to individuals with herpes zoster. Subsequent experimental and clinical observations by

*Corresponding author e-mail: sdfah2525@gmail.com (Abbas Mohammed Alnami).

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Kundratitz in 1922 and Bruusgaard in 1932 confirmed that both conditions were caused by the same viral agent, firmly establishing their shared etiology [1][2][3].

Varicella is transmitted primarily through airborne droplets expelled during coughing or sneezing, as well as through direct contact with vesicular fluid from skin lesions. The virus spreads efficiently in community and household settings, particularly among unvaccinated populations. Following exposure, the incubation period ranges from 10 to 21 days, with an average of approximately 14 days. The prodromal phase may include low-grade fever, malaise, headache, and pharyngitis, followed by the characteristic vesicular rash. Lesions typically appear first on the face, scalp, chest, and back before spreading to the extremities. The rash progresses through stages of macules, papules, vesicles, and crusts, often with lesions present in multiple stages simultaneously. Patients are considered infectious from one to two days before rash onset until all lesions have crusted. Although chickenpox is often self-limiting in children, it can lead to serious complications. These include secondary bacterial skin infections, pneumonia, encephalitis, cerebellar ataxia, and, in rare cases, death. Adults, pregnant individuals, neonates, and immunocompromised patients experience higher morbidity and mortality rates. From a nursing and epidemiological perspective, early identification of high-risk populations and prompt infection control measures are essential to reduce transmission and adverse outcomes. Diagnosis of chickenpox is frequently clinical, based on the typical rash and associated systemic symptoms. Laboratory confirmation may be achieved through polymerase chain reaction testing of vesicular fluid or crusted lesions, which provides high sensitivity and specificity. Serologic testing for varicella-specific antibodies may be used to assess immune status, particularly in healthcare workers, pregnant patients, or immunocompromised individuals. Reinfection with varicella is uncommon and generally mild, reflecting partial immune protection following primary exposure [1][2][3].

The introduction of the live attenuated varicella vaccine in 1995 marked a major public health achievement. Widespread immunization has led to a dramatic reduction in disease incidence, complications, hospitalizations, and mortality. Vaccine effectiveness ranges from 70% to 90% for preventing infection and exceeds 95% for preventing severe disease. Routine childhood immunization is now standard practice in many countries, and post-exposure vaccination within three days may reduce disease severity or prevent infection altogether. From a nursing, laboratory, and epidemiological standpoint, continued surveillance, vaccination advocacy, and education remain central to sustaining control of varicella and minimizing its clinical and societal burden.[1][2][3]

Etiology

Chickenpox, clinically referred to as varicella, is caused by the varicella-zoster virus, a DNA virus belonging to the Herpesviridae family with universal geographic distribution. A defining characteristic of this virus, consistent with other herpesviruses, is its ability to establish lifelong latency following primary infection. After resolution of the initial illness, the virus persists in a dormant state within sensory nerve ganglia and may subsequently reactivate later in life, producing herpes zoster. This dual pathogenic behavior underpins both the acute and long-term clinical relevance of varicella-zoster virus infection and explains its continued importance in clinical medicine and public health.[4] Primary infection with varicella-zoster virus occurs through inhalation of aerosolized respiratory droplets expelled by infected individuals or through direct contact with vesicular fluid from skin lesions. The virus is highly contagious, and transmission efficiency is particularly high in enclosed environments such as households, schools, and healthcare settings. Following inhalation, viral replication begins in the mucosal epithelium of the upper respiratory tract. This localized replication is followed by an initial viremia occurring approximately two to six days after exposure, during which the virus disseminates through the bloodstream to reticuloendothelial tissues. A secondary viremia typically develops between ten and twelve days after infection, coinciding with widespread viral replication and the appearance of the characteristic vesicular rash on the skin and mucous membranes.

The host immune response to varicella-zoster virus involves both humoral and cellular components. During the acute phase of infection, immunoglobulin A, immunoglobulin M, and immunoglobulin G antibodies are produced. Immunoglobulin M antibodies appear early and indicate recent infection, while immunoglobulin A contributes to mucosal immunity. Immunoglobulin G antibodies play a critical role in long-term immune protection and are generally responsible for lifelong immunity following primary infection. Despite this robust immune response, the virus is not completely eradicated from the body. Instead, it migrates along sensory nerve fibers and establishes latency in dorsal root ganglia or cranial nerve ganglia, where it remains clinically silent until immune surveillance declines, allowing viral reactivation. Although chickenpox is often mild in childhood, several factors increase the risk of severe disease, particularly among adolescents and adults. One of the most significant risk factors is systemic corticosteroid therapy. Administration of corticosteroids at doses equivalent to one to two milligrams per kilogram per day of prednisolone for two weeks or longer has been strongly associated with severe and potentially fatal varicella. Importantly, even short courses of corticosteroids at these dosages, when given shortly before or during the incubation period, can markedly impair cellular immune responses and predispose individuals to disseminated infection.[5] This risk highlights the critical role of cell-mediated immunity in controlling varicella-zoster virus replication.

