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Incidence of Bullous Pemphigoid After Herpes Zoster Treatment with Acyclovir: A Rare Case Report in a Rural Area

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Abstract

Background: Bullous pemphigoid is the most common autoimmune subepidermal blistering disorder, accounting for approximately 80% of subepidermal immunobullous diseases. It mainly affects elderly patients aged 60–80 years and typically presents with tense bullae, widespread pruritus, and erosive skin lesions. The disease may arise from genetic predisposition and environmental triggers, including drugs that disrupt immune tolerance to dermal–epidermal junction antigens. **Objective:** This case report aimed to describe the clinical features of bullous pemphigoid, its possible relationship with acyclovir use, and treatment outcomes in a rural health-care setting. **Method:** This study used a qualitative single case report approach based on clinical observation, patient history, physical examination, and treatment evaluation. **Results:** A patient developed generalized tense bullae after receiving acyclovir therapy for herpes zoster. The lesions resembled bullous pemphigoid and were suspected to be related to acyclovir-induced immunological activity and its chemical structure. Management was carried out with systemic corticosteroid therapy and supportive care. **Conclusion:** Acyclovir may act as a rare trigger for bullous pemphigoid. The patient showed improvement in clinical signs and symptoms after seven days of steroid treatment. This finding emphasizes the need for careful monitoring of cutaneous adverse reactions following antiviral therapy, especially in resource-limited rural clinical settings.

Keywords: Bullous Pemphigoid; Herpes Zoster; Acyclovir.

Introduction

Pemphigus is a chronic autoimmune disease caused by IgG autoantibodies against intraepidermal desmoglein, leading to blister formation on the skin and mucous membranes[1]. Bullous pemphigoid (BP) is the most common autoimmune subepidermal blistering disorder and accounts for approximately 80% of subepidermal immunobullous cases[2]. Although BP has a broad clinical spectrum, it typically presents with tense bullae, intense generalized pruritus, and inflammatory skin lesions. In atypical cases, obvious bullae may be absent, requiring a high level of clinical suspicion. Histopathological examination with hematoxylin and eosin staining usually demonstrates subepidermal splitting with eosinophils, while direct immunofluorescence highlights autoantibodies along the basement membrane zone. Treatment depends on disease severity and generally involves topical or systemic immunosuppressive therapy[3].

Bullous pemphigoid occurs more frequently in older adults. Previous studies have shown that BP commonly affects individuals older than 60 years. The annual incidence in the United States is estimated at 6 to 13 new cases per one million people, while in Central Europe it reaches approximately 12 to 13 new cases per one million people. The

disease affects men and women at similar rates and has no clear racial predilection. Although BP may occur in childhood, pediatric cases are rare. Specific HLA class II alleles have been reported in patients with BP, including DQB1*0301 among White patients and DRB1*04, DRB1*1101, and DQB1*0302 among Japanese patients[3].

Bullous pemphigoid is the most common type of subepidermal autoimmune bullous disease. It typically develops in elderly individuals, particularly those older than 70 years. The annual incidence of BP is estimated to range from 2.4 to 21.7 new cases per million population across different populations worldwide. A higher annual incidence of 42.8 cases per million population has been reported in the United Kingdom, although this figure should be interpreted carefully because it was based on a computerized longitudinal general-practice database. Based on health insurance data, the prevalence of BP in Germany was recently estimated at 259 cases per million population, equivalent to approximately 21,000 patients living with BP in 2014. Despite increasing incidence, BP is still considered an orphan disease because it affects fewer than 5 per 100,000 people[4].

Bullous pemphigoid has traditionally been considered a disease of the elderly population. The mean age at presentation ranges from 66 to 83 years in different groups worldwide. Its incidence increases exponentially with age, peaking at 190 to 312 cases per million per year among individuals older than 80 years. In contrast, BP is rarely observed among individuals younger than 50 years, with reported incidence generally below 0.5 cases per million population in this age category. This age distribution is unusual for autoimmune diseases because autoimmunity typically appears in younger adulthood. BP is caused by autoantibody responses against hemidesmosomal proteins BP180 and BP230[5]. Until this case report was prepared, no clear national data regarding the incidence or prevalence of BP in Indonesia were available.

Herpes zoster (HZ) is an infectious disease caused by reactivation of endogenous latent varicella-zoster virus in the sensory dorsal-root ganglia after primary infection. HZ occurs when immune defense mechanisms fail to control latent viral replication. The occurrence of herpes zoster is strongly associated with immune status; individuals with strong immunity are less likely to develop HZ. This infection is not always benign and may present in various clinical forms. Even after the skin lesions heal, many patients continue to experience moderate to severe pain known as postherpetic neuralgia. The global incidence of HZ has been reported at 1.2–3.4 cases per 1,000 persons among healthy young adults and 3.9–11.8 cases per 1,000 persons among individuals aged 65 years and older[6,7].

