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CASE REPORT: STEVENS-JOHNSON SYNDROME INDUCED BY OFLOXACIN, KETOROLAC, AND ACECLOFENAC IN A PLHIV PATIENT WITH HYPOTHYROIDISM

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ABSTRACT

Stevens-Johnson Syndrome (SJS) is a rare, life-threatening mucocutaneous reaction characterized by extensive necrosis and detachment of the epidermis. It is most commonly triggered by adverse drug reactions, particularly in immunocompromised patients. This report details the case of a 43-year-old male with newly diagnosed HIV and hypothyroidism who developed SJS following the administration of ofloxacin, ketorolac, and aceclofenac. The patient presented with persistent oral ulcers and widespread fluid-filled lesions. Prompt recognition, systemic corticosteroid therapy, supportive care, and multidisciplinary management resulted in complete clinical resolution. This case emphasizes the importance of pharmacovigilance and proactive management strategies, especially in immunocompromised individuals.

KEYWORDS: Stevens-Johnson Syndrome, HIV, drug-induced, ofloxacin, ketorolac, aceclofenac, hypersensitivity, adverse drug reaction.

INTRODUCTION

Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN)[1] represent a spectrum of severe mucocutaneous reactions primarily triggered by drugs. These reactions are characterized by epidermal necrosis and detachment, commonly affecting the skin and mucous membranes. Drugs like sulfonamides, NSAIDs, antiepileptics, and antibiotics[2] are frequent offenders. Immunocompromised individuals, including those living with HIV, are at increased risk of developing SJS. The management of SJS is complex and requires early diagnosis, immediate cessation of the offending agents, and initiation of supportive care. Herein, we present a case of SJS in a PLHIV patient, induced by a combination of ofloxacin, ketorolac, and aceclofenac, with a positive clinical outcome after aggressive multidisciplinary intervention.

CASE PRESENTATION

A 43-year-old male presented with painful oral ulcers for two months and multiple fluid-filled vesicles all over the body for three days. He was previously healthy until he was prescribed systemic antibiotics and NSAIDs (ofloxacin, ketorolac, and aceclofenac) at a local clinic for acute febrile illness. Subsequently, he developed widespread erythematous lesions and bullae, especially over the neck, chest, and thighs, with associated lip swelling and mucosal involvement. History of burning sensation, pain over the lesion. No history of respiratory symptoms or systemic complaints were reported. Medical History: The patient had recently been diagnosed with hypothyroidism and was on thyroxine 75 mcg OD. HIV serology turned out to be reactive in a routine blood checkup during admission. Physical Examination: Vital Signs: Blood pressure: 120/80 mmHg; Pulse rate: 95/min; Respiratory rate: 18/min; SpO_2: 98%; Temperature: 97°F. Skin Examination: Multiple well-defined bullae of varying sizes over the neck, chest, right thumb, and inner aspect of the right thigh, the largest measuring 15 x 7 cm. Surrounding erythema was noted, but the Nikolsky sign was negative. Mucosal Examination: Hyperpigmented lips; white patches on the tongue; phimosis with glans inflammation; hyperpigmented patch on the penile shaft; scrotal erythema. Other Systems: Cardiovascular, respiratory, abdominal, and neurological examinations were unremarkable. Investigations: Laboratory Tests: Complete blood count, liver and renal function tests were within normal limits. Serology: HIV-positive. Skin Biopsy: Confirmed diagnosis of SJS with (SCORTEN) score of 2. Hospital Course: The patient was admitted and managed with: **Medications:** Intravenous DEXAMETHASONE 2 cc twice daily for two days, then tapered. Oral PREDNISOLONE (Omnacortil) 20 mg daily, tapered over a week.

ORAL FLUCONAZOLE 150 mg daily. CETIRIZINE 10 mg twice daily. THYROXINE 75 mcg daily. Supportive care with PARACETAMOL, ANTIEMETICS, PROTON PUMP INHIBITORS, MUCAINE GEL, and topical applications. Complications: Developed pain and tenderness over the right gluteal region on the third day of admission. Ultrasound ruled out abscess; cellulitis was diagnosed. Initiated on systemic antibiotics and analgesics (ULTRACET and CHYMORAL FORTE), which were discontinued due to the development of wheals. Switched to Tablet CEFUROXIME 500 mg twice daily with close monitoring. **Outcome:** The patient's condition improved with the above management. Skin lesions began to resolve, and no new lesions appeared. Discharged with instructions for follow-up and continuation of medications as per the tapering schedule.

