ISSN- O: 2458 - 868X, ISSN–P: 2458 – 8687
National Library of Medicine - ID: 101731606

International Journal of Medical Science and Innovative Research (IJMSIR)
IJMSIR : A Medical Publication Hub
Available Online at: www.ijmsir.com
Volume – 10, Issue – 6, December – 2025, Page No. : 23 – 27

Diclofenac Induced Stevens Johnson Syndrome – Toxic Epidermal Necrolysis Overlap In A HIV-AIDS Patient: A Case Report

[1]Dr. Gayathri S, Junior Resident, Department of Pharmacology, B.J. Government Medical College, Pune.

[2]Dr. Rohini Jagtap, Professor, Department of Pharmacology, B.J. Government Medical College, Pune.

[3]Dr. Shekhar Pradhan, Professor, Department of Dermatology, B.J. Government Medical College, Pune.

Corresponding Author: Dr. Gayathri S, Junior Resident, Department of Pharmacology, B.J. Government Medical College, Pune.

Citation this Article: Dr. Gayathri S, Dr. Rohini Jagtap, Dr. Shekhar Pradhan, "Diclofenac Induced Stevens Johnson Syndrome – Toxic Epidermal Necrolysis Overlap In A HIV-AIDS Patient: A Case Report", IJMSIR - December – 2025, Vol – 10, Issue - 6, P. No. 38 – 46.

Type of Publication: Case Report

Conflicts of Interest: Nil

Abstract

Stevens–Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are rare diseases that are characterized by widespread epidermal necrosis and sloughing of skin. They are associated with significant morbidity and mortality. Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are characterised by serious reaction of the skin and mucous membranes that can be caused by certain medications, infections or autoimmune diseases. Pathophysiology of these conditions has not been fully understood. We report the case of a 45-year-old female who experienced an adverse drug reaction subsequent to the administration of diclofenac 50 mg orally, which had been prescribed for genital pain. Within 24 hours, she developed fever, followed by reddish lesions and painful erosions involving most of her body. Diclofenac was discontinued and systemic steroids were initiated. Close monitoring and management of the symptoms was carried out and the patient recovered completely after an in-patient treatment of 9 days.

Keywords: SJS-TEN, Adverse drug reaction, Pharmacovigilance, HIV infection

Introduction

Stevens Johnson syndrome (SJS) and Toxic epidermal necrolysis (TEN) are acute life-threatening mucocutaneous diseases characterized by extensive necrosis, mucous membrane involvement and detachment of epidermis, differing only in their degree of severity.[4] The incidence of SJS has been estimated to be around 1–6/1,000,000 persons per year with a mortality rate of 1%–5%, which rises to 30% in TEN.[3] The classification of Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) is based on the extent of epidermal detachment: involvement of less than 10% of the body surface area defines SJS, more than 30% defines TEN, and 10–30% is categorized as SJS/TEN overlap. Although SJS can occur across all age groups, its incidence increases after the fourth decade of life, with a higher predilection among elderly women. HIV-AIDS patients, immunodeficiency patients, family history of the condition and variation of gene called

HLA-B are at higher risk of developing SJS[4]. The drugs most frequently implicated in the development of Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) include antibacterials such as sulphonamides, anticonvulsants like phenytoin, phenobarbital, and carbamazepine, nonsteroidal anti-inflammatory drugs (particularly oxicam derivatives), and xanthine oxidase inhibitors such as allopurinol. Several nonsteroidal anti-inflammatory agents have also been reported to be associated with SJS and TEN.[3] Here, we report a case of SJS/TEN overlap syndrome due to non-steroidal anti-inflammatory drug Diclofenac.

Case Report

A 45-year-old female patient, a known case of HIV since the last 2 months, reported to the outpatient department of Dermatology department of a tertiary care hospital with multiple reddish maculopapular eruptions over face, abdomen, both limbs, and oral cavity, fluid filled lesion over right hand and oral mucosal erosions. It was associated with itching and pain all over the body. Additionally, she complained of fever and oral intolerance to spicy food.

