International Journal of Contemporary Pediatrics
Salwa Z et al. Int J Contemp Pediatr. 2024 Nov;11(11):1644-1650
http://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291

DOI: https://dx.doi.org/10.18203/2349-3291.ijcp20243093

Case Report

Carbamazepine triggered Stevens-Johnson syndrome with gastrointestinal and renal system involvement: a case report

Zeena Salwa[1]*, Tanha Tazmin[2]

[1]Department of Paediatrics and Child Development Centre, Square Hospitals Limited, Dhaka, Bangladesh
[2]Department of Child Development Centre, Square Hospitals Limited, Dhaka, Bangladesh

Received: 29 August 2024
Revised: 02 October 2024
Accepted: 08 October 2024

***Correspondence:**
Dr. Zeena Salwa,
E-mail: zsalwa100@gmail.com

ABSTRACT

Stevens-Johnson syndrome/toxic epidermal necrolysis (SJS/TEN) is a rare but life-threatening condition characterized by severe skin and mucosal involvement. Recently, we encountered four cases of Stevens-Johnson syndrome (SJS), three of which were associated with carbamazepine use. This report focuses on one of these cases to highlight the risks associated with this medication. Carbamazepine (CBZ) is recognized as one of its common triggers. This report discusses one of these cases involving an eight-year-old boy weighing 23.3 kg, with a one-year history of epilepsy initially managed with oral sodium valproate, experienced frequent seizures. Consequently, sodium valproate was discontinued, and carbamazepine was started at a dose of 150 mg twice daily. After one week, the dosage was increased to 150 mg in the morning and 300 mg at night. Forty-two days post-initiation of carbamazepine, the patient exhibited fever, cough, and malaise, followed by skin rashes affecting the oral mucosa and eyes three days later. He then developed profuse oral secretions, bloody diarrhea, microscopic hematuria, and albuminuria. The administration of carbamazepine was immediately halted, and the patient was managed in the Pediatric Intensive Care Unit (PICU) by a multidisciplinary team. He achieved full recovery after 35 days. The objective of this case report is to raise awareness of carbamazepine as a potential trigger for SJS, differentiate between Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN), and discuss the risk factors, differential diagnosis, and management of SJS/TEN, which often requires a multidisciplinary approach in a pediatric intensive care unit (PICU).

Keywords: Stevens-Johnson syndrome, Toxic epidermal necrolysis, Skin, Mucous membrane, Carbamazepine

INTRODUCTION

Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are very rare, serious vesiculobullous disorder of the skin and mucous membranes. It's usually a reaction to medication that starts with flu-like symptoms, followed by a painful rash that spreads and blisters. Then the top layer of affected skin died, sheds and begins to heal after several days. The hallmark symptom of SJS/TEN is a blistering rash that involves two or more mucous membranes.[1] SJS/TEN are two ends of the same spectrum of disease, distinct from other drug eruptions.[2-4]

SJS is the less severe form, although still a medical emergency, affecting less than 10% of the body surface area, while TEN is severe, life-threatening disorder, affecting more than 30% of the BSA, when 10-30% is affected, the term SJS/TEN overlap is used which is shown in Figure 1 and Table 1.[5] The extent of skin involvement is a major prognostic factor. It should be emphasized that only necrotic skin, which is already detached (e.g. blisters, erosions) or detachable skin (Nikolsky positive) should be included in the evaluation of the extent of skin involvement. Bastuji-Garin et al, proposed classifying patients into three groups according

to the degree of skin detachment.[6] Purpose of this case reporting is to make an awareness about carbamazepine which acts as an offending agent causing Stevens Johnson syndrome (SJS/TEN), make a difference between SJS and toxic epidermal necrolysis (TEN), to discuss about several risk factors and differential diagnosis of SJS/TEN, and as it is a medical emergency, therefore it's management often need a multidisciplinary team approach preferably in pediatric intensive care unit (PICU).

