



Interdisciplinary Management of Varicella-Zoster Virus Infection: Integrating Medical Treatment, Nursing Care, and Health Information Systems

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Abstract

Background: Varicella-zoster virus (VZV) is a highly contagious pathogen causing two distinct clinical entities: primary infection (chickenpox) and reactivation (herpes zoster). While often self-limiting in healthy children, VZV poses significant risks of severe complications in adults, immunocompromised individuals, and pregnant women, leading to hospitalizations and mortality.

Aim: This article aims to provide a comprehensive, interdisciplinary review of VZV management, integrating perspectives from medical treatment, nursing care, and health information systems to optimize patient outcomes and public health control.

Methods: The approach is a synthesis of current clinical guidelines and literature. It details diagnostic methods (clinical assessment, PCR, serology), medical management (antiviral therapy, symptomatic care), and public health strategies (isolation, vaccination, post-exposure prophylaxis). The role of nursing in patient education and supportive care, alongside the contribution of health information systems to surveillance and coordination, is emphasized.

Results: Effective management hinges on risk stratification. Supportive care suffices for immunocompetent children, whereas early antiviral therapy (e.g., acyclovir) is critical for high-risk groups. Vaccination has dramatically reduced disease incidence and severity. Post-exposure prophylaxis with varicella-zoster immune globulin (VZIG) or vaccination can prevent or modify disease. Nursing care is vital for symptom relief and preventing complications like bacterial superinfection.

Conclusion: A successful approach to VZV requires a coordinated, interprofessional model. Integrating timely medical intervention, dedicated nursing support, and robust health information systems is essential for reducing transmission, managing complications, and improving individual and population health outcomes.

Keywords: Varicella-Zoster Virus, Chickenpox, Interdisciplinary Management, Antiviral Therapy, Vaccination, Nursing Care.

1. Introduction

Varicella, commonly known as chickenpox, is an acute, highly transmissible infection produced by the varicella-zoster virus (VZV). The clinical spectrum of VZV encompasses primary varicella infection in seronegative hosts and subsequent reactivation as herpes zoster. Historical delineation of these entities dates to the 18th century when Heberden distinguished chickenpox from smallpox in 1767. The etymology of the term “chickenpox” remains debated; derivations proposed include the French *chiche-pois*, denoting the size resemblance of the vesicles to chickpeas, and the Old English *gigan*, implying pruritus. The epidemiological and etiological link between varicella and herpes zoster emerged in the late 19th and early 20th centuries. Von Bokay observed varicella in children following exposure to individuals with zoster in 1888. Experimental and clinical reports by Kundratitz in 1922 and Bruusgaard in 1932

provided more definitive evidence when varicella developed in vulnerable infants after inoculation with vesicular fluid from zoster patients. Subsequent virological studies confirmed the same viral agent underlies both clinical syndromes, thereby unifying their etiologies [1][2][3].

Transmission of VZV occurs predominantly through respiratory droplets and aerosolized particles generated by coughing and sneezing, as well as via direct contact with exudative skin lesions. The virus maintains global endemicity and demonstrates efficient spread in susceptible populations. The incubation interval ranges from 10 to 21 days, with a mean of approximately 14 days, a temporal pattern that informs both clinical suspicion and public health interventions. Prodromal features are often nonspecific and include fever, malaise, pharyngitis, and cephalalgia. The defining manifestation is a generalized vesicular exanthem. Lesions progress

rapidly from macules to papules to clear, fluid-filled vesicles that later crust. The centrifugal distribution commonly initiates on the trunk and face before disseminating to the extremities. New lesions may continue to appear for several days, so patients are potentially infectious 1 to 2 days before rash onset and remain contagious until all lesions have crusted [1][2][3]. Clinical severity of primary varicella varies with host factors. Children typically experience self-limited disease characterized by extensive but uncomplicated cutaneous involvement and transient systemic symptoms. Adults exhibit higher rates of systemic complications and more pronounced morbidity. Immunocompromised hosts, neonates, and pregnant persons represent high-risk groups for severe or atypical presentations. Complications include viral or secondary bacterial pneumonia, meningoencephalitis, and invasive bacterial soft tissue infections. These outcomes account for the majority of varicella-related hospitalizations and fatalities in settings where immunization is incomplete. The risk profile and spectrum of complications underscore the need for prompt recognition and risk stratification in clinical practice [1][2][3].

Diagnostic confirmation relies principally on clinical assessment supported by laboratory testing in uncertain or severe cases. Polymerase chain reaction identification of VZV DNA from vesicular fluid, crust material, or other clinical specimens provides sensitive and specific confirmation and facilitates genotypic and antiviral-resistance analyses when indicated. Serologic assays quantifying varicella-specific IgG and IgM antibodies assist in determining immune status, documenting prior exposure or vaccination, and guiding postexposure prophylaxis decisions. Reinfection with VZV can occur but tends to present with attenuated clinical features relative to primary disease, often subclinical or manifesting with limited dermatologic involvement. Such occurrences reflect partial immunity conferred by prior infection or immunization rather than true viral persistence in an actively replicative state [1][2][3]. Control of varicella at both individual and population levels has been transformed by immunization. Since licensure of the varicella vaccine in 1995, substantial declines have occurred in incidence, complication rates, and varicella-associated hospitalizations. Vaccination induces robust humoral and cellular responses that reduce susceptibility to primary infection and mitigate disease severity among breakthrough cases. Population-based studies demonstrate vaccine effectiveness in the range of 70 to 90 percent for prevention of infection and approximately 95 percent effectiveness against severe disease, outcomes that have translated into measurable public health benefits in jurisdictions that implemented routine pediatric immunization. Postexposure vaccination administered within a narrow temporal window after exposure can modify the clinical course in susceptible children and

reduce transmission risk, thereby serving as an adjunct to other containment measures [1][2][3].

