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# Ciprofloxacin-Associated Stevens-Johnson Syndrome in A Healthy Young Woman: A Case Report

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## ABSTRACT

**Background:** Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are rare but life-threatening mucocutaneous reactions, primarily triggered by medications. These conditions are characterized by extensive epidermal necrosis, mucosal involvement, and systemic complications. Ciprofloxacin, a fluoroquinolone antibiotic, is an uncommon but recognized cause of SJS. Early recognition and multidisciplinary management are crucial in reducing morbidity and mortality.

**Case Presentation:** We report a case of a previously healthy 23-year-old woman who developed SJS following ciprofloxacin use. The patient presented with widespread erythematous patches, blistering lesions, mucosal erosions, ocular involvement, and urogenital ulcerations. The SCORTEN score was calculated to be 2, indicating an estimated mortality risk of 12.1%.



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Management included immediate discontinuation of the suspected causative drug, systemic corticosteroids, wound care, fluid and electrolyte balance maintenance, and supportive ophthalmologic and urologic treatments. The patient demonstrated clinical improvement and was discharged after a 11-day hospital stay.

**Conclusion:** This case highlights the potential for ciprofloxacin to induce SJS, emphasizing the need for vigilance in drug-induced cutaneous reactions, even in younger patients. Prompt diagnosis, drug withdrawal, and comprehensive supportive care play pivotal roles in patient outcomes. Given the unpredictability of long-term sequelae, continued monitoring is essential.

**Keywords:** Stevens-Johnson Syndrome, Toxic Epidermal Necrolysis, Ciprofloxacin, Drug-induced skin reaction, SCORTEN



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## INTRODUCTION

Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are dermatologic emergencies characterized by widespread epidermal necrolysis and sloughing.They are considered to have the same pathophysiology and are classified based on body surface area (BSA) involved. The syndrome can be subdivided into SJS in cases of involvement of BSA below 10%, SJS/TEN overlap in 10-30% BSA, or TEN when skin detachment extends beyond 30% BSA.[1]

Epidemiologically, the estimated incidence of SJS worldwide is between 1 and 6 cases per million population per year.[2] In the United States, the annual incidence of SJS is approximately 3.4 - 6.1 cases per million population.[3] Females are more commonly affected than males at a ratio of approximately 1.5:1. The mortality rates are 4.8–9% for SJS, 19.4–29% for SJS/TEN, and 14.8–48% for TEN.[1] In Indonesia, the estimated incidence is around 12 cases per year. Epidemiological data from Sanglah General Hospital in Bali reported a total of 40 cases of SJS, 3 cases of SJS/TEN overlap, and 14 cases of TEN from 2014 to April 2018.[4]

Medications are the leading cause of SJS and TEN. The drugs most strongly associated with these conditions include allopurinol, carbamazepine, cotrimoxazole, other sulfonamide antibiotics, sulfasalazine, lamotrigine, nevirapine, oxicam-class nonsteroidal anti-inflammatory drugs (NSAIDs), phenobarbital, and phenytoin. Agents classified as moderate-risk encompass cephalosporins, macrolides, quinolones, tetracyclines, and NSAIDs of the acetic acid subclass. Previous research has indicated that certain medications pose minimal or negligible risk, including beta-blockers, angiotensin-converting enzyme inhibitors, calcium channel blockers, sulfonamide-derived thiazide diuretics, sulfonylurea antidiabetic agents, insulin, and NSAIDs belonging to the propionic acid category.[2] Several risk factors have also been associated with SJS, including HIV infection, active malignancies, and collagen vascular diseases. Genetic predispositions also play a role, such as the presence of the HLA-B*1502 allele, which has been linked to an increased risk of SJS following carbamazepine use. Additionally,



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infectious agents, including the herpes simplex virus, Mycoplasma bacteria, and the measles vaccine, have been implicated as potential triggers.[5]

