



A Life-Threatening Adverse Drug Reaction: Allopurinol-Induced Stevens–Johnson Syndrome in a Patient with Gout and Hypertensive Heart Disease: A Case Report

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ABSTRACT

Background

Stevens–Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are rare but life-threatening immune-mediated mucocutaneous disorders characterized by extensive keratinocyte apoptosis, resulting in epidermal detachment and mucosal involvement. Allopurinol, a xanthine oxidase inhibitor primarily used to manage chronic gout and hyperuricemia, is globally recognized as one of the most frequent causative agents of SJS and TEN. The risk of such adverse drug reactions is increased in patients with comorbidities such

as chronic kidney disease and hypertensive heart disease, where altered drug clearance and cardiovascular fragility complicate both disease pathogenesis and management. This report highlights the clinical challenges of managing allopurinol-induced SJS in a patient with cardiovascular comorbidity.

Case Report

A 58-year-old female with chronic tophaceous gout and hypertensive heart disease developed SJS approximately 18 days after initiating allopurinol 300 mg daily. She presented with high-grade fever, malaise, and rapidly progressive erythematous eruptions with dusky macules and flaccid bullae involving 9% of total body surface area. Severe mucosal involvement affected the oral, conjunctival, and genital regions. Histopathology confirmed full-thickness epidermal necrosis. Management in a high-dependency unit required careful balance between fluid resuscitation and strict volume control. Following drug withdrawal, systemic corticosteroids, and supportive care, complete re-epithelialization was achieved within three weeks.

Conclusion

Early recognition, prompt cessation of the offending drug, and multidisciplinary care are essential. In patients with cardiovascular comorbidities, fluid management must be carefully individualized to prevent complications while maintaining adequate organ perfusion.

Keywords

Adverse Drug Reaction, Allopurinol, Gout, Hypertensive Heart Disease, Stevens–Johnson Syndrome.

INTRODUCTION

The clinical challenge of managing severe cutaneous adverse reactions (SCARs) remains one of the most demanding aspects of modern dermatology and clinical pharmacology.¹ Within this spectrum, Stevens–Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) represent medical emergencies that carry profound morbidity and a significant risk of mortality.² These conditions are characterized by an idiosyncratic, immune-mediated destruction of keratinocytes, resulting in the separation of the epidermis from the dermis.³ While the incidence of SJS is relatively low, ranging from 1 to 6 cases per million

person-years, its impact is catastrophic, often requiring management in intensive care or burn units due to the loss of vital skin functions, including thermoregulation, fluid balance, and protection against microbial invasion.¹

Allopurinol, a cornerstone in the management of gout and hyperuricemia for over five decades, has ironically emerged as the leading cause of SJS/TEN in many parts of the world.⁴ As a purine analogue, allopurinol inhibits xanthine oxidase, the enzyme responsible for the conversion of hypoxanthine and xanthine into uric acid.⁵ While highly effective at reducing serum urate levels and preventing gouty flares, allopurinol is associated with a range of hypersensitivity reactions, collectively known as Allopurinol Hypersensitivity Syndrome (AHS). These reactions can range from mild maculopapular exanthems to life-threatening SJS, TEN, and Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS).⁷

The complexity of these reactions is further intensified by the demographic and clinical profile of the typical patient requiring urate-lowering therapy. Hyperuricemia is frequently comorbid with the metabolic syndrome, including hypertension, obesity, and cardiovascular disease.⁷ Hypertensive heart disease (HHD), characterized by left ventricular hypertrophy and eventual heart failure, is particularly prevalent in this population.⁸ The management of SJS in a patient with HHD presents a clinical paradox: the extensive skin detachment necessitates aggressive fluid resuscitation to prevent hypovolemic shock and acute kidney injury, yet the compromised cardiac structure and function of an HHD patient make them highly susceptible to fluid overload and acute pulmonary edema. Furthermore, the use of diuretics—a mainstay in the treatment of hypertension and heart failure—is a known independent risk factor for the development of allopurinol-induced SJS, likely due to altered renal clearance of the active metabolite, oxypurinol.⁹

Recent genomic research has identified a strong association between the HLA-B*58:01 allele and the risk of allopurinol-induced SCARs.⁵ This genetic predisposition is especially prominent in Asian populations, where the carrier rate can be as high as 10-15%, compared to approximately 1-2% in European populations.¹⁰ Despite the availability of genetic screening, many patients are still initiated on allopurinol without testing or without appropriate dose adjustments for their renal function, as seen in the broader clinical landscape.³

This case report aims to provide an exhaustive analysis of a 58-year-old female with

chronic tophaceous gout and hypertensive heart disease who developed life-threatening SJS following the initiation of allopurinol. By detailing the clinical progression, the diagnostic intricacies, and the multidisciplinary management required to navigate her cardiovascular fragility, this report seeks to offer profound insights into the prevention and treatment of severe adverse drug reactions in patients with complex systemic comorbidities.