Management of varicella encompasses supportive care for uncomplicated disease and targeted antiviral therapy for those at increased risk for complication or with severe manifestations. Antiviral agents directed at VZV replication, when instituted early, reduce lesion formation, duration of fever, and the likelihood of certain complications in selected populations. Preventive strategies emphasize routine childhood immunization, consideration of vaccination for susceptible adolescents and adults, and tailored approaches for high-risk cohorts including the use of varicella-zoster immune globulin in specified exposure scenarios. Surveillance, vaccination policy, and access to diagnostic and therapeutic resources remain central to minimizing the clinical and societal burden of VZV infection.[1][2][3]

Etiology

Varicella, commonly referred to as chickenpox, is an infectious disease caused by the varicella-zoster virus (VZV), a member of the herpesvirus family with global prevalence. VZV possesses the capacity to establish lifelong latency following primary infection, a hallmark characteristic of herpesviruses that distinguishes it from many other viral pathogens [4]. Transmission occurs predominantly through inhalation of aerosolized droplets expelled by infected individuals, although direct contact with vesicular fluid also facilitates viral spread. The virus exhibits high contagiousness, enabling rapid dissemination within susceptible populations. Initial replication occurs in the mucosal epithelium of the upper respiratory tract. Within two to six days of exposure, the virus enters the bloodstream, producing a primary viremia. This is followed by a secondary viremia around 10 to 12 days post-exposure, coinciding with the emergence of the characteristic vesicular rash. The host immune response includes the production of immunoglobulins IgA, IgM, and IgG, with IgG antibodies providing durable, often lifelong, immunity. Following resolution of the primary infection, VZV establishes latency within sensory ganglia, with potential for subsequent reactivation manifesting as herpes zoster in later life [4]. Certain populations demonstrate heightened susceptibility to severe manifestations of varicella, particularly adolescents, adults, immunocompromised individuals, and pregnant women. Exogenous immunosuppression, especially corticosteroid therapy, significantly increases the risk of complicated disease. Administration of prednisolone at doses equivalent to 1 to 2 mg/kg per day for two weeks or longer is recognized as a substantial risk factor. Notably, even short courses of corticosteroids at these dosages, if administered during or shortly before the incubation period of varicella, can precipitate severe or fatal outcomes [5]. Immunocompromised states, including malignancies,

treatment with antineoplastic agents, human immunodeficiency virus infection, and congenital or acquired immunodeficiency disorders, similarly predispose patients to a more aggressive disease course. Within these groups, cellular immune deficits are particularly associated with severe varicella, whereas isolated humoral deficiencies confer comparatively lower risk [4].



Figure-1: Varicella Zoster Infection.

Pregnant women represent another vulnerable cohort. Maternal varicella infection can lead to severe maternal disease, most notably varicella-associated pneumonia, which remains a

leading cause of morbidity and mortality in this population. Furthermore, maternal viremia facilitates transplacental viral transmission, potentially resulting in congenital varicella or neonatal varicella. Infants born within a window of five days before to two days after maternal rash onset are at highest risk for severe disease [6]. The first month of life constitutes a critical period during which neonates are particularly susceptible, especially if maternal seroconversion has not occurred. Premature delivery before 28 weeks of gestation further compounds this risk, as maternal IgG antibodies, which cross the placenta primarily in the third trimester, are insufficiently transferred to confer passive immunity. The pathophysiological progression of varicella reflects the interplay between viral replication, host immune responses, and cellular tropism. The initial mucosal infection allows viral amplification before systemic dissemination, while secondary viremia drives the dermatologic manifestations that characterize the disease. Immunologic memory, established through IgG production, generally prevents reinfection or limits subsequent disease severity. However, in immunologically vulnerable hosts, the inability to mount an effective immune response permits widespread viral replication, systemic involvement, and life-threatening complications. Understanding these etiological mechanisms informs both clinical management and public health strategies, highlighting the importance of vaccination, early antiviral therapy in high-risk groups, and heightened surveillance for pregnant women, immunocompromised patients, and neonates [4][5][6].

Epidemiology

Varicella-zoster virus (VZV) exhibits a pervasive global presence, with seroepidemiological studies indicating that approximately 98% of adults worldwide are seropositive, reflecting previous exposure or immunization. The virus is endemic in all regions, causing an estimated 7,000 deaths annually, predominantly attributable to complications such as pneumonia, encephalitis, or secondary bacterial infections. Seasonal patterns demonstrate higher transmission during winter and spring, consistent with increased indoor crowding and enhanced viral survival in cooler, less humid environments. In the United States, VZV contributes to over 9,000 hospitalizations per year, with the highest disease burden observed among children aged four to ten years, underscoring its predominance as a pediatric infection [7]. The epidemiology of varicella has undergone a substantial shift following the introduction of the live attenuated varicella vaccine in 1995. Nationwide surveillance has documented an approximate 85% reduction in overall incidence, providing evidence of both direct protection and indirect herd immunity. This vaccination-driven reduction altered the age distribution of infection, shifting the peak incidence from the traditional five-to-nine-year range to ten-to-fourteen years. This epidemiological transition also highlighted the

occurrence of breakthrough varicella, defined as infection in previously vaccinated individuals, typically presenting with milder symptoms. Recognition of these breakthrough cases prompted the incorporation of a second varicella vaccine dose into the standard childhood immunization schedule in 2006, which has been shown to enhance long-term immunity and reduce incidence among older children [7].

