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SEVERE STEVENS-JOHNSON SYNDROME INDUCED BY ANTIBIOTIC THERAPY: A CASE REPORT

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ABSTRACT

Background: Stevens–Johnson Syndrome is a rare but life-threatening hypersensitivity reaction commonly triggered by medications, especially antibiotics. It is characterized by widespread skin lesions, mucosal ulceration, and epidermal detachment requiring urgent medical management. **Case Presentation:** A 34-year-old female developed high-grade fever, painful erythematous rash, oral ulcerations, conjunctival redness, and skin peeling following cotrimoxazole therapy prescribed for urinary tract infection. Dermatological examination revealed positive Nikolsky sign and epidermal detachment involving approximately 12% body surface area. Laboratory investigations showed leukocytosis and elevated inflammatory markers. Based on clinical findings and temporal association with drug exposure, a diagnosis of antibiotic-induced Stevens–Johnson syndrome was established. The offending medication was immediately discontinued, and the patient was treated with intravenous fluids, corticosteroids, antihistamines, wound care, nutritional support, and ophthalmic management. Progressive clinical improvement was observed with complete recovery after 14 days of hospitalization. **Conclusion:** Early recognition of Stevens–Johnson syndrome and prompt withdrawal of the causative drug are crucial for reducing morbidity and mortality. Careful antibiotic prescribing and patient awareness regarding adverse drug reactions remain essential for preventing severe complications.

KEYWORDS: Stevens–Johnson syndrome; Cotrimoxazole; Antibiotic-induced hypersensitivity; Adverse drug reaction; Severe cutaneous adverse reaction.

INTRODUCTION

Stevens–Johnson Syndrome is a rare but extremely dangerous disease that primarily impacts skin and mucous membrane tissues. The condition typically occurs as a result of specific medications and infectious diseases. The condition manifests when the immune system of the body begins to attack its own skin cells, which results in the development of painful rashes, blisters, and skin ulcers, and skin peeling.[1] The disease can severely damage the mouth, eyes, throat, and genital regions. Early treatment is essential to prevent the uncommon disease from developing into a life-threatening condition. The initial symptoms that patients experience include fever, body pain, and sore throat, which later develop into skin symptoms. The condition requires fast diagnosis because it progresses quickly, therefore immediate diagnosis and treatment are essential to enhance patient outcomes and minimize complications.[2]

Stevens–Johnson syndrome develops most frequently through medications which include antibiotics and antiepileptic drugs and painkillers as their primary treatment. Researchers found that cotrimoxazole serves as one of the most common antibiotics which results in severe skin reactions. Some people may develop hypersensitivity to a medication even after taking it for a short period.[3] The exact reason why this reaction occurs is not fully understood, but immune system activation and genetic factors are believed to play important roles. The body creates inflammatory substances after an incident which harms skin cells and mucosal tissue. Patients may initially ignore early symptoms because they resemble common viral infections. The rapid spread of skin lesions combined with painful ulcers shows disease progression, which requires urgent hospitalization with specialized medical treatment.[4]

Stevens–Johnson syndrome not only affects the skin but can also involve multiple organs and cause serious complications. Affected patients commonly experience severe dehydration along with secondary infections and breathing difficulties and eye damage and electrolyte imbalance. Eye involvement may lead to long-term complications such as dry eyes, corneal scarring, or even loss of vision if not treated properly.[5] Patients require burn management because their extensive skin loss needs intensive supportive care. The most vital treatment step requires doctors to stop administering the suspected medication. The combination of intravenous fluids with corticosteroids and wound care and pain management and nutritional support creates supportive therapies that help patients recover while decreasing their chances of dying. Hospitals require dermatologists to monitor patients who need skin care along with other specialized medical professionals.[6]

The case reports for Stevens-Johnson syndrome provide critical information which assists healthcare workers in identifying rare yet serious medication side effects. The reporting of such cases helps people understand how to use medications safely while protecting public health through pharmacovigilance efforts. A young female patient experienced a serious case of Stevens-Johnson syndrome which doctors considered to be antibiotic-induced after she received cotrimoxazole treatment for her urinary tract infection.[7] The patient experienced fever along with a complete body skin rash and mouth sores and skin peeling which needed special medical care. The patient achieved total recovery after doctors diagnosed her condition and stopped her medication and provided her with medical support which enabled her to avoid any severe health issues. The case demonstrates that medical professionals need to identify patient symptoms at an early stage while implementing strict rules for antibiotic use and educating patients about potential drug side effects.[8]

CASE PRESENTATION

Patient Demographics

A 34-year-old female weighing 58 kg presented to the emergency department of a tertiary care teaching hospital with complaints of fever, painful skin lesions, oral ulcerations, and redness of both eyes. The patient was a homemaker with no significant occupational exposure to chemicals or allergens. She belonged to a middle socioeconomic background and had no history of smoking, alcohol consumption, or recreational drug use.

