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Case Report

A Fatal Case of Phenobarbital-Induced Stevens-Johnson Syndrome with Erythroderma Associated with Sepsis, Drug-Induced Liver Injury and Normocytic Normochromic Anaemia

Keerthana N K[1], Monika G M[1], Kumaraswamy M[1], Hafis A[2], Pavan Kumar Chikkavalli Muddanna[1,*], Darshan J P[1], Likhith G[1], Yuktha B[1]

[1]Department of Pharmacy Practice, Sri Adichunchanagiri College of Pharmacy, Adichunchanagiri University, B.G. Nagara, Mandya, Karnataka, INDIA.
[2]Department of Pharmacy Practice, Faculty of Pharmacy, Parul University, Vadodara, Gujarat, INDIA.

ABSTRACT

Stevens-Johnson syndrome is a potentially fatal condition characterized by severe adverse drug reactions involving the mucosal surfaces and skin. This study describes an SJS case with erythroderma induced by the medication phenobarbital that resulted in the death of a 17-year-old boy diagnosed with seizures. The patient suffered from fever, rash, oral mucosal ulcers, dysphagia, hematochezia, and hemoptysis after taking phenobarbital. Test results showed neutrophilia, normocytic anemia, hyperbilirubinemia with increased serum transaminase levels (drug-induced hepatotoxicity), electrolyte disturbances, coagulopathy, and markedly high procalcitonin (sepsis). Immediate discontinuation of phenobarbital followed by supportive treatment using broad-spectrum antibiotics, mechanical ventilation, fresh frozen plasma, and metabolic therapy were unsuccessful.

Keywords: Drug-induced Liver Injury, Fatal Case, Phenobarbital, Sepsis, Stevens-Johnson Syndrome.

Correspondence:
Mr. Pavan Kumar Chikkavalli Muddanna
PhD Scholar, Department of Pharmacy Practice, Sri Adichunchanagiri College of Pharmacy, Adichunchanagiri University, B.G. Nagara, Mandya-571448, Karnataka, INDIA.
Email: pavankumarcm15@gmail.com

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INTRODUCTION

Stevens-Johnson Syndrome is a life-threatening inflammatory mucocutaneous drug reaction. SJS exists as a medical condition that falls between mild erythema multiforme and severe toxic epidermal necrolysis because it includes symptoms of both conditions (Hazin *et al*., 2008). SJS and TEN are severe cutaneous adverse reactions. They share the same underlying processes and are classified based on BSA. These conditions are rare, and their incidence varies across different regions (Frantz *et al*., 2021). Erythroderma is a condition characterised by a state of generalised erythema and scaling of the skin (Criado *et al*., 2004).

Mortality rates in SJS and TEN range from 12-49%, with sepsis being the most common cause of death (Stewart *et al*., 2025). Sepsis is a systemic response to infection that progresses through sepsis, severe sepsis, and septic shock, the most advanced stage, causing a widespread inflammatory reaction via endogenous mediators (Matot and Sprung, 2001). A seizure occurs due to abnormal, excessive electrical activity in the brain, affecting specific brain functions. Epilepsy is a disorder characterised by two or more unprovoked seizures occurring on different occasions without a known cause (Marchi *et al*., 2014). DILI is recognised as a significant cause of both acute and chronic liver disease. The most frequently involved substances include paracetamol, antimicrobials, NSAIDs, statins, isoniazid and herbal remedies (Verma and Kaplowitz, 2009). Anaemia is a condition characterised by a decrease in RBCs, haemoglobin levels, or PCV, resulting in reduced oxygen-carrying capacity of the blood. Normochromic, normocytic anaemia is frequently the result of an underlying chronic, non-haematological disease (Bates and Bain, 2012). Normocytic normochromic anaemia has several causes, with chronic diseases and drug therapy being among the most common (Bonnet, 1977).