Prior to widespread vaccination, varicella infection was nearly universal among children in temperate countries, affecting approximately 90% by the age of ten. The clinical presentation is typically symptomatic, characterized by fever, malaise, and the hallmark vesicular rash. In contrast, herpes zoster represents the reactivation of latent VZV within sensory ganglia. It affects around 20% of otherwise healthy adults and approximately 50% of immunocompromised individuals, who experience heightened morbidity and mortality due to severe dermatologic and systemic complications. Importantly, secondary varicella cases among household contacts often demonstrate greater disease severity than primary infections, a phenomenon attributed to increased viral load exposure and host immunologic factors. Geographic and climatic differences influence the epidemiology of varicella. In tropical regions, the disease tends to occur at older ages, often in adolescents and adults, resulting in a higher risk of severe clinical manifestations, including pneumonia, hepatitis, and encephalitis. This delayed acquisition contrasts with temperate regions, where childhood infection predominates. Reports have documented cases of varicella in elderly patients, including individuals as advanced in age as 81 years, highlighting the continued susceptibility in unexposed adults and the potential for severe outcomes in this population [8][9][10][11]. Ongoing epidemiological surveillance remains critical for informing public health strategies, guiding vaccination policies, and monitoring shifts in disease burden. Understanding age-specific incidence, seasonal trends, and regional differences in clinical severity allows healthcare systems to prioritize high-risk populations, optimize immunization schedules, and implement targeted interventions to reduce both morbidity and mortality associated with VZV. Furthermore, the persistent risk of herpes zoster underscores the importance of preventive measures, including vaccination for older adults and immunocompromised individuals, to mitigate reactivation-related complications [10][11].

Pathophysiology

Varicella-zoster virus (VZV), the etiologic agent of chickenpox, is a double-stranded DNA virus within the Alphaherpesvirinae subfamily of human herpesviruses. Like other herpesviruses, VZV exhibits a unique capacity to establish lifelong latency following primary infection. Entry into the host typically occurs through the respiratory tract, via

inhalation of aerosolized particles or contact with conjunctival or upper respiratory mucosa. Once introduced, the virus initiates replication within the epithelial cells of the upper respiratory tract before infecting regional lymphoid tissues. Viral proliferation in these lymph nodes occurs within two to four days post-exposure, providing a reservoir from which systemic dissemination can occur [12]. Approximately four to six days following initial replication, primary viremia ensues. During this phase, VZV circulates through the bloodstream and infects reticuloendothelial tissues, including the spleen, liver, and other lymphoid organs. This stage is critical for the establishment of viral reservoirs and the induction of initial immune responses. Secondary viremia develops around one week after exposure, marking the dissemination of the virus to peripheral tissues, most notably the skin and mucous membranes. The characteristic vesicular lesions of varicella appear during this secondary viremia, coinciding with the peak period of viral shedding from respiratory secretions. This early viremia facilitates the high transmissibility of the virus, allowing transmission to susceptible contacts before visible cutaneous manifestations emerge. In severe or complicated cases, dissemination can extend to internal organs, including the central nervous system, liver, and lungs, resulting in complications such as encephalitis, hepatitis, or varicella pneumonia [12].

The host immune response to VZV involves both humoral and cell-mediated mechanisms. Following infection, the immune system generates IgM, IgA, and IgG antibodies. IgG antibodies persist for life and provide durable protection against subsequent symptomatic infection. Cell-mediated immunity plays a crucial role in controlling viral replication and limiting the severity and duration of primary disease. Recurrent symptomatic varicella is rare among immunocompetent individuals; however, subclinical reinfections can occur and serve to boost immune memory, enhancing the protective response initiated by the primary infection. These immunologic processes are critical in maintaining long-term viral control and preventing widespread dissemination during reexposure [13]. Following resolution of the primary infection, VZV establishes latency within sensory neurons of the dorsal root ganglia. The virus is believed to reach these sites via retrograde transport from mucocutaneous lesions, where it remains transcriptionally inactive under the surveillance of the host immune system. Latent VZV can persist for decades without causing symptoms. Immune senescence, immunosuppression, or other disruptions in cellular immunity can precipitate viral reactivation, manifesting clinically as herpes zoster, or shingles. Unlike primary varicella, herpes zoster presents as a localized, often painful dermatomal eruption, reflecting reactivation from a specific sensory ganglion [14]. Understanding the pathophysiological

processes of VZV infection is essential for guiding clinical management and preventive strategies. The sequential progression from initial mucosal invasion, primary and secondary viremia, cutaneous manifestations, and immune control highlights key stages in disease development. Recognition of these mechanisms informs therapeutic interventions, vaccination strategies, and anticipatory guidance for high-risk populations, including immunocompromised individuals and older adults, in whom viral reactivation or severe disease may occur [14].

