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Available online at: http://www.iajps.com *A Case Report*

DEATH DUE TO STEVENS JOHNSON SYNDROME; A RARE CASE REPORT

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Abstract:

Stevens Johnson's syndrome is a life threatening drug induced hypersensitivity reaction. The drugs that cause SJS commonly are antibacterials (sulfonamides), anticonvulsants (phenytoin, phenobarbital, and carbamazepine), non-steroidal anti-inflammatory drugs (oxicam derivatives) and oxide inhibitors (allopurinol).Clinical symptoms are urticarial skin eruptions, arthralgia or arthritis, lymphadenopathy and fever.Treatment of Stevens Johnsons Syndrome includes systemic steroids, cyclosporine, intravenous immunoglobulin's and supportive therapy.A male patient of 58 years was admitted in cardiology unit with chief complaints of skin reactions (Erythema andrashes) all over the body and oral erosions. The patient was diagnosed to have ***drug induced stevens Johnson's syndrome****andthe suspected drug was amoxicillin potassium clavulanate. By using Naranjo scale and WHO-UMC scale it was found to be a probable ADR and it was managed by drug dechallenge, dexamethasone, dew on ultra-lotion, diprovate ointment. During treatment patientdeveloped severe breathlessness and died.Hence there is a need of clinical pharmacy services to detect, manage and prevent adverse drug reactions.*

Key words: *Stevens Johnson's syndrome, amoxicillin+potassium clavulanate, Naranjo scale, Dechallenge.*

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INTRODUCTION:

Stevens Johnson's syndrome or toxic epidermal necrosis are the terms used to describe the life threatening drug induced hypersensitivity reaction characterized by blistering and epidermal sloughing involves skin, mucous membranes of mouth, conjunctivae, genital and perianal area [1,2].

Table: 1 Drugs causing Stevens Johnson syndrome[1]

DRUGS CAUSING STEVENS JOHNSONS SYNDROME
Allopurinol
Antiretrovirals, for example nevirapine
Co-trimoxazole
Carbamazepine
Dapsone
Gold salts
Leflunomide
Lamotrigine
NSAIDs, for example meloxicam, diclofenac
Penicillins, for example amoxicillin, ampicillin
Phenylbutazone
Phenolphthalein
Phenobarbitone
Phenytoin
Sulphasalazine
Sulphadiazine
Tetracyclines, for example doxycycline

It was firstly described in 1922 by A.M. Stevens and Frank C. Johnson.[8]The overall incidence of Stevens Johnsons syndrome is 1 to 6 cases per million annually. Males are at higher risk than females for Stevens-Johnson syndrome.[9]The condition is fatal in 5% of treated cases and in 15% of untreated cases.[6]

Stevens Johnson syndrome is an Arthus or type III reaction predominantly by IgG,Mechanism involves the generation of antigen–antibody complexes and subsequently fixesthe complement. The complexes are deposited in the vascular endothelium, where a destructive inflammatory response called serum sickness occurs. The signs and symptoms of serum sickness include urticarial skin eruptions, arthralgia or arthritis, lymphadenopathy, and fever[4]. Pathophysiology of Stevens Johnsons Syndrome involves epidermolysis which is due to apoptosis of keratinocyte cell. These series of biochemical reactions leads to changes in cell and death of cell. The cytotoxic T-cell lymphocytes found in Toxic epidermal necrolysis patients' blister fluid is believed to induce a cascade of intracellular enzymes that results in a rapid, triggered cell death.[5]

Clinical symptoms and Treatment:

General symptoms of Stevens Johnsons Syndrome include fever and malaise, dermatologic were Skin lesions, ENT were Oral lesion and pain in throat, gastrointestinal were oesophageal erosions, anorectal lesions, vomitings, musculoskeletal were myalgia, neurologic were headache, ophthalmic were conjunctivitis, lacrimation and lesions in eye, reproductive were Genital ulcer, respiratory were Cough and respiratory tract infections.[9]Treatment of Stevens Johnsons Syndrome includes systemic steroids,cyclosporine,intravenous immunoglobulins and supportive therapy [1].

CASE REPORT:

A male patient of 58 years was admitted in cardiology department with the chief complaints of shortness of breath and chest discomfort which is retrosternal and burning type. On 1st day of admission vitals are found to be normal and treadmill test was positive his coronary angiogram report advises coronary artery bypass grafting surgery. On 2nd day of admission vitals are normal and sinus rhythm was shown by ECG. On 3rd day of admission patient was found to be little hypotensive (100/60 mm of Hg); pulse rate: 106bpm; respiratory system examination shows presence of crypts; Laboratory reports shows abnormal values of haemoglobin (8.6g/dl), serum sodium (130 m mol/L), serum potassium (3.2 m mol/L); 2D Echo shows post op CABG,RWMA present, mild LV dysfunction, trivial MT/TR, No PAH, no PE and clots.On 4th day of admission vitals are normal except pulse rate (127bpm); ECG shows sinus tachycardia, short PR interval, inferior myocardial infarction with posterior extension, Laboratory reports shows abnormal values of haemoglobin (6.3 g/dl), serum sodium (124 m mol/L). On 5th day of admission vitals were found to be normal except pulse rate (134bpm); ECG shows sinus tachycardia, T-wave abnormality.

