

THE PRICE OF A PILL: STEVENS-JOHNSON SYNDROME AFTER SELF-MEDICATION OF FLUOROQUINOLONE

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ABSTRACT

Stevens-Johnson Syndrome (SJS) is a rare and potentially life-threatening mucocutaneous reaction, usually caused by drugs (such as NSAIDs, antibiotics and antiepileptics). In the described case study, a medical house officer who was 28 years old used moxifloxacin and over-the-counter cough medicine, another cause of acute SJS. The patient had progressive worsening of the symptoms including involvement of mucosa, targetoid skin lesions, odynophagia, oliguria, and systemic signs despite the symptom treatment and multiple courses of antimicrobials. The laboratory work revealed elevated levels of hepatic enzymes, neutrophilic leukocytosis, and various markers of inflammations, which suggests multiorgan involvement. Early SJS was diagnosed based on clinical manifestations and all the drugs suspected to have caused it were discontinued immediately. Following commencement of high-dose intravenous dexamethasone the patient improved rapidly and ultimately discharged with a tapering steroid dose. The case highlights the need to provide clinical focus to prescription and self-medication practices and the intense association of fluoroquinolones and SJS, especially moxifloxacin. Recovery is based on early diagnosis of mucocutaneous symptoms, prompt elimination of the offending substance and immediate beginning of immunomodulatory therapy. The case also illustrates the role that corticosteroids along with other interventions such as TNF- alpha blockers are taking in controlling SJS. In an attempt to minimize the morbidity and mortality associated with such a severe drug reaction, knowledge, diagnostic handling, and antibiotic vigilance rate higher as ways to prevent such reactions.

Keywords: Stevens-Johnson Syndrome, Self medication, Diagnostics.

CASE INTRODUCTION & BACKGROUND

Self medication is an emerging menace in the Pakistani population and the factors fueling it include easy availability of over-the-counter medications, non-implementation of controls, healthcare infrastructure shortage and poverty amongst a large portion of the population (Bhatti et al, 2023). People especially those people in the rural regions tend to self-medicate because most of them lack qualified medical practitioners and because medical consultation is expensive. Medical stores and drug stores sell antibiotics, pain medications, sedatives among other drugs in large quantities and without prescription, which is leading to the development of drug resistance, unwanted events and complications, because of the wrong dose or wrong use of the drug. The fact that the general population lacks any knowledge of the possible side-effects of medications, further increases the problem and in many cases, serious illnesses will not be diagnosed in time (Bhatti & Ghufra, 2020). Moreover, the use of misinformation and beliefs comes in the form of cultural beliefs and misinformation that travels within the community or other social networking sites that also encourage abuse of medicines. Effects of this practice are more morbidity, drug addiction, and additional pressure on the already overloaded health system (Bhatti et al., 2024).

The dermatological disorder known as Stevens-Johnson syndrome (SJS) is characterized by severe exfoliative reactions that mostly affect the skin and mucous membranes (1). Both SJS and TEN are

classified on the same disease continuum and are characterized by extensive necrosis and epidermis detachment. SJS is defined by skin detachment of less than 10% of the body surface (BSA), TEN involves skin detachment of more than 30% of the BSA, and SJS/TEN overlap describes patients with skin detachment of 10 to 30% of the BSA (2). SJS is primarily caused by drugs and is linked to high rates of morbidity and death (3). Global data indicates that the incidence of SJS is between 0.4 and 1.2 per million patient-years (4). Antiepileptics, antibiotics, and NSAIDs are the most often implicated medications that induce SJS/TEN, however almost any medication known to man—including non-allopathic medications—has the potential to cause this reaction (5). Furthermore, underreporting and delayed diagnosis of SJS continue to be serious public health issues in nations like Pakistan that lack access to dermatological knowledge or diagnostic resources (6).

CASE PRESENTATION

A 28-year-old male medical house officer presented with an acute febrile illness with sore throat. Initially tried to manage his symptoms conservatively without medication. But when he was unable to control his symptoms, he took 400mg of moxifloxacin and over-the-counter cough syrup, taking both medications for three consecutive days. Despite this, his fever persisted intermittently. He sent laboratory investigations including complete blood count (CBC) and dengue serology returned within normal limits.