She had a similar history around 20 days back on taking an over-the-counter drug for genital pain, which was managed on an OPD basis effectively and oral Diclofenac 50 mg was prescribed for the same. She developed fever within 24 hours of drug intake followed by cutaneous lesions over upper limbs which was sudden in onset, gradually progressed to involve the whole body. Patient was poorly built and well oriented. On examination, she was afebrile with BP: 128/88 mm Hg, Heart rate: 88 bpm, SpO2: 98% on room air. Dermatological examination showed multiple erythematous papules coalescing to form a plaque, multiple vesicles over right upper limb, multiple pustules over right thigh fold and single pustule over labia minora. There were multiple hyperpigmented plaques over oral mucosa. There were no ocular findings.

Laboratory examination showed haemoglobin: 10g/dL, total leucocyte count to be 7000 cells/mm^3 with mild eosinophilia (1380 cells), liver function test and renal function tests were within normal limits but HIV test was reactive with CD4 count of 316 cells. Mantoux tests were sent which were negative. Other serological tests for Hepatitis B and hepatitis C were negative. SCORTEN-Severity of illness gave a score of 2, which is suggestive of 12.1% mortality.

SCORTEN-Severity of illness score

Prognostic Factors	Individual Score	Scorten (sum of individual scores)	Predicted Mortality (%)	Score
Age (>40 years)	Yes=1, No=0	0-1	3.2	1
Malignancy	Yes=1, No=0	2	12.1	0
Tachycardia (>120 bpm)	Yes=1, No=0	3	35.8	0
Initial BSA* detachment (>10% involvement)	Yes=1, No=0	4	58.3	1
Serum urea >28 mg/dL	Yes=1, No=0	>5	>90	0
S. glucose >252 mg/L	Yes=1, No=0			0
S. bicarbonate <20 mmol/L	Yes=1, No=0			0

Upon the diagnosis of SJS-TEN overlap, Diclofenac was immediately discontinued. Supportive measures were initiated including strict infection control, wound care and pain management. The patient was treated with prophylactic antibiotics (Ceftriaxone 1 g IV 12 hourly) to prevent the risk of secondary infection. Intravenous Dexamethasone (4-0-2 mg 12 hourly) was given for 4 days followed by oral Prednisolone 10 mg BD for next 3 days. Oral antihistamines, topical steroids and liquid paraffin were added for symptomatic management. After 9 days of treatment and monitoring, there was improvement in patient's condition, hence was discharged.