CASE REPORT

The patient is an eight-year-old boy weighing 23.3 kg with a one-year history of epilepsy, initially treated with oral sodium valproate (200 mg twice daily). Due to frequent seizure attacks over the past few months, sodium valproate was discontinued, and carbamazepine was introduced at a dose of 150 mg twice daily for the first seven days. The dose was then increased to 150 mg in the morning and 300 mg at night. Forty-two days after starting carbamazepine, the patient developed a high-grade fever (maximum temperature recorded at 104°F), sore throat, malaise, and seizures. He was admitted to a local hospital, where he was managed with rectal diazepam and parenteral ceftriaxone for a suspected infection. Despite three days of treatment, his fever was persisted, and he developed erythematous rashes on various parts of his body, including the oral mucosa.

On the fourth day of his illness, the patient was transferred to Square Hospitals Limited in Bangladesh. Upon admission, he exhibited widespread mucocutaneous rashes involving the oral mucosa, lips, eyes, and erythematous flat bullae on the trunk, limbs, palms, and soles (Figure 3 and 4). Nikolsky's sign was absent. He also had perianal involvement with flat bullae but no urethral abnormalities. His vital signs included a temperature of 101°F, oxygen saturation of 96% on room air, tachycardia at 162 beats per minute (BPM), a capillary refill time of <2 seconds, and a blood pressure of 100/70 mm Hg. His abdomen was soft, non-tender, with normal bowel sounds, and no abnormalities were found in other systems. There was no history of allergies.

Initial laboratory investigations revealed a white blood cell (WBC) count of 9,570 cells/µl, hemoglobin at 12.5 g/dl, red blood cell (RBC) count of 4.98 M/µl, platelet (PLT) count of 242 K/µl, aspartate aminotransferase (AST) at 78 U/l, alanine aminotransferase (ALT) at 38 U/L, urea at 8.2 mmol/l, creatinine at 0.4 mg/dl, C-reactive protein (CRP) at 82.8 mg/l, albumin at 38 g/l, glycemia at 4.5 mmol/l, serum bicarbonate at 8.1 mmol/l, prothrombin time (PT) of 14.3 seconds, activated partial thromboplastin time (APTT) of 36.9 seconds, sodium at 139 mmol/l, and potassium at 3.7 mmol/l. HIV I & II (Ag and Ab) tests were non-reactive (0.19 U). Immediate management included the withdrawal of carbamazepine and switching to levetiracetam for seizure control, along with crystalloid fluid replacement. Systemic steroids (2 mg/kg/day) were initiated. Broad-spectrum antibiotics, including ceftriaxone, flucloxacillin, and metronidazole, were administered to manage the suspected infection. Orofacial care included petroleum jelly for the lips, mupirocin ointment, triamcinolone acetonide gel for the lips and face, chlorhexidine gluconate mouthwash, and miconazole gel. Eye care involved saline washes, hypromellose+dextran eye drops, fluorometholone eye drops, moxifloxacin eye drops, and ciprofloxacin eye ointment. Skin care included mupirocin ointment, fusicort cream, and Ezex cream applied to the whole body, with special attention to affected areas, including the ears, twice daily. Perianal care was provided with triamcinolone acetonide cream, and urethral care was done through careful catheterization. The patient also received a proton pump inhibitor, amino acid injections, and supplemental nutrition. On the second day of hospitalization (the fifth day after cutaneous symptoms appeared), the patient continued to have a high fever, with worsening oral ulcers on the buccal mucosa, tongue, soft palate, and floor of the mouth, leading to continuous drooling and difficulty swallowing. Hemorrhagic crusting developed on the lips, and ophthalmic examination revealed conjunctivitis with hemorrhagic crusting on the eyelids. The patient also experienced blood-tinged loose stools, and the cutaneous lesions began to slough.