Histopathology

Infections caused by herpes simplex virus, varicella-zoster virus (VZV), and herpes zoster share remarkably similar histopathological characteristics, reflecting their close viral relationship within the Herpesviridae family. Histological examination of affected skin typically reveals intraepidermal vesicles accompanied by pronounced ballooning degeneration of keratinocytes. This degeneration is characterized by cytoplasmic swelling and loss of normal cellular architecture, which contributes to vesicle formation. A key microscopic feature is acantholysis, in which individual keratinocytes detach from one another and appear to float freely within the blister cavity. This phenomenon reflects both direct viral cytopathic effects and the disruption of intercellular junctions. Multinucleated giant cells are frequently observed in the epidermal layer, arising from the fusion of adjacent infected keratinocytes. These giant cells serve as a hallmark of herpesvirus infection and are readily identifiable under light microscopy. Within the nuclei of these affected cells, characteristic viral-induced alterations are present. Chromatin margination, in which nuclear chromatin condenses along the periphery of the nucleus, is a consistent finding. In addition, multiple nuclei may be present within a single keratinocyte, reflecting the virus's ability to alter normal nuclear division processes. Nuclear inclusion bodies, both eosinophilic and basophilic depending on staining technique, are also observed and represent accumulations of viral proteins or nucleic acids. These nuclear changes are critical indicators of viral activity and aid in the identification of infected cells [14]. Despite these characteristic findings, histopathology alone is insufficient to distinguish between herpes simplex, varicella, and herpes zoster infections, as the cytopathic features are virtually identical across these viruses. Accurate diagnosis therefore requires correlation with the clinical presentation, including lesion distribution, patient age, and symptom chronology. Ancillary techniques enhance diagnostic specificity. Immunohistochemistry enables visualization of viral antigens within infected tissue, allowing differentiation between viral species. Viral culture can confirm active infection and provide isolates for further study, while polymerase chain reaction (PCR) offers highly sensitive detection of viral DNA, even in

minimal or degraded tissue samples. Combining histopathological analysis with these diagnostic modalities ensures precise identification of the causative virus and informs subsequent management strategies [14].

History and Physical

The clinical presentation of varicella varies with age, immune status, and prior exposure to the varicella-zoster virus (VZV). In adolescents and adults, the disease often begins with a prodromal phase characterized by systemic and nonspecific symptoms. Patients typically report myalgia, nausea, anorexia, headache, and malaise, often accompanied by a low-grade fever. Oral lesions may develop prior to the appearance of cutaneous manifestations, presenting as small, painful, or pruritic ulcers within the oral cavity or on the tonsillar region. These early signs serve as important diagnostic clues, particularly in adults who may experience a more severe disease course. In contrast, children frequently exhibit an abrupt onset of varicella without an identifiable prodromal phase, with the initial clinical evidence often manifesting as skin lesions or oral enanthem [14]. The hallmark cutaneous eruption of varicella progresses in a predictable pattern. Lesions initially appear as pruritic, erythematous macules and papules, typically arising first on the face and scalp before spreading centrifugally to the trunk and extremities. Within approximately 12 hours, these lesions evolve into clear vesicles measuring one to three millimeters in diameter, each surrounded by a narrow erythematous halo, producing the classic "dew drops on a rose petal" appearance. The total number of vesicles varies widely, ranging from a few to several hundred, and frequently involves the oral mucosa. Distal and lower extremities may be relatively spared, although blisters can appear on the palms, soles, and genital region in some patients. Varicella lesions display a characteristic asynchronous development, with vesicles, pustules, and crusted lesions coexisting simultaneously. Each lesion typically resolves within seven to ten days, forming crusts that gradually separate from the skin. Intense pruritus is common during the vesicular stage, contributing to patient discomfort and the potential for secondary bacterial infection. Oral and tonsillar involvement, or enanthem, often precedes cutaneous manifestations by one to three days, presenting as small, painful ulcers that may interfere with oral intake and exacerbate systemic malaise [14][15].

The incubation period for varicella ranges from 10 to 21 days following exposure. During the initial stages of viral replication, patients may be contagious before the onset of visible lesions. Respiratory secretions, particularly watery nasal discharge containing live virus, are a primary source of transmission, rendering individuals infectious one to two days before the eruption of the exanthem. In adults, the disease tends to manifest with a more widespread rash, higher fever, and a greater risk of

complications, the most significant being varicella pneumonia. Other potential complications, although less common, include encephalitis, hepatitis, and secondary bacterial infections. In immunocompetent individuals, the disease course is typically self-limiting, with complete resolution occurring within two to four weeks. The coexistence of lesions at various stages of development remains a defining feature and aids in distinguishing varicella from other vesicular exanthems. Recognition of the prodromal symptoms, lesion progression, and distribution pattern is essential for timely diagnosis, effective isolation to prevent transmission, and initiation of supportive or antiviral therapy when indicated, particularly in adults, immunocompromised patients, and pregnant women [14][15].

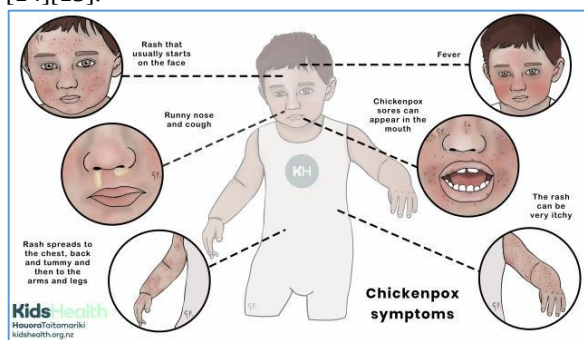


Figure-2: varicella zoster infection symptoms.

Evaluation

The diagnosis of varicella infection is primarily clinical, relying on the characteristic signs and symptoms, including the presence of pruritic vesicular lesions at different stages of development, fever, malaise, and, in some cases, oral enanthem. While clinical evaluation often suffices for diagnosis in typical cases, laboratory confirmation may be necessary in atypical presentations, immunocompromised patients, or pregnant women. Laboratory methods focus on detecting the virus or identifying the host immune response. Direct examination of vesicular fluid or lesion scrapings is commonly employed for confirmation. Vesicular fluid, obtained from lesions that have not yet crusted, can be analyzed for viral presence using polymerase chain reaction (PCR), which demonstrates the highest sensitivity and specificity among diagnostic tests. PCR can also be applied to noncutaneous samples, such as bronchoalveolar lavage or cerebrospinal fluid, which is particularly valuable in cases with respiratory or neurological complications. Direct fluorescent antibody testing has largely supplanted the traditional Tzanck smear, providing more precise detection of VZV antigens within infected cells. Viral culture of vesicular fluid is possible but has limited yield compared to PCR due to slower growth and lower sensitivity [15][16].

