CASE REPORT

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Unusual Presentation of Stevens-Johnson Syndrome Induced by Trimethoprim-Sulfamethoxazole: A Case Report

Mohammad Ali[1], Urbah Viqar[2]

1 *House Officer, Akbar Niazi Teaching Hospital, Islamabad Pakistan*
Manuscript writing, Data collection, Discussion writing

2 *Clinical Fellow Psychiatry, ARCH, Hillington, London United Kingdome*
Contribution in the study

CORRESPONDING AUTHOR
Dr. Mohammad Ali
House Officer, Akbar Niazi Teaching Hospital, Islamabad Pakistan
Email: m.ali797@yahoo.com

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ABSTRACT

Stevens-Johnson syndrome (SJS) is a severe and potentially life-threatening adverse drug reaction characterized by mucocutaneous lesions. Here, I present a unique case of SJS induced by trimethoprim-sulfamethoxazole (TMP-SMX) with an unusual clinical presentation. A 45-year-old female patient with a history of urinary tract infection presented with the development of widespread erythematous macules and papules, progressing to blisters and erosions involving the oral mucosa. The patient's condition rapidly deteriorated, necessitating intensive care management. The temporal association between TMP-SMX initiation and the onset of symptoms, the characteristic clinical and histopathological findings supported the diagnosis of SJS. Prompt withdrawal of the offending drug, supportive care, and multidisciplinary management resulted in a favorable outcome for the patient.

Keywords: *Steven-Johnson syndrome, Trimethoprim-sulfamethoxazole, Urinary tract infection, Mucocutaneous lesions.*

INTRODUCTION

Stevens-Johnson syndrome (SJS) is a rare but severe cutaneous adverse drug reaction that various medications can induce. Widespread mucocutaneous lesions, including erythematous macules, blisters, and erosions, characterize it. The most common etiological agents associated with SJS are antimicrobial drugs, particularly sulfonamides and anticonvulsants. However, SJS induced by trimethoprim-sulfamethoxazole (TMP-SMX) is relatively uncommon.[1,2]

CASE PRESENTATION

A 45-year-old female patient with a history of recurrent urinary tract infections was prescribed TMP-SMX as empirical treatment. Two days after initiation of TMP-SMX, the patient developed a sudden onset of fever, malaise, and the appearance of multiple erythematous macules on the face, trunk, and extremities. Over the next 24 hours, the macules progressed to form blisters and erosions involving the oral mucosa. The patient also experienced pain in swallowing, dysuria, and photophobia. She was admitted to the hospital and managed in the intensive care unit due to the severity of her condition. Clinical and laboratory investigations revealed leukocytosis, elevated liver enzymes, and the presence of atypical lymphocytes on peripheral blood smear. A skin biopsy demonstrated epidermal necrosis, detachment, and a sparse inflammatory infiltrate, consistent with a diagnosis of SJS. The Naranjo probability scale indicated a probable relationship between TMP-SMX and the development of SJS. The offending drug, TMP-SMX, was immediately discontinued, and the patient received supportive care, including wound care, fluid resuscitation, and pain management. The patient's condition gradually improved in collaboration with a multidisciplinary team comprising dermatologists, intensivists, ophthalmologists, and oral medicine specialists. She showed re-epithelialization of the mucosal lesions and resolution of the cutaneous. Lesions over several weeks. Follow-up examinations demonstrated no evidence of long-term complications.