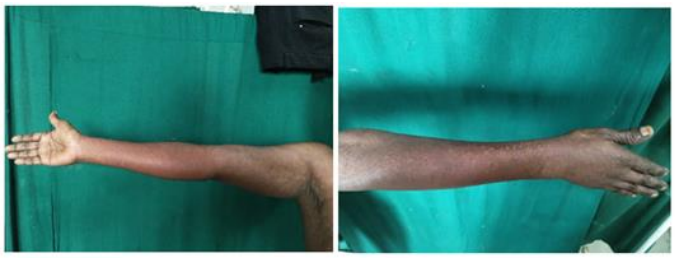

Figure 1: Multiple lesions on the upper limb

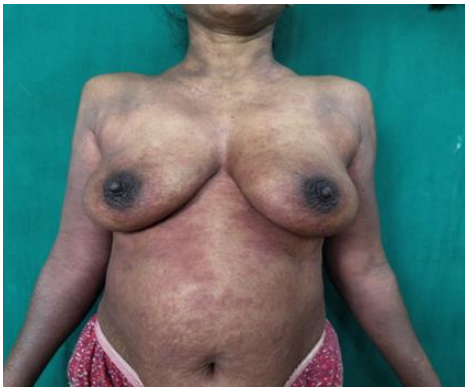

Figure 2: Multiple erythematous lesions on the trunk

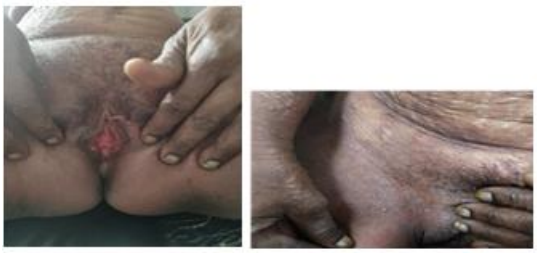

Figure 3: Pustular lesions in the genital region

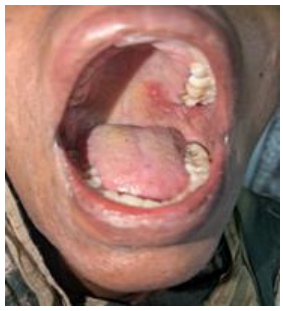

Figure 4: Oral lesions on the hard palate

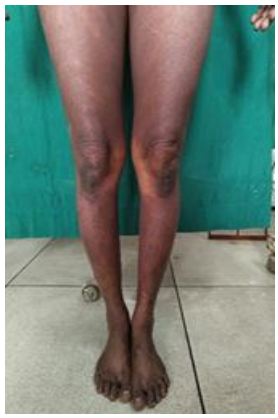

Figure 5: Lesions on the lower limb

Discussion

Stevens–Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are dermatologic emergencies characterized by widespread epidermal necrolysis and sloughing. In the majority of cases, the onset of cutaneous manifestations is preceded by a prodromal phase characterized by non-specific systemic symptoms such as fever, malaise, sore throat, and cough. This is invariably followed by cutaneous and mucosal involvement, initially presented as erythematous macules or atypical target lesions predominantly distributed over the trunk. These lesions subsequently coalesce into confluent erythematous areas with dusky centers and evolve into flaccid blisters demonstrating a positive Nikolsky sign.[1] When gentle lateral pressure is applied to the edge of an intact blister, the surrounding epidermis shears away, causing the blister to extend. This clinical finding is characteristic of pemphigus, toxic epidermal

necrolysis, and staphylococcal scalded skin syndrome (SSSS).[9]

The vast majority of patients with SJS-TEN overlap syndrome have mucosal involvement, with two or more mucosal surfaces being involved in up to 80% of cases. Oral involvement is most common, with mucositis and ulceration occurring in up to 100% of cases. Ocular involvement also occurs frequently, with severity ranging from conjunctival hyperaemia to complete epidermal sloughing of the ocular surface[1].

The pathogenesis of Stevens–Johnson syndrome (SJS) involves a complex interplay between the immune system and the drug or its metabolites. It is hypothesized that certain drugs, including Diclofenac, bind to endogenous proteins and trigger an immune response. This immune activation leads to the destruction of epidermal cells, resulting in the characteristic skin manifestations observed in SJS. The severity of this reaction can vary, with SJS representing the milder form and toxic epidermal necrolysis (TEN) denoting the more severe end of the spectrum.

Dysfunction or depletion of regulatory T cells has been implicated in the pathogenesis of adverse cutaneous drug reactions, particularly in immunosuppressed states such as HIV. In individuals living with HIV, the increased susceptibility to severe drug hypersensitivity reactions is believed to result from a complex interplay of immunologic, metabolic, viral, and host factors. Research has shown that HIV-positive patients with TEN exhibit lower numbers of skin-homing CD4+ T cells, a higher CD8+/CD4+ ratio, and a reduction in CD4+CD25+ Tregs. This immunologic imbalance is thought to impair the regulation of cytotoxic responses, thereby facilitating the development of severe drug-induced cutaneous reactions.

A retrospective study conducted by Mittmann et al. within an HIV-positive population demonstrated the SJS/TEN incidence rate of approximately 1-2 cases per 1,000 individuals, which was over 100 times higher than the incidence rate in the general population. This emphasizes the strong association between HIV infection and the heightened risk of these severe reactions[7].

