

Ciprofloxacin and Tinidazole Induced Steven Johnson Syndrome

Zeenath Unnissa

Deccan School of Pharmacy

Shayesta Nishath

Deccan School of Pharmacy

Sameera Tarannum

Deccan School of Pharmacy

Madiha Nazish

Deccan School of Pharmacy

Ameena Farheen

Deccan School of Pharmacy

Sofia Tabusum

Deccan School of Pharmacy

Mohammed Misbah Ul Haq

drmdmisbah@outlook.com

Deccan School of Pharmacy

Short Report

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Abstract

Background:

Stevens–Johnson syndrome (SJS) is a rare, potentially life-threatening mucocutaneous adverse drug reaction characterized by epidermal necrosis and mucosal involvement. It is most commonly triggered by medications, particularly antibiotics and anticonvulsants.

Objective:

To report a rare case of probable ciprofloxacin- and tinidazole-associated SJS and to describe the diagnostic challenges, management strategy, and clinical outcome.

Design:

We describe a 65-year-old woman with multiple comorbidities who developed painful oral ulcers and erythematous cutaneous lesions involving the palms and soles following recent exposure to ciprofloxacin and tinidazole. Diagnosis was made clinically based on temporal drug association, characteristic mucocutaneous involvement, and exclusion of alternative causes.

Intervention:

The suspected offending drugs were immediately discontinued. The patient received systemic corticosteroids, intravenous fluids, symptomatic antihistamines for pruritus, and meticulous supportive care.

Result:

Disease progression halted within 48–72 hours after initiation of therapy, followed by gradual resolution of mucocutaneous lesions. The patient recovered without major sequelae and was discharged after clinical stabilization.

Conclusion:

This case highlights the importance of early recognition of SJS, prompt withdrawal of suspected drugs, and individualized supportive care. Although ciprofloxacin and tinidazole are uncommon triggers, they should be considered in patients presenting with acute mucocutaneous reactions.

INTRODUCTION

Stevens–Johnson syndrome (SJS) is a rare and severe cutaneous adverse drug reaction (CADR) affecting the skin and mucous membranes. It is most frequently caused by medications, although infections may also serve as triggers.¹ SJS is characterized by epidermal necrosis, mucosal erosions, and systemic symptoms, with an estimated incidence of 2–7 cases per million population per year.^[2,3]

Cutaneous adverse drug reactions encompass a broad clinical spectrum ranging from mild exanthematous eruptions to severe life-threatening conditions such as SJS and toxic epidermal necrolysis (TEN). This spectrum includes maculopapular eruptions, urticaria, fixed drug eruption, erythema multiforme, drug reaction with eosinophilia and systemic symptoms (DRESS), SJS, and TEN.⁴ SJS and TEN are considered variants of the same disease entity and are distinguished primarily by the extent of epidermal detachment⁵.

SJS is defined by epidermal detachment involving < 10% of body surface area (BSA), whereas TEN involves > 30% BSA, with SJS/TEN overlap accounting for intermediate involvement⁶. The pathogenesis involves a type IV hypersensitivity reaction mediated by cytotoxic T lymphocytes and natural killer cells, leading to keratinocyte apoptosis⁷.

More than 80% of SJS/TEN cases are drug-related, with common offending agents including antibiotics (particularly sulfonamides and β -lactams), anticonvulsants, NSAIDs, and allopurinol⁸. Fluoroquinolones and nitroimidazoles are rare causes, making the present case clinically noteworthy.

Both SJS and TEN are considered variants of the same condition, distinguished mainly by the extent of skin involvement⁷. It is a rare, life-threatening acute mucocutaneous Syndrome⁸. It typically results from an adverse Reaction to medication, Causing widespread skin blistering and peeling. The condition affects at least 30% of the body's skin surface, leading to painful, open sores and increased risk of infection⁸.

The condition is characterized by the detachment of the epidermis from the dermis, leading to painful blisters and skin loss. Diagnosis involves a combination of clinical evaluation and laboratory investigations. The hallmark symptoms include extensive skin detachment, mucosal lesions, and systemic symptoms like fever and malaise⁸. Diagnostic tools include skin biopsy, which shows necrosis of keratinocytes, and ruling out other similar conditions like toxic epidermal necrolysis (TEN). Blood tests may reveal elevated inflammatory markers, while a thorough patient history helps identify possible triggers⁸.

