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Steven johnson syndrome induce by carbamazepine in epileptic patient: a case report

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ABSTRACT

Introduction: Steven Johnson Syndrome (SJS) is life-threatening skin reaction, it is a mucocutaneous disorder induced by immune complex-mediated hypersensitivity reaction. Most frequent offending agents are antibiotic, antiretroviral and aromatic anticonvulsants. Problems arise when these drugs are required for long-term use and necessary for several health conditions. These case series aim to describe SJS and provide replacement therapy especially inpatient with epilepsy.

Case report: A 37 years old female was consulted from neurology department with chief complaints an erythematous rash on her chest, back upper and lower extremities accompanied with fever, the patient also complaint erosions on her lips. She had history of seizure and was prescribe Carbamazepine. Carbamazepine was replaced and patient treated with dexamethasone intravenously. After 1 week of admitted there is an improvement.

Conclusion: Steven Johnson Syndrome (SJS) is a life-threatening disease, the replacement of the suspected drugs and appropriate therapy can improve the prognosis of patient.

Keywords: steven johnson syndrome, carbamazepine, drugs

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INTRODUCTION

Stevens-Johnson syndrome (SJS) is one of the most severe types of cutaneous adverse reactions to drugs, with high morbidity and mortality rates.[1] The common cause of SJS is drug exposure. Drugs commonly associated with the development of SJS include antibiotic, antiretroviral and aromatic anticonvulsants such as carbamazepine.[1] It is a challenge to determine the causative agent in treating SJS syndrome, when there are varieties of suspected drugs and problems arise when these drugs are required for long-term use and necessary for several health conditions. We report a SJS induce by carbamazepine in epileptic patient.

CASE REPORT

A 37 years old female was consulted from neurology department with rash on her chest, back, upper and lower extremities accompanied with fever since 2 days ago. Patient also complaint of erosions on her lips, its make patient difficult to eat. History of stoke since 1 year ago and 2 weeks before admitted patient got seizure and treated with carbamazepine two times daily 100 mg. History of hypertension since 1 year ago but didn't take medicine regularly. History of the same symptome was denied, history of applying tradisional medicine was denied.

On the dermatologic status we found erythematous macules well define margin in chest, back, upper and lower extremities and also we found multiple purpuric lesions. On the lip we found a multiple erosions (**Figure 1a-1f**), body surface area (BSA) was 4%. On diascopic test not blanced upon pressure, SCORTEN on first day is 0, we do gram staining on the lip there are no bacteria. We diagnosed this patient with steven johnson syndrome with suspect due to carbamazepine and treated this with stop the suspected drugs, replace with diazepam, dexamethasone 10 mg twice daily intravenously. For the erosions on the lip we give triamcinolone acteonid in ora base and for topical therapy we give open dressing with normal saline 0,9% every 8 hours. After 10 days of hospitalisation there was an improvement, no new lesion appeared.

DISCUSSION

Steven Johnson Syndrome (SJS) is a rare but life-threatening reaction, almost always associated with drugs. Commonly, some of the drugs such as Carbamazepine have a high incidence to cause SJS, and also, these kinds of reactions are independent

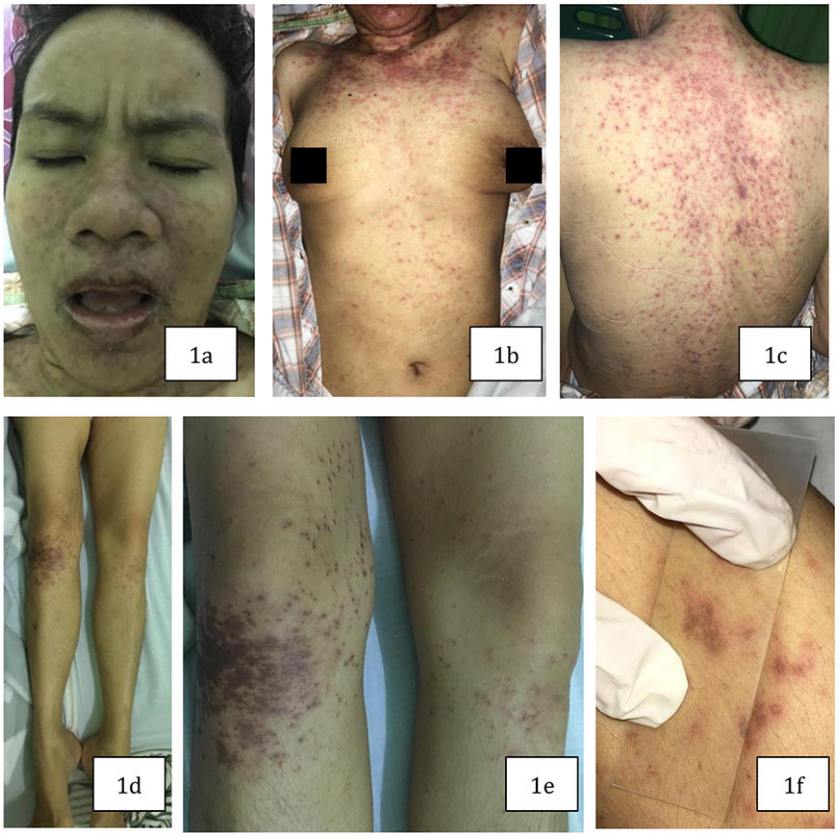

Figure 1. **(a)** Multiple erythematous and purpuric lesions on the face; (**b-e)** multiple purpuric lesions on the chest, back, legs; **(f)** diascopy test not blanched upon pressure

