Print ISSN 2319-2003 | Online ISSN 2279-0780

IJBCP International Journal of Basic & Clinical Pharmacology

doi: 10.5455/2319-2003.ijbcp20140430

Case Report

Nevirapine induced Stevens-Johnson syndrome: a case report

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Received: 02 January 2014
Accepted: 02 February 2014

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ABSTRACT

Stevens-Johnson syndrome (SJS) is a severe mucocutaneous skin reaction with extensive necrosis and detachment of the epidermis. Nevirapine is one of the high risk agents implicated in development of SJS. Here, we present a case of 27 years old male with HIV infection who was diagnosed to have SJS following administration of nevirapine.

Keywords: Stevens-Johnson syndrome, Nevirapine, HIV

INTRODUCTION

Nevirapine, a dipyridodiazepinone non-nucleoside reverse transcriptase inhibitor, has potent activity against HIV-1.[1] According to National AIDS Control Organization guidelines, nevirapine is one of the drugs used as preferred first line regimen against HIV infection.[2] Stevens-Johnson syndrome (SJS) is an acute life-threatening mucocutaneous reaction characterized by extensive necrosis and detachment of the epidermis.[3] SJS is the most often cause by drugs such as sulfonamides, non-steroidal anti-inflammatory drugs, allopurinol, anticonvulsants, etc.[4] Life-threatening SJS is seen as an adverse reaction to nevirapine in about 0.3% of recipients.[1]

CASE REPORT

A 27 years old male patient presented with complaints of miliaria like lesions over the abdomen, redness, pain, swelling, and watery discharge from eyes. He had conjunctival congestion and symblepharon. He also had pain in the throat while swallowing. One day later he developed painful lesions in the oral cavity, reddish-black fluid filled lesions over the trunk, upper limb and lesions over the scrotum with oozing. Ten days before the above reaction he was diagnosed to have retroviral illness with CD4 count of 349 cells/mm^3. He was started on highly active anti-retroviral therapy with tablet zidovudine 300 mg, tablet lamivudine 150 mg, tablet nevirapine 200 mg twice daily.

On examination, he had multiple hemorrhagic vesicles present over forehead, malar area and crusting over pinna with bulla. He had erosions and crusting over lips, angular cheilitis, difficulty in opening mouth and erosions over tongue and buccal cavity. He had multiple hemorrhagic lesions present over trunk, upper and lower limb, purpurea present over palms, soles, legs, and toes. He had erosions with seropurulent discharge over scrotum.

Laboratory investigation revealed the following: hemoglobin - 12.5 g/dl, hematocrit - 37.6%, total WBC count - 4000 cells/mm^3, platelet count - 3,22,000 cells/mm^3, erythrocyte sedimentation rate - 70 mm/hr, aspartate transaminase - 25 IU/L, alanine transaminase - 23 IU/L, alkaline phosphatase - 71 IU/L, serum creatinine - 0.7 mg/dl, urea - 37 mg/dl, serum sodium - 135mEq/L, serum potassium - 4.7 mEq/L, serum chloride - 97.5 mEq/L. Urine analysis showed traces on protein, no sugar and RBCs.

He was admitted to hospital. ART was stopped and he was started on injection dexamethasone 12 mg IV once daily for 3 days, saline compressions for lips and scrotum twice daily, betamethasone and fusidic acid combination cream for local application over lips and scrotum twice daily, chlorhexidine gargles, tablet linezolid 600 mg twice daily for 6 days, ciprofloxacin and carboxymethylcellulose eye drops. His vision was >3 (Counting finger [CF], both eyes) on the day of admission, but after glass rod sweeping was done to break adhesion his vision improved to >6 (CF) in both eyes. He was put on a soft diet during the hospital stay due to restricted opening of mouth and oral ulcers. He responded to the treatment and after 6 days skin lesions improved, congestion in conjunctiva subsided and he had clear corneas, no symblepharon and extra-ocular movements were normal.

He was discharged with tablet prednisolone, topical applications and eye drops as mentioned above. The dose of steroids was gradually tapered. On follow-up after 1 month the rash had subsided, there were no erosions in the oral cavity. Sloughing of skin and new skin formation was seen over chest, nape of the neck and entire back. On ophthalmological examination, there was no symblepharon, lens were clear, pupils were 3 mm in diameter and vision was 6/6 in both eyes.

DISCUSSION

SJS is a rare and life-threatening condition provoked by the activation of a cell-mediated cytotoxic reaction and amplified by cytokines. Drugs are the most important etiologic factors for development of SJS. Nevirapine is considered to be amongst the "high-risk" category of medications implicated in development of SJS.[3] In our patient, the development of mucocutaneous lesions had a temporal relationship with administration of nevirapine and coincides with the time taken for development of this condition following drug exposure which is approximately 4-30 days.

The initial skin lesions in SJS are characterized by erythematous, irregularly shaped purpuric macules which progressively coalesce followed by necrosis and detachment mainly involving the face, trunk and proximal limbs. Mucous membrane involvement (nearly always on at least two sites) is observed in approximately 90% of cases, preceding or following skin eruption. SJS is also associated with extracutaneous symptoms like fever, pain, weakness etc.[3] Our patient had presented with miliaria like lesions which later progressed to hemorrhagic vesicles, crusting and oozing. There was involvement of two mucosal sites namely ocular and oral mucosa. Genital region was also involved and patient had symptom of pain.

SJS is a life-threatening disease that requires optimal management: Early recognition and withdrawal of offending drugs as was done in our patient. Prompt withdrawal of the offending agent is associated with an increased rate of survival in patients with SJS induced by drugs with short elimination half-lives. Because of the importance of immunologic and cytotoxic mechanisms, a large number of immunosuppressive and/or anti-inflammatory therapies have been tried to halt the progression of the disease.[3] Our patient was treated with steroids to control the inflammatory process and antibiotics to prevent the development of infection. However, the use of steroids is still controversial as some studies conclude that steroids did not stop the progression of the disease and were associated with increased mortality and adverse effects, particularly sepsis, but a large cohort study has suggested a possible benefit that should be explored by a prospective study.[5]

There have been previously published case reports that ascertain the relation between nevirapine administration and development of SJS. Nevirapine is amongst the first-line agents in the treatment of HIV infection, which is a growing concern in our country. As it cannot be discarded as a treatment option owing to its efficacy in control of progression of the HIV, necessary precautions need to be taken while introducing nevirapine in any HIV afflicted individuals. It may become necessary to keep patients under observation for a few days following the start of treatment with nevirapine or at least forewarn them about such possible reactions and the importance of seeking medical help immediately if any skin reactions develop.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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doi: 10.5455/2319-2003.ijbcp20140430
Cite this article as: Patel IB, Adiga S, Shetty DJ, BairyKL. Nevirapine induced Stevens-Johnson syndrome: a case report. Int J Basic Clin Pharmacol 2014;3:401-2.