PERINATAL JOURNAL

Original Article

Perinatal Journal 2026; 34(1):1179-1184

https://doi.org/10.57239/prn.26.034100118

# Varicella (Chickenpox) in immunocompromised patients

**Adhyanti[1*], Musofa Rusli[2]**

*[1,2]Universitas Airlangga, Indonesia*

**Abstract**

Varicella, commonly known as chickenpox, is a viral disease caused by the varicella-zoster virus and is easily transmitted from person to person. While the infection usually resolves without significant consequences in individuals with normal immune function, it can become severe and potentially fatal in adults and those with compromised immunity. This report presents the case of a 34-year-old man with long-term glucocorticoid therapy who developed an extensive vesicular rash accompanied by systemic symptoms such as fever, generalized weakness, nausea, and vomiting. Clinical evaluation revealed widespread vesicles with areas of crusting and erosion involving the face and body. Laboratory investigations identified reduced platelet counts, abnormalities in electrolyte levels, and substantially increased liver enzyme values. Imaging of the chest showed pulmonary infiltrates, and cytological examination using the Tzanck test demonstrated multinucleated giant cells, supporting a diagnosis of varicella, while testing for monkeypox was negative. The patient was diagnosed with complicated varicella associated with electrolyte imbalance and liver enzyme elevation. Treatment consisted of oral acyclovir along with supportive measures and correction of electrolyte disturbances. Marked clinical and biochemical improvement was noted within six days, allowing discharge in good condition. This case underscores the critical role of timely diagnosis and early antiviral intervention in immunocompromised patients to reduce the risk of serious complications and achieve favorable outcomes.

**Keywords:** Varicella zoster virus, Immunocompromised patient, Adult chickenpox, Acyclovir therapy

## Introduction

Chickenpox, also known as varicella, is a highly transmissible viral illness marked by a characteristic vesicular rash and general systemic complaints. It is caused by the VZV, which produces chickenpox during primary infection in individuals without immunity and may later reactivate as shingles. The disease typically presents with an itchy rash consisting of small fluid-filled blisters. Systemic manifestations such as fever, tiredness, sore throat, and headache commonly accompany the rash and persist for approximately five to seven days. Although chickenpox is often mild in children, adults tend to experience more pronounced and severe symptoms (Izikson L., Lilly E., 2009).

Meyer and colleagues reported that in the 25-year period preceding the availability of the varicella vaccine in 1995, varicella-related mortality accounted for approximately 47 to 138 deaths annually, corresponding to 0.29–0.46 deaths per million people. Data from 1990 to 1994 showed that adults aged 20 years and older represented the majority of fatalities, accounting for about 54% of deaths. Following the implementation of widespread VZV vaccination programs, age-specific mortality rates associated with varicella declined substantially, with an overall reduction of approximately 66% (Nguyen HQ, et al., 2005).

Adults and individuals with compromised immune systems tend to develop more severe clinical manifestations and complications of varicella. Those with underlying conditions such as malignancy, prolonged corticosteroid use, immunosuppressive treatment, HIV infection, or a history of organ transplantation are particularly vulnerable to disseminated disease as a result of impaired cell-mediated immunity. Compared with immunocompetent individuals, immunocompromised patients with varicella face significantly higher risks of serious morbidity and mortality. Although this population represents only about 0.1% of all varicella cases (Strauss SE, et al., 1988).

Varicella can present with atypical, more severe clinical features, including prolonged formation of vesicular lesions that may persist for several weeks, extensive skin involvement with large or hemorrhagic blisters, and serious systemic complications. Possible complications include lung involvement caused by varicella as well as

**Correspondence:** Adhyanti, Universitas Airlangga, Indonesia, **e-mail:** adhyantiantz@gmail.com, **Received:** November 27, 2025 **Accepted:** January 8, 2025

widespread systemic infection; in more serious situations, these conditions may worsen and lead to the development of disseminated intravascular coagulation (Heininger U., 2006).

## Case report

A 34-year-old male patient, referred to as Mr. P, presented to the emergency department of Dr. Soetomo Hospital with a one-week history of fluid-filled blisters involving the face and subsequently spreading to the entire body. The lesions initially appeared on the face before becoming generalized. The blisters were not pruritic or warm but were associated with pain around the lips. Two days prior to admission, the patient also developed fever, generalized weakness, and nausea accompanied by vomiting up to five times per day, consisting of food contents. He additionally reported decreased urine output, while bowel movements remained normal. The patient denied any recent international travel or contact with individuals who had traveled abroad. He was unsure about his childhood history of varicella vaccination. His medical history was notable for allergic contact dermatitis affecting both hands for the past five years. He routinely received treatment at RSI Jemursari and had a history of long-term methylprednisolone use at a dose of 4 mg once daily. When his prescribed medication was unavailable, he occasionally obtained similar medication from a pharmacy without medical consultation.

