

Multiple Drug Induced Steven Johnson Syndrome- A Case Report

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ABSTRACT

Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are rare, life threatening conditions usually associated with medicines use. They are severe forms of exfoliative dermatitis, and characterized by extensive epidermal erythema and blistering, which leads to necrosis and detachment of the epidermis. They can also cause mucocutaneous lesions. A 28 years old male patient admitted high grade fever associated with chills and rigors and loose stools but not blood stained with Cefotaxim treatment allergic responses like swelling in the B/L legs, rashes with itching followed by skin peeling all over the body, erythema positive on face and hands, multiple painful ulcers in the oral cavity with inflamed borders. Laboratory investigation shown that abnormal values particularly, total count and SGOT (85 U/L), SGPT (108 U/L) levels are elevated abnormally. He is diagnosed as multiple drug induced SJS (Phenytoin, Cefotaxim and native medicines). This case highlights the precipitant of antibiotics for Stevens-Johnson syndrome.

Key words: Steven Johnson syndrome, Toxic epidermal necrosis, Dermatitis, Epidermis and Erythema.

INTRODUCTION

The exact cause of SJS and TEN is indefinite. SJS is estimated to affect 1-6 patients per million population per year and it is up to three times more common than TEN.¹ Pharmacogenomic studies designate that ethnicity and assured human leukocyte antigen (HLA) types may predispose patients to SJS and TEN.² SJS stereotypically involves the skin and the mucous membranes. While minor presentations may occur, significant involvement of nasal, oral, vaginal, urethral, GI, and lower respiratory tract mucous membranes may develop in the course of the illness.³

The modest breaks the disease down as follows⁴⁻⁶

- Stevens-Johnson syndrome - A “minor form of TEN,” with < 10% body surface area (BSA)
- Overlapping Stevens-Johnson syndrome/toxic epidermal necrolysis (SJS/TEN)- Detachment of 10-30% BSA

- Toxic epidermal necrolysis-Detachment of >30% BSA

Antiseizure drugs were the most common drugs causing SJS and TEN, higher chance (81.8%) of causing severe eruption, that is, TEN than NSAIDs (53.84%) and antimicrobials (34.48%). This is higher as compared with the previous report (70%). Average number of drugs to be found as causative for SJS, TEN, and SJS-TEN overlap was 1.81.⁷

CASE DISCUSSION

A 28 years old male patient admitted in the department of General medicine in a tertiary care teaching hospital with the complaints of high grade fever (intermittent rise of temperature in the evening) for 20 days associated with chills and rigors and loose stools but not blood stained since 1 week. Ten days back patient was taken to a nearby hospital and the patient was treated with

Submitted date : 02/11/2015
Accepted date : 10/12/2015

DOI: 10.5530/ijopp.8.4.11

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Figure 1: Skin Ulceration

Table 1: Follow up Examinations vs Choice of Drug Treatment

Days	Examinations	Drug
Day-1	Fever spikes, conscious and oriented Temperature: 100 F, BP: 100/60 mm.Hg PR: 80/min, EEG: Generalized epileptic waves, TC: 25900 mCL	Initially patient treated with symptomatic treatments.
Day-2	Conscious and oriented Temperature: 99 F, BP: 100/60 mm.Hg PR: 80/min, Ulceration in the oral cavity, Erythema positive on face TC: 18700/mm ³	Inj. Dexa 8 mg, i.v, OD Betadine gargle TDS Linocort oral paste E/A, BD Candid B cream as a symptomatic therapy Inj. Rantac 50 mg, i.v, BD for stress ulcer Inj. Ciplox 400 mg, i.v, BD Inj. Metrogyl 500 mg, i.v, BD for sepsis T.Livipil 500 mg, p/o, BD T. Frisium 10 mg, p/o, BD for seizure disorder
Day-3	Normal physical signs and the lab parameters, TC: 18700 mCL, developed oral candidiasis	Same treatment continued. Additionally permitted to do serum procalcitonium for antibiotic escalation. Candid oral mouth paint L/A OD for oral candidiasis.
Day-4	Normal physical signs	Same treatment continued
Day-5	Oral ulcer reduced	T. Ciplox and T. Metrogyl stopped. Inj. Amikacin 500 mg, OD started.