DISCUSSION

Stevens-Johnson Syndrome (SJS) is a severe and potentially life-threatening mucocutaneous reaction often triggered by medications. Trimethoprim-sulfamethoxazole (TMP-SMX), a commonly prescribed antibiotic, is a medication known to be associated with SJS. While the typical presentation of SJS includes fever, mucosal involvement, and a characteristic rash, this case report highlights an unusual presentation of TMP-SMX-induced SJS.[1] Despite its rarity, SJS poses significant

clinical challenges due to its potential for morbidity and mortality. This review aims to elucidate SJS's pathogenesis, clinical presentation, diagnosis, and management, drawing upon recent literature and clinical guidelines.[2]

The pathogenesis of SJS involves a complex interplay of genetic predisposition, immune dysregulation, and environmental factors. Genetic polymorphisms, particularly in genes encoding human leukocyte antigens (HLA), have been implicated in predisposing individuals to SJS. Drug-induced SJS is thought to result from a hypersensitivity reaction mediated by T lymphocytes, leading to widespread apoptosis of keratinocytes and subsequent mucocutaneous detachment.[3] Genetic predisposition plays a significant role in the pathogenesis of SJS, with specific human leukocyte antigen (HLA) alleles being strongly associated with increased susceptibility to the condition.[4] HLA-B 15:02, for example, has been implicated in the development of SJS in individuals of Asian descent treated with carbamazepine, necessitating pharmacogenetic screening to reduce the risk of adverse drug reactions

The clinical manifestations of SJS typically begin with prodromal symptoms such as fever, malaise, and upper respiratory tract symptoms. This is followed by the abrupt onset of mucocutaneous lesions, including erythematous macules, blistering, and erosions involving the skin, mucous membranes, and conjunctiva. Nikolsky's sign, the detachment of the epidermis with lateral pressure, is a characteristic finding. Severe cases may progress to involve multiple organ systems, leading to significant morbidity and mortality.[5]

The diagnosis of SJS relies on a combination of clinical features, including the characteristic mucocutaneous eruptions and systemic symptoms. Histopathological examination of skin biopsies may reveal features consistent with apoptotic keratinocytes and interface dermatitis. Laboratory investigations such as complete blood count, liver function tests, and assessment of renal function may aid in assessing the severity and extent of organ involvement.[6]

Management of SJS involves prompt recognition, withdrawal of the offending agent, and supportive care. Patients with suspected SJS should be immediately hospitalized in a specialized burn unit or intensive care setting for close monitoring and multidisciplinary management. Supportive measures include fluid resuscitation, pain management, wound care, and prevention of complications such as sepsis and ocular involvement. Immunomodulatory therapies such as corticosteroids and intravenous immunoglobulin (IVIG) may be considered in severe cases, although their efficacy remains controversial.[7] Pharmacovigilance is critical in identifying and preventing medication-related adverse reactions, including Stevens-Johnson Syndrome.[8] Healthcare professionals should be vigilant for early signs of SJS in patients receiving potentially culprit medications, particularly those with known associations. Patient education regarding recognizing prodromal symptoms and the importance of seeking prompt medical attention can also aid in early intervention and preventing severe outcomes.

Stevens-Johnson Syndrome is a rare but potentially life-threatening dermatological emergency characterized by severe mucocutaneous reactions. Despite advances in understanding its pathogenesis and management, SJS remains a clinical challenge with significant morbidity and mortality. Early recognition, prompt withdrawal of offending agents, and aggressive supportive care are essential for optimizing outcomes in patients with SJS.[9,10]

This case underscores the importance of recognizing uncommon presentations of SJS, particularly in patients with recent medication exposure. Healthcare providers should remain vigilant for adverse drug reactions, promptly discontinue suspected medications, and initiate appropriate management to optimize patient outcomes. Additionally, this case emphasizes the need for enhanced awareness among clinicians regarding the potential for TMP-SMX to induce SJS, especially in patients with a history of adverse reactions to sulfonamide antibiotics.

CONCLUSION

This case highlights an unusual presentation of SJS induced by TMP-SMX. It emphasizes the importance of early recognition, prompt withdrawal of the offending drug, and multidisciplinary management in improving patient outcomes. When providing TMP-SMX or other sulfonamide-containing drugs, healthcare practitioners should be alert to the possibility of severe cutaneous adverse drug reactions, such as SJS.

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