On physical examination, the patient was fully conscious (compos mentis). The patient weighed 60 kg and measured 155 cm in height, resulting in a body mass index of 25 kg/m$^2$. Examination of the head and neck revealed non-anemic conjunctiva, absence of scleral icterus, no signs of dyspnea or cyanosis, a positive moon face appearance, and enlargement of the right submandibular gland. Thoracic inspection demonstrated a symmetrical chest with equal expansion and no retractions. Palpation and percussion showed normal tactile fremitus and resonant percussion notes, with no evidence of cardiomegaly. Auscultation revealed normal vesicular breath sounds bilaterally without rhonchi or wheezes, as well as normal single S1 and S2 heart sounds without murmurs or gallop rhythms.

Abdominal examination showed a distended abdomen without visible collateral veins. Bowel sounds were normal on auscultation, and percussion revealed a tympanic note throughout the abdomen. Abdominal palpation did not reveal enlargement of the liver or spleen, and there was no evidence of abdominal tenderness. Dermatological examination demonstrated widespread erythematous macules accompanied by crusting and erosions involving the face, anterior and posterior thorax, abdomen, as well as the upper and lower extremities.

Laboratory investigations revealed increased hemoglobin and hematocrit values, while red blood cell indices remained normal. White blood cell counts were within reference limits, although a reduced platelet count was noted. The differential count showed a predominance of neutrophils accompanied by a relative decrease in lymphocytes. Random blood glucose levels, kidney function tests, and serum creatinine were unremarkable. Evaluation of electrolytes demonstrated decreased sodium and potassium concentrations, along with a mildly reduced chloride level. Hepatic function tests showed significant elevations in transaminases, whereas serum albumin levels were preserved. Both total and direct bilirubin values remained within acceptable ranges. Coagulation profiles indicated a prolonged prothrombin time, with a normal activated partial thromboplastin time. Serologic screening for hepatitis B, hepatitis C, hepatitis A, and HIV was negative. Arterial blood gas analysis suggested mild respiratory alkalosis with satisfactory oxygenation. Chest radiography demonstrated patchy infiltrates in the right paracardial region, suggestive of pulmonary inflammation. Cytological examination using the Tzanck test identified multinucleated giant cells, supporting a viral etiology. In addition, a monkeypox PCR swab test was performed and yielded negative results.

Based on the clinical history, physical findings, and diagnostic investigations, the patient was initially assessed as having suspected monkeypox, with differential diagnoses including varicella zoster infection accompanied by hypokalemia (2.8 mmol/L), hypotonic hypovolemic hyponatremia (129 mmol/L), and significantly elevated liver transaminases (AST 787 U/L, ALT 929 U/L) requiring further evaluation. The patient was admitted to an isolation unit in the emergency department, and diagnostic plans included Polymerase Chain Reaction (PCR) testing for monkeypox and a Tzanck smear

examination. Initial management consisted of a high-calorie, high-protein diet providing 2,100 kcal per day, intravenous fluids (WIDA KN2: RD 5 1:1 at 1,000 mL over 24 hours), metoclopramide injections at 10 mg three times daily, oral acyclovir 800 mg five times daily, paracetamol 500 mg three times daily, curcuma tablets three times daily, potassium slow-release 600 mg three times daily, N-acetylcysteine 600 mg twice daily, and topical sodium fusidate cream applied twice daily to erosive skin lesions.

On the second day of hospitalization, the monkeypox PCR test returned negative, while the Tzanck test demonstrated multinucleated giant cells, confirming a diagnosis consistent with varicella zoster infection.

