### ACYCLOVIR INDUCED STEVEN JOHNSON SYNDROME

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**ABSTRACT:** Acyclovir, anti-viral drug rarely causes Stevens-Johnson syndrome (SJS). Steven Johnson syndrome is a rare, life threatening disorder characterized by skin condition with bullous formation, ocular lesions, genital and anal lesions/ulcers. It's usually a reaction to a medication or an infection. Often Steven Johnson syndrome begins with flu-like symptoms followed by a painful red or purplish rash that spreads and blisters. Then the top layer of the affected skin dies and sheds. This case report is about a 40 year old male patient who came to the medicine outpatient department with blisters on palms and soles and characteristic hemorrhagic crusting of mouth and lips. Initial diagnosis of Steven Johnson Syndrome was made and treated with steroids. Eruption usually healed without sequelae.

**KEYWORDS:** Acyclovir, neurological manifestation, Stevens-Johnson syndrome.

**INTRODUCTION:** Acyclovir is used widely in the treatment of different/iral infections. Stevens-Johnsonsyndrome (SJS), life-threatening skin condition is a very rare side effect of Acyclovir,.<sup>(1)</sup> The syndrome is thought to be a hypersensitivity complex that affects the skin and the mucous membrane.<sup>(2)</sup> Incidence ranges from 1.2 to 6 cases per million per year; the condition is fatal in 5% of treated cases and in 15% of untreated cases.<sup>(3)</sup> The condition is more common in adults than in children.

Stevens–Johnson syndrome (SJS) is thought to arise from a disorder of the immune system.<sup>(4)</sup> The immune reaction can be triggered by drugs or infection.<sup>5</sup> Genetic factors are associated with a predisposition to SJS.<sup>(6)</sup> Although Stevens–Johnson Syndrome can be caused by viral infections and malignancies, the main cause in medications.<sup>(5)</sup> The major causative drugs were antimicrobials (37.27%), anti-epileptics (35.73%) and non-steroidal anti-inflammatory drugs (15.93%). Carbamazepine (18.25%), phenytoin (13.37%), fluoroquinolones (8.48%) and paracetamol (6.17%) were most commonly implicated drugs.

It manifests as fever with skin lesions on the trunk, face, neck, andproximal upper extremities including palms and soles sparingrelatively the distal arms and legs. Involvement of buccal, ocular, and genital mucosa is present in more than 90% of patients.<sup>(6)</sup> with characteristic hemorrhagic crusting of mouth and lips.<sup>(7)</sup> Eruption usually heals without sequelae. We present a caseof SJS where the patient had blisters on palms and soles and haemorrhagic mucocutaneous lesions

**CASE REPORT:** A 40-year-old nonalcoholic male was diagnosed withHerpes and prescribed oral vitamins, and oral Acyclovir 400 mg twice daily for 10 days. After 2 days, his developed severe burning sensation and itching all over the body followed by dizziness, confusion. Later he complained of shivering and palpitation, blisters over soles and palms (fig I, II), redness over the faceand neck. He developed fever, ulcers around lips and oral mucosa, difficulty on swallowing

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and burning sensation during micturition. He then consulted a general physician, who withdrew Acyclovir. He had no history of neurological disease.







On physical examination patient was disoriented, his vitals were: temp 102°F, Pulse rate 86 beats/min, Respiratory rate 22/min, Blood pressure 90/60 mm/Hg. On oral examination lips were swollen, cracked, crusted while bleeding was evident (fig III). Intra oral examination reveals mucosal desquamation. Patient's laboratory Investigations showed white blood cell count 7.2 x  $10/\mu$ L, Neutrophils count 82.5%.Patient's renal profile and serum electrolytes were normal.

He was diagnosed clinically and managed as a case of SJS with involvement of approximately 3% of total body surface area. He was treated with Deflazocart 6mg oral twice daily for 7 days, pantoprazole 40 mg once a day, daily for 7 days. Deflazocart was tapered over the next14 days. Pain from oral lesions may be lessened by rinsing with viscous lidocaine the patient recovered without any sequelae in 3 weeks.

**DISCUSSION:** The clinical presentation of this patient with palmar and plantar blisters, hemorrhagic encrustation of lips and oral mucous membrane, and involving 3% of total body surface area following acyclovir use favours the clinical diagnosis of SJS. These preclude the possibility of toxic epidermal necrolysis, staphylococcal scalded skin syndrome, erythema multiforme, and generalized fixed drug eruption. Patient had taken oral vitamins which are not

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related with this type of mucocutaneous lesion and developed SJS within 2days of intake of Acyclovir. On withdrawal of Acyclovir and subsequent treatment, patient had improved remarkably without any sequelae. As per World Health Organization-Uppsala Monitoring Centre (WHO-UMC) standardized case causality assessment criteria.<sup>(8)</sup> and Naranjo's algorithm.<sup>(9)</sup> this event can be considered as a probable reaction due to Acyclovir(Naranjo's score-6).The present study shows that Acyclovir induced SJS may not follow the classical sequence of SJS, rather it may present with blisters of palms and soles followed by rapid mucocutaneous manifestation.

**CONCLUSION:** Stevens-Johnson syndrome is a potentially fatal multi organ disease with a strong etiologic link to some medications. As Acyclovir is a widely used drug, physicians should be aware with this adverse reaction for early detection and intervention. The patient should also be encouraged to report any abnormal manifestation following use of Acyclovir to prevent such potentially life threatening condition. Affected patients and their first-degree relatives should be instructed to avoid any identified drugs or chemicals that may be responsible.

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