Immunocompromised states represent another major determinant of disease severity. Individuals with malignancies, those receiving antineoplastic agents, patients with human immunodeficiency virus infection, and those with congenital or acquired immunodeficiency disorders are at substantially increased risk. Cellular immunodeficiency, rather than humoral immune deficiency, is particularly associated with severe varicella, as T-cell–mediated responses are essential for limiting viral spread. In these populations, varicella may present with extensive cutaneous involvement, visceral dissemination, pneumonia, hepatitis, or encephalitis, often requiring aggressive antiviral therapy and supportive care. Pregnancy constitutes a unique and clinically significant risk factor for severe varicella infection. Pregnant individuals are more susceptible to complications, particularly varicella pneumonia, which carries increased morbidity and mortality. In addition to maternal risks, viremia during pregnancy can result in transplacental transmission of the virus to the fetus. Neonatal varicella is most severe when maternal infection occurs within a narrow window spanning five days before to two days after delivery, as the neonate lacks sufficient time to acquire protective maternal antibodies.[6] The neonatal period, particularly the first month of life, represents a phase of heightened vulnerability, especially when the mother is seronegative for varicella-zoster virus. Prematurity further compounds neonatal risk. Infants delivered before twenty-eight weeks of gestation are less likely to benefit from adequate transplacental transfer of immunoglobulin G antibodies, which predominantly occurs later in pregnancy. Consequently, these neonates may experience more severe manifestations of varicella if exposed. Collectively, these etiological and host-related factors underscore the complex interaction between viral biology and immune competence in determining the clinical severity of chickenpox and reinforce the importance of prevention, vaccination, and early recognition in high-risk populations.[4][5][6]

Epidemiology

Varicella-zoster virus infection demonstrates a truly global epidemiological distribution and remains one of the most prevalent viral infections affecting humans. Serological studies indicate that approximately 98% of adults worldwide possess antibodies against varicella-zoster virus, reflecting either prior natural infection or vaccination. Varicella occurs in virtually all geographic regions and across diverse socioeconomic settings, underscoring its high transmissibility and efficiency of spread. Despite being commonly regarded as a childhood illness, varicella continues to impose a measurable global burden, accounting for an estimated 7000 deaths annually. These fatalities predominantly occur among adults, immunocompromised individuals, pregnant women, and neonates, in whom disease severity is significantly amplified. The epidemiology of varicella exhibits marked seasonal variation, with peak incidence typically observed during the winter and spring months in temperate climates. This seasonal clustering is thought to be related to increased indoor crowding and enhanced viral stability in cooler environments, which facilitate airborne transmission. In the United States, prior to widespread immunization, varicella was nearly universal in childhood. Even in the post-vaccine era, varicella-zoster virus remains a notable cause of morbidity, accounting for more than 9000 hospitalizations annually. Historically, the highest disease prevalence was observed among children aged 4 to 10 years, reflecting early exposure in school and household settings where close contact promotes efficient transmission [7].

The introduction of the live attenuated varicella vaccine in 1995 fundamentally altered the epidemiological landscape of varicella in the United States and other countries that adopted routine immunization programs. Following vaccine implementation, the overall incidence of varicella declined by approximately 85%, providing compelling evidence of both direct protection and herd immunity. This population-level immunity reduced virus circulation, thereby protecting vulnerable individuals who were either ineligible for vaccination or had suboptimal immune responses. As disease incidence declined in younger children, a notable shift in the age distribution of varicella cases was observed. The peak incidence moved from children aged 5 to 9 years toward older children and adolescents aged 10 to 14 years. This epidemiological shift revealed an important limitation of the single-dose vaccination strategy. An increased frequency of breakthrough varicella cases, defined as infection occurring despite prior vaccination, was documented among older vaccinated children. Although these breakthrough infections were generally milder than natural disease, they raised concerns regarding waning immunity and ongoing transmission within partially immune populations. In response to these findings, a second dose of the varicella vaccine was incorporated into the routine childhood immunization schedule in 2006, resulting in further reductions in disease incidence, outbreak frequency, and severity.[7]

Before the availability of vaccination, varicella was almost universally symptomatic, with approximately 90% of children in the United States experiencing infection by the age of 10 years. The near inevitability of childhood infection contributed to the widespread perception of varicella as a benign disease. However, epidemiological data consistently demonstrated that complications, hospitalizations, and deaths occurred across all age groups, with severity increasing substantially with age. Adults were disproportionately affected by complications such as pneumonia, encephalitis, and secondary bacterial infections, emphasizing the public health importance of prevention strategies. Herpes zoster represents the reactivation of latent varicella-zoster virus and constitutes a distinct but epidemiologically related clinical entity. Reactivation occurs in approximately 20% of otherwise healthy adults during their lifetime, while the incidence rises to nearly 50% among immunocompromised individuals. In these populations, herpes zoster is associated with significant morbidity, including postherpetic neuralgia, ophthalmic involvement, and disseminated disease. Mortality and long-term disability are considerably higher in immunocompromised patients, reinforcing the link between immune function and varicella-zoster virus epidemiology [7][8][9].