Serological testing offers an alternative means of evaluation, particularly for assessing immune status or confirming recent infection.

Detection of varicella-specific IgM antibodies indicates acute or recent infection, while IgG antibodies signify past infection or immunity, providing essential information for management and postexposure prophylaxis. These tests are critical in prenatal care, where maternal immunity and fetal risk must be evaluated. Prenatal diagnosis of fetal varicella can be performed using ultrasound, which may identify structural anomalies associated with congenital varicella syndrome. To optimize accuracy, a delay of approximately five weeks following primary maternal infection is recommended before conducting ultrasound assessment. In addition, PCR testing of amniotic fluid can detect fetal VZV DNA, offering a more direct diagnostic approach. However, invasive procedures such as amniocentesis carry a higher risk of spontaneous abortion than the estimated risk of fetal varicella and therefore are reserved for cases in which the diagnostic benefit outweighs procedural risk [15][16]. Overall, the evaluation of varicella combines clinical assessment with targeted laboratory investigations. PCR remains the gold standard for confirming infection, while serology is invaluable for assessing immunity and guiding management in susceptible or high-risk populations. Prenatal evaluation must carefully balance diagnostic accuracy with procedural safety to optimize maternal and fetal outcomes [15][16].

Treatment / Management

Management of varicella primarily focuses on limiting transmission, alleviating symptoms, and preventing complications, particularly in high-risk populations. Infected individuals are generally advised to remain isolated at home while contagious to minimize exposure to susceptible contacts. Personal hygiene measures play an important role in mitigating secondary bacterial infections. Keeping fingernails short and, in some cases, wearing gloves can reduce scratching and prevent inoculation of bacteria into vesicular lesions [17][18]. Daily cleansing with warm water and mild soap helps maintain skin integrity and reduces the risk of superinfection. Pruritus can be managed using topical agents such as calamine lotion, which provides symptomatic relief. Antipyretics, primarily acetaminophen, are indicated to control fever and discomfort. Use of aspirin is contraindicated in children and adolescents due to the risk of Reye syndrome [19]. In pediatric patients, management is largely supportive. Symptomatic relief remains the primary goal, as the disease course in otherwise healthy children is generally self-limiting. Administration of oral acyclovir within 24 hours of rash onset can reduce symptom duration by approximately one day; however, it does not significantly alter the incidence of complications and is therefore not routinely recommended for immunocompetent children. Emphasis is placed on hydration, fever control, and relief of pruritus, with careful monitoring for signs of secondary infection.

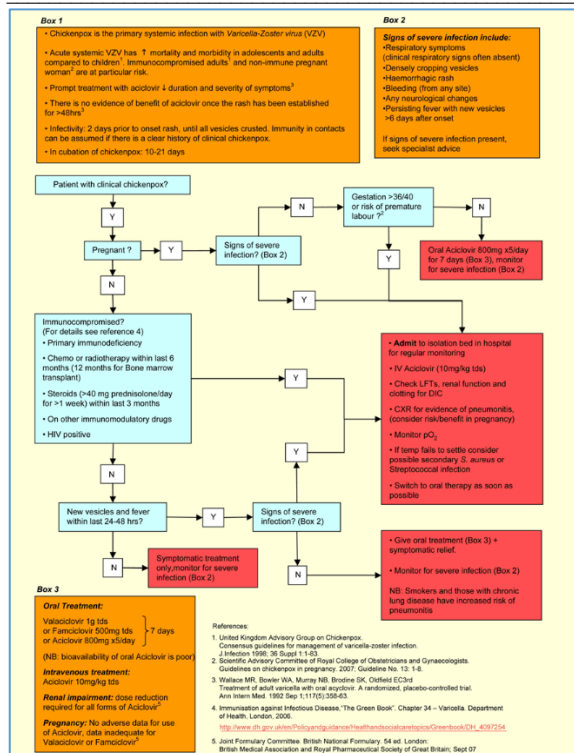


Figure-2: Management of chickenpox in adults.

Adult patients often experience more severe disease and a higher risk of complications, including varicella pneumonia, encephalitis, and hepatitis. In this population, antiviral therapy is advised, ideally initiated within 24 to 48 hours of rash onset. Oral acyclovir or valacyclovir has been shown to reduce both the severity and duration of symptoms when administered early in the course of the disease. For immunocompromised adults or those receiving systemic corticosteroids, intravenous antivirals may be necessary due to the elevated risk of severe disease and complications. Supportive care, including adequate fluid intake, antipyretics, and antihistamines for pruritus, remains integral to management. Postexposure prophylaxis is a critical component of varicella management in high-risk populations, including immunocompromised adults, pregnant women, and susceptible neonates. Varicella-zoster immune globulin (VZIG), administered intramuscularly at 125 U per 10 kg body weight (maximum 625 U), provides passive immunity when given within 96 hours of exposure, with protection lasting approximately three weeks. Intravenous immunoglobulin (IVIG), containing higher concentrations of varicella-specific IgG, is an alternative for postexposure prophylaxis in similar high-risk groups. Oral acyclovir administered prophylactically for one week, starting from 7 to 10 days post-exposure, may also be considered in selected individuals [19]. For immunocompetent individuals aged 12 months and older who lack immunity, postexposure vaccination within 72 to 120 hours can prevent or mitigate disease. This live attenuated vaccine stimulates active immunity,

reducing the severity of breakthrough infections and contributing to overall disease control. Management of varicella thus integrates isolation, symptomatic care, antiviral therapy, and targeted immunoprophylaxis to limit transmission, reduce complications, and protect vulnerable populations [20].