of dose or the drug and are idiosyncratic.[2] Maximal incidences of SJS is in the initial two months of starting the offending medication, with the incidence rate after a period of two months.[3] In our case patient has a history seizure and treated with carbamazepine two times daily 100 mg since 2 weeks ago before the lesions appeared.

The most common clinical features of SJS is marked by symptoms of fever, myalgia, and general weakness for 1 to 3 days before the development of cutaneous lesions. The skin lesions are symmetrically distributed on the face and upper trunk areas. The rash spreads rapidly and is usually maximal within four days, sometimes within hours. The initial skin lesions are usually poorly defined macules with darker purpuric centres that coalesce.[4] In the current case, a typical clinical picture was observed. Conjunctiva lesions such as hyperemia, painful erosions, inflammation are present in about 85% of SJS and TEN patients.[5] Some patients may complain of dysuria and the anogenital region may be involved in pathological process.[6] In our case did not develop any conjunctiva and anogenital lesions.

The differential diagnosis of SJS includes toxic epidermal necrolysis (TEN), SJS/TEN overlap, autoimmunological blister diseases such as pemphigus vulgaris and bullous pemphigoid also Duhring disease. In SJS erosions or blisters involve less than 10% of the body surface covered with atypical target lesions and maculae (mainly on the trunk). The hemorrhagic-erosive lesions are present on at least one mucosal surface. SJS/TEN overlap is characterised by widespread atypical target lesions and maculae. The erosions or blisters involve 10-30% of the body surface. In TEN syndrome body surface is covered with erosions or blisters in more than 30% and the widespread target lesions and maculae are present. Pemphigus vulgaris and bullous Pemphigoid maybe both present with oral blisters and erythematous skin lesions. The clinical course and the histologic and immunofluorescent evaluation produce the answer. The immunopathological differences between autoimmunological blister diseases and SJS syndrome concern deposits of immunoglobulins and complement. In pemphigus deposits are located in intercellular spaces of epidermis, in Pemphigoid in epidermal-dermal junction and in SJS only in mucosal vessels. The deposits consist of IgG and C3 in autoimmunological bister diseases and IgM and C3 in SJS. Dermatitis herpetiformis very rarely has acral and oral involvement.[7]

The SCORTEN scale introduced in 2000 is the most common algorithm used to predict the mortality rate of the SJS/TEN patients. It takes into account the age of the patient, extent of lesions, tachycardia, associated malignancies, metabolic derangements and renal failure. In our case the SCORTEN was 0 indicates a mortality of 3.2% while a maximal score of 5 or above is associated with a mortality rate of 90%.[8,9]

Managing a patient of SJS typically mirrors that of a patient with burns. Fluid and metabolic corrections have to be done, although the deficit is less when compared with that seen in burns.[9] The main therapeutic action in SJS is early recognition of the drug reaction and withdrawal of the drug, since any delay can be seriously deleterious to the patient. Corticosteroid management is highly debated. They may increase the risk of sepsis (primarily due to *Staphylococcus aureus*, *Pseudomonas spp*., Gramm- negative bacilli or candida) and delay healing.[7] On the other hand, some studies reported that early treatment with high doses of systemic steroids ensured a more rapid recovery, mainly in SJS patients where the skin destruction was not too extensive and could be reversed by antiinflammatory effects of steroids.[10] Some studies revealed good therapeutic effect after treatment of SJS patients with systemic steroids

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simultaneously with intravenous immunoglobulin therapy (IVIG) and IVIG alone, however, no randomised clinical trial was published. In our case we treated patient with dexamethasone 10 mg twice daily intravenously and we tapering it every 3 days until the dose of dexamethasone 5 mg, then we switch to oral methylprednisolone. Carbamazepine was replaced with diazepam. We treated patient for 10 days and there was an improvement, no new lesion appears.

CONCLUSION

Steven Johnson Syndrome (SJS) serious dermatological conditions. Since drugs play a major role in the etiopathogenesis of SJS, it is absolutely essential to obtain a detailed medication history in the patient, before initiation of drugs that are known to cause SJS.The replacement of the suspected drugs and appropriate therapy can improve the prognosis of patient.

CONFLICT OF INTEREST

The author declares there is no conflict of interest regarding publication of current report.

ETHIC IN PUBLICATION

The patient had recieved signed inform consent regarding publication of their respective photograph in journal article.

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