Transmission dynamics within households further influence disease severity and epidemiological patterns. Secondary cases among household contacts tend to exhibit more severe clinical manifestations than index cases, likely due to higher viral inoculum and prolonged exposure. Geographic and climatic factors also shape epidemiological trends. In tropical regions, varicella infection often occurs later in life compared to temperate climates, resulting in a higher proportion of adolescent and adult cases. This delayed exposure contributes to increased disease severity and complication rates in these settings. Notably, recent reports have documented varicella infection in very elderly individuals, including an 81-year-old patient, highlighting that susceptibility and clinical impact extend across the entire lifespan.[8][9][10][11] Collectively, the epidemiology of varicella-zoster virus reflects the interplay between viral biology, host immunity, vaccination practices, and environmental factors. Ongoing surveillance and sustained immunization efforts remain essential to further reduce disease burden, prevent outbreaks, and protect high-risk populations worldwide.

Pathophysiology

Varicella is caused by the varicella-zoster virus, a member of the Alphaherpesvirinae subfamily within the Herpesviridae family. Like other human herpesviruses, VZV is an enveloped deoxyribonucleic acid virus characterized by its ability to establish lifelong latency following primary infection. The pathophysiological process of varicella begins when the virus enters the host through the respiratory tract, most commonly via the conjunctival or upper respiratory mucosa. This mode of entry explains the high transmissibility of the virus and its efficient spread in community and household settings. Following initial exposure, VZV establishes local infection within the epithelial cells of the upper respiratory tract. Viral replication occurs early in the regional lymphoid tissue, particularly within lymph nodes draining the site of entry. This phase typically develops within two to four days after infection. The virus then gains access to the bloodstream, leading to a primary viremia approximately four to six days after exposure. During this stage, VZV disseminates to components of the reticuloendothelial system, including the spleen, liver, and other lymphoid tissues, where further viral replication occurs. After an incubation period of approximately one week, a secondary viremia develops, representing a critical step in the pathogenesis of varicella. This secondary viremia results in widespread distribution of the virus to target organs, including the skin and mucous membranes, and gives rise to the characteristic vesicular rash that defines the clinical presentation of chickenpox. The dissemination of the virus to the skin leads to infection of epidermal cells, causing ballooning degeneration, intracellular edema, and the formation of thin-walled vesicles filled with infectious viral particles. Importantly, viral shedding from the respiratory tract also occurs during this phase, facilitating transmission of the virus one to two days before the appearance of the rash. This explains why varicella is often spread before individuals are aware they are infected [12].

During the viremic phases, VZV may also invade internal organs, which accounts for the systemic manifestations and complications of varicella. In some patients, particularly adults, pregnant women, and immunocompromised individuals, viral dissemination can involve the central nervous system, lungs, or liver, leading to encephalitis, pneumonia, or hepatitis.[12] These complications reflect both direct viral cytopathic effects and the host inflammatory response to infection. The host immune response plays a central role in controlling VZV infection and shaping long-term outcomes. Varicella induces both humoral and cell-mediated immune responses, which together contribute to viral clearance and the development of durable immunity. B lymphocytes generate virus-specific immunoglobulins, including IgM, IgA, and IgG. IgM antibodies appear early during acute infection, while IgA contributes to mucosal immunity. IgG antibodies persist for life and are responsible for long-term protection against reinfection. Although reinfections with VZV can occur, they are typically subclinical or very mild in immunocompetent individuals due to preexisting immunity. Cell-mediated immunity is particularly critical in limiting viral replication and determining disease severity. T lymphocytes, especially CD4+ and CD8+ T cells, are essential for controlling primary infection and preventing prolonged viral shedding. Deficiencies in cell-mediated immunity, rather than humoral immunity, are strongly associated with severe or disseminated varicella. This explains the heightened vulnerability of individuals with malignancies, advanced human immunodeficiency virus infection, or those receiving immunosuppressive therapies [12].