Differential Diagnosis

Varicella, or chickenpox, must be distinguished from several other dermatologic conditions that are presented with febrile exanthems and vesicular eruptions. Among these, monkeypox and smallpox are historically significant due to overlapping clinical features such as fever, myalgia, chills, headache, and generalized rash. Despite these similarities, careful assessment of lesion characteristics and progression allows differentiation. In varicella, lesions typically appear asynchronously, with multiple stages of development—macules, papules, vesicles, pustules, and crusts—present simultaneously. This hallmark contrasts with monkeypox, in which lesions are generally uniform in stage, progressing synchronously across the affected areas [21]. Smallpox, although eradicated in natural populations, presents a diffuse, synchronous vesicular eruption. Recognition of these temporal and morphological patterns is critical for accurate diagnosis, particularly in regions where monkeypox remains endemic. Clinical overlap between monkeypox and varicella can occasionally result in diagnostic uncertainty, and reports of coinfection have emerged, particularly in Central African countries, emphasizing the importance of comprehensive assessment and laboratory confirmation [22][23]. Laboratory testing, including polymerase chain reaction (PCR) and viral culture, can distinguish these infections by identifying the specific viral genome. Serological assays may also support diagnosis by demonstrating recent or prior exposure.

Beyond these poxviruses, varicella must be differentiated from other vesicular or pustular eruptions of viral, bacterial, or inflammatory origin. Vesicular exanthems caused by coxsackieviruses, including hand-foot-and-mouth disease, can mimic early varicella lesions but often exhibit a predilection for the distal extremities and oral mucosa rather than the centrifugal distribution seen in chickenpox. Disseminated herpes simplex virus infection, while typically more localized, may be presented with widespread vesicles in immunocompromised patients, requiring PCR for definitive identification. Other inflammatory or infectious conditions that resemble varicella include pityriasis lichenoides et varioliformis acuta, rickettsialpox, and bullous reactions from insect bites. Drug eruptions can produce vesicular or pustular lesions and must be considered in patients with recent medication exposure, while scabies may induce excoriated papules and vesicles, often concentrated in interdigital spaces and flexural areas. Accurate differentiation relies on integrating clinical features, lesion morphology, distribution, temporal

progression, patient history, and relevant laboratory testing. Correct identification not only guides appropriate management but also informs infection control measures, particularly for highly transmissible or potentially severe viral exanthems. Vigilance is essential in distinguishing varicella from these alternative diagnoses, particularly in regions where multiple vesicular illnesses may co-circulate [22][23].

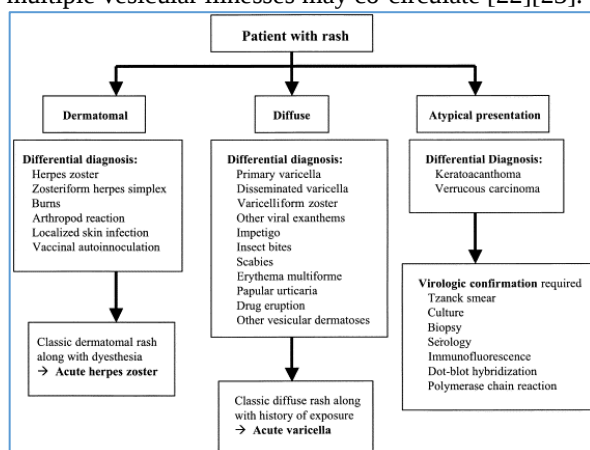


Figure-4: Differential diagnosis of rash.

Prognosis

In immunocompetent children, the prognosis of varicella is generally excellent. The disease is self-limiting, with full recovery expected within two to four weeks. Complications in this population are uncommon and usually mild, including secondary bacterial skin infections or transient febrile episodes. Following primary infection, most children develop durable immunity mediated by IgG antibodies, which provides long-term protection against subsequent symptomatic disease. In contrast, varicella carries significantly higher morbidity in immunocompromised individuals, including those with congenital or acquired immune deficiencies, patients receiving immunosuppressive therapy, or those with hematologic malignancies. In these populations, complications such as varicella pneumonia, encephalitis, hepatitis, or severe skin involvement are more frequent and can be life-threatening. Adults, particularly pregnant women, also experience more severe disease compared to children, with pneumonia representing the most serious complication. Although reinfections are rare in healthy children, cases of clinical varicella following prior infection have been documented, typically presenting with milder symptoms than primary infection. These occurrences suggest that while immunity is generally robust, it may not be absolute, and exposure to VZV can occasionally result in subclinical or symptomatic reinfection [24].

Complications

Varicella, or chickenpox, is typically a mild and self-limiting illness in healthy children, with the vast majority recovering without sequelae. However, in adolescents, adults, pregnant women, and immunocompromised individuals, the disease can be

considerably more severe and is associated with a higher risk of complications. In adults, the illness is characterized by more extensive vesicular eruptions, higher fevers, and prolonged symptom duration, with the potential for systemic involvement that can be life-threatening. One of the most common complications is secondary bacterial infection of the skin. Lesions may become infected with bacteria such as *Staphylococcus aureus* or *Streptococcus pyogenes*, leading to cellulitis, impetigo, or erysipelas. These infections can result in scarring, which tends to be deeper and more pronounced in adults due to the severity of skin involvement. Varicella pneumonia represents a significant risk in adults, with reported mortality ranging from 10% to 30% in untreated cases. Patients may be present with dyspnea, cough, and hypoxia, necessitating prompt antiviral therapy and supportive respiratory care [25].