The main management of HZ is antiviral therapy using nucleoside analogues, one of which is acyclovir. Acyclovir selectively inhibits replication of herpes simplex virus types 1 and 2 (HSV-1 and HSV-2) and varicella-zoster virus. After intracellular uptake, acyclovir is converted into acyclovir monophosphate by viral thymidine kinase. This step occurs minimally in uninfected cells, which provides specificity to the drug's activity. The monophosphate derivative is then converted into acyclovir triphosphate by cellular enzymes. Acyclovir triphosphate competitively inhibits viral DNA polymerase by acting as an analogue of deoxyguanosine triphosphate. Incorporation of acyclovir triphosphate into viral DNA results in chain termination because the absence of a 3-hydroxyl group prevents attachment of additional nucleosides. Acyclovir triphosphate has a much higher affinity for viral DNA polymerase than for the cellular homologue, thereby producing a high therapeutic ratio[8].

In the present case, an adverse reaction occurred after acyclovir treatment in a patient initially diagnosed with herpes zoster. After 10 days of therapy, the patient developed skin lesions resembling bullous pemphigoid. BP is often associated with the use of certain drugs. A systematic review identified several drugs commonly associated with BP induction, including aspirin, linagliptin, furosemide, and ibuprofen; however, acyclovir was not listed as a common cause[9]. A literature search identified one similar case associating acyclovir use with BP in a 65-year-old Filipino male who developed BP after taking acyclovir for 10 days[10]. Because this condition is rarely reported, the present case was considered important to describe and discuss.

This case highlights the successful diagnosis and management of suspected acyclovir-induced bullous pemphigoid in a rural Type D hospital, demonstrating that effective care is achievable in resource-limited settings. The suspected association between acyclovir and bullous pemphigoid is rarely reported, making this case clinically noteworthy. The patient responded well to systemic corticosteroid monotherapy, without additional immunosuppressive treatment. This report describes the clinical presentation, discusses the possible pathogenesis, and outlines practical management strategies for rural healthcare facilities. It also contributes to the growing evidence on rare drug-induced autoimmune diseases and supports evidence-based clinical practice in Indonesia.[29–34]

Materials and Methods

Study Design

This study was a rare single case report using a qualitative observational approach combined with direct clinical assessment and interview of the patient. The case report design was selected because the suspected association between acyclovir therapy and bullous pemphigoid is uncommon and requires detailed description of chronology, clinical manifestations, treatment response, and clinical reasoning. This approach allowed comprehensive documentation of the patient's condition from initial presentation to inpatient management and discharge follow-up[35].

Sample

The subject of this case report was one male patient, identified as K, aged 57 years, who presented to the Emergency Department of Maguan Husada Pracimantoro Hospital, a type D hospital in a rural area, with painful fluid-filled skin lesions distributed throughout the body. The patient was selected because the clinical course was unusual: he had a prior history of facial herpes zoster treated with acyclovir and subsequently developed generalized tense bullae consistent with bullous pemphigoid. The patient had no history of similar disease and no known comorbidities, making the temporal relationship with drug exposure clinically important.

Data Collection Technique

Data were collected through history taking, physical examination, dermatovenereological assessment, clinical observation during hospitalization, medication history review, and photographic documentation of skin lesions. The patient reported that blistering lesions developed several days after initiating acyclovir therapy for facial herpes zoster. Clinical data included vital signs, body mass index, lesion distribution and morphology, associated symptoms, medical history, and treatment response. As the case was managed in a rural Type D hospital and the patient declined referral to a higher-level facility, all diagnostic evaluation, treatment, and follow-up observations were conducted at Maguan Husada Pracimantoro Hospital. This approach enabled continuous monitoring of disease

progression and assessment of the patient's clinical response to therapy.

Data Analysis Technique

The data were analyzed descriptively using a clinical case-report approach. The analysis focused on the chronological relationship between acyclovir administration and the onset of bullous lesions, clinical morphology, differential diagnosis, severity assessment, treatment response, and comparison with relevant literature on drug-induced bullous pemphigoid. The findings were synthesized narratively to explain the suspected pathophysiological mechanism, clinical significance, and management considerations in a rural type D hospital setting.

