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Carbamazepine Induced Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis: A Case Report

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ABSTRACT

Stevens-Johnson syndrome and toxic epidermal necrolysis are acute life-threatening mucocutaneous drug reactions. Early symptoms of this syndrome include fever and blisters that peel and leave painful raw areas. As the infection progresses, consequences like dehydration, sepsis, pneumonia, and multiple organ failure are common. In this report the authors present case of a 28-years-old male patient referred to our hospital after taking carbamazepine for seizures and developing high-grade fever and skin lesion initially on upper trunk maculopapular followed by all over body including eyes and oral cavity. The use of IVIG and plasmapheresis was a good management for our case, helping in our patient's well-being and recovery.

Keywords: Stevens Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), mucocutaneous, plasmapheresis, carbamazepine

INTRODUCTION

Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are acute life-threatening mucocutaneous drug reactions. It has been demonstrated that some antibiotics, nonsteroidal anti-inflammatory drugs (NSAIDs), and antiepileptic medications (AEDs) might cause these effects [1].

SJS and TEN were once treated as distinct illnesses, but they are now recognized as two variations of the same skin ailment. Less than 10% of the entire body surface area (BSA) can be detached as SJS, while more than 30% of the total BSA can be detached as TEN [2].

It affects people of all ages, but it particularly affects those with HIV, autoimmune illnesses, immunocompromised patients, and underlying malignancy. Additionally, it is believed to be an immune-mediated pharmacological reaction that occurs more frequently in some populations (for instance, HLA-B∗1502 has been linked to carbamazepine-associated SJS/TEN in Han Chinese and other Asian groups) [3].

According to reports, patients with SJS/TEN who survive are very susceptible to long-term problems affecting the mucosa, skin, eyes, respiratory, renal, and/or hepatic systems [1].

CASE REPORT

We report a case of 28-years-old male patient brought to hospital with chief complaints of

skin lesion initially on upper trunk maculopapular followed by all over body including eyes and oral cavity, itching and burning sensation all over the body. Difficulty in swallowing, pain in both eyes, burning sensation and ulcers in mouth. These symptoms began after using carbamazepine. The patient had been taking carbamazepine for seizure, he is having no other complications. Additionally, he did not have any drug allergies.

The initial examination of the patient was conscious\coherent and was observed to be febrile. The pulse rate was measured as 70 beats per minute, blood pressure was observed as120/80 mmHg, respiratory rate was 18 beats per minute, P/A is soft, and CNS was NFND. His skin examination revealed multiple maculopapular rashes covering over body surface area. The blistered area was positive for Nikolai's sign.

Initial laboratory results elevated blood urea (61 mg/dl), elevated platelet count (7.41 lakhs), a normal white blood cell count (9000 cells in microliter), RBC (5.1 million), hemoglobin (14.2 g/dl), normal blood sugar levels and normal renal (including electrolytes, creatinine) and liver function tests.

Considering the clinical presentation and involved total body surface area the patient was diagnosed with a suspected case of carbamazepine induces SJS – TEN overlaps.

Table 1: SCORTEN scale

Risk factors	Reference	Score
Age	<40 years	0
Associated malignancy	No	0
Heart rate (beats/min)	<120	0
Serum BUN (mg/dL)	<27	1
Detached or compromised Body Surface	<10%	1
Serum bicarbonate (mEq/L)	>20	0
Serum glucose (mg/dL)	<250	0

SCORTEN scale represents severity of illness score for toxic epidermal necrolysis. By using these criteria, the patient was diagnosed as stevens – Johnson syndrome/ toxic epidermal necrolysis. Prognosis was assessed using score of TEN (SCORTEN) criteria which conferred a score of 2 for the index case. The mortality risk was estimated to be 12.1 %.

A multidisciplinary team was involved in patient management, including those drawn from the fields of ophthalmology, ear, nose and throat (ENT), internal medicine and dermatology. The patient had initially been admitted in MICU (isolation room barrier nursing care) stop the drug carbamazepine and treated with IV fluid, antibiotics inj dexamethasone, symptomatic and supportive therapy. Patient TEN score was high & lesion progressing and then plan for IVIG therapy 0.4 gr/kg/day for 5 days and taken proper skin care general nursing care paraffin coated banana leaves along with skin moisturizer and emollients eye care – lubricating eye drops and antibiotics mouth care before and after taking food. Position changing 4^{th} hourly and mobilization in bed and out of bed. Had reactive thrombocytosis & started on antiplatelets.

Resolving skin lesions- scab formation, no new skin lesion, afebrile hemodynamically stable accepting full diet orally. And condition gradually improved skin lesions / tenderness gradually decreases. Patient on oral liquid along with RT feeds oral ulcer gradually healed and patient shifted on oral feeds eye side congestion improved, pain decreased high protein & high caloric diet taken. Then patient shift to room. Patient gradually improved well during hospital stay course was uneventful. Now patient haemodynamically stable.

DISCUSSION

While there are several causes of SJS and TEN, medications are the most common. The most often linked medications include oxicam, lamotrigine, carbamazepine, phenytoin, and phenobarbital, as well as nonsteroidal anti-inflammatory medicines, sulfa medications, and allopurinol. There have also been reports of other causes, including infection and cancer, and individuals who are immunocompromised

or have HIV have a higher chance of contracting the illness [4].

