CASE REPORT

Fatal blows on a young patient with toxic epidermal necrolysis during his adventurous recovery

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Abstract

Toxic Epidermal Necrolysis (TEN) is a rare, life threatening mucocutaneous disorder most often precipitated by adverse drug reactions, characterized by extensive epidermal detachment and severe mucosal involvement. We describe a patient developing widespread skin and mucosal denudation complicated by sepsis due to barrier loss and ulceration following TEN. *Staphylococcus aureus* (including MRSA), *Pseudomonas aeruginosa,* and *Candida* species were identified as pathogens isolated in this patient. Massive transcutaneous fluid loss led to profound electrolyte disturbances and acute kidney injury, while hepatic dysfunction manifested as deranged transaminases, likely secondary to systemic inflammatory response and possible drug induced hepatotoxicity. Neutropenia further increased susceptibility to infection, delayed wound healing, and prolonged systemic illness. Severe mucosal ulcerations caused odynophagia, dysarthria, and nutritional compromise, while disfigurement, chronic pain, and prolonged hospitalization contributed to significant psychological morbidity.

Management required aggressive fluid resuscitation, steroid pulse therapy, broad spectrum antimicrobial therapy, renal and hepatic monitoring, analgesia, nutritional support, and meticulous wound care, delivered through a multidisciplinary approach involving dermatology, infectious disease, nephrology, hepatology, psychiatry, and rehabilitation. This case underscores the fulminant nature of TEN and highlights the importance of early recognition, withdrawal of the offending agent, vigilant monitoring for systemic complications, and comprehensive supportive care to optimize outcomes.

Keywords: toxic epidermal necrolysis (TEN); lamotrigine; neutropenia; sepsis

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Introduction

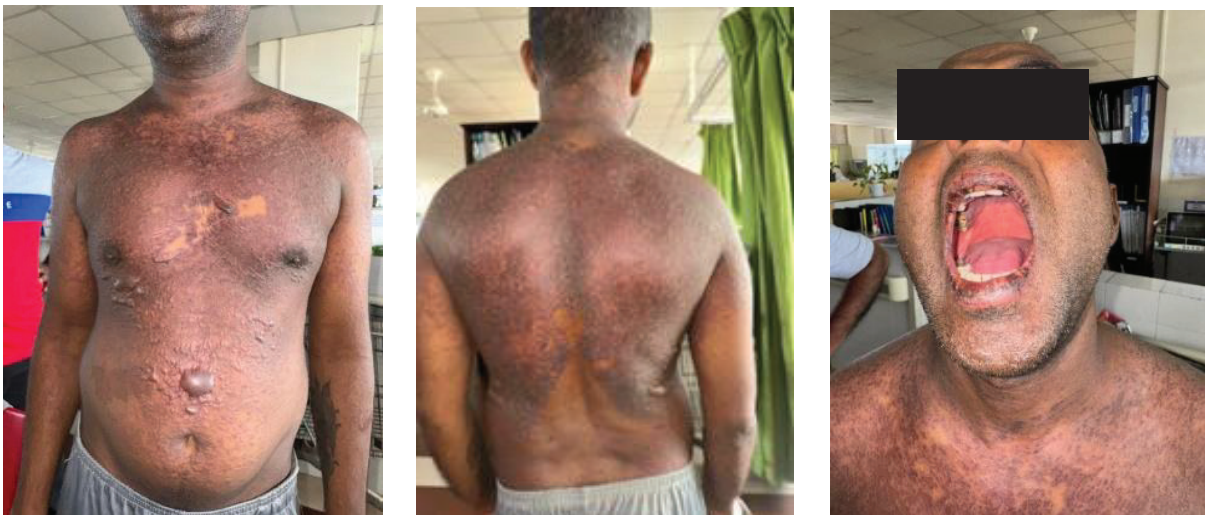
Toxic epidermal necrolysis (TEN) is a rare, life threatening mucocutaneous disorder characterized by extensive epidermal detachment involving more than 30% of the body surface area.[1] It represents the severe end of the spectrum of drug induced cutaneous reactions, with mortality rates ranging from 12% to 49%, due to complications such as sepsis, multiorgan dysfunction, and immunosuppression.[1] The pathogenesis is most commonly linked to adverse drug reactions, with anticonvulsants, antibiotics, and non-steroidal anti-inflammatory drugs frequently implicated.[2] Loss of the epidermal barrier predisposes patients to secondary infections, which significantly influence clinical outcomes.[3] Early recognition, prompt withdrawal of the offending agent, and meticulous supportive care are critical to improving survival.

We present a case of lamotrigine induced TEN complicated by neutropenic sepsis and sequential bloodstream infections, highlighting the temporal pattern of microbial invasion and associated systemic complications. This report underscores the importance of vigilance for evolving infectious spectra in TEN and the need for dynamic antimicrobial strategies tailored to disease progression.

Case Presentation

A 36-year-old male with no significant comorbidities, but with a history of drug addiction and depression, presented to the medical casualty unit with widespread necrotic lesions involving approximately 50% of the upper trunk and back. The lesions were associated with bullae formation, necrotic ulcerations of the lips, and painful ulcerations of the oral mucosa (Figure 1 A-C). Lamotrigine had been initiated 10 days earlier by a psychiatrist for depressive symptoms and behavioral disturbances. The diagnosis of toxic epidermal necrolysis was established based on clinical findings consistent with TEN. Using the SCORTEN severity index, the patient scored 2 points, derived from: Heart rate >120 bpm (tachycardia)/Epidermal detachment >10%.