SJS and TEN are clinical diagnoses without universally established criteria. Histopathological findings, while supportive, are not routinely performed. The diagnosis of SJS is typically considered in patients with a history of exposure to medications known to be associated with the syndrome, particularly when accompanied by malaise and/or a febrile prodromal illness, along with characteristic lesion morphology indicative of SJS.[6] Given the significant morbidity and mortality associated with SJS, early diagnosis, along with prompt identification and discontinuation of the causative drug, is crucial in reducing disease progression and improving patient outcomes.[7] The SCORTEN scale is commonly used to assess prognosis in epidermal necrolysis, with higher scores correlating with increased mortality rates.[8] The management of SJS and TEN begins with the immediate discontinuation of the suspected causative agent, followed by the providing of comprehensive supportive care.

This case report aims to further evaluate a case of SJS in a previously healthy 23-year-old female, suspected to have been triggered by prior medication use, particularly ciprofloxacin.

## CASE REPORT

A 23-year-old Balinese female, an Indonesian citizen, was consulted from the Emergency Department to the Dermatology and Venereology Department at Wangaya General Hospital, Denpasar, with complaints of widespread erythematous patches over the entire body and blistering lesions on the face for the past two days. Upon history-taking, the patient initially experienced facial swelling and a rash, which progressively extended to the neck, chest, abdomen, upper limbs, and back. The erythematous patches were followed by the appearance of painful vesicles on the face the following day. The patient reported thickened, swollen lips with ruptured vesicles that formed dark crusts with purulent discharge, making it difficult to open her mouth. She also experienced extensive oral ulcers and throat pain, causing difficulty in eating. Additionally, the patient



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complained of pruritus, redness, discomfort, and excessive tearing in both eyes, as well as dysuria.

Three days prior, the patient had visited a general practitioner with complaints of fever, generalized pruritus, oral ulcers, vaginal discharge, and genital pruritus. She was prescribed ciprofloxacin 500 mg, Nodrof PE (Paracetamol 650 mg, Phenylephrine 10 mg, Diphenhydramine 15 mg), Methylprednisolone 4 mg, Cetirizine 10 mg, and Acyclovir ointment. Cutaneous lesions began to appear approximately 24 hours after medication intake.

The patient had no prior history of similar skin conditions. She denied any history of prolonged fever, chronic cough, or persistent diarrhea. There was no history of sexually transmitted infections, such as genital or anal vesicular lesions. She also denied any drug or food allergies, malignancies, chemotherapy, or systemic diseases such as asthma, diabetes mellitus, or hypertension. No family members had similar complaints. The patient worked as a beauty store attendant and lived with her parents.

Upon physical examination, the patient was conscious and appeared moderately ill. She was alert, with a Glasgow Coma Scale (GCS) score of E4V5M6. Vital signs were as follows: blood pressure 122/71 mmHg, pulse rate 122 bpm, respiratory rate 20 breaths per minute, axillary temperature 37.6°C, oxygen saturation 98% on room air, and a Visual Analogue Scale (VAS) pain score of 7. General examination revealed bilateral conjunctival hyperemia with mucopurulent discharge. Oral examination was limited due to pain and restricted mouth opening. The lips exhibited multiple erosions covered with dark crusts and serous exudate. Cardiovascular examination revealed normal heart sounds (S1 and S2), with no murmurs. Respiratory examination showed vesicular breath sounds with no rhonchi or wheezing. Abdominal examination was unremarkable, with normal bowel sounds and no distension. The extremities were warm, with no edema or lymphadenopathy.

Dermatologic status were as follows: on the facial region, multiple well-defined, coalescing erythematous papules in a geographic shape were observed bilaterally on the cheeks, accompanied by flaccid vesicles and bullae. The perioral region exhibited



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multiple, well-demarcated, round erosions measuring 0.2-0.5 cm in diameter, covered with purulent discharge and black crusts, along with edema. The neck, chest, back, upper extremities, and bilateral thighs displayed multiple well-defined purpuric lesions ranging from 0.5 to 1 cm in size, symmetrically distributed. In the genital region, multiple well-demarcated erythematous macules and erosions of varying sizes (0.2-0.5 cm) were observed. Diascopy testing indicated non-blanching lesions. The estimated epidermal detachment area was approximately 3%.



