



Rare but Severe: A Case Report of Carbamazepine-Induced Stevens-Johnson Syndrome

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Abstract

This case report describes a rare but serious adverse drug reaction – Steven Johnson Syndrome (SJS) induced by Carbamazepine. A female patient, aged 15, arrived at the Casualty unit complaining of redness and pain in both eyes, a painful lesion over her mouth, and a dusky red discoloration that had been present for six days throughout her face, neck, trunk, and upper limbs. The patient has a history of mental disorders, including temper tantrums, for which she was under medication. For the same initial diagnosis, she was administered a once-daily dose of carbamazepine and she received the medication for about four weeks. Nevertheless, the patient was brought to the Casualty unit four weeks after starting carbamazepine due to the aforementioned concerns. The patient was diagnosed with Carbamazepine-induced SJS and promptly treated with supportive measures followed by discontinuation of the offending drug.

Keywords: Carbamazepine; Stevens-Johnson Syndrome; Severe Cutaneous Reaction; Hypersensitivity Reactions; Adverse Drug Reactions

Introduction

Stevens Johnson Syndrome (SJS) is a rare but life-threatening skin disease characterized by flat, irregular target-shaped lesions and/or scattered blisters that originate in macules [1]. Clinically, it manifests as erythema, necrosis, mucous involvement, systemic symptoms, and significant epidermal sloughing [2].

Drug exposure is one of the most prevalent cause of Stevens Johnson Syndrome (SJS) [1]. Phenobarbital, lamotrigine, and carbamazepine have a high risk of causing epidermal necrolysis [2]. According to reports, the incidence of a major hypersensitivity reaction to carbamazepine ranges from 1/1,000 to 1/10,000 [2]. The aim of this article is to highlight a case of Carbamazepine induced Stevens Johnson syndrome (SJS).

Case Report

A 15-year-old female patient presented to Casualty unit with chief complaints of redness and pain over both eyes, sore lesion over the mouth and dusky red discoloration over the face, neck, trunk and upper limbs since 6 days (Figure 1, Figure 2). The patient additionally complained of difficulty in swallowing and breathing difficultly. According to the patient's prior medical records, she is a known case of mood disorder with temper tantrums for which she was under medication (tablet paroxetine 12.5mg, once daily). The duration for which she was under tablet paroxetine is unknown. However, on 1st January 2024, she was prescribed tablet carbamazepine (100 mg) once daily for the same initial diagnosis and continued the treatment for approximately 4 weeks. However, 4 weeks post-initiation of carbamazepine, the patient was rushed to the Ca-



Figure 1: Dusky red discoloration over neck.



Figure 2: Dusky red discoloration over Trunk.

sualty unit with the above mentioned complaints. On examination she was in minimal respiratory distress, with dusky red coalescent macular discoloration of skin over the face, upper chest and back region noted with desquamation < 5% BSA at presentation. No active blisters were noted, however there was mild skin tenderness on palpation over affected sites. Direct Nikolsky's sign was positive. In addition, hemorrhagic crusting was also noted over the upper and lower lips with erosions over bilateral angles of mouth. Oral mucosal erosions were also observed over the tongue and buccal mucosa. Upon ocular examination, there was bilateral conjunctival congestion. Her temperature was 98°F, with SpO₂ of 100%, a blood pressure recording of 90/60 mmHg and a regular pulse of 68 beats/min.

A diagnosis of Stevens Johnson Syndrome (SJS) secondary to carbamazepine exposure was made. Carbamazepine was immediately discontinued and the patient received intravenous fluid resuscitation and supportive treatment. She was subsequently transferred to dermatology ward where the patient was kept warm, had careful protection of the eroded areas with intravenous hydration. Treatment for Stevens Johnson Syndrome included IV Corticosteroids, Oral Antibiotics, Mouth gel, Topical antibiotic ointment, and antiseptic mouth wash. Routine laboratory assessments showed normal blood counts without hypereosinophilia. Serum electrolyte levels, renal function, hepatic enzyme levels, and serological test results were all negative and within normal ranges. Evaluation of the SCORE of Toxic Epidermal Necrosis (SCORTEN) score on day 1 indicated a score of 1. Punch biopsy taken for histopathology findings were s/o lichenoid pattern of inflammation seen in drug reactions. But since the patient presented almost 1 week after stopping the drug, lesions were already healing and findings were not significant. Overall, majority of the test were normal. While on medication, the patient showed improvements with each coming day and two weeks post admission, the patient was discharged with instructions to follow up in dermatology outpatient department after a week.

Discussion

Carbamazepine is a well-known psychoactive drug that is responsible for drug-induced Stevens Johnson Syndrome (SJS).^[2] Following a seven-year study, Devi et al. recently concluded that

anticonvulsants were the main cause of SJS, especially in the first eight weeks of treatment, and that carbamazepine was the main medication accountable for over 80% of drug-induced SJS [3]. Individual variations in drug metabolism or clearance, HIV-1 seropositivity, polypharmacy, and competitive drug inhibition are other factors that may be important in the development of SJS when using carbamazepine [4].

In light of current literature, no Stevens Johnson Syndrome (SJS) cases involving the co-administration of carbamazepine and Selective serotonin reuptake inhibitor (SSRI) have been documented. In the present case, the patient had been on carbamazepine for approximately four weeks. She had previously received a prescription for an SSRI paroxetine, but the relevant prescription documents are missing, so it is unclear how long she took the medication and when she quit. The finding that the onset of SJS and the administration of carbamazepine correlated temporally suggests that the latter was the causative agent. Despite the fact that the usual latency period for the onset of SJS with carbamazepine is 15 days (12–20), four weeks have also been recorded as latency intervals [5]. The latency period for SSRI-caused SJS is three weeks. The immune-mediated carbamazepine-induced SJS in our patient may have been caused by an increase in carbamazepine plasma concentration brought on by paroxetine's suppression of CYP3A4, given the current level of knowledge surrounding idiosyncratic reactions. Elevations in plasma carbamazepine or its metabolites, including phenytoin, can increase the likelihood of adverse effects, including SJS.⁴ The administration of paroxetine earlier could have potentially increased our patient's susceptibility to SJS. We could not, however, ascertain whether earlier paroxetine administration may have raised the risk of carbamazepine-induced SJS because information regarding the time of ceasing paroxetine was not available.

Conclusion

Early recognition and prompt discontinuation of the offending drug are crucial in improving patient outcomes and preventing progression to more severe forms of SJS.

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