TABLE 01 DENGUE SEROLOGY

Test	Result	Reference Range
Malaria Antibodies (IgG/IgM)	Negative	Negative

TABLE 02 COMPLETE BLOOD COUNT TEST RESULTS

Parameter	Result	Reference Range
WBC Count	6,450	4,000 - 11,000 /mm ³
RBC Count	4.45 ↓	4.5 - 6.0 mil/mm ³
Hemoglobin	13.5	13.0 - 17.0 g/dL
Hematocrit	40	40 - 50 %
MCV	89	80 - 95 fL
MCH	30	27 - 31 pg
MCHC	34	32 - 36 g/dL
RDW-CV	14	11 - 16 %
Platelet Count	164,000	140,000 - 425,000 /mm ³

On the fourth day, the patient developed diffuse erythema and hyperemia of the oral mucosa, prompting a consultation with an internal

medicine specialist. He was tested there for EBV which come out negative.

TABLE 03 MONOSPOT TEST RESULTS

Test Name	Result	Reference Range	Date
Monospot (EBV)	1.5	Negative: <20 Borderline: 20-25 Positive: >25	23-Nov-24

He was administered intramuscular dexamethasone 4 mg twice daily and 1g of intravenous ceftriaxone for 2 days. Despite treatment, his symptoms remained unchanged. The following day, he was evaluated by an otolaryngologist due to his sore throat who prescribed 400mg of oral azithromycin bid and 4mg of oral corticosteroids TID.

grade fever, intensified sore throat, and severe odynophagia, rendering him unable to speak. Pain was rated 10/10 on the visual analog scale. He was admitted and started on intravenous meropenem and intravenous azithromycin by the internal medicine team. His CBC report at this time showed neutrophilia and lymphopenia.

The patient's condition worsened the next day. He presented to the emergency department with high-

TABLE 04 CBC TEST RESULTS

Parameter	Result	Reference Range
WBC Count	17,780	4,000 - 11,000 /mm ³
RBC Count	4.55	4.5 - 6.0 mil/mm ³
Hemoglobin	13.8	13.0 - 17.0 g/dL
Hematocrit	40	40 - 50 %
MCV	88	80 - 95 fL
MCH	30	27 - 31 pg
MCHC	35	32 - 36 g/dL
RDW-CV	14	11 - 16 %
Platelet Count	280,000	140,000 - 425,000 /mm ³

TABLE 05 CELL TYPE TEST RESULTS

Cell Type	Percentage
Neutrophils	90%
Lymphocytes	6%
Monocytes	3%
Eosinophils	1%

On hospital day two, he developed a severe cutaneous drug reaction characterized by targetoid lesions—erythematous to violaceous macules with central dusky zones—initially on his back, later on

face with red conjunctive and lips skin start desquamating suggesting early Stevens-Johnson Syndrome (SJS).

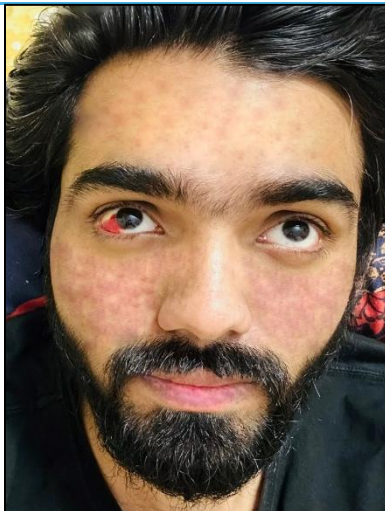


FIGURE 01 PATIENT WITH VISIBLE EFFECTS OF STEVENS-JOHNSON SYNDROME



FIGURE 02 DURING TREATMENT RESULTS

He concurrently developed burning micturition, oliguria, and urethral pain independent of urination.

