



CASE REPORT

CARBAMAZEPINE INDUCED TOXIC EPIDERMAL NECROLYSIS IN A CASE OF TRIGEMINAL NEURALGIA – AN ATYPICAL CASE REPORT

Dr. Tamasi Choudhury¹, Dr. Lopamudra (Dhar) Chowdhury², Dr. Koustuv Chowdhury³ and Mr. Abhik Saha^{4*}

¹3rd Year Post Graduate Trainee, Department of Pharmacology, R. G. Kar Medical College and Hospital, Kolkata

²Professor and Head, Department of Pharmacology, R. G. Kar Medical College and Hospital, Kolkata

³Assistant Professor, Department of Pharmacology, R. G. Kar Medical College and Hospital, Kolkata

⁴Junior Pharmacovigilance Associate, Indian Pharmacopoeia Commission, National Coordination Centre, R. G. Kar Medical College and Hospital, Kolkata

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*Corresponding author:

Mr. Abhik Saha

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ABSTRACT

Introduction: Toxic epidermal necrolysis (TEN) is a severe cutaneous adverse drug reaction characterized by widespread erythema, necrosis, exfoliation and bullous formation of epidermis and mucous membranes which may cause sepsis and death. Medications are the most common causative factor for TEN. **Case Details:** A 31 years old male with past history of bipolar disorder was on Lithium and Sodium valproate. Due to elevation of liver enzymes discontinuation of lithium and valproate done. After 1 year patient developed right-sided facial pain with hyperalgesia, attended to Neuromedicine OPD and diagnosed as a case of Trigeminal neuralgia and advised to take Carbamazepine. After 2 weeks the patient developed bilateral conjunctivitis, generalized erythematous rash, exfoliation, mucosal surface involvement of mouth and genitalia with subsequent ulceration. The patient was diagnosed as a case of toxic epidermal necrolysis, liver enzymes were found elevated for which Carbamazepine was discontinued immediately, and treated conservatively. Adverse drug reaction was reported to ADR monitoring center. Causality assessment was found probable by WHO (UMC) scale and Naranjo scale. **Conclusion:** Toxic Epidermal Necrolysis is an emergency condition having high rate of morbidity and mortality. Early diagnosis and intervention with timely withdrawal of the offending drug can save the life of patients.

INTRODUCTION

- Toxic epidermal necrolysis (TEN) is a severe cutaneous adverse drug reaction characterized by widespread erythema, necrosis, exfoliation and bullous formation of epidermis and mucous membranes which may result in sepsis and death. Medications being the most common causative factor for TEN, but it may be idiopathic, infection or vaccine induced.^[1]
- Trigeminal neuralgia is a chronic pain disorder associated with sudden attacks of severe unilateral pain in head and face, occurs due to involvement of trigeminal nerve.^[2]
- Carbamazepine is an anti-epileptic drug used for treatment of trigeminal neuralgia. Carbamazepine causes T-cell mediated delayed hypersensitivity reaction which results in development of TEN.

CASE PRESENTATION

A 31 years old male with past history of bipolar disorder was on Lithium and Sodium valproate for 2 years. Patient has

history of elevated liver enzymes for which discontinuation of lithium and valproate was done. After 1 year, patient developed right-sided hemifacial pain with hyperalgesia which was not controlled by analgesics. Patient was referred to Neuro medicine OPD and diagnosed as a case of Trigeminal neuralgia and advised to take Carbamazepine. Pain was controlled significantly but after 2 weeks the patient developed bilateral conjunctivitis, generalized erythematous rash, exfoliation, mucosal surface involvement of mouth and genitalia with subsequent ulceration.

The patient was diagnosed as a case of toxic epidermal necrolysis and admitted to Medicine ward. Liver enzymes were found elevated. Carbamazepine was discontinued immediately and patient was treated with injectable ceftriaxone, metronidazole, methylprednisolone, topical calamine lotion and moxifloxacin eye drop. Adverse drug reaction was reported to ADR monitoring center. Causality assessment was found probable by both WHO (UMC) scale and Naranjo scale.



Fig. Bilateral conjunctivitis, generalized erythematous rash, exfoliation and mucosal involvement of mouth and genitalia seen after taking Carbamazepine – corroborate with the features of Toxic Epidermal Necrolysis

PATHOPHYSIOLOGY

- TEN is primarily a drug induced phenomenon which is caused by T-cell mediated delayed hypersensitivity reaction.^[1] Fas is a membrane-bound protein that triggers a sequence of intra-cellular processes that lead to apoptosis. Fas ligand, often generated by cytotoxic T-cells and natural killer cells, tends to attach to target cell Fas and trigger apoptosis. A high level of keratinocyte localization of FasL was found, which suggested that keratinocytes may be responsible for their own death.^[3]
- The etiology of TEN is mainly due to oxidative stress. A high level of Glutathione S-transferase-p has been detected in TEN patients which is a biomarker of oxidative stress in keratinocytes.^[3]

DIFFERENTIAL DIAGNOSIS

- Staphylococcal scalded skin syndrome.
- Erythema multiforme major.
- Generalized fixed drug eruption.
- Pemphigus vulgaris.
- Bullous pemphigoid.^[4]

DISCUSSION

- Toxic epidermal necrolysis involves more than 30% of the total body surface area.^[1]
- Most cases of TEN are drug induced immune reactions which occurs within 1-3 weeks of initiation of treatment.^[3]

- Majority are caused by anti-epileptic, antibiotics, anti-viral and non-steroidal anti-inflammatory drugs.^[3]
- Starts with Flu like symptoms - fever, malaise, sore throat and conjunctivitis.^[1]
- Cutaneous presentation starts with an exanthematous rash over face and thorax and then gets symmetrically distributed to other areas.^[1]
- Early lesions begin with ill-defined, coalescing, erythematous macules which develop dusky erythema and purpuric spots after 1-2 days.^[1]
- Mucosal affection (erythema and erosion) in oropharynx, eye and genitalia were found.^[3]
- In routine blood investigations anemia and raised liver enzymes were noted.
- Monitoring of the patient who is on anti-epileptic drugs, antibiotics or non-steroidal anti-inflammatory drugs must be continued for 1-3 weeks from the start of medicine.

CONCLUSION

Toxic Epidermal Necrolysis is an emergency condition having high rate of morbidity and mortality. Life-threatening complications include infection, pneumonia, shock, acute respiratory distress syndrome, gastrointestinal bleeding and multiple organ failure. Early diagnosis and intervention with timely withdrawal of the offending drug can save the life of patients.

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