Physical examination revealed a body temperature of 101°F, tachycardia at 126 BPM, blood pressure of 105/70 mm Hg, and an oxygen saturation of 97% on room air. The abdomen was soft, non-distended, and without pain. Further investigations showed a platelet count of 223 K/µl, hyponatremia at 123 mmol/l, hypokalemia at 2.8 mmol/l, hypoalbuminemia at 2.8 g/dl, a low hemoglobin level of 9.7 g/dl, and urine analysis revealed plenty of RBCs and albuminuria. CRP remained elevated at 82.8 mg/l (Tables 2-4). The severity score for toxic epidermal necrolysis was 1 point (tachycardia>120, serum urea <10 mmol/l, and initial detachment <10%), indicating a 3.2% probability of in-hospital mortality.[6] The patient's tachycardia responded well to 20 ml/kg of crystalloid fluid replacement over 30 minutes and 2.0 l of daily fluid compensation. Steroid therapy was continued (2 mg/kg/day) for a total of seven days. The patient also required electrolyte infusion, PRBC transfusion for correction of dyselectrolytemia and anemia, and total parenteral nutrition (TPN) supplementation to maintain daily nutrition. Given the clinical presentation, he was diagnosed with "Carbamazepine-triggered Stevens-Johnson Syndrome with gastrointestinal and renal system involvement."

Over the following days, the patient gradually improved, with a reduction in fever, mucocutaneous lesions, drooling, and bloody stools. By the eighth day after admission, he was afebrile, his eyelids had normalized with mild conjunctival congestion, his lips were mildly swollen with some bleeding from cracked points, and the oral mucosa and tongue appeared pale without bleeding or erosion.

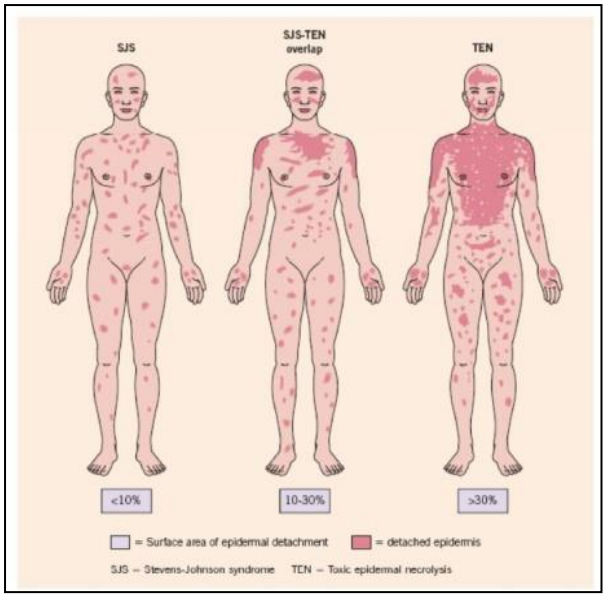

Figure 1: Picture representation of SJS, SJS-TEN overlaps and TEN.

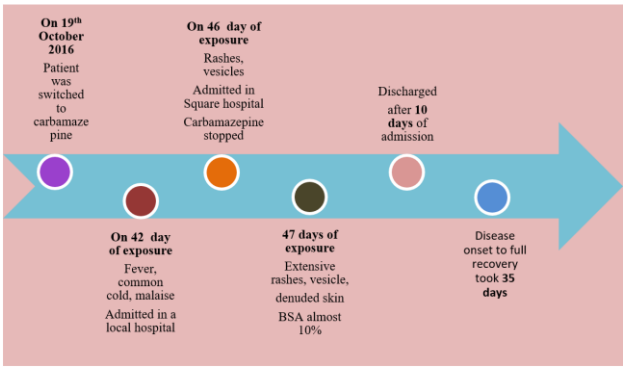

Figure 2: First sign of SJS was seen after 42 days of intake and worst involving <10% BSA after 47 days of taking CBZ and disease onset to full recovery took 35 days.