Reactivation of latent varicella-zoster virus is another important sequela. Individuals with prior varicella infection have an estimated 20% lifetime risk of developing herpes zoster. Rarely, concurrent primary varicella and zoster can occur, sometimes accompanied by neurological manifestations such as facial nerve palsy [25]. Central nervous system involvement in varicella is infrequent, occurring in less than one per 1,000 cases, but can include serious conditions such as encephalitis, acute cerebellar ataxia, and Guillain-Barré syndrome. VZV may also induce vasculopathy affecting cerebral and cervical arteries, increasing the risk of stroke. Reye syndrome, historically associated with aspirin use during varicella infection, has become rare due to widespread avoidance of aspirin in pediatric populations. Maternal varicella poses significant fetal and neonatal risks. Infection during the first 20 weeks of gestation carries approximately a 2% risk of congenital varicella syndrome, which may manifest as low birth weight, cutaneous scarring, ocular defects, cortical atrophy, hypoplastic limbs, and psychomotor delay. Children born to mothers infected during pregnancy may develop herpes zoster early in life without experiencing extrauterine varicella. Neonatal varicella can occur when maternal infection arises between five days before and two days after delivery, often resulting in severe illness due to the absence of maternal antibody protection. Maternal herpes zoster generally poses minimal risk to the neonate, likely due to passive immunity conferred by maternal antibodies [13]. Immunocompromised individuals are particularly high risk of severe varicella. These patients often develop widespread and atypical cutaneous eruptions, including hemorrhagic or purpuric lesions, with systemic involvement of the lungs, liver, and central nervous system. Additional fewer common complications may include thrombocytopenia, hepatitis, glomerulonephritis, optic neuritis, keratitis, arthritis, myocarditis, pancreatitis, orchitis, and vasculitis. Collectively, these complications

underscore the importance of early recognition, vigilant monitoring, and prompt antiviral and supportive interventions in high-risk populations [13].

Patient Education

Patient education plays a crucial role in reducing the morbidity associated with varicella infection and preventing secondary complications. Parents and caregivers of children diagnosed with chickenpox should be instructed to maintain proper hygiene and minimize behaviors that increase the risk of skin injury or infection. Keeping the child's fingernails short can reduce scratching, which helps prevent bacterial superinfections such as cellulitis or impetigo. Additionally, caregivers should avoid administering aspirin to young children during febrile episodes due to the risk of Reye syndrome, a rare but potentially fatal complication. Supportive measures to relieve discomfort, such as applying cold compresses to pruritic areas and using moisturizers to prevent skin dryness and irritation, should also be encouraged. Clinicians should emphasize the importance of vaccination as the most effective preventive strategy. Routine immunization of children with the varicella vaccine significantly reduces the incidence of disease and its associated complications. During clinical encounters, the characteristic rash and fever of varicella can be identified, and careful evaluation may reveal early signs of pulmonary involvement, neurological complications, or bacterial superinfection. Early recognition allows for prompt intervention and reduces the risk of severe outcomes [13].

For adult populations, education should include the importance of herpes zoster vaccination. The Advisory Committee on Immunization Practices in the United States recommends vaccination for all adults over 60 years of age to prevent shingles, particularly in individuals who experienced chickenpox during early childhood. Approximately one in five adults will develop herpes zoster, with immunocompromised individuals at higher risk. Shingles is most common in adults over 60 and can occur in those who had chickenpox before the age of one. Educating patients that herpes zoster can transmit varicella to nonimmune individuals through direct contact with vesicular fluid reinforces the importance of isolation and hygiene measures. Overall, patient education should focus on preventive strategies, early recognition of complications, safe symptomatic management, and vaccination awareness. Empowering parents and adult patients with this knowledge not only mitigates the severity of infection but also limits transmission, protecting vulnerable populations within the community [13].

Other Issues

Management of varicella in immunocompetent children is primarily supportive, focusing on symptom relief and prevention of secondary complications. Standard measures include antipyretics such as acetaminophen to control fever,

antihistamines to reduce pruritus, application of calamine lotion, and tepid baths to soothe irritated skin. These interventions aim to improve comfort while the immune system clears the virus. Although antiviral therapy can reduce the risk of severe complications, its routine use is not indicated for otherwise healthy children, as the disease is typically self-limiting and the benefits of antiviral treatment are limited. Antiviral therapy is recommended, however, for healthy adolescents and adults, children with chronic dermatological or pulmonary conditions, or those receiving medications such as salicylates, inhaled corticosteroids, or intermittent oral corticosteroids. Early initiation of antivirals can mitigate the severity and duration of varicella and reduce the likelihood of complications in these higher-risk populations [26]. Pregnant women represent a special group in which varicella can have serious consequences. Infection during pregnancy carries three primary risks: maternal varicella complications, congenital varicella syndrome in the fetus, and increased susceptibility to herpes zoster later in life. Nonimmune pregnant women exposed to VZV should receive prophylaxis with anti-VZV immunoglobulins within ten days of exposure, administered intravenously at 0.5 to 1 ml/kg. Maternal varicella occurring within seven days before or after delivery can result in severe neonatal varicella. In such cases, timely administration of anti-VZV immunoglobulins to the newborn within ten days is essential. For preterm infants born before 28 weeks of gestation or weighing less than 1,000 g at birth, the prophylactic window extends to 21 days to provide adequate protection [26].