CASE REPORT

A 58-year-old female presented to the Emergency Department with an acute onset of painful, blistering skin lesions and severe oral erosions. The patient's symptoms had begun five days prior with a high-grade fever reaching 39.5°C, accompanied by a persistent sore throat, non-productive cough, and generalized malaise. Initially, she attributed these symptoms to a common viral infection. However, 48 hours into the febrile period, she noticed a faint, erythematous rash on her chest and upper back. This rash rapidly evolved into dusky, purpuric macules that spread centripetally to involve her face, neck, and all four extremities. By the morning of admission, the patient reported the formation of flaccid blisters and the "sliding off" of her skin upon gentle contact, particularly on her torso.

The patient's medical history was significant for long-standing chronic tophaceous gout, diagnosed 12 years ago. She experienced frequent polyarticular flares, for which she had historically used non-steroidal anti-inflammatory drugs (NSAIDs) on an as-needed basis. She also had prominent tophi over both olecranon processes and the Achilles tendons. In addition to her gout, the patient had a documented history of hypertensive heart disease, diagnosed 8 years prior. Her hypertension was managed with Lisinopril 20 mg daily and Amlodipine 5 mg daily. A routine echocardiogram performed six months earlier had revealed concentric left ventricular hypertrophy with an ejection fraction of 50%, indicating a heart with limited diastolic reserve. She also had stage 2 chronic kidney disease (CKD), with a baseline serum creatinine of 1.3 mg/dL and an eGFR of approximately 62 mL/min/1.73 m².

Three weeks prior to the current presentation, the patient had experienced a severe gout flare involving her right ankle. During a visit to a primary care clinic, she was prescribed a 7-day course of Celecoxib and was newly initiated on allopurinol 300 mg daily for long-term urate management. She had completed the Celecoxib course ten days before the rash appeared and had continued the allopurinol until the day of admission. She denied any other new

medications, herbal supplements, or recent vaccinations. She had no known drug allergies.

On admission, the patient was in significant distress due to pain and difficulty swallowing. She was febrile, with a temperature of 39.2°C. The general examination was remarkable for the absence of peripheral lymphadenopathy. The cardiovascular examination confirmed a laterally displaced apex beat at the 6th intercostal space, consistent with her known cardiomegaly. Auscultation revealed a prominent S4 gallop and a Grade II/VI systolic murmur at the apex, which was interpreted as functional mitral regurgitation secondary to left ventricular hypertrophy. There were no signs of acute heart failure, such as jugular venous distention or pulmonary crackles, at the time of initial assessment.

The dermatological examination revealed a widespread, symmetrical eruption involving approximately 65% of the total body surface area in terms of erythema, with actual epidermal detachment and erosions involving 9% of the BSA. This placed the patient at the upper limit of the SJS classification (< 10% BSA detachment).

The clinical examination revealed primary lesions characterized by dusky to violaceous macules with necrotic centers, accompanied by atypical targetoid lesions predominantly located on the palms and soles. Secondary lesions evolved into large, flaccid bullae distributed over the trunk and proximal limbs, with extensive areas of denuded dermis that appeared moist and weeping. The Nikolsky sign was positive, as firm lateral pressure applied to erythematous areas resulted in immediate epidermal separation.

Significant mucosal involvement was observed in multiple sites. Oral examination demonstrated severe hemorrhagic crusting of the lips and extensive, painful erosions affecting the buccal mucosa, tongue, and hard palate. Ocular involvement was evident with bilateral purulent conjunctivitis, chemosis, and marked photophobia. In addition, erythematous erosions were present on the genital mucosa, particularly involving the glans penis and the perianal region. As shown in Figure 1, the cutaneous lesions were distributed across the face, neck, trunk (both anterior and posterior), and the upper and lower extremities. The palms and soles were also involved; however, the skin over these areas remained intact without widespread epidermal detachment.



