Delayed antiviral therapy in adult varicella: Severe infection and aesthetic implications

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SUMMARY

Background: Varicella-zoster virus infection is commonly self-limiting in childhood. However, adults may present with increased severity, higher complication rates, and long-term cutaneous sequelae. Early initiation of antiviral therapy within 72 hours of rash onset is important in reducing disease burden. Case Presentation: A man in his 20s, a college student residing in a campus hostel, presented with a 7-day history of fever, malaise, and a progressively worsening generalised pruritic vesiculopustular rash. He reported recent exposure to a classmate diagnosed with chickenpox and had no prior history of childhood varicella or varicella vaccination. He reported no other relevant medical history. He initially sought medical attention on day 3 of illness and was managed symptomatically, without initiation of antiviral therapy. The clinical condition subsequently deteriorated, with increasing lesion burden, pustulation, and severe pruritus, particularly over the face. On day 7, he was clinically diagnosed with varicella with severe cutaneous involvement complicated by secondary bacterial infection. He was treated with oral valaciclovir, amoxicillin-clavulanate, and bilastine for 5 days. Although acute lesions resolved, extensive post-inflammatory hyperpigmentation and atrophic scarring developed over the face and body, resulting in significant psychosocial distress. Patient-reported outcome assessment (SCAR-Q) demonstrated marked improvement in appearance and symptoms after treatment but persistent psychosocial impact. Conclusion: This case highlights the importance of timely antiviral therapy in adult varicella to reduce complications and long-term aesthetic sequelae. Prompt recognition of secondary bacterial infection and timely antibiotic treatment are essential. Early intervention is particularly important in cases with facial involvement to reduce cosmetic and psychosocial impact.

INTRODUCTION

Varicella-zoster virus (VZV) infection is commonly regarded as a benign, self-limiting illness characterised by a prodromal fever and generalised vesicular rash. However, in adults, the clinical course may be substantially more severe, with higher rates of complications and morbidity — particularly among immunocompromised individuals — although severe disease may also occur in otherwise healthy adults.

Early initiation of antiviral therapy, such as acyclovir or valaciclovir, within 24–72 hours of rash onset is recommended to reduce viral replication, limit disease severity, and prevent complications.[1,2] Delayed presentation or initial under-recognition in primary care may result in missed therapeutic opportunities.

In the absence of timely antiviral therapy, ongoing viral replication may lead to more extensive disease and increased susceptibility to complications, including secondary bacterial infection and, less commonly, systemic involvement such as pneumonia or neurological complications.[2]

We report a case of adult varicella with delayed initiation of antiviral therapy, resulting in severe cutaneous involvement complicated by secondary bacterial infection and subsequent aesthetic sequelae. This case highlights the importance of early recognition and prompt antiviral treatment in primary care to reduce both clinical complications and long-term morbidity.

CASE PRESENTATION

A man in his 20s presented with a 7-day history of fever, malaise, and a generalised pruritic rash. The eruption initially appeared as papules over the trunk and subsequently spread to the back, chest, and face, progressing to vesicles and subsequently pustules. He was a college student residing in a campus hostel. The patient reported recent exposure to a classmate diagnosed with chickenpox. The patient reported no prior history of chickenpox during childhood and had not received varicella vaccination. The patient had no known immunocompromising condition, malignancy, chronic systemic illness, or regular immunosuppressive medication use.

He first sought medical attention on day 3 of illness and was clinically diagnosed with varicella. He was managed symptomatically with antipyretics and antihistamines, without initiation of antiviral therapy.

Despite initial treatment, the condition worsened, with an increasing number of lesions, progression to pustules, and severe pruritus, particularly over the face. He sought a second opinion on day 7 of illness. Cutaneous examination revealed widespread polymorphic lesions involving the face and trunk, comprising papules, vesicles, and pustules at varying stages of development. Multiple lesions demonstrated central crusting, particularly over the facial region. The distribution predominantly involved the face, anterior chest, and

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Table I: Recommended antiviral therapies for adult varicella[2]

Clinical Scenario	Drug	Adult Dose	Duration	Major Toxicities
Immunocompetent patients	Acyclovir	800mg po 5 times daily	5-7 days	None; minor nausea or headache
	Valaciclovir	1g po three times daily	5-7 days	None; minor nausea or headache
	Famciclovir	500mg po three times daily	5-7 days	None; minor nausea or headache
	Brivudin	125mg po once daily	5-7 days	Potentially lethal interaction with fluropyrimidines (e.g, 5-fluorouracil)
Immunocompromised patients / disseminated VZV syndrome (e.g., encephalitis, pneumonitis)	Intravenous Acyclovir	10-15mg/kg every 8 hours	7-10 days	Nephrotoxicity (rare); CNS disturbances
Acyclovir-resistant VZV	Intravenous Foscarnet	60-90mg/kg every 12 hours until healed	>10days	Nephrotoxicity (common); electrolyte disturbance (common), seizure, arrhythmias, anaemia, genital ulcers