Chief Complaints

The patient presented with the following complaints:

- High-grade fever for 3 days
- Painful red skin rash over the body for 2 days
- Peeling of skin over chest and upper limbs for 1 day
- Severe burning sensation of skin
- Painful oral ulcers with difficulty swallowing
- Redness and watering of both eyes
- Generalized weakness and malaise

History of Present Illness

The patient was apparently healthy until five days before admission, when she developed burning micturition and lower abdominal discomfort. She consulted a local practitioner and was diagnosed with urinary tract infection. She was prescribed oral cotrimoxazole (trimethoprim 160 mg + sulfamethoxazole 800 mg) twice daily along with paracetamol for fever.

After three days of antibiotic therapy, the patient developed sudden onset high-grade fever associated with malaise, headache, and sore throat. Within 24 hours, erythematous maculopapular rashes appeared over the trunk and upper limbs. The lesions rapidly progressed to involve the face, neck, back, and thighs. She complained of severe burning sensation and tenderness over the lesions.

Over the next day, multiple fluid-filled blisters developed followed by peeling of skin. Simultaneously, painful ulcerative lesions appeared inside the oral cavity, causing difficulty in eating and swallowing. Redness, watering, and photophobia of both eyes also developed. Due to worsening symptoms and progressive skin detachment, she was brought to the emergency department for further evaluation and management.

Medication History

The patient had received the following medications before onset of symptoms:

Medication	Dose	Frequency	Duration	Indication
Cotrimoxazole	160/800 mg	Twice daily	5 days	Urinary tract infection
Paracetamol	500 mg	As needed	3 days	Fever

No history of recent use of antiepileptics, NSAIDs, herbal medications, corticosteroids, or over-the-counter supplements was reported. There was no previous exposure to cotrimoxazole according to patient history.

Past Medical History

The patient had no history of:

- Diabetes mellitus
- Hypertension
- Bronchial asthma
- Tuberculosis
- Autoimmune disorders
- Previous adverse drug reactions
- Chronic dermatological illness
- Epilepsy or psychiatric illness

She had not undergone any major surgery in the past.

Family History

There was no family history of:

- Drug allergies
- Stevens–Johnson syndrome
- Autoimmune disorders
- Dermatological diseases
- Genetic disorders

Personal History

- Appetite: Reduced
- Sleep: Disturbed due to pain and burning sensation
- Bowel habits: Normal
- Bladder habits: Burning micturition initially present
- Addictions: None
- Diet: Mixed diet

General Physical Examination

On admission, the patient appeared toxic, dehydrated, and severely ill.

Parameter	Findings
Temperature	39.1°C
Pulse Rate	112 beats/minute
Blood Pressure	100/60 mmHg
Respiratory Rate	24 breaths/minute
Oxygen Saturation	97% on room air

Additional findings included:

- Pallor: Absent
- Icterus: Absent
- Cyanosis: Absent
- Clubbing: Absent
- Lymphadenopathy: Mild cervical lymph node enlargement present
- Edema: Absent

Dermatological Examination

Cutaneous examination revealed multiple irregular erythematous to violaceous macules distributed over the trunk, upper limbs, face, and neck. Several lesions had coalesced forming large areas of epidermal detachment. Multiple flaccid bullae were present over the chest and back.

Key dermatological findings included:

- Positive Nikolsky sign
- Epidermal detachment involving approximately 12% body surface area
- Tender skin lesions with burning sensation
- Crusted hemorrhagic erosions over lips
- Extensive oral mucosal ulceration
- Bilateral conjunctival congestion and photophobia

No genital mucosal involvement was observed.

Systemic Examination

Cardiovascular System

- S1 and S2 heard normally
- No murmurs detected

Respiratory System

- Bilateral air entry present
- No wheeze or crepitations

Central Nervous System

- Conscious and oriented
- No focal neurological deficits

Gastrointestinal System

- Mild epigastric tenderness
- No hepatosplenomegaly

Laboratory Investigations

Investigation	Result	Reference Range
Hemoglobin	12.4 g/dL	12–15 g/dL
Total Leukocyte Count	15,800 cells/mm³	4,000–11,000
Platelet Count	2.8 lakh/mm³	1.5–4 lakh
ESR	42 mm/hr	<20 mm/hr
C-Reactive Protein	48 mg/L	<10 mg/L
Serum Sodium	131 mEq/L	135–145
Serum Potassium	3.4 mEq/L	3.5–5.0
Blood Urea	28 mg/dL	15–40
Serum Creatinine	0.8 mg/dL	0.6–1.2
AST	68 IU/L	<40
ALT	72 IU/L	<40