Figure 1. Clinical Presentation of the patient demonstrate widespread cutaneous involvement affecting the face (A, B, C) and posterior (D) of trunk, as well as the upper (E) and lower (F) extremities. The lesions are characterized by dusky to violaceous macules with necrotic centers and atypical targetoid appearance, accompanied by areas of epidermal detachment forming flaccid bullae and denuded, moist dermis.

A comprehensive set of laboratory tests was performed to assess the degree of systemic inflammation, renal function, and the presence of any secondary complications. Laboratory investigations revealed several abnormalities consistent with systemic inflammation and organ involvement. Hemoglobin level was 12.8 g/dL, slightly below the normal reference range, suggesting mild anemia of chronic disease or inflammation. The white blood cell count was elevated at $14.2 \times 10^9/L$, indicating leukocytosis in the context of a systemic inflammatory response, with a corresponding neutrophil count of $11.5 \times 10^9/L$ reflecting neutrophilia, which is commonly observed in acute severe cutaneous adverse reactions. In contrast, the eosinophil count remained within normal limits at $0.2 \times 10^9/L$, a finding that helps differentiate this condition from DRESS. Renal function tests demonstrated impairment, with a serum creatinine level of 1.8 mg/dL and blood urea nitrogen (BUN) of 32 mg/dL, consistent with acute kidney injury, likely superimposed on underlying chronic kidney disease and associated with dehydration and increased catabolic state. Serum uric acid was elevated at 8.4 mg/dL despite ongoing allopurinol therapy, indicating suboptimal control of hyperuricemia. Inflammatory markers were markedly increased, as evidenced by a C-reactive protein (CRP) level of 145 mg/L, reflecting significant systemic inflammation. Serum albumin was reduced to 3.1 g/dL, suggesting hypoalbuminemia likely due to protein loss through compromised skin integrity. Liver enzymes were mildly elevated, with ALT and AST levels of 48 U/L and 52 U/L, respectively, indicating mild transaminitis and possible visceral involvement.

Imaging and specialized tests were performed to further evaluate the patient's condition. Electrocardiogram (ECG) demonstrated sinus tachycardia at 115 beats per minute

with findings consistent with left ventricular hypertrophy based on the Sokolow–Lyon criteria (S in $V1 + R$ in $V5 > 35$ mm), accompanied by a left ventricular strain pattern characterized by ST-segment depression and T-wave inversion in leads I, aVL, V5, and V6 (LV strain pattern). Chest X-ray revealed an enlarged cardiac silhouette, indicated by an increased cardiothoracic ratio.

A skin biopsy was obtained using a 4-mm punch biopsy from a dusky macule on the lateral thigh. Histopathological examination showed full-thickness necrosis of the epidermis with formation of a subepidermal bulla. The dermis contained a sparse inflammatory infiltrate composed mainly of lymphocytes and macrophages. Direct immunofluorescence testing was negative for IgG, IgA, IgM, and C3 deposition, effectively ruling out autoimmune bullous diseases.¹

To estimate the risk of hospital mortality, the SCORTEN was calculated within the first 24 hours of admission.¹ The patient was 68 years old, which contributed one point to the score. There was no history of malignancy, therefore no additional point was assigned for this parameter. The recorded heart rate was 112 beats per minute, which did not meet the threshold of greater than 120 beats per minute and thus contributed no points. The initial body surface area (BSA) of epidermal detachment was estimated at 9%, which was below the cutoff of 10% and therefore did not add to the score. Laboratory evaluation showed a serum urea level of 11.4 mmol/L, exceeding the threshold of 10 mmol/L and contributing one point. The serum glucose level was 7.8 mmol/L, which was below the cutoff value of 14 mmol/L and therefore added no points. Serum bicarbonate was measured at 22 mEq/L, which was above the threshold of 20 mmol/L and did not contribute to the score. Based on these parameters, the patient's total SCORTEN was calculated to be 2, which corresponds to a predicted mortality rate of 12.1%.² Given the patient's heart disease, her actual risk was considered higher than the score suggested.