On 6th day of admission vitals are normal except blood pressure (150/90); serum sodium (126 m mol/L), serum potassium (2.8 m mol/L); ECG shows sinus tachycardia, left atrial enlargement, T-wave abnormality, possible lateral ischemia. On 7th day of admission vitals are normal except pulse rate (110bpm); ECG shows abnormal junctional ST depression and nonspecific T-wave abnormality; Laboratory reports shows abnormal values of haemoglobin (10.2g/dl). On 8thand 9thday of admission vitals are normal and2D Echo shows post op CABG,RWMA present, hypokinetic inferior and posterolateral wall, mild LV dysfunction, trivial MT/TR, No PAH, no PE and clots.

Based on subjective and objective evidence patient was diagnosed to have CORONARY ARTERY DISEASE; UNSTABLE ANGINA.

Treatment includes Tab.Isosorbide mononitrate 40mg at bed time, Tab.atorvastatin 40mg OD, Inj.Cetriaxone sulbactum 1.5g OD, Inj. Ofloxacin 200mg BD, T.pantoprazole, Tab. Paracetamol 650mg OD, Tab.Clopidogrel+aspirin 75mg+75mg OD,Syp.Potassium chloride, Nebulization: Levosalbutamol + Ipratropiumbromide + Budesonide - 2.5mg + 500mcg + 200mcg, Coronary artery bypass grafting surgery on 3rd day and blood transfusion on 5th day of admission. On 9th day patient was advised for discharge and discharge medication includes Tab.Clavix OD (Clopidogrel-75mg), Tab.Atorvas OD (Atorvastatin-40mg), Tab. Rantac OD (Ranitidine-150mg), Tab. Augmentin BD (Amoxicillin + potassium clavulanate-500mg+125mg), Tab.Linozid OD (Linezolid 600mg).

By using these medications within a week patient developed skin reactions (Erythema and rashes) all over the body and oralerosions and admitted into the hospital.The patient was diagnosed to have **drug induced stevens johnsons syndrome**.On day1 temperature was 101^0F. On day 2 patient was hypotensive(90/70 mm of Hg) and pulse rate was found to be 125bpm. On day 3 vitals are normal.

Causality assessment of ADR:

S.No	Suspected ADR	Suspected drug	Naranjo scale	WHO-UMC
1	SJS	Tab.Augmentin (Amoxicillin 500mg+Potassium Clavulanate 125mg)	Probable	Probable

Dechallenge of suspected drug was done.

Management of ADR:

On day of admission for ADR management drugs prescribed to the patient was dexamethasone 100mg OD and Supportive treatment includes dew on ultra-lotion, diprovate ointment. On second day same therapy was continued and on 3rd day methyl prednisolone at the dose of 1g ODwas administered. On 4th day same therapy was continued.

At evening the patient developed severe breathlessness and immediately shifted to ICU and oxygenation was done. At night patient was died.