Vaccination remains the cornerstone of varicella prevention. The live attenuated varicella vaccine (Oka strain), approved by the United States Food and Drug Administration in 1995, demonstrates high efficacy, achieving seroprotection rates of approximately 85% after a single dose and 99% following two doses in healthy pediatric populations. The recommended two-dose schedule, administered at 12 to 15 months and 4 to 6 years, enhances long-term protection and reduces the risk of waning immunity. Despite its effectiveness, the vaccine is contraindicated in specific populations. Administration is not recommended during pregnancy or in individuals with significant immunosuppression, including those with hematologic malignancies, advanced HIV infection (CD4⁺ T cell levels <15% or <200 cells/ μ L), other T cell deficiencies, or ongoing systemic immunosuppressive therapy, including high-dose corticosteroids or tumor necrosis factor inhibitors [27][28]. Overall, varicella management integrates supportive care, targeted antiviral therapy for at-risk groups, immunoglobulin prophylaxis in exposed neonates and pregnant women, and vaccination to prevent infection and reduce disease burden. These measures collectively address both individual patient outcomes and broader public health considerations,

emphasizing the importance of early intervention, risk stratification, and adherence to immunization guidelines [27][28].

Enhancing Healthcare Team Outcomes

Varicella, commonly known as chickenpox, is a highly contagious disease primarily transmitted via inhalation of aerosolized droplets from infected individuals. The majority of cases occur in children under the age of ten, in whom the disease is generally mild and self-limiting. Most children recover without complications, with fever and pruritic vesicular rash resolving within two to four weeks. However, certain populations—including immunocompromised individuals, neonates, and adults—experience higher morbidity and mortality rates, making careful management crucial [29][30][31]. Effective management of varicella requires a coordinated, patient-centered, interprofessional approach. Physicians and advanced practice clinicians are responsible for accurate diagnosis, early detection of complications, and formulation of individualized treatment plans. These plans may include antiviral therapy, symptom management, isolation guidance, and vaccination counseling. Physicians must also identify high-risk patients, such as immunocompromised children or seronegative pregnant women, who require closer monitoring or referral to specialists [29][30][31]. Nurses play a pivotal role in direct patient care, symptom monitoring, and education. They provide hands-on support, including the application of topical treatments, monitoring hydration and fever, and preventing secondary infections through hygiene measures. Nurses also educate patients and caregivers about infection control strategies, such as isolation, hand hygiene, and environmental cleaning, which are critical in limiting the spread of VZV within households and healthcare settings. Pharmacists contribute to safe and effective care by ensuring appropriate dosing of antiviral medications, monitoring for drug interactions, supporting adherence, and advising on vaccination protocols for patients and at-risk populations. Infection control specialists and public health professionals assist with outbreak management, contact tracing, and community vaccination campaigns, mitigating broader transmission risks [29][30][31].

Health information workers play a crucial and often underrecognized role in optimizing team outcomes. They manage and analyze patient data, maintain accurate electronic health records, and facilitate communication across the healthcare team. By ensuring that vaccination history, laboratory results, and antiviral treatment records are up to date, health information professionals support evidence-based decision-making and continuity of care. They also contribute to public health surveillance by reporting varicella cases, identifying trends, and aiding in the rapid identification of outbreaks. Their

expertise enhances workflow efficiency, reduces errors, and supports data-driven interventions for both individual patients and population-level disease control [29][30][31]. Education remains a cornerstone of interprofessional management. Parents of children with varicella should be instructed to trim fingernails to prevent scratching, avoid administering aspirin due to the risk of Reye syndrome, and use cold compresses or moisturizers to alleviate itching and dryness [32][33][34]. Clinicians should emphasize the importance of vaccination, which is highly effective in preventing severe disease and its associated complications. Immunocompromised children require specialized care under the guidance of infectious disease specialists, and seronegative pregnant women should avoid exposure to infected individuals. Collaborative care teams are essential for pregnant patients who develop varicella, as coordinated management decisions ensure both maternal and fetal safety. Interprofessional collaboration also improves outcomes when managing complications. Physicians, nurses, pharmacists, and allied health professionals must maintain open communication and shared decision-making with patients and families. This promotes trust, ensures informed choices, and allows early intervention for complications such as secondary bacterial infections, pneumonia, or neurological sequelae. By integrating clinical expertise, ethical responsibility, and communication, healthcare teams optimize patient safety and quality of care while supporting public health initiatives. A holistic, team-based approach enhances patient outcomes, reduces disease severity, and strengthens community-wide efforts to control varicella transmission [32][33][34].

Conclusion:

In conclusion, the management of varicella-zoster virus infection demands a cohesive and interdisciplinary strategy to address its wide clinical spectrum. While chickenpox is typically a mild, self-limiting illness in children, it can lead to severe complications in adolescents, adults, and immunocompromised hosts. The cornerstone of effective management includes timely diagnosis, appropriate use of antiviral medications for at-risk populations, and robust supportive care to alleviate symptoms and prevent secondary infections. Crucially, vaccination remains the most powerful public health tool, significantly reducing the incidence and severity of both primary varicella and herpes zoster. Ultimately, optimal patient outcomes are achieved through a synergistic model where physicians, nurses, pharmacists, and health information professionals collaborate seamlessly. This team-based approach ensures comprehensive care, from accurate diagnosis and treatment to patient education, infection control, and systematic surveillance, thereby mitigating the individual and societal burden of VZV.

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