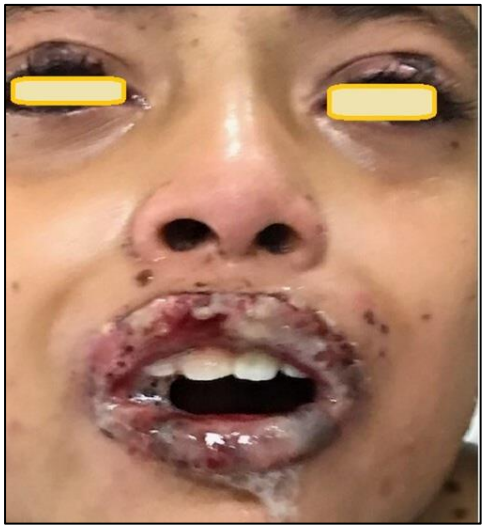

Figure 3: Acute phase of SJS.

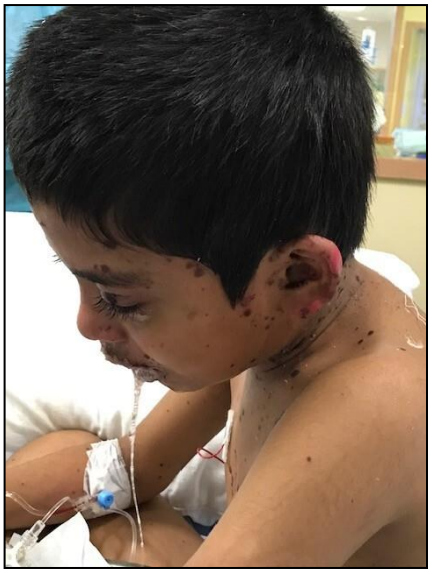

Figure 4: Acute phase of SJS.

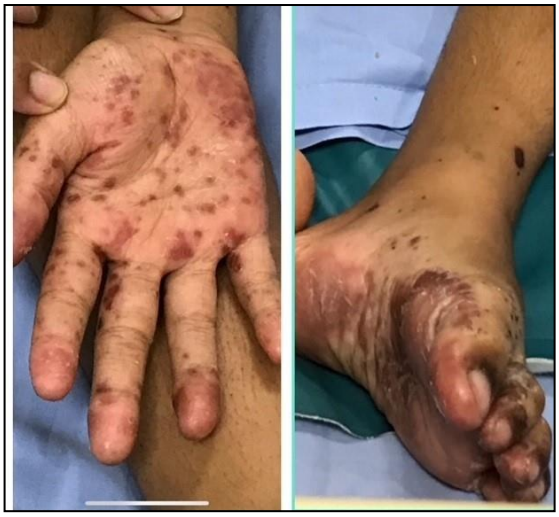

Figure 5: Reepithelialisation phase of SJS.

Figure 6: Complete recovery of SJS.

He tolerated small oral feeds, and his skin lesions had dried, with desquamation of the palms and soles (Figure 5) and healing of the perianal area. The catheter was carefully removed eight days after admission.

The patient was slowly reintroduced to normal feeds and was discharged after ten days of hospitalization with a plan for long-term follow-up. He took total 35 days from disease onset to full recovery (Figure 6). Disease course of the patient is shown in figure 2 by Shrestha O, et al.[7]

Table 1: Classification of SJS and TEN by percentage of skin involvement.

Classification	Body surface area
Stevens- Johsons syndrome	<10%
Overlap SJS/TEN	10-29%
Toxic epidermal necrolysis	>30

Table 2: Blood test reports.

Date	02.12.2016	03.12.2016	08.12.2016
Haemoglobin (gm/dl)	12.5	9.70	12.3
Total WBC count (K/µl)	9.57	5.70	5.53
Neutrophils (%)	71.7	55.1	53.5
Platelets (K/µl)	242	202	241
CRP (mg/l)	82.8		12
Albumin (mg/dl)	2.8		3.8
Creatinine (mg/dl)	0.4		
BUN (mmol/l)	7.4		

Table 3: Urine routine examination test.