The final diagnosis was established as SJS with epidermal detachment involving 9% of the body surface area, which was considered secondary to allopurinol exposure. In addition, the patient was diagnosed with chronic tophaceous gout characterized by inadequately controlled hyperuricemia. Cardiovascular evaluation indicated hypertensive heart disease with evidence of left ventricular hypertrophy and stage C heart failure in a compensated state. The patient also developed acute kidney injury (AKI), which was considered likely pre-renal in origin, associated with insensible fluid losses and systemic inflammation.

The patient was admitted to the High Dependency Unit (HDU) to facilitate intensive monitoring of both her dermatological and cardiovascular status. The most critical step was the immediate cessation of allopurinol. This was documented in her medical records as a life-threatening allergy.¹

Fluid resuscitation represented the primary management challenge in this patient. Based on her 9% skin loss, he required approximately 2-3 liters of isotonic saline daily. However, to avoid exacerbating her hypertensive heart disease and triggering pulmonary edema, fluids were administered in 250 mL boluses with frequent auscultation of the lungs. Urine output was targeted at 0.5 mL/kg/hour.

Pharmacological management included the administration of systemic corticosteroids, with intravenous methylprednisolone given at a dose of 1 mg/kg/day for the first three days in an effort to halt immune-mediated keratinocyte destruction. Adequate pain control was achieved using intravenous morphine delivered through a patient-controlled analgesia (PCA) pump, which was essential for managing the severe pain associated with extensive skin denudation and oral lesions. Prophylactic antibiotic therapy was deliberately avoided to reduce the risk of additional drug-related reactions; however, close monitoring for infection was maintained through skin swabs and blood cultures obtained every 48 hours.

Supportive care played a crucial role in overall management. Daily ophthalmologic evaluation was conducted, and the patient received preservative-free artificial tears along with topical moxifloxacin drops to prevent secondary infection and the development of symblepharon. Skin care was carefully managed by gently cleansing denuded areas with sterile saline, followed by the application of non-adherent dressings, such as petrolatum-impregnated gauze, over the torso and extremities to promote healing and minimize further trauma.

By Day 6, the patient's fever had resolved, and no new skin lesions had appeared. Her oral intake remained poor due to the erosions, so a nasogastric tube was placed for high-protein, high-calorie liquid nutrition. By day 7, skin detachment had stabilized at approximately 10% of the body surface area, with early re-epithelialization evident on the face. Laboratory parameters showed improvement, with serum creatinine decreasing to 1.6 mg/dL and CRP to 82 mg/L. At this stage, methylprednisolone was tapered by 50%, and nasogastric tube feeding had reached the full nutritional target.

By day 10, mucosal recovery became more apparent, as oral lesions began to crust over and conjunctival injection was significantly reduced. Renal function continued to improve, with serum creatinine at 1.4 mg/dL, while serum albumin increased to 3.3 g/dL. Pain management was adjusted by discontinuing morphine via PCA and transitioning to oral paracetamol. On day 14, substantial cutaneous healing was noted, with large areas of the trunk covered by newly formed thin epidermis, although desquamation persisted on the fingers and toes. Serum creatinine returned to baseline at 1.3 mg/dL, and CRP further declined to 18 mg/L. Methylprednisolone was discontinued, and lisinopril was reintroduced at a dose of 5 mg.

By day 18, the patient was able to tolerate soft oral intake, and overall skin and mucosal healing exceeded 90%. Laboratory values remained stable, with serum creatinine at 1.3 mg/dL and hemoglobin at 11.8 g/dL. The patient began mobilization with the assistance of physical therapy, and the nasogastric tube was subsequently removed. At day 21, complete re-epithelialization had been achieved, with no evidence of ocular adhesions. Laboratory findings had normalized, and the patient was deemed clinically stable and prepared for discharge.

The patient was discharged on Day 22. At discharge, her skin appeared erythematous with significant post-inflammatory hyperpigmentation but no evidence of hypertrophic scarring. She was strictly counseled to never take allopurinol or any related medications (such as oxypurinol) again. Her gout was managed with a low-purine diet and low-dose Colchicine (0.5 mg daily) as a bridge, with the future consideration of Febuxostat only under specialist supervision once he had fully recovered from the hypersensitivity state.⁷ Her heart disease management was resumed with Lisinopril 20 mg and Amlodipine 5 mg, with her blood pressure stabilized at 134/82 mmHg.

DISCUSSION

The development of SJS in this patient illustrates the profound danger inherent in the routine prescription of allopurinol, particularly when underlying comorbidities such as hypertensive heart disease and renal impairment are present. This case warrants a deep analysis of the pharmacological, immunological, and cardiovascular intersections that dictate the outcome of such a life-threatening reaction.

