

Carbamazepine Induced Stevens Johnson Syndrome - A Case Report

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Abstract: Carbamazepine is considered to be the first-line treatment for trigeminal neuralgia. It may rarely lead to complications like Stevens Johnson syndrome (SJS). Initially thought to be a dose-independent, idiosyncratic, unpredictable adverse event specific to an individual, SJS is now considered a delayed hypersensitivity immune reaction involving human leukocyte antigen (HLA) alleles specific for carbamazepine and other drugs in defined populations. In the present case, carbamazepine was immediately withdrawn upon diagnosis of SJS and the patient was managed conservatively using corticosteroids and oral anti-histaminic agents.

Keywords: Carbamazepine, Stevens Johnson Syndrome, Human Leukocyte Antigen

1. Introduction

Carbamazepine is an iminostilbene group of anti-seizure drug used in partial and tonic-clonic seizures [1]. It is also used in the treatment of other conditions like trigeminal neuralgia and in manic-depressive disorder. The primary mechanism of action of carbamazepine is by slowing the rate of recovery of voltage-activated sodium channels from inactivation.

Stevens-Johnson syndrome (SJS) is an immune cutaneous reaction, characterised by development of blisters on target lesions, dusky or purpuric macules and with significant mucosal involvement [2]. The total body surface area and eventual detachment is <10%. SJS is a rare complication of carbamazepine therapy.

SJS is often considered as a delayed hypersensitivity immune reaction involving human leukocyte antigen (HLA) alleles specific for carbamazepine [3]. In particular, it is observed that HLA-B*1502, can significantly increase a person's susceptibility to developing SJS when exposed to specific drugs, such as carbamazepine, in Asian populations [4].

2. Case

A 40 years old male patient with no history of any pre-existing medical or surgical co-morbidities or documented drug allergies, was being treated with oral carbamazepine and paracetamol (1000 mg) for Trigeminal Neuralgia. He received a dose of 200 mg/day of carbamazepine for 2 weeks, increased to 400 mg/day for the next one week and finally up titrated to 600 mg/day. After 5 days of receiving 600 mg/day oral dose of carbamazepine, the patient presented with a 2 days history of multiple itchy and painful skin eruptions.

It was observed that the patient developed multiple itchy and painful erythematous patches with vesicles and bullae of varying sizes. There was sloughing of skin on the back and chest along with oral ulcerations and conjunctival hyperaemia with difficulty in opening the eyes. A diagnosis

of Stevens Johnson Syndrome was made clinically and carbamazepine was immediately withdrawn and the patient was managed conservatively with steroids, anti-allergic drugs and others.

3. Result

A diagnosis of Stevens-Johnson syndrome was made clinically.

A detailed medication history showed the absence of any co-administered drugs that had strong association to causing immune cutaneous reactions.

Therefore, carbamazepine was ruled as the probable offending agent, using the World Health Organisation – Uppsala Monitoring Centre (WHO-UMC) Causality Assessment Scale.

4. Treatment

Upon diagnosis of SJS, carbamazepine was immediately withdrawn.

The patient was admitted in a tertiary care hospital and adequate hydration was maintained using normal saline and Ringer's lactate infusion in 1:1 ratio, given 8 hourly.

He also received intravenous corticosteroid, Hydrocortisone 100 mg thrice daily for a period of 3 days.

After this he was switched over to oral prednisolone therapy, given at a dose of 60 mg/day for 10 days, tapered to 40 mg/day for the next 10 days and finally down titrated to 20 mg/day for the next 10 days. This was supplemented with oral levocetirizine 10 mg/day for 10 days.

Chloramphenicol eye drop was instilled in both eyes twice daily until the conjunctival hyperaemia subsided. Clotrimazole ointment was applied locally over the sloughed skin and ulcers, to prevent secondary fungal infection.

The patient gradually recovered without no sequelae.

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5. Conclusion

Carbamazepine is considered to be the first-line treatment for trigeminal neuralgia. It may rarely lead to complications like Stevens Johnson syndrome. Early diagnosis and initiation of treatment is of utmost importance in treating such serious adverse reactions. It is essential to take proper drug history and regularly monitor patients to prevent such adverse reactions. Screening of patients for HLA-B*1502 allele should ideally be implemented to prospectively identify individuals at genetic risk for developing SJS [5].

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