The main etiologic categories include infections, drug-induced reactions, malignancy-related factors, genetic predisposition, and idiopathic cases. Infections like viral, fungal, and bacterial infections are known contributors. Drug-induced SJS can result from antibiotics (e.g., penicillin, sulpha drugs, ciprofloxacin), anticonvulsants (such as phenytoin, valproic acid, and carbamazepine), antiretrovirals like indinavir, as well as NSAIDs, analgesics, antigout medications, and other drugs (e.g., tramadol, pantoprazole, sertraline, and allopurinol)⁹. Genetic factors also play a role, as certain human leukocyte

antigens (HLAs) increase the risk of severe skin reactions. Additionally, in 25–50% of cases, SJS remains idiopathic, with no identifiable cause¹⁰.

Clinical pharmacists play a critical role in the management of Stevens-Johnson Syndrome (SJS) by ensuring prompt identification and cessation of offending drugs, optimizing pharmacological therapy, and monitoring for drug interactions and adverse effects. They assist in selecting appropriate immunosuppressive agents, such as corticosteroids or IVIG, and ensure accurate dosing. Pharmacists also provide patient education on drug allergies and preventive measures to avoid future triggers. Through collaboration with the healthcare team, they help enhance treatment efficacy and improve patient outcomes using evidence-based approaches¹¹.

CASE REPORT

History of Present illness: A 65-year-old woman presented with a 5-day history of painful erosions involving the upper and lower lips, accompanied by erythematous discoloration of the palms, soles, and lower limbs, associated with swelling and pruritus. The symptoms began approximately 3–5 days after initiation of ciprofloxacin and tinidazole, which were prescribed for a presumed gastrointestinal/urinary tract infection. The initial symptom was a burning sensation in the oral cavity, followed by the development of mucosal ulcers and cutaneous erythema.

Medication History: A detailed medication review revealed that the patient had been receiving antidiabetic, antihypertensive, and psychiatric medications for several years without prior adverse reactions. Ciprofloxacin and tinidazole were the only newly introduced drugs, establishing a strong temporal relationship with symptom onset.

Past Medical history: The patient had a history of a similar episode several years earlier, characterized by erythematous lesions over the palms and soles, skin peeling, and periocular hyperpigmentation. However, details regarding medications used during that episode were unavailable. Comorbidities included type 2 diabetes mellitus, chronic kidney disease (CKD), and hypertension.

Clinical Examination: Dermatological examination revealed erythematous, tender lesions over the palms and soles with mucosal ulceration of the lips. Epidermal detachment was limited (<10% BSA), with no target lesions suggestive of erythema multiforme. Ocular and genital mucosa were spared. A skin biopsy and clinical photographs could not be obtained due to patient refusal and acute clinical instability.

Laboratory Test Performed:

Differential diagnosis: The differential diagnoses considered included erythema multiforme, generalized fixed drug eruption, and drug-induced mucositis. The presence of painful mucosal involvement, systemic symptoms, and characteristic distribution favored a diagnosis of SJS.

The laboratory findings are summarized in Table 1.^(Table 1) On the day of admission and during the treatment based on subjective evidence, the following laboratory tests have been performed.

Table 1: Laboratory Investigations

<i>Lab Parameter</i>	DAY-1	<i>Normal value</i>
HB	11.6	13-18gms%
WBC	9700	4000-11000c/cmm
RBC	3.9	4.3-5.7c/mcl
DLC=N+L+E+M+B	81+16+2+1	
PLATELETS	3.24	1.5-4.5 lakhs/cumm
Viral markers: HIV 1&2 , HBsAg, HCV	Negative	
HbA1C	8.7	
Sr. Creatinine	5.8	<200 IU/M
Urea	128	
Uric acid	9.6	
Total serum IGE	22.74	0-100
Protein creatinine ratio: urine protein	23.9	0-15
Urine creatinine	39.4	15-278
Protein creatinine ratio	0.6	
Sodium	130.9	136-145
Potassium	4.56	3.5-4.5
Chloride	100.3	95-105

USG OF ABDOMEN & PELVIS: Normal

ULTRASONOGRAM FOR ABDOMEN : Fatty liver grade 1, Bilateral mild atrophic kidneys

ECG: Sinus tachycardia, Abnormal Q-wave III

X-RAY LUMBAR SPINE AP & LATERAL VIEW: Features suggestive of end plate osteophytes are seen in the lower lumbar vertebrae

2D ECHO: Normal

AEROBIC CULTURE AND SENSITIVITY URINE:

SUSCEPTIBILITIES – Cefoperazone Sulbactam, Piperacillin-Tazobactam, Colistin, Meropenem

MANAGEMENT AND OUTCOME:

Ciprofloxacin and tinidazole were immediately discontinued. The patient was managed in a monitored setting with systemic corticosteroids, intravenous fluids, and supportive care. Antihistamines were administered only for symptomatic relief of pruritus and were used judiciously considering the underlying CKD. Disease progression ceased within 48–72 hours, with gradual improvement of mucocutaneous lesions. The patient was discharged after stabilization with advice for lifelong avoidance of both ciprofloxacin and tinidazole, given the severity of the reaction and inability to identify a single culprit drug.