Following resolution of the primary infection, VZV is not eradicated from the body. Instead, the virus migrates from mucocutaneous lesions to adjacent sensory nerve endings and is transported retrogradely to the dorsal root ganglia and cranial nerve ganglia. Within these sensory neurons, VZV establishes a latent state in which viral replication is suppressed but the viral genome persists. The host immune system, particularly cell-mediated immunity, maintains this latent state and prevents viral reactivation. Over time, waning immunity, immunosenescence, or immunosuppression can permit reactivation of latent VZV. Reactivation results in herpes zoster, a distinct clinical syndrome characterized by painful, unilateral vesicular eruptions in a dermatomal distribution.[13][14] Thus, the pathophysiology of varicella and herpes zoster represents a continuum of primary infection, immune control, latency, and potential reactivation, underscoring the complex interaction between viral biology and host immune defense.

Histopathology

The histopathological features of varicella infection closely resemble those observed in herpes simplex and herpes zoster, making microscopic differentiation among these herpesvirus infections inherently challenging. All three conditions demonstrate characteristic epidermal changes that reflect the cytopathic effects of viral replication within keratinocytes. One of the most prominent findings is the formation of intraepidermal vesicles. These vesicles arise due to ballooning degeneration of keratinocytes, a process in which infected cells undergo marked intracellular edema, leading to cellular swelling and disruption of normal epidermal architecture. Within the vesicular lesions, multinucleated giant cells are frequently observed. These cells result from the fusion of adjacent keratinocytes that have been infected by the virus. The presence of multinucleated giant cells is a classic feature of herpesvirus infections and reflects virus-induced alterations in cell membranes that promote cell-to-cell fusion. In addition, acantholysis represents a key histological hallmark. This process involves the loss of intercellular connections between keratinocytes, causing individual cells to separate and float freely within the blister cavity. The detached keratinocytes often show pronounced viral cytopathic changes. Nuclear alterations are particularly distinctive in affected cells. These include margination and clumping of chromatin along the nuclear membrane, enlargement of nuclei, and the presence of multiple nuclei within a single cell. Eosinophilic intranuclear inclusion bodies may also be identified, representing accumulations of viral particles and altered host nuclear material. Despite these characteristic findings, histopathology alone cannot reliably distinguish varicella from herpes simplex or herpes zoster. Accurate diagnosis therefore requires correlation with clinical presentation and confirmation using ancillary techniques such as immunohistochemistry, viral culture, or polymerase chain reaction testing [13][14].

History and Physical

The clinical presentation of varicella varies according to age and immune status, with notable differences between children and adults. In adolescents and adults, the illness commonly begins with a prodromal phase characterized by nonspecific systemic symptoms. These include myalgia, headache, nausea, anorexia, malaise, and a low-grade fever. This prodrome usually precedes the appearance of cutaneous manifestations by one to two days and reflects early viral replication and systemic dissemination. Oral discomfort or mucosal lesions may develop during this phase and can precede the skin rash, serving as an early clinical indicator of infection. In children, the prodromal phase is often absent or minimal. The first noticeable sign may be the sudden onset of a rash or the appearance of lesions within the oral cavity. The characteristic exanthem begins as pruritic, erythematous macules and papules, initially involving the scalp, face, and upper trunk. The rash then spreads centrifugally to involve the trunk and, to a lesser extent, the extremities. Within approximately 12 hours, these lesions evolve rapidly into small, clear vesicles measuring 1 to 3 mm in diameter, surrounded by a narrow erythematous halo. This classic appearance is often described clinically as “dew drops on a rose petal.” The number of lesions is variable, ranging from a limited eruption to several hundred vesicles, and mucosal involvement is common. Lesions may be present on the oral mucosa, palate, tonsils, and occasionally the genital area. Involvement of the palms and soles can occur but is less frequent. Over time, older vesicles become cloudy, transform into pustules, and subsequently crust over. Each lesion typically heals within 7 to 10 days without scarring unless secondary bacterial infection occurs. A defining feature of varicella is the simultaneous presence of lesions at different stages of development, including macules, papules, vesicles, pustules, and crusts. Pruritus is most intense during the vesicular stage and may lead to excoriation. Adults often experience a more extensive rash, higher fever, and a longer disease course. They are also at increased risk of complications, particularly varicella pneumonia. Patients are contagious from one to two days before rash onset until all lesions have crusted. In most uncomplicated cases, the disease is self-limiting and resolves completely within two to four weeks [12][13][14].

Fig. 1: Chickenpox infection (Varicella).