The Pharmacological Trigger: Allopurinol and Oxypurinol

Allopurinol is an analogue of the purine hypoxanthine. Its therapeutic utility in gout is derived from its ability to competitively inhibit xanthine oxidase (XO), thereby preventing the production of uric acid.⁹ However, the drug's metabolism is a key factor in its toxicity. Allopurinol has a short half-life of 1-2 hours and is converted by XO into its active metabolite, oxypurinol. Oxypurinol has a significantly longer half-life (18-30 hours) and is excreted primarily by the kidneys.⁵

The presence of stage 2 CKD (eGFR 62 mL/min) in the patient likely reduced the clearance of oxypurinol. When she was initiated on a standard dose of 300 mg daily, her plasma levels of oxypurinol likely exceeded the safe threshold. High serum oxypurinol concentrations are strongly correlated with the development of Allopurinol Hypersensitivity Syndrome.⁹ This highlights the critical importance of the "start low, go slow" strategy recommended by rheumatology guidelines.³ Starting at 100 mg daily—or even 50 mg in patients with more advanced CKD—and titrating based on serum urate levels can significantly lower the risk of SCARs by allowing the immune system to tolerate gradual increases in drug concentration.⁶

Immunopathogenesis of SJS: The HLA-B*58:01 Link

SJS is an idiosyncratic reaction, meaning it is not dose-dependent in the traditional sense, but rather an immune-mediated response occurring in susceptible individuals.¹¹ The current understanding of allopurinol-induced SJS centers on a Type IV delayed hypersensitivity reaction mediated by T-cells.³

The most potent risk factor identified to date is the presence of the HLA-B*58:01 allele. This Human Leukocyte Antigen molecule possesses a specific binding pocket that can present allopurinol or its metabolites directly to CD8⁺ cytotoxic T-lymphocytes (the p-i concept) or after binding to an endogenous peptide (the hapten concept). Once these T-cells are activated, they orchestrate a massive release of cytotoxic mediators, most notably granulysin.⁵ Granulysin is a cationic protein that directly induces the death of keratinocytes by disrupting their cell membranes and activating apoptotic pathways.¹²

This case emphasizes the public health importance of pre-treatment genetic screening. In many countries, particularly in Southeast Asia where the HLA-B*58:01 allele is common, screening for this marker before prescribing allopurinol is now considered a standard of care and is highly cost-effective.⁶ For patients who carry the allele, allopurinol should be avoided

entirely, and alternative agents such as febuxostat should be utilized.⁶

Cardiovascular Interactions in SJS Management

The patient's hypertensive heart disease (HHD) significantly complicated her management. SJS is effectively a "medical burn," where the loss of the epidermis leads to massive evaporative water loss and the shifting of fluid from the intravascular to the interstitial compartment.⁹ In a young, healthy patient, aggressive fluid resuscitation is straightforward. However, in our 58-year-old patient with LVH, her heart was structurally "stiff" (diastolic dysfunction).⁸

The Hemodynamic Tightrope:

Aggressive fluid administration can quickly lead to an increase in left ventricular end-diastolic pressure, resulting in backward failure and acute pulmonary edema.⁹ Conversely, under-resuscitation would have exacerbated her AKI, potentially leading to permanent renal damage and an inability to clear the remaining oxypurinol from her system.³ This necessitated a meticulous "micro-bolus" approach to fluids, guided by clinical markers such as lung auscultation and urine output rather than standard volume-replacement formulas.

Uric Acid as a Cardiovascular Stressor:

Interestingly, hyperuricemia is not just a driver of gout; it is also an independent risk factor for the progression of HHD and heart failure.¹² High uric acid levels promote oxidative stress, impair endothelial function, and activate the renin-angiotensin-aldosterone system.¹² In this patient, her uncontrolled gout and baseline hyperuricemia (8.4 mg/dL) may have contributed to her underlying cardiovascular strain. The irony is that the treatment meant to reduce this cardiovascular risk—allopurinol—precipitated a crisis that nearly killed her.