TREATMENT:

The main aim of the treatment is to treat the disease, to stabilise the patient and to reduce the ulcers and discoloration of palm, feet and lower limbs.

Treatment Options:

1. Non-Pharmacological:

- a. **Hospital Environmental Care:** Patients should undergo interprofessional assessment in a specialized – Intensivist, Dermatologist, Plastic surgery or burns specialist, Ophthalmologist, Gynecologist, Urologist, Respiratory physician, Physical therapist, Nutritionist ¹².
- b. **Patient Care:** Cessation of the suspected causative drug(s), Hospital admission: preferably to an intensive care and/or burn unit, Fluid replacement (crystalloid), Nutritional assessment: may require nasogastric tube feeding, Temperature control: warm environment, emergency blanket, Pain relief, Supplemental oxygen and, in some cases, intubation with mechanical ventilation, Sterile/aseptic handling ¹³.
- c. **Skincare:** requires daily examination of skin and mucosal surfaces for infection, non-adherent dressings, and avoidance of trauma to the skin. Mucosal surfaces require careful cleansing and topical anesthetics, Gentle removal of necrotic skin/mucosal tissue, Culture of skin lesions, axillae, and groins every two days ^{12,13}.
- d. **Fluid and electrolyte balance:** Peripheral intravenous line can be left in place for a long duration and should not be replaced before 96 h unless there is evidence of phlebitis, local infection or malfunction. Routine culture of vascular catheter tip is not recommended. Patients of toxic epidermal necrolysis lose a significant amount of fluid as blister fluid and insensible fluid loss and should be assumed to be hypovolemic. Adult patients having involvement of 50% of body surface area lose around 3–4 L of fluid every day ¹⁴.

2. Pharmacological:

- a. **Immediate Actions:** Treatment of Stevens-Johnson Syndrome (SJS) starts with identifying and stopping the drug causing the reaction, as this is critical to prevent further progression. Isolating the

patient is also important to reduce the risk of infection, given the extensive skin barrier damage associated with SJS.

- b. **Supportive Care:** Essential supportive measures include administering IV fluids to maintain hydration and corticosteroids to manage inflammation. If available, intravenous immunoglobulin (IVIG) may be used to modulate the immune response. Antibiotics are given only if there are secondary infections to control, while using the minimum necessary medications to avoid triggering further adverse reactions.
- **Corticosteroids:** Corticosteroids may help reduce inflammation in early stages of SJS, though their use is controversial due to possible risks of infection and delayed healing ¹⁵.
 - **Intravenous Immunoglobulin (IVIG):** IVIG is used for its immunomodulatory effects, especially in patients with severe and rapidly progressing cases of SJS ¹⁶.
 - **Cyclosporine:** Cyclosporine, an immunosuppressant, has been shown to halt the progression of SJS by inhibiting T-cell activation ¹⁷.
 - **Thalidomide:** Thalidomide has been tested as a treatment due to its anti-inflammatory properties, though its use is limited because of concerns about worsening outcomes in certain cases ¹⁸.
 - **Plasmapheresis:** Plasmapheresis helps to remove circulating toxins and drugs in severe SJS cases, and may improve outcomes in rapidly progressing patients ¹⁹.
 - **N-Acetylcysteine:** N-Acetylcysteine (NAC) has been used experimentally as an antioxidant and mucolytic agent to mitigate oxidative stress in SJS/TEN ²⁰.
- c. **Management of Lesions:** Oral lesions are managed with mouthwashes and topical anesthetics to reduce pain and enable the patient to stay hydrated. Skin lesions are carefully treated by covering exposed areas with saline compresses to promote healing and protect against infection. Tetanus prophylaxis is also provided if needed, given the increased risk from open wounds.
- d. **Ocular Care:** For ocular involvement, treatment focuses on protecting the eyes with aggressive lubrication to maintain moisture, while topical steroids and antibiotics manage inflammation and prevent scarring. Symblepharon lysis is used as necessary to prevent adhesions and reduce long-term complications.