Evaluation

The clinical evaluation of varicella infection relies predominantly on characteristic signs and symptoms, which in most cases are sufficient to establish a presumptive diagnosis. The presence of a generalized vesicular rash with lesions at varying stages of development, accompanied by systemic features such as fever and malaise, provides strong diagnostic indicators. However, laboratory confirmation is recommended in atypical presentations, immunocompromised patients, adults with severe disease, or cases complicated by visceral involvement, where diagnostic certainty is essential for appropriate management. Definitive confirmation of varicella may be achieved through the analysis of vesicular fluid obtained from fresh, uncrusted lesions or through scraping of the lesion base. Blood samples may also be utilized to detect immunologic markers of acute or prior infection. Among available diagnostic modalities, polymerase chain reaction testing offers the highest sensitivity and specificity. PCR enables rapid detection of viral deoxyribonucleic acid and is applicable not only to skin-derived specimens but also to nonskin samples, including bronchoalveolar lavage fluid and cerebrospinal fluid, making it particularly valuable in cases of pneumonia or central nervous system involvement. This method has become the diagnostic standard in many clinical settings. Direct fluorescent antibody testing is another option for detecting viral antigens in lesion samples and has largely supplanted the historically used Tzanck test, which lacks specificity and cannot reliably differentiate among herpesviruses. Viral culture of vesicular fluid remains possible; however, its diagnostic yield is comparatively low and results are delayed, limiting its clinical utility when rapid diagnosis is required. Serologic testing further contributes to evaluation by identifying immunoglobulin M antibodies indicative of recent infection and immunoglobulin G antibodies reflecting past exposure or established immunity. In the context of pregnancy, assessment of suspected fetal varicella requires particular caution. Prenatal diagnosis may be supported by ultrasonographic evaluation to identify structural abnormalities consistent with congenital infection, although such findings typically emerge several weeks after maternal illness. A waiting period of approximately five weeks following primary maternal infection is recommended before further investigation. Polymerase chain reaction testing of amniotic fluid can confirm fetal infection; however, this approach carries procedural risks. Notably, the likelihood of spontaneous abortion associated with amniocentesis exceeds the overall risk of fetal varicella, necessitating careful risk–benefit assessment prior to intervention.[15][16]

Treatment / Management

Management of varicella focuses on both infection control and symptomatic relief. Patients are generally advised to remain isolated during the infectious period to prevent transmission. Preventive measures include maintaining short fingernails and using gloves to reduce scratching and minimizing the risk of secondary bacterial infections. Topical applications, such as calamine lotion, may relieve pruritus, while daily cleansing with warm water helps prevent skin complications. Fever can be managed with acetaminophen, but aspirin should be avoided due to the risk of Reye syndrome [17][18][19]. In children, treatment is primarily supportive. Symptomatic relief remains the cornerstone, as antiviral therapy is not routinely recommended for immunocompetent pediatric patients. Acyclovir may modestly reduce symptom duration if administered within 24 hours of rash onset, though it does not significantly affect complication rates. In contrast, adults experience more severe disease and benefit from early antiviral therapy. Acyclovir or valacyclovir, ideally initiated within 24 to 48 hours of rash appearance, reduces both the severity and duration of symptoms. Immunocompromised adults, pregnant women, and those on chronic corticosteroid therapy often require intravenous antivirals due to increased risk of complications. Supportive care, including hydration, antipyretics, and antihistamines, remains essential. Postexposure prophylaxis is recommended for high-risk populations. Varicella-zoster immune globulin administered intramuscularly within 96 hours provides passive immunity lasting approximately three weeks. Intravenous immunoglobulin may also be used for nonimmune, high-risk individuals, offering higher varicella-specific IgG concentrations. Oral acyclovir can be considered 7 to 10 days post-exposure. Additionally, postexposure vaccination within 72 to 120 hours may prevent or reduce disease severity in nonimmune, immunocompetent individuals aged 12 months and older [20].

Differential Diagnosis

Chickenpox shares clinical features with other vesicular rashes, including monkeypox and smallpox, which can complicate diagnosis due to overlapping symptoms such as fever, rash, myalgia, chills, and headache. Accurate differentiation relies on careful clinical evaluation and, when needed, diagnostic testing. Varicella is distinguished by the presence of lesions at multiple stages simultaneously—macular, papular, vesicular, pustular, and crusted—whereas monkeypox lesions are typically uniform, progressing in synchrony. Coinfections with both viruses have been reported, particularly in Central Africa, further complicating clinical assessment [21][22][23]. Smallpox, although now eradicated, also presents with widespread vesicular eruptions. Additional conditions that may mimic varicella include other vesicular viral exanthems such as coxsackievirus infections, disseminated herpes simplex virus, pityriasis lichenoides et varioliformis acuta, rickettsialpox, drug-induced eruptions, bullous insect bite reactions, and scabies. Distinguishing between these conditions is critical to ensure appropriate management and prevent misdiagnosis.