**Figure 1a.** Well-defined erythematous huge oozing plaques in a geographic shape were



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observed bilaterally on the cheeks and chin, accompanied by flaccid vesicles and bullae. Multiple well-demarcated and round erosions covered with purulent discharge and dark crusts, accompanied by edema. **Figure 1b, 1c, 1d and 1e.** Multiple well-demarcated irregularly purpuric shaped lesions symmetrically distributed in both arms, upper trunk and back.

Additional examinations, including a complete blood count (February 4, 2025), revealed the following results: erythrocytes 5.12 $10^6$/µL (4.2–5.4), hemoglobin 12.1 g/dL (12.0–16.0), hematocrit 37.6% (37.0–47.0), platelets 244 $10^3$/µL (150–440), leukocytes 15.89 $10^3$/µL (4–10), neutrophils 76.1% (50–70), lymphocytes 12.2% (20–40), monocytes 10.2% (2–8), eosinophils 1.4% (0–4), and basophils 0.1% (0–1). Liver function tests showed SGOT 30 U/L (0–37.0) and SGPT 24 U/L (0–42). Renal function tests revealed a BUN level of 26 mg/dL (10–50) and creatinine 0.9 mg/dL (0.3–1.2). Electrolyte levels included sodium at 133 mmol/L (130–145), potassium at 3.2 mmol/L (3.5–5.5), and albumin at 3.6 g/dL (3.8–5.1).The blood sugar level is 76 mg/dL, and the bicarbonate ($HCO_3^-$) concentration is 28 mmol/L.

Based on the patient's history, physical examination, and laboratory findings, a diagnosis of SJS, suspected to be caused by ciprofloxacin, was established. The dermatovenereologist managed the patient by discontinuing the suspected causative drug and initiating intravenous normal saline infusion at a rate of 20 drops per minute. Topical therapy consisted of twice-daily application of triamcinolone acetonide to erosive lesions on the lips and a combination cream containing fusidic acid and desoximetasone to erythematous lesions on the body. Additionally, 0.9% normal saline compresses were applied to the lips and genital area every 6 hours for 10 minutes. The patient also received intravenous dexamethasone at a dose of 10 mg every 12 hours with a gradual tapering dose, with subsequent transition to oral corticosteroids.

For the patient's conjunctivitis, the ophthalmologist prescribed Lyteers eye drops and Dexamethasone, Neomycin, and Polymyxin B eye drops, with one drop instilled in both eyes six times daily. The internal medicine team administered intravenous potassium



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chloride (KCl) 25 mEq to correct hypokalemia and intravenous gentamicin 8 mg for seven days to address the elevated white blood cell count. Beyond pharmacological interventions, the patient's family was provided with comprehensive education regarding the condition's etiology, progression and management plan.

Over 11 days of hospitalization, the patient demonstrated continuous clinical improvement and was subsequently discharged. At the time of discharge, dermatological examination revealed multiple areas of hyperpigmentation across the body, along with a large erythematous patch on both cheeks and the chin, and residual erosions on the lips. The patient was prescribed oral methylprednisolone, starting at 16 mg every 12 hours on the first day, with a gradual taper over six days. A combination cream containing gentamicin and desoximetasone was prescribed for application twice daily to erythematous lesions, while topical triamcinolone acetonide was to be applied twice daily to the lips. Ocular symptoms and genital erosions had completely resolved at the time of discharge. A follow-up visit at the dermatology and venereology outpatient clinic was scheduled for five days later.