Treatment given:

- The patient was given the **Inj. Hydrocortisone (Dose:200mg)** through intravenous route, 50 mg IV every 6 hours to reduce inflammation in the body, and to prevent the progression of the condition, and reduce the skin and mucosal lesions.
- **Inj. Avil (Dose: 2ml)** was given twice a day for 5 days through intravenous and intramuscular route to help reduce itching sensation and provide relief from the allergic reaction
- **Inj. Decadron (Dose: 0.5cc)** through intravenous route, twice a day for 5 days to reduce the inflammation, and suppress the discoloration and swelling.

- **Tab. Allegra (Dose:120mg)** once a day through oral route for 4 days to reduce itching, skin irritation and discomfort to the patient.
- **Mucopain Gel (Dose: 5ml)** is a local anesthetic applied on the affected areas inside the mouth of about 5 ml, 3-4 times a day to reduce painful sores and ulcers in the mucosal area making it easier for the patient to eat and drink.
- **Chlorhexane mouthwash (Dose: 10-15ml)** is orally rinsed in the mouth for about 30-45 seconds, to prevent infections and oral lesions. It was prescribed to reduce oral; ulcers.

DAY WISE ASSESSMENT:

Day 1: The patient had a temperature of 98.5°F, blood pressure of 110/80 mmHg, and a pulse rate of 100 beats per minute. They presented with a burning sensation in the mouth and absence of bowel movements for two days, leading to their transfer to the Medical ICU. No issues with urination or flatulence were noted, but the patient exhibited pallor and bilateral pedal edema. Dermatology confirmed a diagnosis of Candida in the mouth, while the orthopaedic consultation indicated lower back pain due to a fall a month ago. Treatment included intravenous fluids, antihistamines, diuretics, corticosteroids, and supportive medications such as topical agents and nebulization.

Day 2: The patient's vitals included a temperature of 98°F, blood pressure of 110/70 mmHg, and a pulse rate of 115 beats per minute with a respiratory rate of 14 breaths per minute. The patient was diagnosed with acute kidney injury (AKI), chronic kidney disease (CKD), uncontrolled diabetes mellitus (DM), and major depression disorder (MDD). A more severe condition, Stevens-Johnson Syndrome (SJS), was identified as a result of a reaction to specific antibiotics. The treatment plan focused on corticosteroid therapy, along with the administration of antipsychotic drugs.

Day 3: The patient's vitals were a temperature of 98°F, blood pressure of 130/80 mmHg, pulse rate of 120 beats per minute, and a respiratory rate of 18 breaths per minute. The patient's condition, including SJS, MDD, CKD, and newly diagnosed type 2 DM, was stable with no fresh complaints. The treatment plan continued with corticosteroid therapy, immunosuppressants, and intravenous fluids to manage the symptoms and provide supportive care.

Day 4: On this day, the patient had a temperature of 98°F, blood pressure of 130/80 mmHg, and a pulse rate of 122 beats per minute, with a respiratory rate of 21 breaths per minute. Steroid-induced hyperglycaemia and hyponatremia were noted, along with persistent tachycardia and issues with urination. The treatment regimen included antibiotics, continued corticosteroid therapy, and electrolyte management through intravenous fluids.

Day 5: On this day, the patient had a temperature of 98°F, blood pressure of 130/80 mmHg, and a pulse rate of 122 beats per minute, with a respiratory rate of 21 breaths per minute. Steroid-induced hyperglycaemia and hyponatremia were noted, along with persistent tachycardia and issues with urination. The treatment regimen included antibiotics, continued corticosteroid therapy, and electrolyte management through intravenous fluids.

DISCUSSION

Stevens-Johnson Syndrome (SJS) is a rare but severe disorder of the skin and mucous membranes, most often triggered by a hypersensitivity reaction to medications or, in some cases, infections. The disease is characterized by widespread skin blistering, mucosal involvement, and extensive epithelial detachment, resembling second-degree burns in severe cases.

The pathophysiology of SJS involves a delayed hypersensitivity reaction that leads to keratinocyte apoptosis, driven by the interaction between drug metabolites and the immune system. T-cell activation, cytokine release, and cytotoxic effects on skin cells result in the detachment of the epidermis. The primary triggers of SJS include antibiotics (especially sulfonamides, penicillins, and quinolones), anticonvulsants, and nonsteroidal anti-inflammatory drugs (NSAIDs).