Ethical Consideration

This case report was prepared by maintaining the principles of medical ethics, patient confidentiality, and respect for patient autonomy. The patient and family received an explanation regarding the purpose of documentation and publication of the case. Consent was obtained for the use of clinical information and photographs while ensuring that personal identifiers were not disclosed. The report was prepared solely for scientific and educational purposes, and all clinical management was performed according to the patient's condition, available resources, and applicable hospital procedures.

Results

Case Presentation and Clinical Findings

It is well established that a combination of genetic predisposition, such as human leukocyte antigen (HLA) class II alleles (e.g., HLA-DQβ1*0301), and environmental factors, including ultraviolet (UV) radiation, trauma, and certain medications, can contribute to the loss of immune tolerance toward dermal–epidermal junction antigens. An imbalance between autoreactive T helper (Th) cells and regulatory T (Treg) cells, together with systemic Toll-like receptor (TLR) activation independent of T cells, may stimulate B-cell activation, leading to the production of bullous pemphigoid (BP) autoantibodies. Concurrently, activation of the Th17 pathway appears to sustain the inflammatory cascade initiated by humoral immune hyperactivity by promoting Th2 responses, recruiting neutrophils and eosinophils, and stimulating the release of pro-inflammatory cytokines and proteolytic enzymes. Although the roles of IgA and IgM remain poorly understood and their deposition does not appear to influence disease progression, the pathogenic role of IgE remains controversial.[11]. A 57-year-old male patient presented to the Emergency Department of Maguan Husada Pracimantoro Hospital with painful fluid-filled lesions over the body for five days before admission. The patient initially had facial herpes zoster and was treated as an outpatient with acyclovir for 10 days. On the seventh day of treatment, he began to notice fluid-filled lesions on the back and trunk. The lesions were initially painless and did not interfere with activity, but by the ninth day similar lesions appeared on both legs and gradually spread. Several lesions ruptured and caused mild pruritus and discomfort. The extensive distribution of lesions made mobilization difficult and limited daily activities. Physical examination showed blood pressure of 129/79 mmHg, pulse rate 88 beats/minute, respiratory rate 20 breaths/minute, temperature 36.1°C, oxygen saturation 97%, and body mass index 21.7 kg/m². Dermatological examination revealed multiple generalized tense bullae containing clear fluid with well-defined borders, as well as erosions on the anterior cruris. The patient was diagnosed with bullous pemphigoid caused by suspected drug allergy with secondary infection and a history of facial herpes zoster. The patient

refused referral to a higher-level facility; therefore, inpatient treatment was performed at Maguan Husada Pracimantoro Hospital.

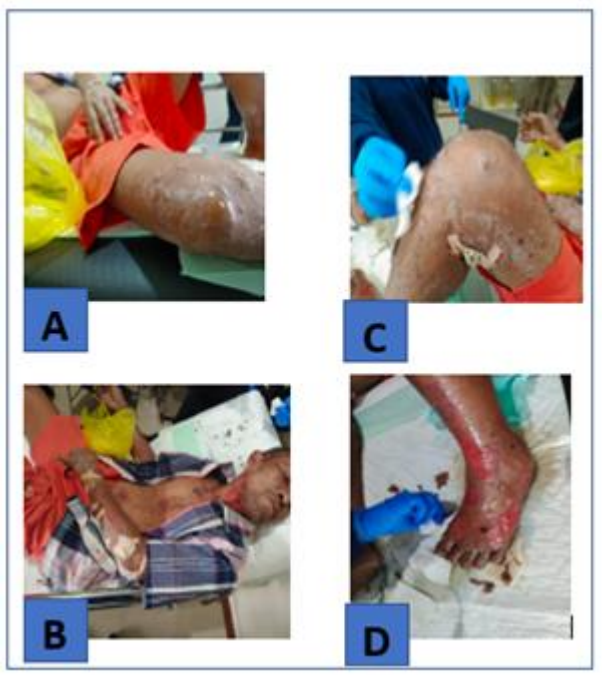

Figure 1. A–C. In the generalized region, multiple bullae with clear fluid, thick and tense walls, variable sizes, and well-defined borders were observed. D. In the anterior cruris region, erosions with a reddish erosive base, irregular shape, and well-defined margins were observed; several lesions had dried into crusts.

Pathogenesis of Bullous Pemphigoid

It is known that a combination of genetic predisposing factors, such as HLA class II alleles including HLA-DQβ1*0301, and environmental influences, such as ultraviolet radiation, trauma, and drugs, may contribute to loss of immune tolerance against dermal-epidermal junction antigens[11,12]. This process results in autoantibody formation against structural proteins of the basement membrane zone, especially BP180 and BP230. Binding of autoantibodies activates inflammatory pathways that lead to separation between the epidermis and dermis and the formation of tense subepidermal bullae.