TABLE 06 MULTIPLE URINE TEST RESULTS

Specimen	Urine for Culture and Sensitivity
Pus Cells	4-6 / HPF
Quantitative Culture	No growth after 48 hours at 37°C
Colony Count	Nil
Comments	Please correlate clinically

Examination	Results	Reference Ranges
Physical Examination		
Color	Yellow	
Appearance	Transparent	Clear, Transparent
Chemical Examination		
Specific Gravity	1.020	1.003 - 1.025

pH	5.0	4.5 – 7.5
Protein	Negative	Negative
Glucose	Negative	Negative
Ketone	Negative	Negative
Bilirubin	Negative	Negative
Nitrite	Negative	Negative
Hemoglobin	Negative	Negative
Urobilinogen	Normal	Normal: 3-25 $\mu\text{mol/L}$ Increased: >25 $\mu\text{mol/L}$
Microscopic Examination		
W.B.C./HPF	1-2	0 – 5
R.B.C./HPF	1-2	Nil
Epithelial Cells	NIL	0 – 10
Crystals	NIL	Nil
Casts/LPF	Granular cast 2-4	Granular cast 4-6

Additional symptoms included intractable vomiting, subconjunctival hemorrhages, and constipation, with the anal examination reported as normal.

TABLE 07 ADDITIONAL TESTS RESULTS 01

Test	Now	Prev	Range
aPTT (s)	24	26	24-35
PT (s)	9.8	9.5	9.5-14
INR	0.9	0.9	–

Comments: INR is only helpful in patients on long-term anticoagulants. Therapeutic range: 2.0-3.0. For artificial valves: 2.5-3.5. INR >5.5 = high bleeding risk (WHO). **Note:** Test performed on "Thrombolyzer", Behnk Elektronik GmbH & Co., Germany. Given the rapid mucocutaneous and systemic involvement.

TABLE 08 ADDITIONAL TESTS RESULTS 02

Test	Result
Anti AMA-M2	0.1
Anti SP-100	0.1
Anti LKM1	0.1
Anti Gp-210	0.2
Anti LC1	0.1
SLA	0.2

All antimicrobial agents were immediately discontinued, and the patient was started on intravenous dexamethasone 8 mg three times daily.

TABLE 09 MEDICATION FOLLOWED

Test	Result	Unit	Reference Range	Interpretation
Total Bilirubin	0.5	mg/dL	0.1 – 1.1	Normal
SGPT (ALT)	268 ↑	U/L	5 – 55	Critical High
SGOT (AST)	89 ↑	U/L	9 – 40	Abnormal
Alkaline Phosphatase	87	U/L	30 – 115	Normal
Gamma GT	170 ↑	U/L	Male: <55	Critical High
Total Protein	6.4	g/dL	6.0 – 8.7	Normal

Over the subsequent 48 hours, the patient showed significant clinical improvement. On hospital day four, he was discharged in stable condition on a tapering course of oral dexamethasone for three weeks.

DISCUSSION

The severe immune-mediated mucocutaneous reaction known as Stevens-Johnson Syndrome (SJS) is mainly brought on by drugs. Although less prevalent than sulfonamides or antiepileptics, fluoroquinolones, such as the moxifloxacin this patient was taking, have been linked more and more to SJS. Given their widespread use in respiratory and urinary infections, fluoroquinolones were found to be responsible for approximately 4% of antibiotic-related SJS/TEN cases in a multi-country systematic study (7) (8).

This patient's organ involvement—high liver enzymes and renal signs—reflects systemic issues that are frequently observed in SJS/TEN. GGT and ALT/AST elevations are well-documented and may indicate hepatic stress in severe medication responses. Granular casts, oliguria, and dysuria suggest that there may be renal involvement, supporting new research showing multiorgan involvement in SJS/TEN (9).

New management guidelines emphasize the importance of early drug discontinuation. After stopping all antibiotics and switching to high-dose intravenous dexamethasone, our patient became well. Supportive care in conjunction with immunomodulatory therapy, such as systemic corticosteroids and ciclosporin, is becoming more and more recommended, despite the lack of high-quality trials.

Although the data is still weak, recent developments also indicate that adjuvant therapies, such as TNF- α inhibitors (e.g., etanercept or adalimumab), can hasten re-epithelialization and shorten hospital stays. For severe or refractory cases, such combined therapies are advised by a global recommendation for 2024 (10).