Microbiological and Serological Investigations

- Blood culture: No growth
- Urine culture: No growth
- HIV serology: Negative
- Herpes simplex virus serology: Negative
- Hepatitis B and C screening: Negative

Causality Assessment

Causality assessment was performed using the Naranjo Adverse Drug Reaction Probability Scale. The patient obtained a score of 7, indicating a "probable" association between cotrimoxazole therapy and the development of Stevens–Johnson Syndrome. The temporal relationship, clinical improvement after drug withdrawal, absence of alternative causes, and characteristic manifestations strongly supported the diagnosis of antibiotic-induced Stevens–Johnson syndrome.

Diagnosis

The diagnosis of Stevens–Johnson Syndrome was established based on clinical presentation, medication exposure history, and dermatological findings. The patient developed high-grade fever, widespread erythematous targetoid lesions, mucosal ulceration, conjunctival involvement, and epidermal detachment involving approximately 12% of body surface area following cotrimoxazole therapy. Positive Nikolsky sign and rapid progression of skin lesions further supported the diagnosis. Laboratory investigations revealed leukocytosis and elevated inflammatory markers, while infectious causes were excluded through negative microbiological and serological tests. Dermatology consultation confirmed antibiotic-induced Stevens–Johnson syndrome secondary to cotrimoxazole administration.

Treatment and Clinical Management

Day 1: Immediately after diagnosis of Stevens–Johnson Syndrome, cotrimoxazole was discontinued permanently. The patient was shifted to the intensive care unit for close monitoring. Intravenous fluids (normal saline and Ringer lactate) were initiated to correct dehydration and electrolyte imbalance. Intravenous dexamethasone 8 mg twice daily was started to control inflammation and halt disease progression. Intravenous chlorpheniramine 25 mg twice daily was administered for symptomatic relief of hypersensitivity reactions. Pantoprazole 40 mg intravenously once daily was given for gastric protection. Strict aseptic wound care with sterile saline dressings and topical mupirocin ointment was initiated to prevent secondary bacterial infection.

Day 2: The patient continued to experience fever, oral pain, and conjunctival irritation, but no rapid progression of skin detachment was noted. Intravenous paracetamol 1 g every 8 hours was administered for fever and pain control. Oral cavity care was maintained using chlorhexidine mouthwash and topical lignocaine gel before meals to reduce pain during swallowing. Ophthalmology consultation recommended preservative-free lubricating eye drops every 4 hours along with topical moxifloxacin eye drops twice daily to prevent ocular infection. Nutritional supplementation through high-protein liquid diet and multivitamin therapy was initiated due to reduced oral intake.

Day 3: Clinical stabilization was observed with reduction in fever spikes and burning sensation. Dexamethasone 8 mg intravenously twice daily was continued. Intravenous fluids were adjusted according to urine output and electrolyte monitoring. Serum potassium correction was performed using potassium chloride supplementation. Daily wound

dressing and topical emollients were continued to maintain skin hydration and promote healing. No signs of secondary sepsis or respiratory complications were observed. The patient reported mild improvement in swallowing and oral discomfort.

Day 4: No new lesions appeared, indicating arrest of disease progression. Existing erosions started showing early re-epithelialization. Intravenous dexamethasone dosage was tapered to 4 mg twice daily. Antihistamine therapy with chlorpheniramine was continued for persistent itching and irritation. Oral feeding improved gradually with semisolid diet. Supportive nursing care including temperature maintenance, infection prevention, and pressure sore prevention was continued. Regular ophthalmic examination revealed improvement in conjunctival congestion without corneal involvement.

Day 5: The patient showed marked clinical improvement with reduction in erythema and tenderness. Intravenous corticosteroids were continued at tapering doses, and transition to oral prednisolone 30 mg/day was planned. Intravenous fluids were reduced as oral intake became adequate. Topical liquid paraffin and emollient creams were applied regularly to prevent excessive dryness and crusting. Liver function tests showed gradual normalization. Pain was managed with oral paracetamol 500 mg as required.

Day 6–7: Progressive healing of oral and cutaneous lesions was noted. Oral prednisolone 30 mg/day was initiated and gradually tapered over the next week. Lubricating eye drops and topical ocular antibiotics were continued. The patient remained afebrile and hemodynamically stable. Daily dermatological assessment showed healthy re-epithelialization without further epidermal detachment. Nutritional rehabilitation and psychological reassurance were provided to improve recovery and reduce emotional stress associated with extensive skin lesions.