Components appearance	06.12.2016	10.12.2016
Colour	Straw	Straw
pH	6.0	6.5
Albumin	(++)	Nil
Blood	Present	Trace
Ketones	(++)	Nil
Epithelial cells	0-2	0-3
RBC	Plenty	10-20
Pus cells	1-3	1-3

Table 4: Electrolyte report.

Date	2.12. 16	3.12. 16	4.12.16	9.12.16
Sodium	139	123	135	136
Potassium	3.7	2.8	3.6	3.9
Chloride	101	93	103	104
TCO2	22	21	26	25

Table 5: Multiple drugs and different virus, bacteria and protozoa are responsible for SJS/TEN.

	Etiologies of SJS/TEN
Drugs	Anticonvulsants: carbamazepine, lamotrigine, phenytoin, phenobarbitone
	Allopurinol, especially in doses of more than 100 mg per day
	Sulfonamides: cotrimoxazole, sulfasalazine
	Antibiotics: penicillins, cephalosporins, quinolones, minocycline
	Paracetamol/acetaminophen
	Nevirapine (non-nucleoside reverse-transcriptase inhibitor)
	Nonsteroidal anti-inflammatory drugs (NSAIDs) (oxicam type mainly)
	Contrast media
Viral	AIDS, herpes simplex virus, Epstein-Barr, influenza, coxsackie, lymphogranuloma venereum, and variola
Bacterial	Mycoplasma pneumoniae, typhoid, tularemia, diphtheria, and group A streptococci
Protozoal	Dermatophytosis, histoplasmosis, and coccidiomycosis Protozoal Trichomoniasis, plasmodium

Table 6: Different risk factors that may develop SJS/TEN.

An HIV infection	Among people with HIV, the incidence of Stevens-Johnson syndrome is about 100 times greater than among the general population
A weakened immune system	The immune system can be affected by an organ transplant, HIV/AIDS and autoimmune diseases
Cancer	People with cancer, particularly blood cancer, are at increased risk of Stevens-Johnson syndrome
A history of Stevens-Johnson syndrome	If you've had a medication-related form of this condition, you are at risk of a recurrence if you use that drug again
Genetic factors	Having certain genetic variations puts an individual at increased risk of Stevens-Johnson syndrome, especially if you're also taking drugs for seizures, gout or mental illness

Table 7: Different investigations with probable interpretations.

Variables	Interpretations
Complete blood count (CBC)	Anemia, lymphopenia, neutropenia, eosinophilia, atypical lymphocytosis
Liver function tests (LFT)	Elevated transaminases, hypoalbuminemia
Renal function	Microalbuminuria, renal tubular enzymes in urine, reduced glomerular filtration, rising creatinine and urea, hyponatremia
Pulmonary function	Interstitial infiltrates on chest x-ray, bronchial mucosal sloughing on bronchoscopy
Cardiac function	Abnormal ECG and imaging
Urgent frozen sections of skin biopsy for histopathology	Full-thickness skin necrosis (necrosis of keratinocytes, epidermal (or epithelial) necrosis, and mild lymphocytic dermal infiltration) and direct immune fluorescence is negative

Table 8: SCORTEN severity of illness score of SJS/TEN.

SCORTEN parameter	Units	Individual score		SCORTEN (sum of individual score)	Predicted mortality (%)
Age	>40 year	Yes-1,	No-0	0–1	3.2
Malignancy		Yes-1,	No-0	2	12.1
Hear rate	(>120 per minute)	Yes-1,	No-0	3	35.3
Body surface area involved at day 1	>10%	Yes-1,	No-0	4–5	58.3
Serum bicarbonate	<20 mmol/	Yes-1,	No-0	>5	90
Serum glucose	>14 mmol/l	Yes-1,	No-0		
Serum blood urea nitrogen (BUN)	>10 mmol/l	Yes-1,	No-0		

