Allopurinol Induced Stevens – Johnson Syndrome: A Rare Case Report

NS Neki, Jasbir Kumar, Ankur Jain, Rohit Bajaj, Mohit Manav, Harsh Kumar, Vishwanath Chavan
Department of Medicine, Government Medical College/GND Hospital, Amritsar*

ABSTRACT

Adverse drug reactions occur in about 3-5% of all hospital admissions and are associated with increased morbidity and mortality. Drug induced allergic reactions are categorised as IgE mediated and non IgE-mediated hypersensitivity reactions. These reactions vary from mild rashes to severe life threatening reactions like Stevens Johnson syndrome (SJS). SJS is a rare but potentially fatal adverse drug reaction. It can be associated with infection or combination of infection and adverse drug reactions. A case of 50 year old male normotensive, non diabetic, non alcoholic suffering from gouty arthritis is reported here. Diagnosis of SJS was made on history and clinical examination and the patient was successfully treated with parenteral antibiotics, antihistamines, corticosteroids and IV fluids.

Keywords: Stevens-Johnson Syndrome (SJS), Allopurinol, Adverse Drug Reaction

INTRODUCTION

Adverse cutaneous drug reactions occur in about 3-5% of hospitalized patients with approximately 1 in 1000 hospital patients suffering from life threatening drug reactions. Most commonly observed drug reactions are cutaneous drug reactions in 65-85% of cases. SJS is induced by various drugs like trimethoprim-sulfamethaxazole, cephalosporines, quinolones sulphonamides, antibiotics, carbamazepine, phenytoin, valproic acid, phenobarbital, non steroidal anti-inflammatory drugs (NSAIDs), allopurinol etc.

Allopurinol, a uric acid lowering drug, can cause life threatening severe adverse reactions in the form of hypersensitivity syndrome, Stevens-Johnson syndrome (SJS) and toxic epidural necrolysis. SJS is diagnosed clinically by fever (100.4°F), skin tenderness, mucositis and blistering.

CASE REPORT

A 50 years old man, non diabetic, non hypertensive, non alcoholic presented with joint symptoms in the form of pain and tenderness mainly of the lower limbs especially left great toe. He was diagnosed as a case of gouty arthritis on clinical examination and laboratory profile. He was put on tablet allopurinol 300 mg daily. After 2 weeks, he complained of itchy diffuse skin lesions with burning sensation involving face, chest, abdomen, eyes, upper and lower extremities. He also complained of nausea, headache, burning micturition and constipation. After 2 days, there was involvement in mucosal areas in the form of erythema of the conjunctiva, nasal mucosa, buccal mucosa in the form of eye discharge, lid swelling, dysphagia, angular stomatitis and crusted lips. The skin lesions were erythematous macules and papules of varying size with dark brown centres bilaterally. Nikolsky’s sign was positive. Systemic examination was unremarkable. Laboratory investigations revealed total leucocyte count 12300/mm³, DLC P=77, L=23, E=0, B=0, ESR 25 mm at the end of first hour. C-reactive protein 6 IU/L, serum sodium 135 meq/L, serum potassium 5.6 meq/L, FBS 110 mg/dl, RA factor -ve, HIV non reactive and X-ray chest normal. Based on history of allopurinol exposure, clinical examination and laboratory investigations, a diagnosis of SJS was made. Allopurinol was withdrawn immediately and the patient was put on parenteral antibiotics, corticosteroids, IV fluids, nutritional support, oral and ocular care. The skin lesions


Corresponding Author:
Dr NS Neki. Professor, Junior Residents, Department of Medicine, Government Medical College/GND Hospital, Amritsar (India)-143001. Email: drneki123@gmail.com
The incidence of allopurinol-induced SJS has increased due to its increased use and dosage. Other cases of SJS include infections. The pathogenesis of SJS is poorly understood. But the hypothesis is that drug-induced hypersensitivity reaction causes keratinocyte death, occurring as a result of role played by CD8+ cytotoxic T cells, fatty acid synthetase (FAS) and fatty acid synthetase ligand (FASL). This ultimately results in epidermal sloughing and detachment. Apoptosis also plays a role in keratinocyte cell death. The onset of symptoms following initial drug administration varies from few hours to 3 weeks but symptoms reappear after rechallenge within 48 hours or less in an acquired immune response. Once the diagnosis is made, the offending drug must be stopped immediately in order to reduce mortality. Higher antibiotics are used in SJS patients in view of dreadful complications of sepsis and lung involvement. Successful therapeutic management includes parenteral administration of antibodies, cyclosporins, corticoids, N acetylcysteine, plasmapheresis thalidomide, anti-TNF alpha, pentoxyfylline, IO IV Immunoglobulins although none of the pharmacological agent has conclusively been shown to be beneficial as treatment modality.

CONCLUSION

SJS is a rare, serious systemic reaction. Early identification and withdrawal of the offending drug improves the prognosis of SJS, especially after exposure to drug. Efficient pharmacovigilence is the need of hour so that occurrence of adverse drug reactions can be reduced and prevented. For this, proper history must be taken before prescribing drugs like antibiotics, anticonvulsants, NSAIDs, pyrazoles and anti-rheumatic drugs. Reporting of such events is encouraged.

END NOTE

Author Information

1. NS Neki, Professor, Junior Residents, Department of Medicine, Government Medical College/GND Hospital, Amritsar (India)-143001

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