Fig 1: Shows Oral Erosions

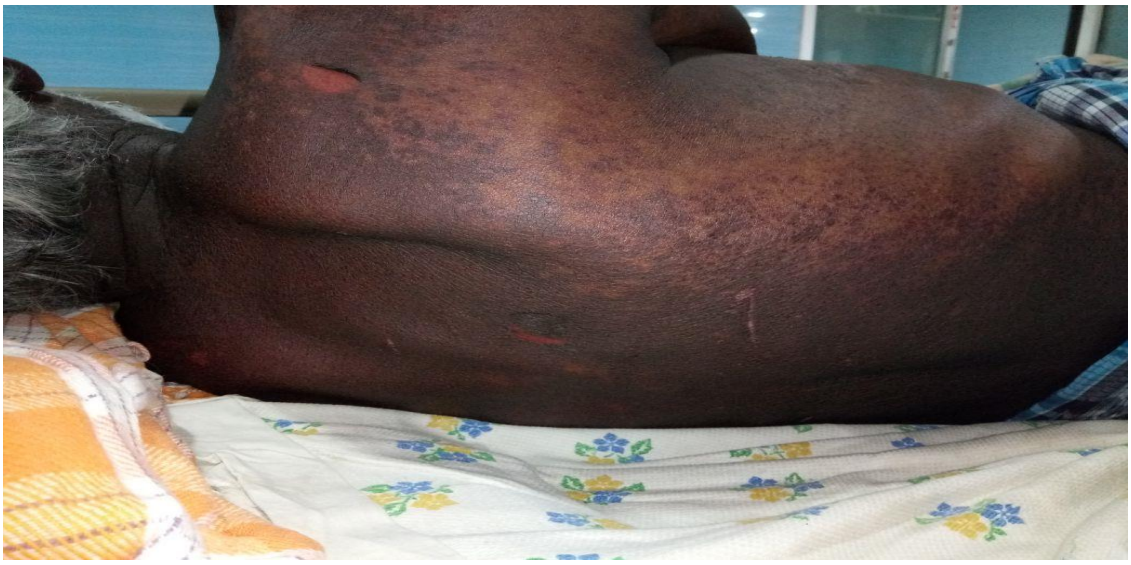

Fig 2: Shows Erythema and Skin Eruptions on Posterior Side

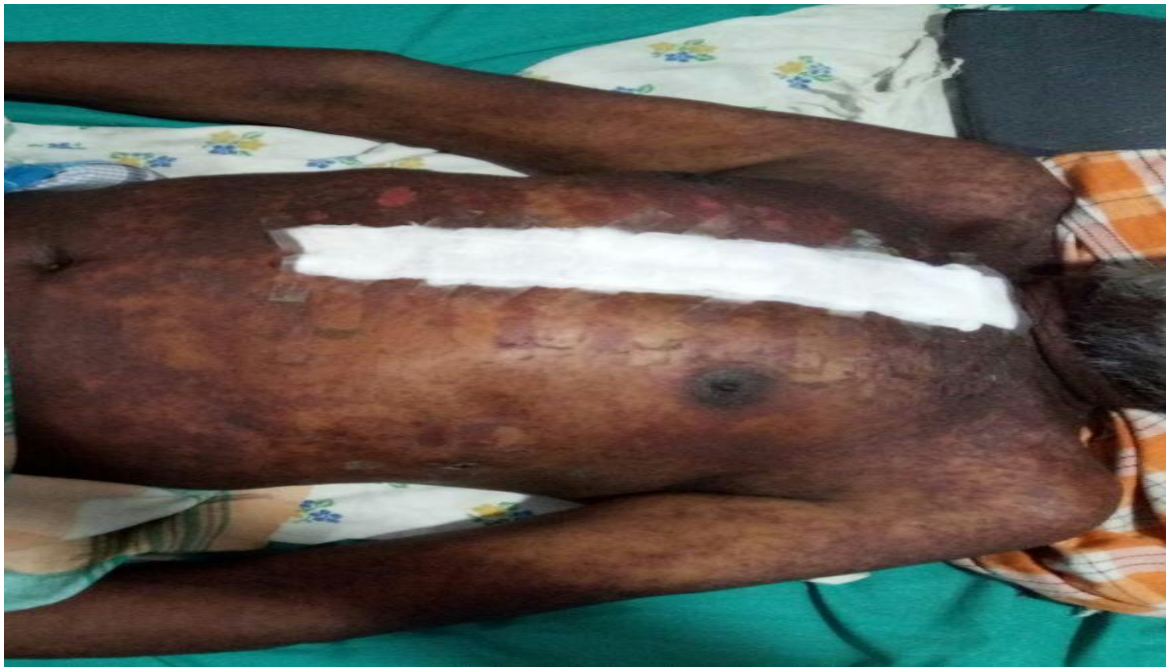

Fig 3: Shows Erythema and Skin Eruptions on Anterior Side

DISCUSSION:

The suspected drug for the occurrence of Stevens Johnson syndrome was amoxicillin which is penicillin like drug acts by inhibiting the biosynthesis of cell wall mucopeptide. Common adverse effects includes Diarrhea, nausea, vomiting, headache, vulvovaginitis and serious adverse effects includes Erythema multiforme, Stevens-Johnson syndrome(SJS), Toxic epidermal necrolysis (TEN) [9]. Erythema multiforme is an acute syndrome mainly characterized by variety of skin lesions. Lesions may typically starts as a macro popular eruptions that may be accompanied by bullous lesions that break down into erosions on mucous membranes, Thus more extensive form of this disease is called multiforme major Stevens Johnsons Syndrome, If the skin lesions resembling 3rd degree burns are called toxic epidermal necrosis (Mortality rate - 40%) [3]. According to A.P. Shah, H. Xu, P.J. Sime and D.R. Trawick, Respiratory pathology in Stevens Johnsons Syndrome occurs in up to 25–45% of cases, and may include mucosal involvement of the upper airway and respiratory tree, pneumonia, pneumothorax and mediastinal emphysema. Up to 30% of cases are preceded by an atypical pneumonia and an association with Mycoplasma pneumonia infection is well established. Persistent pulmonary sequelae

following recovery from Stevens Johnsons Syndrome are rare. However, the incidence of complications may be higher, and the consequences more severe in toxic epidermal necrolysis, the more extensive form of Stevens Johnson Syndrome.[7]

CONCLUSION:

Stevens Johnson Syndrome is a rare drug induced hypersensitivity reaction. In severe cases it may lead to respiratory failure and sometimes fatal. Hence there is a need of clinical pharmacy services to detect, manage and prevent adverse drug reaction.

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