Systemic Inflammation and Multi-organ Involvement

The systemic nature of SJS was evident in the patient's elevated CRP, leukocytosis, and mild transaminitis. This "cytokine storm" imposes a high metabolic demand on the body, similar to sepsis.⁵ For a patient with HHD, this state of hypermetabolism and tachycardia (112-115 bpm) can be deleterious, as it reduces the time for coronary artery perfusion during diastole and can precipitate myocardial ischemia.¹³

The decision to use systemic corticosteroids in this case was influenced by the need to

rapidly dampen this systemic inflammation.¹ While the use of steroids in SJS remains debated due to concerns about infection risk, several studies suggest that "pulse" therapy (high doses for a very short duration) early in the disease course can improve survival without significantly increasing the risk of sepsis.¹ In our patient, the rapid resolution of her fever and the stabilization of her skin lesions following Methylprednisolone suggests a positive therapeutic effect.

Long-term Considerations and Alternative Therapies

Survival is only the first hurdle for an SJS patient. Long-term sequelae are common and can be debilitating.

Ocular Complications:

Up to 50-70% of SJS/TEN survivors experience long-term ocular issues, including chronic dry eye, symblepharon, and even blindness.⁸ The aggressive ocular care our patient received—daily ophthalmological exams and frequent lubricant/antibiotic drops—was instrumental in preventing these outcomes.

Future Urate Management:

How should this patient's gout be managed moving forward? Allopurinol is now strictly contraindicated for her. Febuxostat, a non-purine xanthine oxidase inhibitor, is a potential alternative.⁵ However, there have been rare reports of SJS in patients taking febuxostat, and some cases suggest a possibility of cross-sensitivity.¹⁰ Most experts recommend a "washout" period of several months after an SJS episode before starting any new urate-lowering therapy, and then only with extreme caution and patient education.⁶ Uricosuric agents (like probenecid) are another option, but their efficacy is limited by the patient's underlying CKD.¹⁴

The Role of Education:

The patient and her family were educated on the importance of checking all future medications for potential cross-reactivity and informing every healthcare provider of her history of SJS. A medical alert bracelet was strongly recommended.

CONCLUSION

This case of allopurinol-induced SJS in a patient with hypertensive heart disease serves as a profound reminder of the complexities inherent in managing adverse drug reactions in the elderly and comorbid population. The successful outcome was the result of rapid diagnostic recognition, immediate cessation of the offending agent, and a multidisciplinary management strategy that respected the patient's cardiovascular and renal limitations.

The central insight derived from this case is the avoidable nature of many such reactions. Adherence to established dosing guidelines—starting allopurinol at doses no higher than 100 mg daily in patients with renal impairment—and the adoption of pre-treatment HLA-B*58:01 screening could have prevented this life-threatening episode. As the global burden of gout and its associated cardiovascular comorbidities continues to rise, it is imperative that clinicians remain vigilant. The "safe" profile of common drugs like allopurinol should never blind us to the potential for catastrophic idiosyncratic reactions. For the patient with hypertensive heart disease, the "heart" of the matter lies in balancing the need for intensive supportive care with the reality of a compromised cardiac reserve, a task that requires both clinical skill and pharmacological foresight.

This case report has several limitations. First, as a single-patient observation, the findings cannot be generalized to broader populations, particularly regarding the safety and efficacy of management strategies such as short-course systemic corticosteroids. Second, genetic testing for HLA-B*58:01 was not performed, which limits the ability to confirm a pharmacogenomic predisposition to allopurinol-induced SJS. Third, the absence of long-term follow-up restricts the assessment of delayed complications, especially ocular sequelae and recurrence risk. Additionally, potential confounding factors such as prior drug exposures and exact cumulative oxypurinol levels were not evaluated, which may influence the interpretation of causality.

ETHIC IN PUBLICATION

Written informed consent was obtained from the patient for the publication of this case report, including the use of clinical photographs. The patient was assured that all identifying information would be kept confidential.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest regarding the publication of this case report.

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AUTHOR CONTRIBUTIONS

Stella Jessica Paulus contributed to data collection, manuscript drafting, and literature review. Ketut Kwartantaya Winaya contributed to case supervision, clinical management, and critical revision of the manuscript. Pande Agung Mahariski and Tjokorda Dalem Pemayun contributed to patient management, data interpretation, and manuscript review. All authors read and approved the final manuscript.

GENERATIVE AI DISCLOSURE

No artificial intelligence (AI) tools were used in the preparation, writing, or editing of this manuscript. All content was developed solely by the authors.

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