Prognosis

The prognosis for varicella is generally favorable in healthy children, with most experiencing a self-limited course and recovering fully without significant complications. Lifelong immunity usually develops following primary infection, although rare cases of clinical reinfection have been documented in otherwise healthy individuals [24]. In contrast, immunocompromised patients, including those with congenital or acquired immunodeficiencies, are at heightened risk for severe disease, prolonged illness, and complications. Adults also tend to experience more intense symptoms, including widespread vesicular eruptions and higher rates of systemic complications. The overall outcome in these populations is less predictable, with morbidity and mortality risks significantly elevated. Early identification, vigilant monitoring, and timely initiation of supportive care and antiviral therapy are essential to improving prognosis in high-risk groups. The availability of vaccination has further enhanced prognostic outcomes by reducing disease incidence, severity, and related complications in both pediatric and adult populations, demonstrating the critical role of preventive interventions in mitigating varicella-related morbidity [24].

Complications

Although chickenpox is typically mild in children, complications can be serious in adolescents, adults, immunocompromised patients, and pregnant women. Secondary bacterial infections, including cellulitis, impetigo, and erysipelas, are common and may result in permanent scarring, particularly in adults. Varicella pneumonia carries a mortality risk of 10% to 30% if untreated. Neurological sequelae, though uncommon, may include encephalitis, acute cerebellar ataxia, Guillain-Barré syndrome, and stroke secondary to vascular involvement [13][25]. Neonates born to mothers infected near delivery are particularly vulnerable to severe varicella due to absent maternal antibodies, while maternal infection during early gestation can result in congenital varicella syndrome with low birth weight, ocular anomalies, and limb hypoplasia. Immunocompromised individuals often present with atypical, widespread, hemorrhagic lesions affecting the skin, lungs, liver, and CNS. Additional rare complications include hepatitis, myocarditis, nephritis, keratitis, and vasculitis. Recognizing at-risk populations and monitoring for early signs of complications are vital in preventing morbidity and mortality.

Patient Education

Patient and caregiver education is central to reducing varicella-associated morbidity. Parents should maintain short fingernails to prevent secondary bacterial infections and avoid administering aspirin to children, minimizing the risk of Reye syndrome. Topical measures such as cold compresses and moisturizers can alleviate pruritus and skin irritation. Clinicians should strongly encourage vaccination for children to prevent primary infection and for adults over 60 to reduce the risk of herpes zoster [13]. A thorough assessment during illness, including evaluation for pulmonary or neurological complications, supports early intervention. Education should also extend to immunocompromised patients and pregnant women, emphasizing avoidance of exposure, early antiviral therapy, and administration of varicella zoster immune globulin when indicated. Comprehensive education and preventive strategies empower patients and caregivers, mitigate complications, and strengthen public health outcomes [13].

Other Issues

Management of varicella in immunocompetent children focuses on symptomatic relief, including antipyretics, antihistamines, topical agents, and tepid baths. Antiviral therapy is reserved for high-risk children, adolescents, adults, and those with comorbidities or prolonged corticosteroid exposure. Pregnant women and neonates require specialized interventions, including postexposure immunoglobulin administration to prevent severe disease. The live attenuated varicella vaccine (Oka strain) demonstrates high efficacy, with two-dose immunization recommended at 12–15 months and 4–6 years to maintain protection [26][27][28].

Enhancing Healthcare Team Outcomes

Optimal management of varicella requires an interprofessional, patient-centered approach. Physicians, nurses, pharmacists, and allied health professionals collaborate to ensure accurate diagnosis, early recognition of complications, antiviral therapy administration, and supportive care. Nurses play a pivotal role in monitoring, patient education, and infection control, while pharmacists support vaccination protocols and medication adherence. Public health teams facilitate outbreak surveillance, contact tracing, and immunization strategies. Interprofessional collaboration fosters shared decision-making, improves patient safety, and strengthens disease prevention. Education on vaccination, hygiene, and postexposure prophylaxis reduces morbidity, while coordinated care enhances clinical outcomes, particularly in high-risk populations such as immunocompromised children, adults, and pregnant women. This team-based approach ensures comprehensive management of varicella, mitigates complications, and promotes public health [32][33][34].

Conclusion:

Varicella remains an important infectious disease despite major advances in vaccination and public health control. Although often perceived as a benign childhood illness, the disease can result in significant morbidity and mortality among adults, pregnant women, neonates, and immunocompromised individuals. The ability of the varicella-zoster virus to establish lifelong latency and later reactivate as herpes zoster further underscores its long-term clinical significance. Accurate clinical recognition, supported by laboratory confirmation when indicated, is essential for timely management and prevention of complications. Antiviral therapy plays a critical role in high-risk populations, while supportive care remains the cornerstone of management in uncomplicated cases. The introduction of routine childhood immunization and two-dose vaccination schedules has dramatically reduced disease burden, highlighting the effectiveness of preventive strategies. Continued surveillance, patient education, vaccination advocacy, and interprofessional healthcare collaboration are essential to sustain disease control, protect vulnerable populations, and improve overall health outcomes related to varicella infection [33][34].

