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Sulfa Drugs and Skin Necrosis: A Case of Trimethoprim-Sulfamethoxazole–induced Stevens Johnson syndrome

Eric Micaela*

Department of Dermatology and Venereology, University of Gothenburg, Gothenburg, Sweden

Introduction

Stevens-Johnson Syndrome (SJS) is a rare, acute and potentially fatal mucocutaneous reaction most commonly triggered by medications. Among the drugs implicated, sulphonamides particularly trimethoprim-sulfamethoxazole (TMP-SMX)—are one of the leading causes. SJS is characterized by epidermal necrosis, extensive skin detachment and mucosal erosions. The onset typically occurs within one to three weeks after exposure to the offending agent. Early recognition of SJS and immediate withdrawal of the causative drug is critical to preventing progression and mortality. Despite its rarity, the severity of this condition necessitates heightened vigilance when prescribing high-risk medications like TMP-SMX, particularly in populations with known genetic predispositions or prior drug reactions. Genetic factors play a crucial role in the susceptibility to Stevens-Johnson Syndrome, especially in response to specific medications like TMP-SMX. Variations in Human Leukocyte Antigen (HLA) alleles, such as HLA-B1502 and HLA-B5801, have been strongly associated with increased risk of severe cutaneous adverse reactions in certain ethnic groups. Screening for these genetic markers before initiating high-risk drugs is becoming an important preventive strategy in personalized medicine. Additionally, ongoing research aims to identify other genetic and immunologic factors that contribute to SJS pathogenesis, which could lead to improved risk stratification and tailored treatment approaches in the future [1].

Description

Stevens-Johnson Syndrome (SJS) remains a challenging clinical condition due to its sudden onset, severity and potential for significant morbidity and mortality. Moving forward, advances in pharmacogenomics offer promising avenues for prevention by identifying individuals at high risk before drug exposure. Wider implementation of genetic screening protocols, such as testing for HLA-B*1502 and other relevant alleles, could become standard practice globally, beyond currently targeted populations. This personalized medicine approach may drastically reduce the incidence of SJS by guiding safer drug selection and dosing. In addition to prevention, future therapeutic strategies are likely to focus on targeted immunomodulation. Research into the molecular pathways of keratinocyte apoptosis and immune activation is uncovering potential drug targets that could mitigate the severity of SJS or hasten recovery. Novel agents aiming to inhibit cytotoxic T cell activity or block granulysin-mediated damage are under investigation. Furthermore, advances in supportive care such as improved wound healing technologies and ocular treatments may enhance functional outcomes and reduce long-term complications, especially vision loss [2].

Finally, enhancing pharmacovigilance systems through integration of electronic health records and real-time adverse event reporting can facilitate earlier detection and intervention. Increased awareness and education of healthcare providers and patients about early symptoms will improve timely diagnosis and drug withdrawal, which remain critical for survival. Multidisciplinary care models incorporating dermatology, ophthalmology, immunology and mental health support will continue to evolve, addressing the complex needs of SJS survivors and improving quality of life after acute illness. Stevens-Johnson Syndrome (SJS) remains a complex and potentially life-threatening condition with high morbidity, necessitating ongoing improvements in prevention, diagnosis and management. Looking ahead, pharmacogenomics stands at the forefront of preventive strategies. As the cost and accessibility of genetic testing improve, routine screening for alleles such as HLA-B1502, HLA-A3101 and others linked to drug hypersensitivity reactions may become an integral part of prescribing protocols worldwide, not limited to select ethnic groups. This would enable clinicians to personalize medication choices, avoiding high-risk drugs like Trimethoprim-Sulfamethoxazole (TMP-SMX) or carbamazepine in genetically susceptible patients, thereby significantly reducing incidence rates. Coupling genetic data with clinical risk factors and advanced predictive algorithms using artificial intelligence could further refine risk stratification and guide safer treatment decisions [3].