Table II: Impact of antiviral therapy timing on clinical and aesthetic outcomes in adult varicella

Timing of antiviral therapy	Clinical outcomes	Aesthetic outcomes
≤72 hours of rash onset	Reduced lesion formation, faster recovery	Reduced risk of post-inflammatory hyperpigmentation (PIH) and scarring
Delayed (>72 hours)	Progressive lesion development and increased risk of complications	Increased risk of PIH, atrophic scarring, and possible keloid formation in susceptible individuals

posterior trunk (Figure 1). No signs of systemic involvement were noted.

A diagnosis of varicella with extensive cutaneous involvement complicated by secondary bacterial infection was established, and the patient was commenced on oral valaciclovir 500 mg twice daily, amoxicillin-clavulanate 625 mg twice daily, and bilastine 20 mg twice daily for 5 days.

The diagnosis was made clinically, based on the characteristic polymorphic eruption consisting of papules, vesicles, pustules, and crusts at different stages of evolution in the presence of fever and malaise. Laboratory confirmation with polymerase chain reaction (PCR) testing was not performed.

At the one-week follow-up, clinical improvement was noted with resolution of active lesions. However, extensive post-inflammatory hyperpigmentation and atrophic scarring developed over the face and body, negatively affecting self-esteem. Patient-reported outcome assessment using the SCAR-Q demonstrated substantial improvement in appearance (25 to 75) and symptom domains (33.3 to 83.3). However, psychosocial scores showed only modest improvement (33 to 41.6), suggesting persistent psychological impact despite clinical recovery. Cutaneous findings at 1 week post-treatment are shown in Figure 2.

At three weeks post-treatment, persistent post-inflammatory hyperpigmentation and atrophic scarring were noted over the face, anterior trunk, and posterior trunk, with no active lesions. These findings are illustrated in Figure 3.

DISCUSSION

Varicella is a highly contagious infection caused by the varicella-zoster virus (VZV), typically presenting with a prodrome of pyrexia and malaise, followed by a polymorphic eruption consisting of macules, papules, vesicles, and crusts at different stages of evolution, with possible residual scarring. Although generally self-limiting, varicella in adults is associated with a more severe clinical course, increased lesion burden, and a higher risk of complications.[1]

In routine primary care practice, varicella is commonly diagnosed clinically. Laboratory confirmation is generally reserved for atypical presentations, immunocompromised patients, or cases where the diagnosis is uncertain. Antiviral therapy should not be delayed while awaiting confirmatory testing when clinical suspicion is high.[1,2] The patient in this case presented with clinical features consistent with varicella; however, initiation of antiviral therapy was delayed. Although varicella vaccination is cost-effective for prevention, it has no role in the treatment of established infection. In symptomatic patients, antiviral therapy remains essential. In Malaysia, although there is no single unified national guideline dedicated exclusively to uncomplicated varicella, current practice in primary care is aligned with Ministry of Health formularies and international recommendations. Oral acyclovir remains the mainstay of treatment and is commonly prescribed at a dose of 800 mg five times daily in adults. Recommended antiviral therapies for adult varicella are summarised in Table I.

Early initiation of antiviral therapy is critical, as viral replication typically peaks within the first 24–72 hours of rash onset. Initiation of treatment within this window has been shown to reduce the duration of fever, shorten the time to crusting from 7.4 to 5.6 days (approximately 2 days), reduce the peak lesion count by 46%, and decrease overall disease severity.[3,4] In this case, delayed antiviral initiation beyond the optimal therapeutic window likely contributed to increased lesion burden and subsequent aesthetic sequelae. The impact of timing of antiviral therapy on clinical and aesthetic outcomes is summarised in Table II.[3,4]

Delayed initiation of antiviral therapy results in diminished therapeutic efficacy. Continued viral replication leads to progression of cutaneous lesions and increased inflammatory burden, contributing to more extensive disease involvement.