Conclusion

This example illustrates how quickly and severely medication-induced SJS can develop in a young adult after being exposed to fluoroquinolones. Important lessons consist of:

Recovery is greatly impacted by early detection of prodromal mucocutaneous symptoms, prompt discontinuation of probable medications, and early beginning of supportive plus immunomodulatory therapy. The positive outcome this patient experienced following systemic steroids highlights their continued use in acute care, in accordance with current consensus and tactics recommended by guidelines (11). Since SJS is often accompanied by hepatic, renal, and coagulation dysfunction, multi-organ examination is essential. With improved clinical trial confirmation, future therapy procedures may benefit from incorporating biologics (TNF- α inhibitors) and new diagnostic-scoring systems. In order to stop more instances and improve results, clinical vigilance is crucial, particularly with regard to the use of fluoroquinolones, self-medication, and antibiotic stewardship.

REFERENCES

1. Fakoya AOJ, Omenyi P, Anthony P, Anthony F, Etti P, Otohinoyi DA, et al. Stevens- Johnson Syndrome and Toxic Epidermal Necrolysis; Extensive Review of Reports of Drug- Induced Etiologies, and Possible Therapeutic Modalities. Open Access Macedonian Journal of Medical Sciences. 2018;6(4):730-8.
2. Meledathu S, Gordon M, Thornton M, Ashinoff R. Management of Stevens-Johnson Syndrome/Toxic Epidermal Necrolysis: A Case Report and Literature Review. Journal of Drugs in Dermatology: JDD. 2023;22(11):e24-e8.
3. Chang W-C, Abe R, Anderson P, Anderson W, Ardern-Jones MR, Beachkofsky TM, et al. SJS/TEN 2019: from science to translation. Journal of dermatological science. 2020;98(1):2-12.
4. Parida JR, Tripathy SR. Stevens-Johnson syndrome/toxic epidermal necrolysis spectrum for the rheumatologist. Indian Journal of Rheumatology. 2019;14(Suppl 1):S67-S75.
5. Downey A, Jackson C, Harun N, Cooper A. Toxic epidermal necrolysis: review of pathogenesis and management. Journal of the American Academy of Dermatology. 2012;66(6):995-1003.
6. Hafeez H, Ahmed T, Anwar P. Stevens-Johnson syndrome and toxic epidermal necrolysis:

A review. Pakistan Journal of Medical Sciences. 2021;37(3):285-91.

7. Liu J, Zhou M, Li T, Xu T. Adalimumab combination with corticosteroid therapy for Stevens-Johnson syndrome/toxic epidermal necrolysis. Archives of Dermatological Research. 2025;317(1):694.

8. Lee EY, Knox C, Phillips EJ. Worldwide prevalence of antibiotic-associated Stevens-Johnson syndrome and toxic epidermal necrolysis: a systematic review and meta-analysis. JAMA dermatology. 2023;159(4):384-92.

9. Shah H, Parisi R, Mukherjee E, Phillips EJ, Dodiuk-Gad RP. Update on Stevens-Johnson syndrome and toxic epidermal necrolysis: diagnosis and management. American Journal of Clinical Dermatology. 2024;25(6):891-908.

10. Murillo-Casas AD, Zwiener R, Giavina-Bianchi P, Pardo-Manrique V, Villarreal-González R, Monge-Ortega OP, et al. Latin American guidelines for the diagnosis and treatment of Stevens-Johnson syndrome and toxic epidermal necrolysis. World Allergy Organization Journal. 2025;18(4):101046.

11. Hotha PP, Naidu CDM, Zachariah NE. Stevens Johnson Syndrome Associated with Fluoroquinolones: A Case Series. Journal of Pharmaceutical Care. 2022.

12. Bhatti, O. S., Ghufuran, M. A., & Shah, A. U. (2023). Transforming adversity into opportunity: Assessing user satisfaction in hospital transformation in lieu of a pandemic through the multi-corridor expansion model for epidemic management and environmental design enhancement. Nakhara: Journal of Environmental Design and Planning, 22(2), 308-308.

13. Bhatti, O. S. (2020). Healthcare Infrastructure and COVID-19: A Case for Isolation and Quarantine Facilities Optimization in the Context of Pakistan. J Res Archit Plan, 29, 36-45.

14. Bhatti, O. S., Mujahid, S., & Mughal, N. I. (2024). Unveiling Climate Consciousness: Assessing Perceptions based on Social Media and Mass Communication Strategies among Balochistan University Students. Journal of Development and Social Sciences, 5(2), 33-44.