By the fourth day of therapy, the patient showed marked clinical improvement, with no appearance of new skin lesions and significant drying and crust formation of the existing ones. Gastrointestinal symptoms had resolved, as the patient no longer experienced nausea or vomiting and was able to tolerate oral intake well. Subsequent laboratory assessments indicated a general trend toward hematologic stabilization. Hemoglobin and hematocrit values shifted closer to physiological limits, while red cell indices showed no significant variation. White blood cell counts remained slightly above normal, accompanied by an increase in platelet numbers. Analysis of the leukocyte differential revealed a decline in neutrophil dominance alongside a proportional increase in lymphocytes. Differential counts showed neutrophils at 60.9% and lymphocytes at 24.2%. Serum glucose levels were within the lower end of the normal range. Mild electrolyte imbalances persisted, characterized by reduced sodium and chloride concentrations with potassium remaining near normal limits. Liver function demonstrated significant improvement, as reflected by decreased transaminase levels, while serum albumin was slightly reduced. Bilirubin levels were within normal limits, including the direct fraction. Antiviral therapy with acyclovir was continued at the same dosing schedule, accompanied by ongoing intravenous fluid administration to correct electrolyte disturbances (WIDA KN2: RD 5 1:1 at 1,000 mL over 24 hours).

On the sixth day of treatment, repeat electrolyte analysis showed further improvement. All vesicular lesions had completely crusted, the patient reported no nausea or vomiting, and overall clinical condition was significantly improved. The patient was discharged in stable condition for outpatient management, with oral acyclovir continued until day seven. A follow-up evaluation two days after discharge revealed no residual complaints.

## Discussion

VZV infection may manifest either as a primary illness, or as a secondary reactivation referred to as herpes zoster or shingles. Transmission occurs via direct contact with vesicular fluid or through airborne spread beginning one to two days before the appearance of the rash. In individuals with impaired immune function, viral shedding and infectivity may persist for several weeks. After binding to specific receptors on host cells, the virus enters the cytoplasm and initially replicates within regional lymph nodes, leading to a first, subclinical phase of viremia that lasts approximately 4–6 days. A second viremic phase typically occurs around two weeks after infection and may persist for 10–21 days, reflecting viral dissemination to the nasopharynx and skin and the subsequent development of the characteristic maculopapular and vesicular rash (Heininger Seward, 2006). The ability of the virus to spread ends when every skin lesion has formed a crust. Although the infection has been widely investigated, the exact roles played by antibody-mediated and cellular immune responses in the development of disease and in limiting varicella-zoster virus infection are still not fully clarified (Levin et al., 2016).

*"The patient complained of blisters appearing on the face, then spreading throughout the body. The blisters were not itchy or hot, but felt painful around the lips. The patient also complained of fever, weakness, and vomiting 5 times a day since 2 days before admission to the hospital. Physical examination revealed multiple vesicles with erosion and crusting."*

*Immunocompromised* states reduce immune function to varying degrees, thereby increasing vulnerability to infections and other illnesses. These conditions may be congenital, acquired, or iatrogenic, resulting from medical interventions designed to intentionally suppress immune responses. Prolonged glucocorticoid therapy, as observed in this patient, is classified as an iatrogenic cause of immunodeficiency (Mack, 2014).

In people with normal immune function, varicella is generally a self-resolving disease marked by low-grade fever, general discomfort, and an intensely itchy rash. Subtle early symptoms often appear one to two days prior to the skin manifestations. The infection is characteristically identified by a pruritic rash that first emerges on the scalp or facial area and then progressively extends to the torso and limbs. The skin lesions develop sequentially, starting as red flat spots that advance into raised papules. Over time, these blisters turn pustular and subsequently dry out. In adults, varicella is usually more widespread, often presents with more pronounced systemic manifestations, and carries a greater likelihood of complications than infections seen in children (Grotto et al., 2008).

Individuals with underlying conditions such as malignancy, prolonged corticosteroid use, immunosuppressive treatment, HIV infection, or a history of solid organ transplantation are at increased risk of developing disseminated varicella as a result of impaired cell-mediated immunity. Compared with immunocompetent individuals, patients with compromised immune function who develop varicella face substantially higher rates of severe morbidity and mortality. In this population, the disease may present with atypical and aggressive features, including prolonged emergence of vesicular lesions lasting for weeks, extensive or hemorrhagic skin involvement, varicella-associated pneumonia, and severe systemic dissemination that may progress to complications such as disseminated intravascular coagulation (Heininger U., 2006).