DISCUSSION

The incidence of Stevens-Johnson syndrome/toxic epidermal necrolysis (SJS/TEN) is estimated to be about 2-7 cases per million people each year, with Stevens-Johnson syndrome being three times more common than toxic epidermal necrolysis.[8,9] SJS/TEN can affect individuals of any age, sex, or race, especially those with a genetic predisposition. This illness is both rare and unpredictable, with drugs responsible for about 80% of cases. However, other factors such as viral, bacterial, and protozoal infections can also trigger the condition. (Table 5).[7,10]

The risk of developing SJS/TEN is approximately 100 times greater in people with HIV compared to the general population. Individuals with weakened immune systems, such as organ transplant recipients, patients with autoimmune diseases, and those with cancer-especially hematological malignancies-are at an increased risk (Table 6). A history of SJS/TEN due to a specific drug puts a person at risk of recurrence if that drug is used again. Additionally, a positive family history of SJS/TEN significantly increases the risk for immediate blood relatives.[11]

Genetic variations play a crucial role in the development of SJS/TEN, particularly in genes that influence the immune system's function. The most significant genetic association with SJS/TEN involves variations in the HLA-B gene, which is part of the human leukocyte antigen (HLA) complex. The HLA complex is essential for helping the immune system differentiate between the body's own proteins and those made by foreign invaders, such as viruses and bacteria. The HLA-B gene has many normal variations, enabling each person's immune system to respond to a wide range of foreign proteins. However, certain variations in this gene are much more common in people with SJS/TEN than in those without the condition.

Research suggests that HLA-B gene variations linked to SJS/TEN cause an abnormal immune response to specific medications. Though the exact mechanism is not fully understood, these drugs appear to trigger immune cells specifically cytotoxic T cells and natural killer (NK) cells to release a substance called granulysin.

It is responsible for the destruction of cells in the skin and mucous membranes, leading to the blistering and peeling that are characteristic of SJS/TEN.[12,13] Although SJS/TEN itself is not inherited, the genetic changes that increase the risk of developing the condition can be passed from one generation to the next. The clinical presentation of Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) typically unfolds in three phases.[14]

Prodromal phase

This phase begins 2–3 days before the acute phase, characterized by fever (present in 100% of cases), flu-like symptoms such as cough, malaise, myalgia, arthralgia, and anorexia. Other possible symptoms include conjunctivitis (seen in 32% of cases) and pharyngitis (observed in 25% of cases).

Acute phase

This phase is marked by tender, painful macular eruptions with non-blanching "targetoid lesions," which typically appear symmetrically on the face and trunk before spreading to the extremities. These lesions may become bullous and rupture, leaving the skin vulnerable to secondary infection. Mucosal involvement is common, affecting the pharyngeal, tracheal, bronchial, gastrointestinal, and vaginal regions.

Reepithelialization period

This phase may last 3–6 weeks, during which the risk of secondary infection remains high, potentially lead to scar formation.

Patients with TEN may develop multi-organ involvement, leading to complications in the renal, gastrointestinal, respiratory, and cardiovascular systems. However, sepsis resulting from secondary infection is the most common cause of death.[10,15]

Disorders with similar symptoms includes erythema multiforme (EM), autoimmune blistering diseases, staphylococcal scalded skin syndrome (SSSS), Generalized morbilliform drug eruption, generalised bullous fixed drug eruption (GBFDE), acute generalized exanthematous pustulosis (AGEP), drug-induced hypersensitivity syndrome/drug reaction with eosinophilia and systemic symptoms (DRESS).[16] No specific laboratory tests (other than a biopsy) can definitively diagnose SJS or TEN, making clinical diagnosis particularly in relation to recent drug use crucial. Skin, urine, and blood tests are advocated to prevent serious bacterial bloodstream infections, which significantly contribute to morbidity and mortality (Table 7).[17]