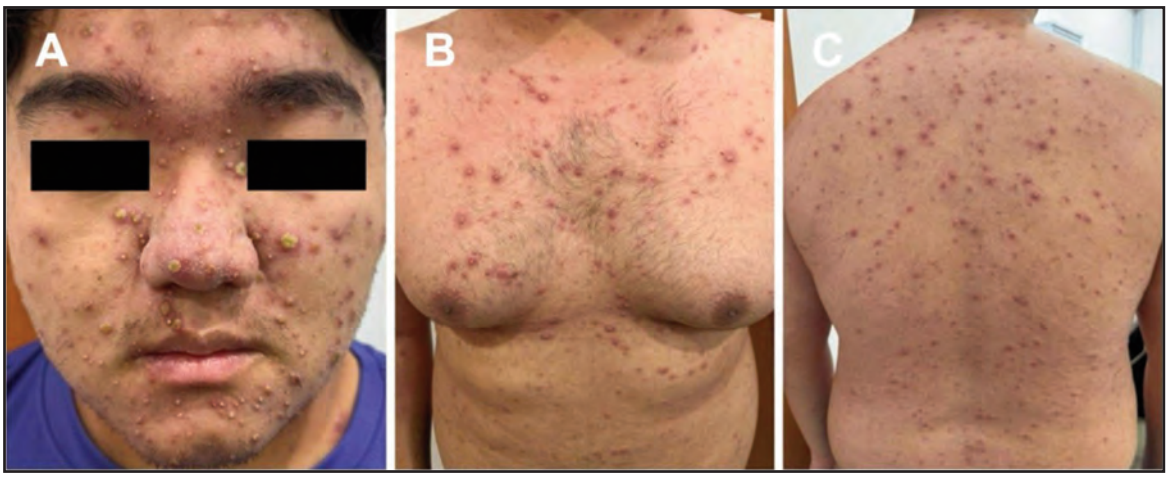

Fig. 1: Acute cutaneous involvement demonstrating (A) facial, (B) anterior trunk, and (C) posterior trunk distribution of multiple vesiculopustular lesions

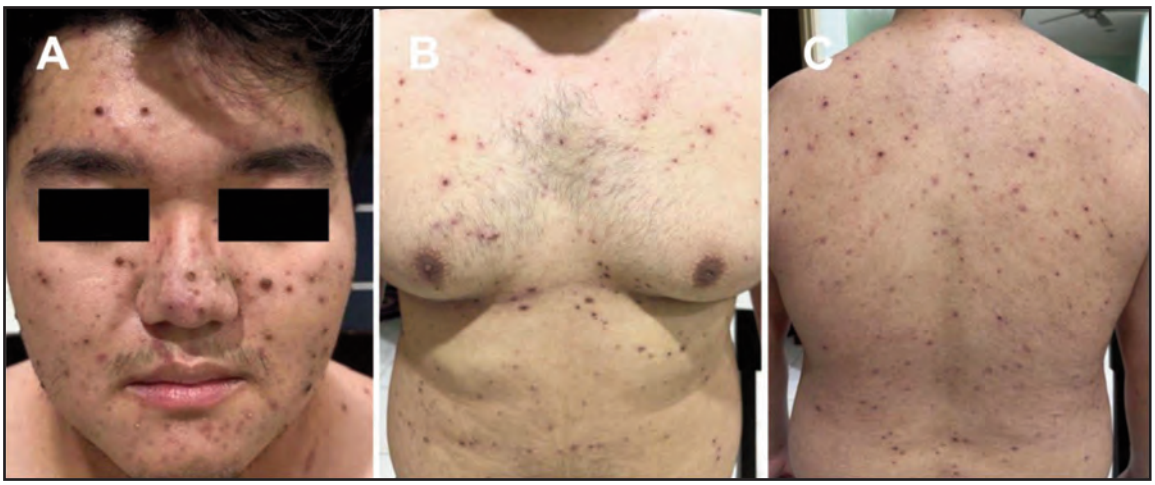

Fig. 2: Cutaneous findings at 1 week post-treatment demonstrating (A) facial, (B) anterior trunk, and (C) posterior trunk distribution of resolving lesions with residual crusting and post-inflammatory changes