Several authors have shown that immunoglobulin deposition along the basement membrane zone or serum autoantibody levels correlate with certain clinical features of BP, including more infiltrated lesions such as urticarial plaques or nodules. Autoantibody profiles may also influence the degree of inflammation and clinical severity. These findings indicate that BP is not a single uniform condition but a heterogeneous autoimmune disorder with different immunological patterns.

Ramcke et al. demonstrated that IgG1 and IgG4 are the IgG subtypes most frequently found along the dermal-epidermal junction in BP. Through cryosection assays, the same authors showed that IgG1 promotes blister formation through complement fixation, whereas IgG4 may contribute through complement-independent mechanisms[24]. This distinction is clinically relevant because different immunoglobulin subclasses may influence disease phenotype, inflammatory intensity, and response to therapy.

IgG antibodies directed against BP180 domains other than NC16A appear to be responsible for less inflammatory manifestations because they induce only limited BP180 depletion compared with anti-BP180-NC16A IgG in experimental studies. These findings suggest that the specific antigenic target and epitope location may influence clinical presentation. Autoantibody diversity may therefore explain why some BP cases present with classical tense bullae, while others appear with atypical or less inflammatory skin lesions.

Anti-BP180 IgE antibodies also show pathogenic characteristics. The pathogenicity of anti-BP180 IgE has been demonstrated using IgE hybridoma against LABD97, a component of the BP180 ectodomain, injected into immunodeficient mice grafted with human

skin[16,19]. In addition, IgE autoantibodies may contribute to eosinophil recruitment, pruritus, and inflammatory amplification. This mechanism may explain the intense pruritus frequently reported in patients with BP.

Although the pathogenic role of anti-BP230 autoantibodies remains debated, recent studies have documented that IgG reactivity with intracellular BP230 epitopes correlates with disease severity. Autoantibodies against BP230 may also participate in maintaining inflammation or act as markers of broader autoimmune activation. Therefore, both BP180 and BP230 should be considered important antigenic targets in understanding BP pathogenesis[24,25].

Drug-Induced Bullous Pemphigoid

In this case, bullous pemphigoid was suspected to be induced by acyclovir, an antiviral therapy used for herpes zoster. Until now, few reports have clearly explained the mechanistic relationship between acyclovir use and the development of bullous pemphigoid. Nevertheless, the temporal relationship between drug exposure and lesion onset supports the possibility of drug-associated bullous pemphigoid (DABP), particularly when other clear triggers are absent.

In general, the pathogenesis of drug reactions in DABP has begun to be understood, although specific causal pathways remain incompletely explained. Drugs are thought to act as triggers in genetically or immunologically predisposed individuals. They may alter antigenic structures in the basement membrane zone, act as haptens, modify immune regulation, or reveal a subclinical autoimmune process. These mechanisms may lead to autoantibody formation and subsequent blister formation[9,22,23].

Thiol-based drugs are among the drug groups most often discussed in relation to drug-induced autoimmune blistering diseases. Most thiol drugs contain or release sulfhydryl groups in their precursors or metabolites. These compounds can structurally modify molecules and allow them to act as haptens, thereby inducing immune recognition. Although acyclovir is not classically categorized as a thiol-based drug, this mechanism illustrates how drug metabolites may alter immune tolerance and contribute to blistering disorders.

Phenol-based drugs are drugs known to contain a phenyl group attached to a hydroxyl group in their molecular structure. Phenol drugs associated with DABP include cephalosporins and aspirin. These drugs are thought to interact with basement membrane components and modify antigen presentation. Acyclovir has chemical characteristics that may be discussed in relation to such drug-reaction mechanisms, although direct evidence remains limited and requires further investigation.

DABP may also occur due to dysregulation of tumor necrosis factor alpha (TNF-α) in an immune system that has already been disturbed by a previous disease process, or this dysregulation may reveal a subclinical autoimmune condition. One theory suggests that drug exposure may shift immune balance and promote autoreactive responses. In patients with recent infection, such as herpes zoster, immune activation may further increase susceptibility to drug-triggered autoimmune reactions[20,21].

Several studies have also suggested the possibility of underlying neurological abnormalities in the development of BP. Neurological disorders have been shown in case-control studies to be associated with a higher risk of BP compared with patients without neurological disease. This association may be related to shared antigens between the skin and nervous system, chronic

inflammation, or immune dysregulation. Although the present patient did not have a known neurological disorder, the relationship between infection, immune activation, and BP remains clinically relevant.