SJS/TEN is a medical emergency best managed in a pediatric intensive care unit (PICU) by a multidisciplinary team. Management includes the immediate discontinuation of the offending drug, maintaining adequate hydration and nutrition, controlling infection and fever, and providing pain relief. Skin care should be performed with great care and aseptic technique. Although the benefits of systemic corticosteroids are uncertain, they are often administered in high doses during the first three to five days of hospitalization. Granulocyte colony-stimulating factor (G-CSF) may benefit patients with severe neutropenia. Other treatments reported to be effective in severe cases include systemic corticosteroids, ciclosporin, TNF-alpha inhibitors, N-acetylcysteine, and intravenous immunoglobulins, though their roles remain controversial.[18]

The severity and prognosis of SJS/TEN are assessed using the SCORTEN severity of illness score. Each of the seven criteria at admission scores one point, with the total number of points predicting patient mortality (Table 8).[7,19]

In the case presented, the patient exhibited all three phases, prodromal, acute, and re-epithelialization. The condition was triggered by carbamazepine, which the patient had been taking for 42 days before the onset of symptoms (Figure 3 and 4). The patient was fully recovered from the disease after 35 days (Figure 6), consistent with literature suggesting that SJS recovery typically begins within 2-3 weeks unless complicated by infection, with full recovery potentially taking weeks or months.[14]

During the illness, the patient developed anemia, dyselectrolytemia, microscopic hematuria, and albuminuria, managed with electrolyte infusion, packed red blood cell (PRBC) transfusion, and total parenteral nutrition (TPN). Given the characteristic vesicobullous rashes, haematuria, albuminuria, bloody diarrhea, and affected body surface area, the patient was diagnosed with "Carbamazepine-triggered Stevens-Johnson syndrome (SJS) with gastrointestinal and renal system involvement." According to the SCORTEN score, his predicted mortality was 3.2%. (Table 8).[6]

SJS/TEN is a potentially severe disease with high morbidity and mortality, which are often predicted by the extent and severity at presentation. In our country, there is no consensus data available. However, data from the USA indicates that the mean adjusted mortality reported for the nationwide inpatient sample (2009-2012) was 4.8% for SJS, 19.4% for SJS/TEN overlap, and 14.8% for TEN.[20]

Preventive measures include considering genetic testing before prescribing certain drugs. The U.S. food and drug administration recommend screening people of Asian and South Asian ancestry for the HLA-B-1502 gene variation before starting treatment with carbamazepine. Avoiding the drug that triggered the condition is crucial to prevent recurrence. Immediate blood relatives of patients with SJS/TEN should also avoid the offending drug, as this condition can sometimes run in family.[19]

Limitations of this case report include the absence of a skin biopsy, histopathology, and genetic screening.

CONCLUSION

SJS/TEN is an unpredictable and life-threatening medical emergency that often requires management in the PICU. Flu-like symptoms following carbamazepine exposure

should not be overlooked, as they may be prodromal indicators of SJS/TEN. A multidisciplinary approach and early intervention are crucial for preventing secondary infections and reducing mortality and morbidity. While screening for the HLA-B-1502 variant allele is recommended, close monitoring for adverse reactions after initiating the offending drug, along with thorough counselling of the patient's parents or caregivers, is essential, especially given an idea about the high mortality and morbidity associated with SJS/TEN. An awareness of carbamazepine as a potential SJS trigger is vital for clinicians, as early recognition and intervention can significantly impact patient outcomes.