DISCUSSION

This case underscores the significance of early identification and management of drug-induced SJS, particularly in immunocompromised patients. OFLOXACIN (a FLUOROQUINOLONE), KETOROLAC (an NSAID), and ACECLOFENAC (another NSAID) have each been implicated in rare cases of SJS, but their combined administration in a PLHIV patient with hypothyroidism likely potentiated the reaction.

Patients with HIV are known to have a 100- to 1000-fold higher risk[3] of developing SJS. The proposed mechanisms include altered drug metabolism, impaired detoxification, and immune dysregulation.[4] Hypothyroidism may further compound drug metabolism pathways.

Prompt drug withdrawal and steroid initiation are the cornerstones[5] of therapy. Antiseptic and antifungal therapies help prevent secondary infections. Close monitoring and a multidisciplinary approach—dermatology, infectious disease, and surgery—are vital. This patient’s favorable prognosis highlights the importance of early intervention.

The SCORTEN score is widely used to predict **mortality-12%**[6] in SJS/TEN. With a score of 2, this patient had an intermediate risk but benefited from rapid initiation of therapy and careful monitoring.

PROACTIVE MANAGEMENT

A. Drug Allergy Documentation and Reporting

The patient was advised to carry a documented list of allergies, including OFLOXACIN, KETOROLAC, ACECLOFENAC, ULTRACET, and CHYMORAL FORTE. This case was

reported to the pharmacovigilance center to contribute to the adverse drug reaction (ADR) database[7] and given a printed drug allergy card.

B. Patient Education

Counseling on recognizing early symptoms of hypersensitivity. Advised to seek immediate medical care if symptoms reappear.

C. Regular Follow-Up

Dermatological and infectious disease follow-up for HIV management. Thyroid function tests for ongoing hypothyroidism treatment monitoring. Psychological counseling, for SJS patients often experience trauma related to their condition and hospitalization.

D. ART Initiation

Since the HIV diagnosis was made during admission, coordination with an ART center was done for the timely initiation of antiretroviral therapy.

FUTURE RECOMMENDATIONS

Vigilant Use of NSAIDs and Antibiotics in High-Risk Populations

Patients with HIV or other immunocompromised states should be prescribed medications with lower hypersensitivity risks, and only when clinically indicated.

Development of Clinical Algorithms

Hospital settings should adopt standardized protocols to promptly identify and manage suspected SJS cases, especially in patients with comorbidities.

Genetic Screening

Screening for HLA-B alleles (e.g., HLA-B15:02, HLA-B58:01) known to predispose to SJS with certain drugs might be beneficial in populations with a high incidence.[8]

Integrate ADR Awareness in Clinical Training

Include real-world case discussions on ADRs and SJS in undergraduate and postgraduate medical and pharmacy education.

CONCLUSION

This case emphasizes the significance of drug vigilance in immunocompromised individuals.[9] SJS induced by ofloxacin, ketorolac, and aceclofenac in a PLHIV patient with

hypothyroidism was successfully managed with early corticosteroids, supportive care, and drug discontinuation. Timely diagnosis, multidisciplinary collaboration[10], and patient education are crucial in preventing morbidity and mortality associated with SJS. Early recognition of mucocutaneous symptoms and timely withdrawal of the offending agents played a crucial role in preventing further disease progression. Systemic corticosteroids, careful monitoring for secondary infections, and supportive care formed the foundation of effective management. The patient's recovery was further supported by close follow-up, appropriate antifungal therapy, and coordinated efforts from dermatology, surgery, and internal medicine teams. This case underscores the importance of Individualized risk-benefit evaluation before prescribing drugs known to have the potential for causing SJS, especially in high-risk populations such as PLHIV. Proactive pharmacovigilance, documentation of drug allergies, and effective patient education. Routine use of scoring systems like SCORTEN for risk stratification and management planning. The need for stronger preventive measures, such as pharmacogenetic screening and electronic alert systems, becomes evident in such scenarios. Ultimately, the case reiterates that SJS, though rare, should always be a differential when evaluating acute-onset skin and mucosal lesions post-drug exposure, especially in vulnerable patient populations.

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