Prompt recognition and timely management of Stevens–Johnson syndrome (SJS) are essential to prevent complications and improve clinical outcomes. In the present case, the patient presented with fever, mucosal involvement, and characteristic cutaneous lesions consistent with an SJS–TEN overlap. Immediate withdrawal of Diclofenac, along with intensive supportive care and symptomatic management, played a pivotal role in the patient's recovery. Early identification of the condition and institution of appropriate therapeutic measures remain critical in minimizing the morbidity and mortality associated with SJS[2].

The severity-of-illness score for TEN (SCORTEN) is a mortality prognostication tool for epidermal necrolysis.[8] In our case, SCORTEN score is 2 which is suggestive of 12.1% mortality. According to the WHO-UMC causality assessment criteria, the ADR was categorized as 'probable' with diclofenac, as the reaction occurred after drug intake, improvement was observed on dechallenge, and rechallenge was not performed. Hence, the SJS/TEN overlap is considered 'probable' with the suspected drug.

Conclusion

This case highlights the critical need for healthcare professionals to remain vigilant regarding the potential risk of Stevens–Johnson Syndrome associated with diclofenac use. Patient education on recognizing early signs and symptoms, along with the importance of seeking immediate medical attention upon onset of adverse reactions, is essential for timely intervention.

Clinicians should exercise caution or consider alternative therapeutic options in patients with a history of hypersensitivity. Furthermore, strengthening pharmacovigilance systems through proactive reporting and monitoring of adverse drug reactions is vital to improving drug safety and preventing similar events in the future.

References

1. Frantz, R.; Huang, S.; Are, A.; Motaparthi, K. Stevens–Johnson Syndrome and Toxic Epidermal Necrolysis: A Review of Diagnosis and Management. Medicina 2021, 57, 895.
2. Navid Faraji, Rasoul Goli Naser Parizad, Pariya Mohsennezhad, Elaheh Salamat et.al 'NSAID (nonsteroidal anti-inflammatory drugs) Induced Stevens Johnson Syndrome in a 50-year-old woman: A case study' Toxicology Reports-Elsevier 2024
3. Chindhalore C, Dakhale GN, Emperumal TS, Dhal S. Stevens–Johnson syndrome/ toxic epidermal necrolysis overlap syndrome due to a fixed-dose combination of aceclofenac, serratiopeptidase, and paracetamol. Natl J Pharmacol Ther 2025; 3:69-71.
4. Juchitra Deuri, Sahid Aziz, Dipjyoti Deka A rare case report of diclofenac induced Steven Johnson syndrome Int J Basic Clin Pharmacol. 2024 Nov;13(6):910-914
5. Sateesh Kumar Reddy K, Shanmuga Kumar SD, Vijay Raghavendra NC, Sudheer Kumar K. A Case Report on Diclofenac Induced Stevens Johnson Syndrome. J Basic Clin Pharma 2018; 9:109 110.
6. Moitra S, Sen S, Banerjee I, Das P, Tripathi SK. Diclofenac-serratiopeptidase combination induced Stevens – Johnson syndrome - a rare case report with review of literature. J Clin Diagn Res. 2014 Jul;8(7): YD08-11. doi: 10.7860/JCDR/2014/ 9509. 4610. Epub 2014 Jul 20. PMID: 25177625; PMCID: PMC4149131.
7. Martínez Jiménez H M (July 15, 2025) Toxic Epidermal Necrolysis in an HIV-Infected Patient. Cureus 17(7)
8. Dobry AS, Himed S, Waters M and Kaffenberger BH (2022) Scoring Assessments in Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis
9. Maity S, Banerjee I, Sinha R, Jha H, Ghosh P, Mustafi S. Nikolsky's sign: A pathognomic boon. J Family Med Prim Care 2020; 9:526-30.
10. Rajput R, Sagari S, Durgavanshi A, Kanwar A. Paracetamol induced Steven-Johnson syndrome: A rare case report. Contemp Clin Dent. 2015 Sep;6 (Suppl 1): S278-81. doi: 10.4103/0976-237X.166838. PMID: 26604588; PMCID: PMC4632237.