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

REFERENCES

1. Joyee JC. Vesiculobullous Disorders, In Nelson textbook of Pediatrics, 21st edition. 2020:3080-90.
2. Jha N, Alexander E, Kanish B, Badyal DK. A study of cutaneous adverse drug reactions in a tertiary care center in Punjab. Indian Dermatol Online J. 2018;9(5):299-303.
3. National organization for rare diseases. fact sheet: Stevens Johnson syndrome. Toxic epidermal necrolysis. 2024. Available at https://rarediseases.org. Accessed on 6th July, 2024.
4. Oakley AM, Krishnamurthy K. Stevens-Johnson Syndrome. National institutes of health (NIH) (.gov); available at https://www.ncbi.nlm.nih.gov. Accessed on 3rd July, 2024.
5. Harr T, French LE. Toxic epidermal necrolysis and Stevens-Johnson syndrome. Orph J Rare Dis. 2010;5:39.
6. Bastuji-Garin S, Fouchard N, Bertocchi M. SCORTEN: A severity-of-illness score for toxic epidermal necrolysis. J Invest Dermatol. 2000;115(2):149–53.
7. Shrestha O, Pant P, Devkota N, Gurung D, Shrestha D B. Carbamazepine induced toxic epidermal necrolysis and Stevens-Johnson syndrome overlapping during pregnancy in a South-East Asian patient: A case report. Anna Med Surg. 2021;68(10):10-6.
8. Yang SC, Hu S, Zhang SZ, Huang JW, Zhang J, Ji C, Cheng B. Corrigendum to The Epidemiology of Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis in China. J Immunol Res. 2018;8:415-45.
9. Safiri S, Ashrafi-Asgarabad A. The risk of Stevens-Johnson syndrome and toxic epidermal necrolysis in new users of antiepileptic drugs: Comment on data sparsity. Epilepsia. 2018;59(5):1083-4.
10. Parrillo SJ, Parrillo CV. Stevens-Johnson syndrome. Available at: http://www.emedicine.com. Accessed on 17TH December 2020.
11. Mayo Clinic. Stevens-Johnson syndrome. 2010 Available at: https://www.mayoclinic.org. Accessed on 17th March 2024.
12. Stevens-Johnson syndrome/toxic epidermal necrolysis. 2020. Available at: www.medlineplus.gov. Accessed on 17th March 2024.
13. Tangamornsuksan W, Lohitnavy M. Association between HLA-B*1301 and dapsone-induced cutaneous adverse drug reactions: a systematic review and meta-analysis. JAMA Dermatol. 2018;154(4):441-6.
14. Nasir SA, Tan HL, Tan HJ, Hussaini HM, Ramli R, Stevens-Johnson syndrome following failure of genetic screening prior to carbamazepine prescription, Case Reports in Dentistry. 2017;5:4201357.
15. Ferguson NN, Rosenbach K, Wanat R, Micheletti L. Taylor, Stevens-Johnson syndrome/toxic epidermal necrolysis inpatient dermatology. Springer. 2018;10:978-9.
16. National Organization for Rare Disorders. 2020. Available at: https://rarediseases.org/rare-disease. Accessed on 17th March 2024.
17. Wong A, Malvestiti AA, Hafner MFS. Stevens-Johnson Syndrome and toxic epidermal necrolysis: a review, Revista da Associação Médica Brasileira. 2016;62(5):468-73.
18. Zhang S, Tang S, Li S, Pan Y, Ding Y. Biologic TNF-alpha inhibitors in the treatment of Stevens-Johnson syndrome and toxic epidermal necrolysis: a systemic review. J Dermatolog Treat. 2020;31(1):66-73.
19. Guegan S, Bastuji-Garin S, Poszepczynska-Guigne E, Roujeau JC, Revuz J. Performance of the SCORTEN during the first five days of hospitalization to predict the prognosis of epidermal necrolysis. J Invest Dermatol. 2006;126:272–6.
20. Hsu DY, Brieva J, Silverberg NB, Silverberg JI. Morbidity and mortality of Stevens-Johnson syndrome and toxic epidermal necrolysis in united states adults. J Invest Dermatol. 2016;136(7):1387-97.

Cite this article as: Salwa Z, Tazmin T. Carbamazepine triggered Stevens-Johnson syndrome with gastrointestinal and renal system involvement: a case report. Int J Contemp Pediatr 2024;11:1644-50.