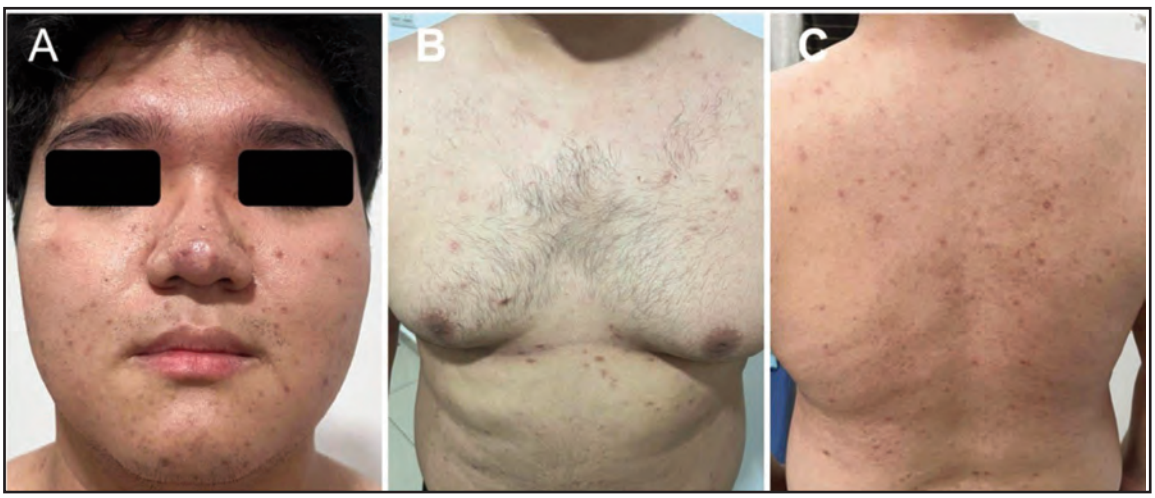

Fig. 3: Cutaneous findings at 3 weeks post-treatment demonstrating residual post-inflammatory hyperpigmentation and atrophic scarring involving (A) face, (B) anterior trunk, and (C) posterior trunk

The widespread vesiculopustular lesions observed in this patient reflect a more severe manifestation of varicella, consistent with findings reported in adult cases.[1,5]

Secondary bacterial infection represents one of the most frequently encountered complications of varicella. Disruption of the epidermal barrier due to vesicle formation and scratching predisposes to bacterial colonisation and superinfection. These infections may manifest as pustulation, crusting, cellulitis, or more severe soft tissue involvement.[1,6]

The presence of purulent crusting and pustular lesions in this patient is consistent with secondary bacterial superinfection, which further exacerbates tissue inflammation and delays healing. In severe cases, life-threatening complications such as varicella gangrenosa and sepsis have been reported.[7] Importantly, complications of varicella are not limited to cutaneous involvement. Systemic complications, including neurological, respiratory, and ocular manifestations, have been described, even in immunocompetent individuals.[8]

From an aesthetic perspective, the severity and extent of inflammation play a pivotal role in determining long-term cutaneous outcomes. Extensive inflammatory lesions, particularly when complicated by secondary bacterial infection, are associated with increased risks of post-inflammatory hyperpigmentation (PIH), atrophic scarring, and, less commonly, keloid formation. Post-varicella scarring has been reported in up to 7–18% of patients and may persist into adulthood.[9] PIH is especially prevalent in individuals with darker skin phototypes, which are common in the Malaysian population.

Facial involvement, as demonstrated in the present case, carries significant psychosocial implications. Visible scarring and pigmentation may negatively impact self-esteem, social interactions, and overall quality of life, particularly in young adults.[10] Patient-reported outcome measures such as SCAR-Q may be used to assess the psychosocial burden associated with scarring. In this case, improvement in scar appearance and symptoms was accompanied by only modest psychosocial recovery, reflecting the recognised lag between physical and psychological adaptation.

Management of post-varicella aesthetic complications frequently requires multimodal therapy. For PIH, first-line treatment includes topical agents such as hydroquinone, retinoids, and azelaic acid, combined with strict photoprotection. Procedural options include chemical peels and pigment-targeting laser therapies such as Q-switched or picosecond lasers. For atrophic scars, treatment options include microneedling, fractional laser resurfacing, subcision, and chemical reconstruction of skin scars (CROSS technique using trichloroacetic acid). Combination therapy is often required to achieve satisfactory outcomes.[9,10]

In the Malaysian private healthcare setting, the estimated cost of managing post-varicella aesthetic complications can be substantial, often requiring multiple treatment sessions.
This case highlights an important gap in real-world clinical practice. Varicella is frequently perceived as a benign condition and managed symptomatically, particularly in primary care. However, adult varicella should not be underestimated. Delayed antiviral initiation may result in preventable complications, prolonged disease course, and significant aesthetic and psychosocial burden.

Accordingly, general practitioners and aesthetic physicians should adopt a proactive approach to the management of varicella. Early antiviral therapy should be strongly considered in adult patients, particularly those presenting within 72 hours of rash onset, with extensive disease or facial involvement. Timely intervention not only improves clinical outcomes but also plays a crucial role in preventing long-term aesthetic sequelae.

In conclusion, this case underscores the importance of early recognition and timely antiviral treatment in varicella infection. Early intervention may significantly minimise complications and long-term aesthetic burden, thereby improving both clinical and psychosocial outcomes.

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