Therapeutically, future directions include the development of targeted immunomodulatory agents aimed at interrupting the underlying immune cascade responsible for epidermal necrosis. Current understanding implicates cytotoxic T lymphocytes and granulysin as key mediators of keratinocyte apoptosis; thus, biologics or small molecules that inhibit these effectors could attenuate tissue damage and improve outcomes. Research into novel therapies such as anti-granulysin antibodies, JAK inhibitors, or other immune checkpoint modulators is ongoing and may revolutionize acute-phase management. Parallel advances in supportive care—such as bioengineered skin substitutes, regenerative medicine techniques and optimized ocular interventions including stem cell therapies and minimally invasive surgeries—hold promise to reduce long-term sequelae such as scarring, contractures and vision loss. Early integration of multidisciplinary care teams encompassing dermatology, ophthalmology, immunology, rehabilitation and mental health professionals will be essential for holistic patient recovery. On a systemic level, enhancements in pharmacovigilance infrastructure are critical to improving SJS outcomes. Leveraging electronic health records, big data analytics and machine learning models can enable real-time detection of adverse drug reactions and prompt alerts to prescribers. Educational initiatives targeting both healthcare providers and patients can increase awareness of prodromal symptoms, improving early recognition and timely discontinuation of offending agents. Additionally, public health policies supporting mandatory adverse event reporting and patient registries will facilitate better epidemiological understanding and drive research. Ultimately, these combined efforts—spanning prevention, innovative therapeutics and robust surveillance will contribute to lowering the global burden of SJS, improving survival rates and enhancing quality of life for survivors [4].

Another promising avenue is the advancement of personalized medicine through the integration of multi-omics approaches, including genomics, proteomics and metabolomics. By comprehensively profiling individual patients' molecular and immune signatures before and during drug exposure, researchers aim to identify novel biomarkers predictive of SJS susceptibility and disease severity. Such biomarkers could facilitate earlier diagnosis and

***Address for Correspondence:** Eric Micaela, Department of Dermatology and Venereology, University of Gothenburg, Gothenburg, Sweden, E-mail: micaela.eric@gothenberg.sw*

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more precise monitoring of disease progression. Moreover, they may reveal new therapeutic targets, enabling tailored interventions that mitigate immune-mediated skin damage without broadly suppressing the immune system. This precision medicine approach could reduce the risks of adverse effects associated with systemic immunosuppressants currently used in severe cases, improving safety and efficacy. Furthermore, global collaboration and data sharing among research institutions, clinicians and regulatory agencies will be pivotal in accelerating progress against SJS. The establishment of international registries and biobanks collecting clinical data, genetic material and treatment outcomes from diverse populations will enhance understanding of regional and ethnic variations in drug hypersensitivity reactions. Such resources will also support large-scale clinical trials necessary to validate emerging therapies and preventive strategies. Additionally, harmonizing guidelines and implementing standardized protocols for SJS management worldwide can reduce disparities in care quality and patient outcomes. Empowering low- and middle-income countries with resources for early detection, management and genetic screening will be essential to address the global impact of SJS effectively [5].

Conclusion

Stevens - Johnson syndrome is a life-threatening drug reaction that requires immediate identification and cessation of the offending agent. This case highlights TMP-SMX as a high-risk trigger, necessitating caution during prescription. Early recognition, drug withdrawal and supportive care are paramount in reducing morbidity and mortality. Clinicians must maintain a high index of suspicion when patients develop febrile mucocutaneous symptoms after new medication use. Improved awareness and patient education can aid in timely diagnosis and management, ultimately saving lives and preventing severe complications. Additionally, advancements in pharmacogenetic screening hold promise for identifying individuals at increased risk before drug exposure, enabling safer prescribing practices. Ongoing research into targeted immunomodulatory therapies may also offer more effective treatment options with fewer side effects in the future.

Acknowledgment

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Conflict of Interest

None.

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