SJS and TEN are severe mucocutaneous drug reactions. The type of skin lesions and the proportion of BSA involvement determine how these two disorders differ from one another. SJS is characterized by the detachment of 10% BSA along with flat, atypical target lesions or extensive erythematous, purpuric macules. SJS/TEN, which is an overlap of the two disorders, is characterized by extensive erythematous, purpuric macules or unusual target-like circular regions together with a 10-15% BSA detachment. When TEN is present, there is a 30% BSA detachment combined with unusual target lesions or extensive erythematous, purpuric macules. Large epidermal sheets with roughly 10% BSA and no purpuric macules or target lesions are the result of TEN without spots [5,6].

Certain HLA alleles and drug-induced SJS/TEN have a high correlation. Among Asians, HLA-B15:02 is the most significant factor in SJS caused by carbamazepine. It is possible to do a genetic screening for HLA-B15:02 before prescribing carbamazepine, particularly in Southeast Asia where this variant is quite prevalent. Since a genetic test was not carried out in our instance, we are sadly unable to establish that the allele is the cause of TEN [7].

A biopsy is the only particular laboratory test that can be used to confirm the diagnosis of SJS and TEN. As a result, clinical diagnosis is made, emphasizing prior drug use. A significant WBC spike may suggest a superimposed bacterial infection, yet CBC may show normal or nonspecific leucocytosis. Skin, urine, and blood cultures have therefore been recommended because sepsis and severe bacterial bloodstream infections are major causes of morbidity and mortality.

The most critical step in managing patients with SJS or TEN is the prompt withdrawal of the medication, which has a substantial impact on morbidity and mortality. Then, supportive and symptomatic care—which includes maintaining body temperature, hydration, and electrolyte replacement—should be administered in intensive care units or burn centers. Other care should include maintaining venous access far from the affected areas, controlling pain, preventing secondary infection, controlling body temperature, providing early oral or parenteral nutrition as needed, and administering anticoagulation [8]. Skin lesions are treated in the same way as large-scale burns. Topical antiseptics are acceptable, but because SJS and sulfonamides are related, silver sulfadiazine should be avoided [5].

Commercial IVIG preparations were found to contain anti-Fas (anti-CD95) antibodies, which prevented the Fas-FasL interaction and influenced the pathophysiology of the disease. Treatment success is mostly dependent on dosage and early application [9]. In our instance, the patient's condition significantly improved with IVIG and plasmapheresis.

CONCLUSION

Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are the most severe adverse drug reactions. Characterized by necrosis and epidermal release. We experienced a patient who was diagnosed as SJS and TEN overlap. In our instance, intravenous immunoglobulin therapy and plasmapheresis treatment proved to be effective means of managing the patient. additionally, diseases are severe and potentially fatal, and they also lack a conclusive treatment. Therefore, quick management is required.

Declaration by Authors

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REFERENCES

1. Borrelli E. P., Lee E. Y., Descoteaux A. M., Kogut S. J., Caffrey A. R. Stevens-Johnson syndrome and toxic epidermal necrolysis with antiepileptic drugs: an analysis of the US food and drug administration adverse event reporting system. *Epilepsia.* 2018;59(12): 2318–2324. Doi: 10.1111/epi.14591.
2. Huang L.-Y., Liao W.-C., Chiou C.-C., Lou J.-P., Hu P., Ko F.-C. Fatal toxic epidermal necrolysis induced by carbamazepine treatment in a patient who previously had carbamazepine-induced Stevens-Johnson syndrome. *Journal of the Formosan Medical Association.*2007;106(12):1032–1037. Doi: 10.1016/S0929-6646(08)60079-0.
3. Masuka J. T., Khoza S., Chibanda D. An interesting case of carbamazepine-induced stevens-Johnson syndrome. *Drug Safety—Case Reports.* 2018;6(1):p. 1. doi: 10.1007/s40800-018-0095-y.
4. Abuzneid, Y. S., Alzeerelhouseini, H. I., Rabi, D., Hilail, I., Rjoob, H., Rabee, A., Amro, N., Qafisheh, Q., & Kharraz, M. (2022). Carbamazepine induced Stevens-Johnson syndrome that developed into toxic epidermal necrolysis: Review of the literature. *Case Reports in Dermatological Medicine*, *2022*, 1–4. https://doi.org/10.1155/2022/6128688
5. Schwartz R. A., McDonough P. H., Lee B. W. Toxic epidermal necrolysis. *Journal of the American Academy of Dermatology.* 2013;69(2):p. 173.e1. Doi: 10.1016/j.jaad.2013.05.003.
6. Thong B. Stevens-Johnson syndrome / toxic epidermal necrolysis: an Asia-Pacific perspective. *Asia Pac Allergy*. 2013;3:215–23. Doi: 10.5415/apallergy.2013.3.4.215.
7. Nasir S. A., Tan H. L., Tan H. J., Hussaini H. M., Ramli R. Stevens-Johnson syndrome following failure of genetic screening prior to carbamazepine prescription. *Case Reports in Dentistry*. 2017;2017:5. Doi: 10.1155/2017/4201357.4201357
8. Wong A., Malvestiti A. A., Hafner M. d F. S. Stevens-Johnson syndrome and toxic epidermal necrolysis: a review. *Revista da Associação Médica Brasileira (1992).* 2016;62(5):468–473. Doi: 10.1590/1806-9282.62.05.468.
9. Viard I., Wehrli P., Bullani R., et al. Inhibition of toxic epidermal necrolysis by blockade of CD95 with human intravenous immunoglobulin. *Science.* 1998;282(5388): 490–493. Doi: 10.1126/science.282.5388.490.

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