Carbamazepine Induced Stevens-Johnson Syndrome (SJS)

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INTRODUCTION

Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN), are dermatologic emergencies characterised by a blistering rash that appears in a variable percentage of the body-surface. The mortality risk increases with increasing epidermal detachment. TEN is the rarest of these phenotypes and is associated with mortality of up to 30 %.¹ Exposure to certain drugs, some infections, contrast media, and vaccines can predispose to development of SJS. We report a case of carbamazepine induced SJS.

PRESENTATION OF CASE

A 35-year-old male, chronic alcoholic presented to us with a generalised symmetrical, erythematous rash, which was fast spreading and affected the face, upper limbs, chest and had breathlessness of 3 days duration. He had been drinking alcohol daily for last 3 months. Because of some problem he didn't get alcohol for 3 days and had a status generalised tonic clonic seizure for which he was taken to a private hospital where he received IV lorazepam and carbamazepine 200 mg t.d.s. per orally. He developed dyspnoea and skin rash 3 days after oral carbamazepine.

On examination, facial swelling was present; bullae were present on neck and chest; there were ulcerations in the mouth associated with conjunctival injection and difficulty in opening eyes. Pulse - 118 / min, temperature - 39.2° C, with SpO₂ of 87 % in ambient air, and blood pressure of 92 / 50 mmHg. Vesiculobullous lesions developed over the course of the admission period which was associated with sloughing of skin on the chest, face and lips (Fig. 1, 2).

Investigations: CBC-Hb - 9.2 gram %, TLC- 2,500 / mm³, Neutrophils- 88 %. Liver and renal function was normal. A diagnosis of carbamazepine induced Steven-Johnson syndrome was made. Evaluation of Toxic Epidermal Necrosis (SCORTEN) score on admission was 2. CXR (chest X-ray) PA revealed right lower lobe consolidation.

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DOI: 10.14260/jemds/2020/852

How to Cite This Article:

*Acharya S, Hulkoti V, Andhale A, et al.
Carbamazepine induced stevens-Johnson
syndrome (SJS). J Evolution Med Dent Sci
2020;9(51):3892-3893, DOI:
10.14260/jemds/2020/852*

*Submission 07-04-2020,
Peer Review 28-10-2020,
Acceptance 04-11-2020,
Published 21-12-2020.*

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Figure 1.
*Ulcerations Over Mouth, Lips
Face and Neck*

Figure 2.
*Bullous Lesions and
Pigmentation Over the Body*

DISCUSSION OF MANAGEMENT

Carbamazepine was discontinued and the patient was treated with intravenous fluids, IV antibiotics, IV dexamethasone, and antiseptic mouth wash. The patient recovered in 2 weeks.

DISCUSSION

Anti-epileptic drugs (AEDs) that can cause SJS / TEN are phenytoin, phenobarbital, lamotrigine, and carbamazepine.² Two to five percent of patients receiving AED develop cutaneous adverse drug reactions (CADRs).³ According to a study conducted by Lange-Asschenfeldt et al. phenytoin, phenobarbital, lamotrigine, and carbamazepine are the most common AEDs that develop SJS. Carbamazepine is a well-known causative agent of drug induced SJS among anti-epileptic drugs.³

SJS is an unpredictable adverse effect of carbamazepine. It is usually idiosyncratic and dose independent. In Asian and Caucasian populations carbamazepine associated SJS is linked to HLA-B (human leukocyte antigen) * 15:02 and HLA-B * 31:01.⁴ We could not perform genotype study in our patient for HLA alleles. Other factors that can play adjuvant role in development of SJS / TEN due to carbamazepine are, individual variability in drug metabolism, HIV (Human Immunodeficiency Virus) infection, polypharmacy, and competitive drug inhibition.⁵

Financial or other competing interests: None.

Disclosure forms provided by the authors are available with the full text of this article at jemds.com.

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