During the follow-up examination, a dermatological assessment showed that the erythematous patch on the patient's face had begun to fade, while the erosions on the lips had almost completely resolved. The crusted lesions on the chest, back, and arms had spontaneously detached, leaving residual hyperpigmented areas. No new lesions were observed. The combination cream containing gentamicin and desoximetasone was continued for the remaining erythematous lesions.



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**Figure 2a.** A fading erythematous patch on the face accompanied by a few papules. **Figures 2b and 2c.** Multiple erythematous macules observed after spontaneous detachment of the crusts.

## DISCUSSION

SJS and TEN are rare, acute, and potentially fatal mucocutaneous adverse reactions, marked by extensive blistering and erosion of the skin and mucous membranes. While medications are the most common triggers, these conditions can also be induced by certain infections.[9] This condition can affect individuals of all ages; however, the risk increases in those over 40 years old. It is more frequently observed in females than in males, with a reported incidence ratio of approximately 1.5:1.[10]

A systematic review by Lee EY et al. included a meta-analysis evaluating the prevalence of specific antibiotics associated with SJS/TEN. The analysis estimated that 28% of SJS/TEN cases were attributed to antibiotic use. Among it, sulfonamides were the most frequently associated (32%), followed by penicillins (22%), cephalosporins (11%), fluoroquinolones (4%), and macrolides (2%).[11]

The precise pathophysiology of SJS and TEN remains incompletely understood. These conditions are primarily associated with drug-induced hypersensitivity reactions in genetically predisposed individuals. The underlying mechanism involves both type III and



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type IV hypersensitivity reactions, which lead to widespread epidermal necrosis through immune-mediated processes.[12] Epidermal necrosis is thought to occur via two primary mechanisms: the extrinsic and intrinsic pathways. The extrinsic pathway is driven by cytotoxic lymphocytes, monocytes, and key mediators such as granulysin, perforin, granzyme, and Fas/Fas ligand interactions, ultimately inducing keratinocyte apoptosis. Meanwhile, the intrinsic pathway involves the release of toxic substances from keratinocytes, resulting in the generation of reactive oxygen species, which in turn stimulate tumor necrosis factor-alpha (TNF-α) production, further amplifying tissue damage.[13]

The case presented in this report is particularly intriguing, as the patient is a healthy young woman with no underlying comorbidities, which deviates from the typical demographic profile of SJS/TEN patients commonly described in the literature. Moreover, the characteristic skin lesions of SJS in this patient emerged within 24 hours of ciprofloxacin consumption, which is significantly faster than the typical onset reported in most SJS cases.

A history of exposure to high-risk medications is generally limited to the initial eight weeks of treatment, with most causative drugs being administered continuously for a period of 4 to 28 days prior to the onset of symptoms. SJS/TEN typically presents with non-specific prodromal symptoms, including fever, malaise, sore throat, headache, rhinorrhea and cough in some cases, which emerge one to three days before skin lesions develop.[4,13] Skin and mucosal involvement are almost universal, initially presenting as erythematous macules or atypical target lesions on the trunk, which later evolve into widespread erythematous patches with darkened centers, flaccid blisters, a positive Nikolsky sign, and extensive epidermal detachment. Mucosal involvement is highly prevalent in SJS/TEN, with two or more mucosal surfaces affected in up to 80% of cases. The oral mucosa is the most frequently involved site, with nearly all patients experiencing mucositis and ulceration. In addition to oral involvement, ocular and gynecologic manifestations are also commonly observed.[1,13] Other complications include dehydration and severe malnutrition, along with shock and multiple organ dysfunction, such as renal



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failure, thromboembolism, sepsis, and disseminated intravascular coagulation.[13]

Patients suspected of having SJS/TEN require immediate hospital evaluation, as management of this condition is multidisciplinary. Assessing prognosis is a crucial component in the management of SJS/TEN, as it aids in guiding therapeutic decisions and appropriate medical settings. The SCORTEN remains the most widely utilized tool for predicting outcomes in patients with SJS/TEN.[1,14] The SCORTEN score for this patient was determined to be 2, based on the presence of tachycardia and elevated urea levels, correlating with an estimated mortality rate of 12.1%.