Day 8–10: Significant improvement in mucosal lesions and skin integrity was observed. Most erosive lesions healed with residual hyperpigmentation. Corticosteroids were tapered gradually to avoid rebound inflammation. Routine laboratory investigations showed normalization of inflammatory markers and electrolyte levels. The patient regained adequate oral intake and mobility. No secondary infections, respiratory complications, or ocular sequelae developed during hospitalization.

Day 11–14: Complete clinical stabilization was achieved. Skin lesions healed substantially with minimal residual crusting. Oral prednisolone was tapered further and planned for discontinuation after discharge. The patient and family were counseled regarding lifelong avoidance of sulfonamide-containing medications and the risk of recurrence with re-exposure. A drug allergy card documenting cotrimoxazole-induced Stevens–Johnson syndrome was issued. The patient was discharged on day 14 with oral antihistamines, topical emollients, lubricating eye drops, and follow-up advice with dermatology and ophthalmology departments.

DISCUSSION

Stevens–Johnson Syndrome is a rare but potentially fatal mucocutaneous disorder commonly triggered by medications, particularly sulfonamide antibiotics. In the present case, cotrimoxazole was identified as the probable offending agent based on temporal association and clinical presentation. The patient developed fever, erythematous rash, mucosal ulceration, and epidermal detachment within five days of antibiotic initiation, which is consistent with the typical onset of drug-induced SJS. Similar observations were reported by Roujeau JC, who identified sulfonamide antibiotics as

major causes of severe cutaneous adverse reactions. Likewise, Mockenhaupt M reported that antimicrobial agents remain one of the leading causes of SJS worldwide, especially in hospitalized patients receiving broad-spectrum therapy.[9,10]

The pathogenesis of SJS involves immune-mediated destruction of keratinocytes leading to epidermal necrosis and mucosal damage. Activated cytotoxic T lymphocytes and natural killer cells release granulysin and inflammatory cytokines, causing widespread apoptosis of epidermal cells. In the present case, extensive oral and ocular involvement reflected severe mucosal inflammation commonly associated with antibiotic-induced SJS. Chung WH demonstrated the important role of immune dysregulation and genetic susceptibility in severe drug hypersensitivity reactions. Similarly, Harr T described granulysin-mediated keratinocyte apoptosis as a central mechanism responsible for epidermal destruction in SJS and TEN. These studies support the immunological basis observed in the current patient.[11,12]

Early withdrawal of the offending medication and prompt supportive care significantly influence prognosis in SJS patients. In this case, immediate discontinuation of cotrimoxazole along with intensive care management contributed to successful recovery without major complications. The patient received intravenous fluids, corticosteroids, wound care, nutritional support, and ophthalmological management, resulting in progressive re-epithelialization and stabilization.

Comparable outcomes were reported by Schneck J, who observed reduced mortality with early supportive treatment and specialized care. Additionally, Creamer D emphasized that multidisciplinary management involving dermatologists, intensivists, ophthalmologists, and pharmacists improves survival and minimizes long-term sequelae in patients with severe cutaneous adverse reactions.[13,14]

The use of systemic corticosteroids in SJS remains controversial due to concerns regarding infection risk and delayed wound healing. However, several recent studies suggest that early administration of corticosteroids may reduce inflammatory progression and improve recovery in selected patients. In the current case, intravenous dexamethasone followed by oral prednisolone was associated with rapid clinical stabilization and complete healing without secondary infection. Similar findings were reported by Zimmermann S, who demonstrated improved outcomes with immunomodulatory therapies in SJS/TEN patients. Furthermore, Kirchhof MG reported that individualized corticosteroid-based treatment strategies may decrease disease progression and hospitalization duration in severe cutaneous adverse reactions.[15,16]

CONCLUSION

Stevens–Johnson Syndrome is a rare but extremely serious reaction that may occur after the use of certain medications, particularly antibiotics such as cotrimoxazole. The present case highlights the importance of recognizing early warning signs including fever, skin rashes, oral ulcers, eye redness, and skin peeling after starting a new medicine. Rapid diagnosis, immediate discontinuation of the offending drug, and proper supportive treatment were essential in achieving complete recovery in this patient. Intensive care management, wound care, hydration, corticosteroid therapy, and ophthalmic monitoring helped prevent severe complications. This case emphasizes the need for cautious prescribing of antibiotics, patient counseling regarding adverse drug reactions, and prompt medical attention to reduce morbidity and improve overall patient outcomes.

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