CASE PRESENTATION

Patient Information

A 17-year-old male (body weight: 42 kg) was admitted to the emergency department on 17 January 2026 with complaints of fever with chills, productive cough with blood-tinged sputum for

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more than one week, generalized skin rash, painful oral ulcers, dysphagia, and haematochezia. The patient had a history of seizure disorder, for which he had been receiving oral phenobarbital 50 mg once daily at bedtime for approximately two months before admission.

The patient reported diminished appetite and disturbed sleep. His family history was significant for seizure disorder, and he had a history of occasional cigarette smoking. There was no documented history of drug allergy, food allergy, previous cutaneous adverse drug reactions, or other significant comorbidities.

Clinical History

Approximately one month before admission, the patient initially developed fever and productive cough, followed by the appearance of progressive erythematous scaly skin lesions involving the face, trunk, upper and lower limbs, genital region, lips, and tongue. The lesions gradually worsened and were associated with painful mucosal ulceration and dysphagia. According to the patient's attendants, mud had been applied over the skin lesions before hospital presentation, which may have increased the risk of secondary infection.

Prior to admission, the patient received treatment at another healthcare facility with intravenous pantoprazole, ceftriaxone-sulbactam, hydrocortisone, ondansetron, multivitamins, followed by oral pantoprazole, paracetamol, diclofenac-SP (Serratiopeptidase, Paracetamol) and levocetirizine. However, despite treatment, his symptoms progressively worsened, leading to referral to our tertiary care hospital (Table 1).

Diagnostic Assessment

Magnetic Resonance Imaging (MRI) and Electroencephalography (EEG) performed during evaluation of the seizure disorder were unremarkable. However, imaging demonstrated bilateral maxillary and ethmoidal mucosal pathology.

Laboratory investigations revealed neutrophilic leucocytosis, normocytic normochromic anaemia, thrombocytosis, elevated Erythrocyte Sedimentation Rate (ESR), electrolyte disturbances, hyperbilirubinemia, hypoalbuminemia, deranged coagulation parameters, elevated serum procalcitonin levels, and abnormal liver function tests, suggestive of severe sepsis with drug-induced liver injury.

The diagnosis of Stevens-Johnson syndrome was established based on the temporal relationship between phenobarbital exposure and symptom onset, characteristic mucocutaneous lesions with oral mucosal involvement, documented involvement of approximately 10% of the total Body Surface Area (BSA), dermatological evaluation, and supportive laboratory findings.

Although the lesions appear extensive in the photograph (Figure 1), the diagnosis was confirmed by the dermatologist as Stevens-Johnson Syndrome (SJS), as only approximately 10% of the Body Surface Area (BSA) was affected. The image may overestimate the severity and does not reflect the actual BSA involvement.

The causality was assessed using the Naranjo causality assessment scale (Table 2). The patient had a total score of 5, indicating a probable adverse drug reaction. This suggests that Phenobarbital was probably responsible for causing Stevens-Johnson syndrome.

Therapeutic Intervention

Considering the strong temporal association between phenobarbital therapy and the onset of Stevens-Johnson syndrome, phenobarbital was immediately discontinued.

Although discontinuation of the offending drug was considered essential, an alternative antiepileptic medication could not be initiated because the patient's condition deteriorated rapidly, requiring intensive supportive management in the intensive care unit.

Supportive treatment included intravenous fluids, systemic corticosteroids, broad-spectrum antimicrobial therapy, electrolyte correction, blood product support, nutritional care, wound care, and ventilatory support as clinically indicated.

Clinical Outcome

Despite immediate withdrawal of the suspected offending drug and aggressive multidisciplinary management, the patient's clinical condition progressively deteriorated because of severe sepsis and multiorgan dysfunction. The patient subsequently succumbed to the illness. The final diagnosis was phenobarbital-induced Stevens-Johnson syndrome with erythroderma complicated by severe sepsis, drug-induced liver injury, and normocytic normochromic anaemia.

Patient Perspective

Owing to the patient's death during hospitalization, a patient perspective could not be obtained.