*"The patient had a history of glucocorticoid use over the past 5 years, which classified them as immunocompromised. The patient exhibited longer-lasting and more severe symptoms compared to immunocompetent patients, including transaminitis and electrolyte disturbances. However, no signs of secondary infection, meningitis, pneumonia, or coagulation abnormalities were observed during the course of the illness."*

VZV infection can be supported by exposure to VZV, the presence of a characteristic rash, particularly with a dermatomal distribution, and serological testing. Clinically, varicella shares similarities with herpes simplex infection; however, herpes simplex is distinguished by its much higher tendency for recurrence. In cases where skin lesions are atypical or the clinical presentation of an exanthematous illness is unclear, further diagnostic evaluation is required.

Serologic approaches, including ELISA, indirect fluorescent antibody tests. Of all diagnostic options, polymerase chain reaction analysis of lesion specimens offers the highest sensitivity and specificity. Additionally, histopathologic evaluation together with PCR testing of samples such as blood can help confirm VZV involvement in internal organs, including cases of pneumonitis, encephalitis, or retinitis. In contrast, cytologic techniques have limited reliability in the diagnosis of VZV infection (Leung et al., 2010).

The Tzanck test is a diagnostic procedure in which material is gently scraped from the base of a cutaneous lesion to identify distinctive cytological features. Although it is a rapid and inexpensive test, its sensitivity is relatively low, approximately 60%, compared with more advanced diagnostic techniques, and it cannot differentiate between VZV and HSV. The presence of Tzanck cells, which are multinucleated giant cells, is not specific to varicella and may also be observed (Gershon et al., 2015).

*"The Tzanck test is performed on patients showing giant cell findings. Varicella diagnosis is established clinically and by a positive Tzanck test, but viral culture or PCR is not performed due to cost limitations."*

The management of varicella is primarily supportive and focuses on relieving symptoms and preventing complications. During the contagious period, infected individuals are generally advised to remain at home to reduce transmission. Measures such as keeping fingernails trimmed and wearing gloves can help minimize scratching and lower the risk of secondary bacterial infection. Pruritus may be alleviated with topical agents such as calamine lotion, while daily bathing with warm water supports skin hygiene and helps prevent superimposed infections. In people who are at increased risk of developing severe illness and who have experienced substantial exposure to the virus, passive immunization with intramuscular varicella-zoster immunoglobulin, which contains high titers of antibodies against VZV, may be administered to reduce the likelihood or severity of infection (Hayward K., 2018; Jam et al., 2018).

Varicella-zoster virus infection in individuals older than 13 years is linked to a higher risk of both morbidity and mortality. As a result, antiviral therapy is advised for individuals in this population, including healthy adolescents and adults without underlying conditions. For patients with normal immune function, oral antivirals such as acyclovir, famciclovir, or valacyclovir. Since prompt treatment has been demonstrated to lessen both the length and intensity of fever and skin manifestations. Treatment courses of five and seven days have demonstrated comparable effectiveness. In patients with compromised immune function, antiviral therapy is essential, with acyclovir, famciclovir, or valacyclovir forming the cornerstone of management to prevent severe disease and complications (Balfour H.H., 2001).

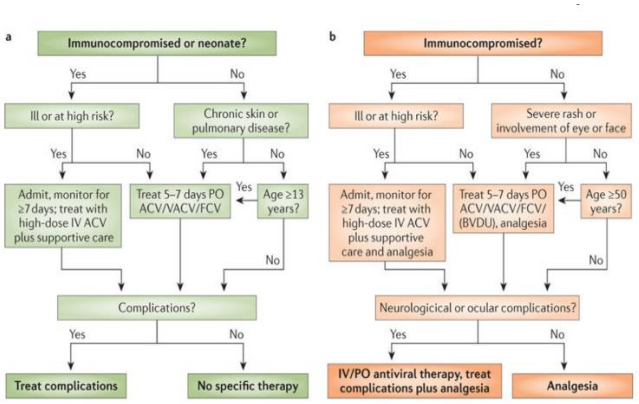


**Figure 1.** Antiviral therapy for VZV infection

For uncomplicated cases of varicella, antiviral therapy may be administered using oral agents such as valacyclovir or famciclovir for an appropriate treatment period. Oral acyclovir can also be considered, with dosing adjusted according to body weight and clinical condition. In immunocompromised patients who develop severe varicella, intravenous acyclovir is recommended as the primary treatment. After clear clinical improvement and in the absence of visceral organ involvement, therapy can be transitioned from intravenous administration to oral antiviral medication (Wittek et al., 2010).