The primary goal of BP treatment is to control clinical manifestations effectively. Treatment choice depends on disease extent, severity, patient age, comorbidities, and overall health status. BP may be categorized as mild, moderate, or severe. Mild localized disease may be treated with high-potency topical corticosteroids, while more extensive or severe disease often requires systemic corticosteroids or additional immunosuppressive therapy. In rural health-care settings, treatment decisions must also consider available resources and referral feasibility[26,27].

Systemic steroids such as oral prednisone at 0.5–1 mg/kg/day should be considered when needed. Oral corticosteroids are widely used but carry risks including hypertension, diabetes, osteoporosis, infection, and other metabolic complications. Additional therapies such as azathioprine, mycophenolate mofetil, methotrexate, doxycycline, nicotinamide, omalizumab, rituximab, or intravenous immunoglobulin may be used depending on disease severity and treatment response. In the present case, systemic corticosteroid therapy was chosen because the disease was considered severe and the patient refused referral.

Immunosuppressive therapy is used when steroids cannot control the disease or when patients have contraindications to systemic corticosteroid therapy. Alternative agents include azathioprine, mycophenolate mofetil, methotrexate, chlorambucil, and cyclophosphamide. If these treatments fail, intravenous immunoglobulin, anti-CD20 therapy such as rituximab, or omalizumab may be considered for refractory cases. Serum IgG autoantibody levels against BP180 have been shown to correlate with disease severity in several ELISA-based studies. In addition, high BP180-NC16A ELISA scores and positive direct immunofluorescence at the end of treatment may indicate a higher likelihood of recurrence[28].

Treatment of classical bullous pemphigoid has historically relied on uncontrolled studies using oral and topical corticosteroids and adjuvant drugs. Management of DIBP includes immediate discontinuation of the suspected causative drug. Patients with DIBP generally respond well to low-dose oral corticosteroids such as prednisone 0.5 mg/kg/day and potent topical corticosteroids. Gradual tapering of corticosteroids may be performed by decreasing 10 mg/day and then reducing more slowly at lower doses, eventually tapering by 1 mg/week when the dose is below 10 mg/day. Studies show that systemic glucocorticoids control moderate or severe BP in 95% and 91% of cases after 21 days of treatment. In this case, because the disease was classified as severe, injectable methylprednisolone at 1 mg/kg/day, equivalent to 62.5 mg/day for a 62-kg patient, was administered for four days. The dose was then reduced to 48 mg/day orally on days five to seven and subsequently continued as 12 mg/day after discharge.

Discussion

This case supports the importance of considering drug-induced bullous pemphigoid in patients who develop widespread tense bullae after exposure to a new medication. Although acyclovir is widely used and generally considered safe, rare hypersensitivity or autoimmune blistering reactions may occur. The temporal relationship between acyclovir use for herpes zoster and the onset of generalized bullous lesions suggests a possible association. The case is clinically important because it was diagnosed and managed in a rural type D hospital, where access to advanced diagnostic tools and subspecialty referral may

be limited. Clinical recognition, careful history taking, discontinuation of the suspected drug, management of secondary infection, and timely corticosteroid therapy were essential to prevent worsening disease. This report also highlights the role of case reports in documenting rare adverse events, particularly in settings where local data remain limited. Healthy Tadulako Journal has contributed to the dissemination of Indonesian case reports and clinical studies, including reports related to internal medicine, infection, antibacterial activity, exercise, and public health, which support the value of local scientific publication in strengthening clinical awareness and evidence-based practice[29–34].

Conclusion

Bullous pemphigoid is an autoimmune-based disease that may be triggered by various factors, including certain medications. In this case, acyclovir, which is a main therapy for herpes zoster, was suspected to trigger bullous pemphigoid, possibly through immune dysregulation and mechanisms involving TNF-α alteration or changes in basement membrane integrity. The main treatment for BP is corticosteroid therapy, either topical or systemic. Systemic corticosteroids should be used particularly in moderate and severe cases, with dosage adjusted according to disease severity, blood pressure condition, and patient comorbidities.

This case demonstrates that even patients without comorbidities may experience adverse drug reactions from medications that are generally considered safe, such as acyclovir. Therefore, periodic evaluation of patients after initiation of therapy is important so that bullous pemphigoid as a possible drug-related adverse event can be detected earlier and treated promptly before progressing to a severe condition. Further case documentation and larger studies are needed to clarify the relationship between acyclovir and bullous pemphigoid, especially in rural health-care settings.

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