The management of SJS/TEN begins with the immediate discontinuation of the suspected causative agent, followed by comprehensive supportive care. This includes wound care, maintenance of fluid and electrolyte balance, nutritional support, temperature regulation, pain management, and infection control. In conservative wound care approaches, detached epidermis may be left in place as a biological dressing, with additional non-adhesive dressings applied to promote healing. Gentle cleansing with sterile water or diluted chlorhexidine solution is recommended during dressing changes. Large blisters may be drained with a sterile needle while preserving the overlying epidermis. Maintaining strict aseptic conditions is critical in wound care.[15] Oral nutrition may be challenging in cases with extensive oral mucosal involvement. If necessary, nasogastric feeding should be initiated early and continued throughout the acute phase of SJS/TEN. Patients with SJS/TEN are at high risk of infection, with bacteremia occurring in 30–50% of cases. Sepsis is the leading cause of mortality.[16]

In this case, supportive treatment included intravenous administration of normal saline with strict fluid balance monitoring. Wound care of the face consisted of aspirating the bullae, cleaning hemorrhagic and serous crusts on the lips with sterile NaCl solution, applying NaCl compresses 4-6 times a day, and administering topical triamcinolone acetonide ointment for erosive lesions. A combination cream containing fusidic acid and desoximetasone was applied twice daily to erythematous lesions on the body. All wound care procedures were performed under sterile conditions.

Urogenital involvement occurs in up to 70% of SJS/TEN cases, presenting as



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erosions on the scrotum/labia and penis/vulva, often accompanied by dysuria and urinary retention.[17] In this patient, erosive lesions were present in the genital region, and management included multiple daily applications of normal saline compresses.

Acute ocular involvement occurs in approximately 80% of patients, presenting with a spectrum of manifestations, including conjunctival erythema, excessive tearing, ocular discharge, pseudomembrane formation, and, in severe cases, total corneal epithelial loss. The severity of ocular complications does not necessarily correlate with the SCORTEN score or the extent of skin detachment. Since chronic ocular sequelae remain unpredictable, prompt and appropriate management during the acute phase is essential to minimize long-term complications.[18] Conjunctivitis was observed in this patient, and the ophthalmologist prescribed a combination of corticosteroid, antibiotic, and lubricating eye drops.

Currently, no pharmacological treatment has been definitively proven to be effective for SJS/TEN. Various systemic immunosuppressive and immunomodulatory agents, including systemic corticosteroids, intravenous immunoglobulin (IVIG), cyclosporine, anti-TNF agents, and plasmapheresis, have been explored as therapeutic options. However, evidence supporting their efficacy remains inconclusive, and none can be universally recommended.[15] In this case, the patient was initially administered 20 mg of intravenous dexamethasone on the first day, followed by a gradual tapering of the dose over the next seven days before transitioning to oral methylprednisolone. The patient exhibited significant clinical improvement and was eventually discharged from the hospital on the 11th day.

## CONCLUSION

SJS/TEN is a severe life-threatening condition with skin, mucosa, and multisystem involvement. This condition results from a hypersensitivity reaction, mostly triggered by medication use. Early diagnosis and treatment are critical for reducing mortality, with SCORTEN being a widely used scoring system to assess prognosis and estimate mortality risk in epidermal necrolysis. Our patient is a healthy young woman with no known



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comorbidities who developed SJS/TEN following ciprofloxacin use. The affected body surface area was 6%. The patient was hospitalized and received multidisciplinary supportive and symptomatic treatment from dermatologist, ophthalmologist, and internist for seven days, resulting in clinical improvement.

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### Conflict of Interest

The authors affirmed that there were no conflicts of interest in this study.

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### Ethical Clearance

Authors have secured informed consent regarding patient medical record for this case report.

### Author Contribution

All authors contributed equally in this research and publication of this manuscripts.

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