DISCUSSION

SJS is a rare but life-threatening hypersensitivity reaction causing severe skin and mucosal damage (Frantz *et al.*, 2021; Hazin *et al.*, 2008). It belongs to the SJS/TEN spectrum classified by body surface area involvement (Frantz *et al.*, 2021).

The condition occurs due to cytotoxic T-cell-mediated destruction of keratinocytes with release of granulysin and inflammatory cytokines, leading to inflammation and skin damage (Chung *et al.*, 2008). SJS has 10-40% mortality rate, which rises further with complications like sepsis and multiorgan failure (Hazin *et al.*, 2008; Stewart *et al.*, 2025).

Figure 1: Generalised erythema and scaling with hemorrhagic lip crusting and areas of epidermal detachment.

Table 1: Chronological timeline of the patient's clinical course.

Time	Clinical Events
Approximately 2 months before admission	Diagnosed with seizure disorder but type of seizure was not mentioned and started on oral phenobarbital 50 mg once daily at bedtime.
Approximately 1 month before admission	Approximately 1 month before admission
Following few days	Progressive erythematous scaly skin lesions appeared over the face, trunk, upper and lower limbs, genital region, lips, and tongue, with painful oral mucosal involvement.
Before hospital admission	Mud was applied over the skin lesions by the patient's attendants. The patient received treatment with intravenous pantoprazole, ceftriaxone-sulbactam, hydrocortisone, ondansetron, multivitamins, followed by oral pantoprazole, paracetamol, diclofenac+ Paracetamol+ Serratiopeptidase, and levocetirizine without clinical improvement.
17th January 2026 (Hospital admission)	Admitted with fever with chills, productive cough with blood-tinged sputum, generalized skin lesions, mucosal involvement, reduced appetite, and worsening general condition.
During hospitalization	Laboratory investigations revealed neutrophilic leukocytosis, normocytic normochromic anaemia, thrombocytosis, elevated ESR, electrolyte disturbances, hyperbilirubinaemia, hypoalbuminaemia, deranged coagulation profile, elevated serum procalcitonin, severe sepsis, and drug-induced liver injury. MRI and EEG were normal, while bilateral maxillary and ethmoidal mucosal pathology was observed.
After diagnosis	Phenobarbital was immediately discontinued. Intensive supportive management was initiated, including corticosteroids, broad-spectrum antibiotics, intravenous fluids, electrolyte correction, nutritional support, wound care, blood product administration, and ventilatory support as clinically indicated.
Final outcome	Despite aggressive management, the patient's condition progressively deteriorated because of severe sepsis and multiorgan dysfunction, resulting in death.

Table 2: Naranjo Causality Assessment Scale.

Question	Answer	Score
1. Are there previous conclusive reports on this reaction?	Do not know	0
2. Did the adverse event appear after the suspected drug was administered?	Yes	+2
3. Did the adverse reaction improve when the drug was discontinued, or a specific antagonist was administered?	No	0
4. Did the adverse reaction reappear when the drug was re-administered?	Do not know	0
5. Are there alternative causes that could have caused the reaction?	No	+2
6. Did the reaction reappear when a placebo was given?	Not applicable	0
7. Was the drug detected in blood or body fluids at toxic concentrations?	Not done	0
8. Was the reaction more severe when the dose was increased or less severe when the dose was decreased?	Do not know	0
9. Did the patient have a similar reaction to the same or similar drugs in the past?	No	0
10. Was the adverse event confirmed by objective evidence?	Yes	+1
Total Score	5	
Interpretation	Probable Adverse Drug Reaction	

Table 3: Alden Score Table.

Parameter	Assessment	Score
Delay from drug intake	Compatible	+2
Drug present at reaction onset	Definite	0
Rechallenge	Not done	0
De-challenge	Neutral	0
Previous exposure	None documented	0
Type of drug (Notoriety)	Strongly associated with SJS/TEN	+3
Other causes	Possible	-1
Total Score		4

Drugs are the major cause of SJS, and in this case phenobarbital was suspected (Hazin *et al.*, 2008). Early identification and immediate withdrawal of the offending drug improve prognosis (Frantz *et al.*, 2021). Immediate cessation of the suspected offending drug is the mainstay of the management of SJS, resulting in better prognosis (Frantz *et al.*, 2021; Hazin *et al.*, 2008). ALDEN score is used for assessment of drug causality in SJS/TEN (Sassolas *et al.*, 2010) and the GCS score can also be obtained as shown in Tables 2 and 3.