References:

1. Shrim A, Koren G, Yudin MH, Farine D. No. 274-Management of Varicella Infection (Chickenpox) in Pregnancy. Journal of obstetrics and gynaecology Canada : JOGC = Journal d'obstetrique et gynecologie du Canada : JOGC. 2018 Aug:40(8):e652-e657. doi: 10.1016/j.jogc.2018.05.034.
2. Pereira L. Congenital Viral Infection: Traversing the Uterine-Placental Interface. Annual review of virology. 2018 Sep 29:5(1):273-299. doi: 10.1146/annurev-virology-092917-043236.
3. Kasabwala K, Wise GJ. Varicella-zoster virus and urologic practice: a case-based review. The Canadian journal of urology. 2018 Jun:25(3):9301-9306
4. Al-Turab M, Chehadeh W. Varicella infection in the Middle East: Prevalence, complications, and vaccination. Journal of research in medical sciences : the official journal of Isfahan University of Medical Sciences. 2018:23():19. doi: 10.4103/jrms.JRMS_979_17.
5. Dowell SF, Bresee JS. Severe varicella associated with steroid use. Pediatrics. 1993 Aug:92(2):223-8
6. Longbottom K, Lyall H. Neonatal varicella. Archives of disease in childhood. 2024 Jul 18:109(8):610-615. doi: 10.1136/archdischild-2022-324820.
7. Marin M, Meissner HC, Seward JF. Varicella prevention in the United States: a review of successes and challenges. Pediatrics. 2008 Sep:122(3):e744-51. doi: 10.1542/peds.2008-0567.
8. Haider M, Novice T, Engers D, Burman I. Never Too Old: A Case of Chicken Pox in an Eighty-One-Year-Old Patient. The American journal of medicine. 2022 Jul:135(7):851-852. doi: 10.1016/j.amjmed.2022.02.031.
9. Rice ME, Bannerman M, Marin M, Lopez AS, Lewis MM, Stamatakis CE, Regan JJ. Maritime varicella illness and death reporting, U.S., 2010-2015. Travel medicine and infectious disease. 2018 May-Jun:23():27-33. doi: 10.1016/j.tmaid.2018.04.001.
10. Schmader K. Herpes Zoster. Clinics in geriatric medicine. 2016 Aug:32(3):539-53. doi: 10.1016/j.cger.2016.02.011.
11. Bakker KM, Martinez-Bakker ME, Helm B, Stevenson TJ. Digital epidemiology reveals global childhood disease seasonality and the effects of immunization. Proceedings of the National Academy of Sciences of the United States of America. 2016 Jun 14:113(24):6689-94. doi: 10.1073/pnas.1523941113.
12. Rockley PF, Tyring SK. Pathophysiology and clinical manifestations of varicella zoster virus infections. International journal of dermatology. 1994 Apr:33(4):227-32
13. Freer G, Pistello M. Varicella-zoster virus infection: natural history, clinical manifestations, immunity and current and future vaccination strategies. The new microbiologica. 2018 Apr:41(2):95-105