antibiotic (Cefotaxim) for health issues. Hence, the patient developed with allergic responses like swelling in the B/L legs, rashes with itching followed by skin peeling all over the body, erythema positive on face and hands, multiple painful ulcers in the oral cavity with inflamed boundaries. Past medical history revealed that patient already a known case of Seizure disorder, under the treatment with Tab. Eptoin and Tab. Frisium since past 1 year. The patient looked conscious, oriented but weak. Family history discloses the grandfather had a seizure disorder. On systemic examination, temperature- 102 F, pulse rate- 100/min, RR-15/min, normal BAE, S₁S₂ and abdomen soft. Furthermore, total count and SGOT (85 U/L), SGPT (108 U/L) levels are elevated. He is diagnosed as multiple drug induced SJS (Phenytoin, Cefotaxim and native medicines). Immediately all the drug precipitants was with drawn (Figure 1 and Table 1).

On the sixth day patient referred to get dermatology opinion regarding the continuation of steroids and other therapies. 7th day Inj. Dexa stopped and switched to oral prednisolone 5 mg OD for one week. The same therapy continued for the next three days and all the sign and symptoms were reduced. The lotion Zensoft max E/A BD started and candid B cream is stopped. After 11 days of therapy, all the skin reactions were subsided. Patient was referred for ophthalmology opinion before discharge.

CONCLUSION

Corticosteroids are a choice of therapy for SJS in most of the cases. SJS and TEN are severe ADR usually caused by NSAIDs, antibiotics and antiseizure drug and the

overall cost of management is higher than other ADRs. Despite approximately half of cases of Stevens-Johnson syndrome being idiopathic, and some literatures reported that the industrial chemicals are causing SJS most frequently. Timely diagnosis with the prompt recognition and withdrawal of all potential causes is essential for a positive outcome. Symptomatic management and nutritional balance is the importance of reducing the intensity of reactions. A risk assessment is required for preventing the additional tissue damage.

ACKNOWLEDGEMENT

I take this opportunity to thank all the department faculty members for their help and support to publish this case study.

REFERENCES

1. Roujeau JC, Kelly JP, Naldi L. Medication use and the risk of Stevens-Johnson syndrome or toxic epidermal necrolysis. *N Engl J Med.* 1995;333(24):1600-8.
2. Yip VL, Alfirevic A, Pirmohamed M. Genetics of immune-mediated adverse drug reactions: a comprehensive and clinical review. *Clin Rev Allergy Immuno.* 2014;48(2-3):198-206.
3. SVSG Nirmala, R Dadeepiya. A case of Steven Johnson syndrome. *E Mednifico Journal.* 2014;2(3):299-300.
4. Satyanand Tyagi, Sachin Kumar, Amit Kumar, MohitSingla and Abhishek Singh. Stevens-Johnson syndrome-A life threatening skin disorder: A review. *J. Chem. Pharm. Res.* 2010;2(2):618-26.
5. M Barvaliya, J Sanmukhani, T Patel, N Paliwal, H Shah, C Tripathi. Drug-induced Stevens-Johnson syndrome (SJS), toxic epidermal necrolysis (TEN), and SJS-TEN overlap: A multicentric retrospective study. *J Postgrad Med.* 2011;57(2):115-9.
6. House RA, Jakobovic H, Wong L, Holness DL. Work-related toxic epidermal necrolysis. *J Occup Med.* 1992;34(2):135-9.
7. Sharma VK, Sethuraman G, Minz A. Stevens Johnson syndrome, toxic epidermal necrolysis and SJS-TEN overlap: A retrospective study of causative drugs and clinical outcome. *Indian J Dermatol Venereol Leprol.* 2008;74(3):238-40.

CONFLICT OF INTEREST

The author declare no conflict of interest.

ABBREVIATION USED

NSAID:	Non-Steroidal Anti-Inflammatory Drug
ADRs:	Adverse Drug Reaction
TEN:	Toxic Epidermal Necrosis
SGPT:	Serum Glutamic-pyruvic Transaminase
SGOT:	Serum Glutamic-oxaloacetic Transaminase