Treatment failure due to resistance of VZV to acyclovir is uncommon but should be considered when there is no clinical improvement within approximately 10 days of initiating therapy or when unusual cutaneous manifestations, such as verrucous lesions, are observed. In such cases, viral culture is recommended to confirm ongoing viral replication. If the culture yields positive results, antiviral susceptibility testing should be performed to guide alternative treatment options. For patients with confirmed or suspected acyclovir-resistant VZV infection, foscarnet is an effective alternative antiviral agent and may be administered as rescue therapy (Seang et al., 2014).

*"The patient showed significant improvement after receiving acyclovir tablets 5x800 mg for 7 days. Therefore, it can be concluded that there is no resistance to acyclovir (treatment failure)."*

## Conclusion

A 34-year-old man was admitted with a one-week history of cutaneous lesions accompanied by generalized weakness. On examination, the skin showed extensive crusting and erosions. Evaluation of clinical findings and laboratory results led to a diagnosis of varicella complicated by electrolyte disturbances. The patient demonstrated significant improvement with appropriate treatment during hospitalization. Although the short-term prognosis was favorable, the patient remains at potential risk for future reactivation of the virus in the form of herpes zoster..

## References

Balfour HH, et al. 2001. *Controlled Trial of Acyclovir for Chickenpox Evaluating Time of Initiation and Duration of Therapy and Viral Resistance*. Pediatr Infect Dis J. 20:919–926. [PubMed: 11642624]

Gershon, A. A., Breuer, J., Cohen, J. I., et al. 2015. *Varicella zoster virus infection*. Nat Rev Dis Primers, 1, 15016.

Grotto, I., Balicer, R. D., Smetana, Z., et al. 2008. *Immunity to Varicella Zoster Virus Among Young Adults: A Decline Prior to Widespread Uptake of Varicella Vaccines*. Infection, 36, 130-4.

Hayward K., Cline A., Stephens A., Street L. 2018. *Management of Herpes Zoster (shingles) During Pregnancy.* J Obstet Gynaecol. Oct;38(7):887-894. [PubMed: 29565203]

Heininger U., Seward J.F. 2006. *Varicella*. Lancet;

368:1365.

Jam, F. A., Singh, S. K. G., Ng, B. K., & Aziz, N. (2018). The interactive effect of uncertainty avoidance cultural values and leadership styles on open service innovation: A look at malaysian healthcare sector. International Journal of Business and Administrative Studies, 4(5), 208.

Izikson L., Lilly E. (2009). *Primary Varicella in an Immunocompetent Adult*, J Clin Aesthetic Dermatol; 2(8):36–38.

Leung, J., Harpaz, R., Baughman, A. L., Heath, K., Loparev, V., Vazquez, M., Watson, B. M. & Schmid, D. S. 2010. *Evaluation of Laboratory Methods for Diagnosis of Varicella*. Clin Infect Dis, 51, 23-32.

Levin, M. J., Weinberg, A., & Schmid, D. S. 2016. *Herpes Simplex Virus and Varicella-Zoster Virus*. Microbiol Spectr, 4.

Meyer P.A., Seward J.F., Jumaan A.O., Wharton M. 2000. *Varicella Mortality: Trends Before Vaccine Licensure in The United States, 1970–1994*. J Infect Dis; 182:383–390.

Nguyen H.Q., Jumaan A.O., Seward J.F. 2005. *Decline in Mortality Due to Varicella After Implementation Of Varicella Vaccination in The United States*. N Engl J Med; 352(5):450–458.

Rice M.E., Bannerman M., Marin M., Lopez A.S., Lewis M.M., Stamatakis C.E., Regan J.J. 2018. *Maritime Varicella Illness and Death Reporting, U.S., 2010-2015*. Travel Med Infect Dis; 23:27-33. [PubMed: 29621623]

Seang, S., Boutolleau, D., Burrel, S., et al. 2014. *Long-term follow-up of HIV-infected patients once diagnosed with acyclovir-resistant herpes simplex virus infection*. Int J STD AIDS, 25, 676-82.

Straus S.E., Ostrove J.M., Inchauspé G., et al. NIH conference. 1988. *Varicella-zoster Virus Infections. Biology, Natural history, Treatment, and Prevention*. Ann Intern Med; 108:221.

Mack, M. 2014. *Diagnosis and Management of Infections in Hospitalized Immunocompromised Patients*. Hospital Medicine Clinics, 3, e378-e395.

Wittek, M., Doerr, H. W., & Allwinn, R. 2010. *Varicella and herpes zoster. Part 2: therapy and prevention*. Med Klin (Munich), 105, 399-403.