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Stevens Johnson Syndrome Induced by Phenytoin: A Case Report







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Keywords: Stevens Johnson Syndrome, Toxic Epidermal Necrolysis, phenytoin, adverse effects

ABSTRACT

Two severe skin responses linked to antiepileptic medicines include Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN). Phenytoin, one of the antiepileptic medications that is most frequently used, has a long list of negative side effects. Clinical features of SJS and TEN include fever, malaise, face puffiness, mucous membrane eruptions, skin lesions, vomiting, and skin eruptions. For individuals with SJS/TEN, a multidisciplinary management approach and supportive care are required. This care entails quitting any suspected drugs that may be the cause of the ailment, hospitalization, replacing lost crystalloid fluid, having a nutritional assessment done, controlling the temperature, managing discomfort, and taking additional steps.

INTRODUCTION:

"Any response to drug which is noxious or unintended and occurs at a dose normally used in man for prophylaxis, diagnosis or treatment of diseases or for modification of physiological function" is the definition of an adverse drug reaction, according to the World Health Organization. Stevens Johnson syndrome (SJS) and Toxic epidermal necrolysis (TEN) are two severe skin reactions associated with antiepileptic medications ^[1]. One of the most often prescribed antiepileptic drugs is phenytoin and is known to cause a plethora of adverse effects ^[1,2]. An estimated one to six cases of SJS and TEN occur for every million person-years. It can occur at any stage, and after the fourth decade, the incidence rises ^[2]. A detachment of less than 10% of the body surface area is characteristic of SJS^[3]. Fever, malaise, facial puffiness, mucous membrane eruptions, skin lesions, vomiting, and skin eruptions are the symptoms of both SJS and TEN^[1]. Whether or not all AEDs can cause SJS is still up for debate. Recent research has examined the contribution of genetic factors, specifically the HLAB*1502 allele, to the development of AED-induced SJS in Asian heritage patients^[4].

Case Report:

A 68-year-old female was admitted to the emergency department with chief complaints of reddish discoloured lesions and peeling of skin since 4 days and shortness of breath for 4 days. The patient has a history of 1 episode of seizure-like activity 15 days ago and was administered with Tab. Phenytoin 100mg OD since 15 days presented with blisters B/L over face. Blisters were ruptured, releasing watery fluid. Multiple reddish discoloured raised lesions associated with minimal burning sensation and whitish discoloured lesions over the tongue with excessive secretions. The patient has a history of hypertension for 20 days and was administered with Tab. Nifedipine SR 20mg OD. Her blood pressure was 160/70mmHg, pulse rate- 90 beats per minute, respiratory rate- 21 cycles per minute. Her oxygen saturation (SpO2) was 98%. On examination, the patient was conscious and coherent. Cutaneous examination reveals multiple targetoid lesions, multiple erosions over face on cheeks, forehead, eye and lips. Oral cavity examination shows whitish plaques on tongue and angles of mouth. CT Brain reveals chronic lacunar infarct. Blood samples were drawn for ABG, CBP, LFT, RFT. Her arterial blood gas analysis (ABG) reported, pH: 7.492, cHCO3: 24.6 mole/L. The ECG report was normal. The WBC count was 2.29, RBC: 3.83, ALT: 270, AST: 560 and urea:80. The evaluation of SCORTEN SCORE for this patient was 1. The patient was treated with Inj. Dexamethasone 2cc OD, Tab. Fluconazole 150mg stat, Candid mouth

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paint for E/A BD, Liq. Paraffin for E/A QID, Mupirocin ointment for E/A BD over the erosions, Betamethasone cream for E/A HS over lesions, Tab. Levipil 500mg BD, Tab. Amlong 5mg BD and by stopping the offending drug(phenytoin). Inj. Ceftriaxone 1gm BD was added along with Tab. Cyclosporine 100mg BD on Day 2. Tab. Fluconazole was reduced to once weekly after 1 week. The patient was discharged after 15 days of treatment.

Discussion:

According to World Health Organisation, the definition of adverse reaction is "any response to drug which is noxious or unintended and occurs at a dose normally used in man for prophylaxis, diagnosis or treatment of diseases or for modification of physiological function" ^[1]. Patient was admitted in the emergency department for the management of multiple reddish discoloured lesions and peeling of skin after the administration of Tab. Phenytoin 100mg. it was a delayed type of hypersensitivity reaction. The patient had initial exposure to Tab. phenytoin at a private hospital for the treatment of seizures. The most frequent type of adverse medication reaction among those that have been recorded is cutaneous. SJS is regarded as a severe form of erythema multiform spectrum, despite its rarity^[5]. The underlying pathophysiology of SJS-TEN involves keratinocyte apoptosis, which causes epidermolysis and subsequent blistering^[6]. With a mortality incidence of up to 40%, SJS and TEN are characterized by rapidly developing blistering exanthema with purpuric macules and target-like lesions together with mucosal involvement and skin detachment^[5]. The primary symptoms of SJS-TEN are fever, headache, coughing, malaise, and rhinorrhoea. Prodromal "target"-type skin lesions also appear, and these eventually progress into a diffuse erythematous rash that affects the skin and mucous membranes. The epidermis experiences widespread necrosis as the disease progresses, and it typically separates from the underlying dermis with minimal effort^[7]. The skin detachment seen in our patient was 10%. According to a case-control study, taking phenytoin for a brief period of time raises your risk of developing SJS and TEN within less than eight weeks. The offending medication should be stopped in such a situation. Most of the time, there is a 1-4 week lag between the first administration and SJS/TEN development. In the present case, the blisters started appearing after 10 days of administration of the drug phenytoin.

A multidisciplinary management strategy and supportive care are necessary for patients with SJS/TEN. This care includes stopping any suspected drug(s) that may be causing the condition, hospitalization (ideally to an intensive care unit), crystalloid fluid replacement,

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nutritional assessment, temperature control, pain alleviation, and other measures ^[6]. In the current case, the suspected drug (phenytoin) was immediately withdrawn and the patient was managed symptomatically. Tab. Cyclosporin 100mg an immunosuppressant drug was given orally twice a day. In the treatment of simple cases of SJS, SJS-TEN overlap, or TEN, cyclosporine plays a promising role. It is also possible to use cyclosporine (3-5 mg/kg/day) in addition to or instead of corticosteroids for a duration of 10-14 days.

The best course of treatment for SJS/TEN is still up for debate because there is so few highquality research comparing the benefits of various targeted therapies. However, the expert panel advises the swift discontinuation of the offending medication, careful supportive care, and the prudent and timely introduction of moderate to high doses of oral or parenteral corticosteroids (ideally within 72 hours)^[2].

Conclusion:

In conclusion, we present a rare case of phenytoin-induced SJS. In this case, the offending drug phenytoin was discontinued and the patient was administered with cyclosporine. Early diagnosis, careful monitoring of complications, and supportive care play major roles in the treatment of SJS.

Conflict of interest:

There is no conflict of interest.

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