14. Dayan RR, Peleg R. Herpes zoster - typical and atypical presentations. Postgraduate medicine. 2017 Aug:129(6):567-571. doi: 10.1080/00325481.2017.1335574.
15. Inata K, Miyazaki D, Uotani R, Shimizu D, Miyake A, Shimizu Y, Inoue Y. Effectiveness of real-time PCR for diagnosis and prognosis of varicella-zoster virus keratitis. Japanese journal of ophthalmology. 2018 Jul:62(4):425-431. doi: 10.1007/s10384-018-0604-7.
16. Onyango CO, Loparev V, Lidechi S, Bhullar V, Schmid DS, Radford K, Lo MK, Rota P, Johnson BW, Munoz J, Oneko M, Burton D, Black CM, Neatherlin J, Montgomery JM, Fields B. Evaluation of a TaqMan Array Card for Detection of Central Nervous System Infections. Journal of clinical microbiology. 2017 Jul:55(7):2035-2044. doi: 10.1128/JCM.02469-16.
17. Harrington D, Haque T. Varicella Zoster Immunoglobulin G (VZIG)-Do current guidelines advocate overuse? Journal of clinical virology : the official publication of the Pan American Society for Clinical Virology. 2018 Jun:103():25-26. doi: 10.1016/j.jcv.2018.03.007.
18. Poole CL, James SH. Antiviral Therapies for Herpesviruses: Current Agents and New Directions. Clinical therapeutics. 2018 Aug:40(8):1282-1298. doi: 10.1016/j.clinthera.2018.07.006.
19. Hayward K, Cline A, Stephens A, Street L. Management of herpes zoster (shingles) during pregnancy. Journal of obstetrics and gynaecology : the journal of the Institute of Obstetrics and Gynaecology. 2018 Oct:38(7):887-894. doi: 10.1080/01443615.2018.1446419.
20. Maranich AM, Rajnik M. Varicella-specific immunoglobulin G titers in commercial intravenous immunoglobulin preparations. Pediatrics. 2009 Sep:124(3):e484-8. doi: 10.1542/peds.2009-0047.
21. Rasizadeh R, Shamekh A, Shiri Aghbash P, Bannazadeh Baghi H. Comparison of human monkeypox, chickenpox and smallpox: a comprehensive review of pathology and dermatological manifestations. Current medical research and opinion. 2023 May:39(5):751-760. doi: 10.1080/03007995.2023.2200122.
22. Buerke M, Böttger P, Lemm H. [Mpox-diagnosis, treatment, immunization, and prognosis]. Medizinische Klinik, Intensivmedizin und Notfallmedizin. 2025 Mar:120(2):141-144. doi: 10.1007/s00063-024-01198-w.
23. Onukak AE, Nga CN, Ubani BC, Umoinwek E, Umoette N. Mpox and Chicken Pox Coinfection Complicated by Acute Urinary Retention: A Case Report. West African journal of medicine. 2023 Aug 28:40(8):873-876
24. Hall S, Maupin T, Seward J, Jumaan AO, Peterson C, Goldman G, Mascola L, Wharton M. Second varicella infections: are they more common than previously thought? Pediatrics. 2002 Jun:109(6):1068-73
25. Bhardwaj LM, Borthakur S, Bhattacharyya PC. Simultaneous Occurrence of Chicken Pox and Herpes Zoster with Facial Nerve Palsy in Immunocompetent Patient. The Journal of the Association of Physicians of India. 2022 Aug:70(8):11-12. doi: 10.5005/japi-11001-0075.
26. Charlier C, Anselem O, Caseris M, Lachâtre M, Tazi A, Driessen M, Pinquier D, Le Cœur C, Saunier A, Bergamelli M, Gibert Vanspranghels R, Chosidow A, Cazanave C, Alain S, Faure K, Birgy A, Dubos F, Lesprit P, Guinaud J, Cohen R, Decousser JW, Grimprel E, Huissoud C, Blanc J, Kayem G, Vuotto F, Vauloup-Fellous C. Prevention and management of VZV infection during pregnancy and the perinatal period. Infectious diseases now. 2024 Jun:54(4):104857. doi: 10.1016/j.idnow.2024.104857.
27. Tommasi C, Drousioti A, Breuer J. The live attenuated varicella-zoster virus vaccine vOka: Molecular and cellular biology of its skin attenuation. Human vaccines & immunotherapeutics. 2025 Dec:21(1):2482286. doi: 10.1080/21645515.2025.2482286.
28. Wooding EL, Kadambari S, Warris A. Varicella: is it time for a global vaccination programme? Archives of disease in childhood. 2025 Jul 18:110(8):586-591. doi: 10.1136/archdischild-2024-327593.
29. Wang X, Zhang X, Yu Z, Zhang Q, Huang D, Yu S. Long-term outcomes of varicella zoster virus infection-related myelitis in 10 immunocompetent patients. Journal of neuroimmunology. 2018 Aug 15:321():36-40. doi: 10.1016/j.jneuroim.2018.05.005.
30. McRae JE, Quinn HE, Macartney K. Paediatric Active Enhanced Disease Surveillance (PAEDS) annual report 2015: Prospective hospital-based surveillance for serious paediatric conditions. Communicable diseases intelligence quarterly report. 2017 Sep 1:41(3):E275-E283
31. Dommasch ED, Joyce CJ, Mostaghimi A. Trends in Nationwide Herpes Zoster Emergency Department Utilization From 2006 to 2013. JAMA dermatology. 2017 Sep 1:153(9):874-881. doi: 10.1001/jamadermatol.2017.1546.
32. Ogunjimi B, Van den Bergh J, Meysman P, Heyndericky S, Bergs K, Jansens H, Leuridan E, Vorsters A, Goossens H, Laukens K, Cools N, Van Tendeloo V, Hens N, Van Damme P, Smits E, Beutels P. Multidisciplinary study of the secondary immune response in grandparents re-exposed to chickenpox. Scientific reports. 2017 Apr 24:7(1):1077. doi: 10.1038/s41598-017-01024-8.
33. Ogunjimi B, Van Damme P, Beutels P. Herpes Zoster Risk Reduction through Exposure to Chickenpox Patients: A Systematic Multidisciplinary Review. PloS one.

2013:8(6):e66485. doi: 10.1371/journal.pone.0066485.

34. Meylan P, Gerber S, Kempf W, Nadal D, Swiss Herpes Management Forum. [Swiss recommendations for the management of varicella-zoster virus infections]. Revue medicale suisse. 2007 Sep 19:3(125):2116-22, 2124-9