Managing SJS requires a multidisciplinary approach due to the potential for severe systemic involvement, including the skin, mucous membranes, and internal organs. The treatment plan in this case consisted of corticosteroids, which are commonly used to suppress the inflammatory response and reduce the progression of skin damage in SJS. Additionally, the use of intravenous fluids was critical for maintaining hydration, especially given the risk of dehydration due to extensive skin loss. Antibiotics were included to prevent or treat secondary infections, a common complication in SJS due to compromised skin integrity.

This case underscores the importance of early recognition and aggressive management of SJS, especially in patients with multiple comorbidities. Supportive care, including nutritional support and electrolyte balance, along with precise pharmacological interventions, are key to managing such a critical condition. Regular monitoring and a tailored approach to each complication are crucial in improving the patient's prognosis.

LITERATURE REVIEW

Stevens-Johnson Syndrome (SJS) is a rare, acute, and potentially fatal mucocutaneous reaction, most often associated with adverse drug reactions. It lies on the severe end of the spectrum of cutaneous adverse drug reactions, along with Toxic Epidermal Necrolysis (TEN), with which it shares clinical and pathological features. The worldwide incidence of SJS is estimated at 1 to 7 cases per million annually, and mortality rates range from 5% to 10%, depending on the extent of skin involvement and patient comorbidities.¹

Several classes of medications have been implicated in the etiology of SJS, particularly antibiotics (e.g., sulfonamides, aminopenicillins, cephalosporins), anticonvulsants (e.g., phenytoin, carbamazepine), nonsteroidal anti-inflammatory drugs (NSAIDs), and allopurinol.² While sulfonamide antibiotics are well-documented triggers, fluoroquinolones such as ciprofloxacin are much less frequently reported.³ Tinidazole, a nitroimidazole antimicrobial, is also a rare cause of SJS, with only a few isolated case reports found in the literature.⁴

In a review by Mockenhaupt et al., ciprofloxacin was implicated in less than 1% of all reported SJS/TEN cases, though it remains a recognized cause.⁵ Tinidazole has even fewer associations, with most literature highlighting metronidazole more commonly.⁶ Despite the widespread use of both agents, severe cutaneous reactions such as SJS remain exceedingly rare, making this case report particularly noteworthy due to the dual involvement of ciprofloxacin and tinidazole.

The pathophysiological mechanism of drug-induced SJS involves a Type IV hypersensitivity reaction mediated by cytotoxic T lymphocytes and natural killer (NK) cells, which release granulysin—a key molecule in inducing keratinocyte apoptosis.⁷ Genetic susceptibility, particularly certain HLA alleles such as HLA-B15:02 and HLA-A31:01, has also been associated with higher risk, particularly in certain ethnic populations.⁸

Management protocols for SJS remain varied due to limited randomized controlled trials. Supportive care remains the mainstay, including withdrawal of the offending drug, fluid and electrolyte balance, infection prevention, and wound care.⁹ The role of systemic corticosteroids, as employed in this case, remains controversial but is often used in early stages to suppress inflammation.¹⁰ Other therapies such as intravenous immunoglobulin (IVIG), cyclosporine, and plasmapheresis have shown promise in selected cases.¹¹

This case contributes to the growing body of literature highlighting the need for vigilance with all antimicrobials, not just those traditionally known to cause SJS. It reinforces the importance of early recognition, discontinuation of suspected agents, and individualized supportive care in managing adverse drug reactions.

CONCLUSION

Stevens–Johnson syndrome is a dermatological emergency requiring prompt recognition and immediate withdrawal of offending agents. Clinicians should remain vigilant for rare drug associations, including fluoroquinolones and nitroimidazoles. Early multidisciplinary management and individualized supportive care remain central to favorable outcomes.

Declarations

Ethics approval and consent to participate:

Not applicable.

Consent to Publish:

Written informed consent for publication of this case report and accompanying clinical imaging and details was obtained from the patient’s legal guardian.

Availability of data and materials:

Not applicable.

Clinical trial number:

Not applicable.

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The authors declare that they have no competing interests.

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Author Contributions:

Zeenath Unnissa: Conceptualization, data collection, manuscript drafting.

Shayesta Nishath: Literature review, data interpretation, manuscript preparation.

Sameera Tarannum: Clinical data collection and manuscript review.

Madiha Nazish: Literature review and manuscript editing.

Ameena Farheen: Data acquisition and manuscript review.

Sofia Tabusum: Case documentation and critical revision of the manuscript.

Mohammed Misbah Ul Haq: Study supervision, manuscript conceptualization, critical revision, final approval of the manuscript, and corresponding author responsibilities.

All authors read and approved the final manuscript.

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