The patient presented with erythematous lesions, mucosal involvement, dysphagia, and erythroderma, consistent with severe cutaneous adverse reactions (Frantz *et al.*, 2021; Sigurdsson *et al.*, 2001). Extensive skin involvement increases fluid loss, electrolyte imbalance, and infection risk (Frantz *et al.*, 2021).

Sepsis is a life-threatening condition that occurs when the body's abnormal response to an infection leads to organ dysfunction (Singer *et al.*, 2016). Sepsis is the main cause of death in SJS (Stewart *et al.*, 2025). High procalcitonin levels and neutrophilic leucocytosis strongly indicate severe bacterial sepsis (Becker *et al.*, 2004).

Significant liver dysfunction with high bilirubin and transaminase levels indicates hepatocellular injury. DILI can occur with several medications, including anticonvulsants (Verma and Kaplowitz, 2009). Electrolyte imbalance is common in critically ill patients and may occur due to systemic inflammation and fluid shifts (Funk *et al.*, 2010). Normocytic normochromic anaemia is often seen in acute infection and inflammation. Elevated LDH levels indicate cellular injury and tissue damage (Bonnet, 1977).

CONCLUSION

Supportive care is the main treatment, ideally given in an ICU/burn unit, and includes fluids, electrolyte balance, temperature control, nutrition, wound care, and infection prevention (Frantz *et al.*, 2021).

This case highlights that phenobarbital can trigger severe Stevens-Johnson syndrome with rapid systemic complications. Early recognition and prompt withdrawal of the suspected drug are essential to prevent progression. However, complications like sepsis and multiorgan dysfunction can still lead to fatal outcomes despite intensive care.

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ABBREVATIONS

ACLS: Advanced Cardiac Life Support; **BP:** Blood Pressure; **BSA:** Body Surface Area; **CPR:** Cardiopulmonary Resuscitation; **DILI:** Drug-Induced Liver Injury; **ECG:** Electrocardiogram; **EEG:** Electroencephalogram; **ESR:** Erythrocyte Sedimentation Rate; **ETT:** Endotracheal Tube; **FFP:** Fresh Frozen Plasma;

ICU: Intensive Care Unit; **IV:** Intravenous; **LDH:** Lactate Dehydrogenase; **MRI:** Magnetic Resonance Imaging; **MV:** Mechanical Ventilation; **NIV:** Non-Invasive Ventilation; **NS:** Normal Saline; **NSAID:** Non-Steroidal Anti-Inflammatory Drug; **OD:** Once Daily; **PCV:** Packed Cell Volume; **PEA:** Pulseless Electrical Activity; **RBC:** Red Blood Cells; **RL:** Ringer Lactate; **ROSC:** Return of Spontaneous Circulation; **SOS:** If Necessary/As Needed; **SJS:** Stevens-Johnson Syndrome; **TEN:** Toxic Epidermal Necrolysis.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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AUTHORS CONTRIBUTION

Keerthana N K: Data Curation, Investigation, Writing-Original draft; Monika G M: Data Curation, Writing-Original draft; Kumaraswamy M: Supervision, Validation, Clinical guidance; Hafis A: Validation, Clinical support, Review, Supervision; Pavan Kumar Chikkavalli Muddanna: Conceptualization, Supervision, Writing-review and editing, Final approval; Darshan J P: Literature review, Writing-review and editing; Likhith G: Literature review, Writing-review and editing; Dr. Yuktha B: Clinical guidance, Writing-review and editing.

AI DISCLOSURE STATEMENT

AI-assisted language editing tools were used only for grammatical refinement and language improvement. All scientific content, interpretation, and final manuscript approval were performed by the authors.

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