IJBPAS, November, 2023, 12(11): 5072-5076

ISSN: 2277–4998

International Journal of Biology, Pharmacy and Allied Sciences (IJBPAS)

'A Bridge Between Laboratory and Reader'

www.ijbpas.com

A CASE REPORT OF TOXIC EPIDERMAL NECROLYSIS CAUSED BY CARBAMAZEPINE

RAULJI A[*1], CHANIYARA H[*1], TANDEL D[1] AND KAKADIYA J[2]

1: Pharm. D, Scholar, Department of Pharmacy Practice, Parul Institute of Pharmacy and Research, Parul University, Vadodara, Gujarat, India

2: Associate Professor, Department of Pharmacology, Parul Institute of Pharmacy and Research, Parul University, Vadodara, Gujarat, India

[*]**Corresponding Author: Mr. Abhijitsinh Raulji: E Mail: himachaniyara1111@gmail.com**

Received 19th Sept. 2022; Revised 16th Oct. 2022; Accepted 27th Feb. 2023; Available online 1st Nov. 2023

https://doi.org/10.31032/IJBPAS/2023/12.11.7547

ABSTRACT

Rare skin responses that can be fatal include toxic epidermal necrolysis (TEN) and Stevens-Johnson syndrome (SJS), which are frequently brought on by specific drugs. All age groups are affected, although those with HIV, autoimmune illnesses, immunocompromised patients, and those with underlying malignancies are more severely affected. SJS and TEN are signs of a medication reaction, and carbamazepine is frequently to blame. The most frequently implicated medicines in Southeast Asia were all carbamazepine-related. According to data from the Food and Drug Administration, carbamazepine and allopurinol were the second- and third-most popular medications in the Philippines, respectively. Phenytoin. Purpuric macules, target-like lesions, skin detachment, and a blistering exanthema with fast development are the hallmarks of SJS and TEN. Oxcarbazepine (OXC) cross-sensitivity was present in around 25–33% of patients with Carbamazepine (CBZ) hypersensitivity, while CBZ hypersensitivity was present in about 27%–70% of patients with OXC hypersensitivity.

Keywords: Carbamazepine, Epidermal necrolysis

INTRODUCTION:

Drugs or their metabolites are most likely to blame for TEN, which is characterised by widespread, full-thicknes TEN **[1]** which is also known as SJS. Bipolar illness, trigeminal neuralgia, chronic pain, seizures, and neuropathic pain are all treated with

carbamazepine (CBZ). According to earlier research, CBZ frequently causes Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN).

The majority of the time, TEN is an unusual, drug-induced reaction, but it can also be caused by a number of illnesses, such as HIV or Mycoplasma pneumoniae, or it could be caused by an unidentified cause. TEN can be brought on by a number of medications, the most frequently mentioned ones are non-steroidal anti-inflammatory medications, sulfonamides, and anticonvulsants. SJS and TEN incidence rates in the United States range from 2.6 to 7.1 per million people per year and in Germany have annual rates of 1 and 0.93 per million, respectively **[2]**. There is yet no known aetiology for these illnesses **[3]**. HLA-B1502 appears to be intermediately prevalent in South Asians, including Indians, with an average prevalence of 2% to 4%, but greater in other groups. In Japan and Korea, less than 1% of the population carries HLA-B1502. Keratinocyte apoptosis and cytotoxic damage are the pathophysiology of TEN. Tumor necrosis factor (TNF), interferon, and inducible nitric oxide synthase are examples of proapoptotic chemicals that may relate medication-induced immune responses to keratinocyte destruction. The relationship between HLA genotype and CBZ-induced SJS/TEN has been demonstrated in numerous investigations; causes drug antigens in keratinocytes in some people to have a T-cell mediated cytotoxic response. Significant blister-forming mediators have been shown to include CD8+ T lymphocytes **[4]**. Particular HLA alleles have been revealed to be potent prognostic indicators for SJS/TEN. HLA-B 15:02 is the most prevalent marker observed for CBZ-induced SJS/TEN **[5]**.

Case Report:

32 year male was relatively asymptomatic and admitted to the hospital with complain of (c/o) fluid filled lesion on lips which gradually involved the face, chest, abdomen, Bilateral upper limb, back and genitals. prior patient had toothache and he went to the hospital for treatment where he was prescribed with carbamazepine 100 mg twice a day. He took the tablet for 2 weeks and after he was developed skin lessions. these lessions thin brust on their own spontaneously to leave behind raw areas and dark in coloured. The patient also developed c/o erosions face and genitals that gradually increased in size and number swallowing difficult. At the time patient having a c/o fever which was occasional in nature, not associated with chills and rigors and was relieved by medications. The use of carbamazepine was promptly stopped and the patient underwent urgent intravenous fluid resuscitation. Patient also developed c/o pain mild to moderate

intensity and was not relived by medication.

On examination and by the specific diagnostic test patient diagnosed with carbamazepine induced toxic epidermal necrosis which is also known as Stevens Johnson Syndrome. After diagnosis done patient was then referred for further management. Treatment for SJS included Cap. Cyclosporine 100 mg twice a day, Tab. Azithromycine 500 mg for 5 days, Fluconazole 200 mg IV and additional some creams for symptomatic treatment.

DISCUSSION:

Carbamazepine is a well-known psychotropic medicine that causes drug-induced SJS **[6]**. SJS is a type of immune system condition, and the immune system's response can be set off by a variety of things, including infections, illnesses, and negative drug side effects. Some medications, including carbamazepine, are known to frequently induce SJS. These reactions are also idiosyncratic and unrelated to dose or medication **[7]**. In our cases, carbamazepine had been started two weeks before the commencement of the symptoms, and microbiologic tests came up empty on signs of infections. Unknown is the pathophysiology underlying SJS/TEN-related GI involvement, however it is likely comparable to T-cell-mediated hypersensitivity reactions that cause skin lesions **[8]**. According to reports, pharmacological side effects get worse as people get older. As a cytotoxic immunological response, SJS/TEN is thought to kill keratinocytes that carry drug-related antigens. By causing epidermal cells to die or by drawing cytotoxic effector cells, or both, TNF-a produced by macrophages and keratinocytes may be a significant factor in the pathogenesis. The initial step in treating SJS-TEN overlap is discontinuing all probable drugs, then administering steroids, broad-spectrum antibiotics, and supportive therapies. Some people describe the use of steroids in the treatment of SJS-TEN overlap "detrimental" **[9]**, while others refer to it as "life-saving". Prodromal symptoms and indicators of SJS in patients typically include fever, cough, sore throat, headache, vomiting, myalgia, polyarthralgia, diarrhoea, and lethargy. These signs emerge one to fourteen days prior to the onset of mucocutaneous lesions. When a person has TEN syndrome, their body surface is covered in erosions or blisters in more than 30% of cases, and they also have widespread target lesions and maculae **[10]**. Patient had Fluid-filled lesion on the lips that spread to the face, chest, belly, B/L upper limb, back, and genitals over time it will get worse by passing certain period of time. After further management and therapies with a decreased fever, bubbling

on the skin, and other symptoms, the patient's health improved.

CONCLUSION:

The higher incidence of SJS/TEN caused by the same medicine may be related to the increased usage of carbamazepine, particularly for the management of pain. Prior to administering medications known to cause SJS, it is imperative to have a thorough understanding of the patient's medical history. The prognosis of the patient can be improved by substituting the suspicious medications and receiving the proper therapy.

CONFLICT OF INTEREST:

The author declares no conflict of interest.

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