On the first day of admission, the patient experienced a fever spike, followed by a 6-day afebrile period. Subsequently, he developed recurrent high-grade fever for 5 days, with progressive necrosis extending to the face and painful lesions over the soles (Figure 2 A-B). Feeding was significantly impaired due to mucosal involvement. With the onset of fever, the patient developed septic shock, initially managed with fluid resuscitation. He required Intensive Care Unit (ICU) care for neutropenia and mild renal impairment. Microbiological evaluation revealed blood culture positive for Staphylococcus aureus (MRSA), wound swab positive for Pseudomonas aeruginosa, and urine culture positive for lactose-fermenting coliforms. He was treated with intravenous vancomycin and meropenem. A subsequent blood culture grew Candida species, for which oral fluconazole was initiated. During the course, he developed a derangement of liver enzymes.



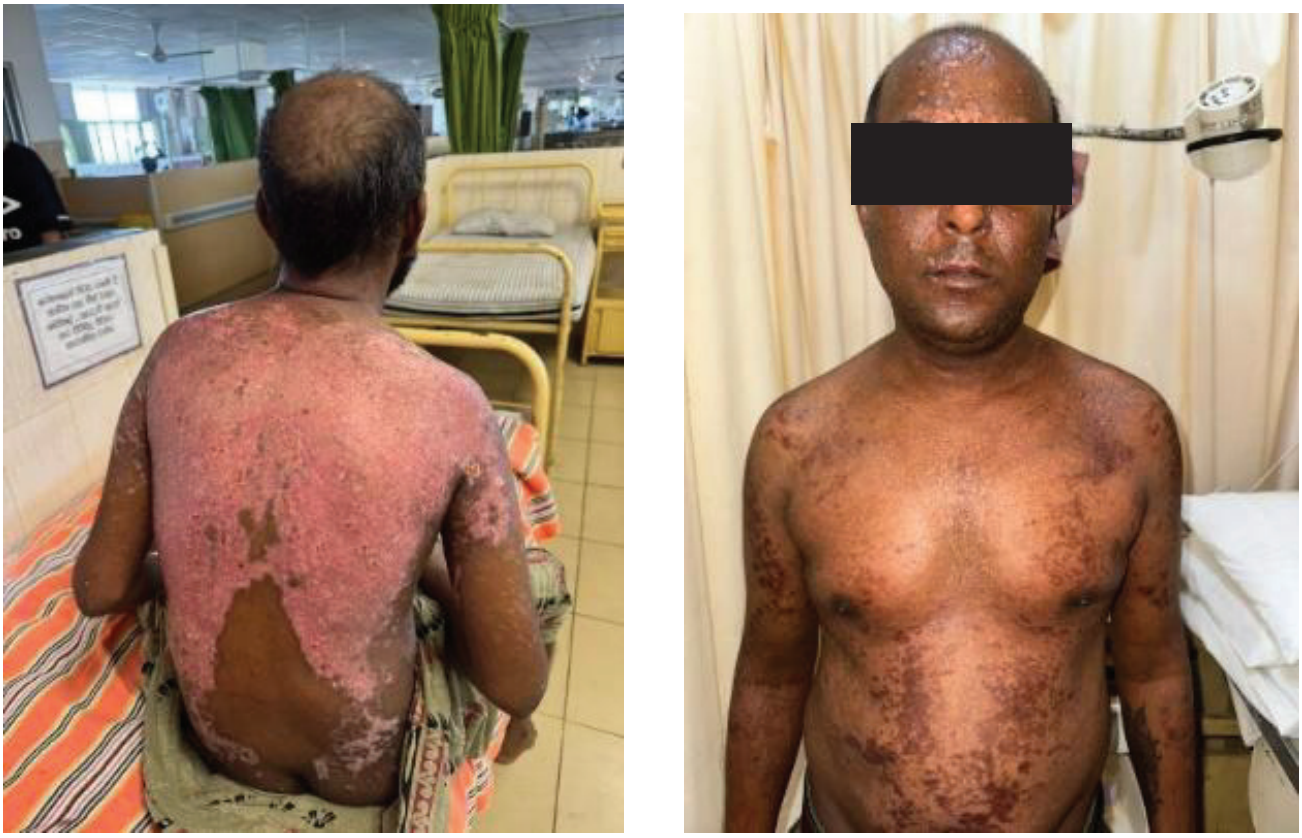
A B C

Figure 1. Initial clinical presentation.

A B

Figure 2. Clinical outlook on the 11th day of presentation.

Dermatologically, the patient experienced severe burning pain over necrotic skin, followed by desquamation during the healing phase. With continuation of antimicrobial therapy and supportive care, the skin lesions gradually resolved with replacement by normal skin (Figure 3 A-B). Liver enzymes trended downward, and the patient was in stable condition.



A B

Figure 3. Progressive re-epithelialization of the necrosed skin towards the end of the disease course.

Clinical Course and Laboratory Findings: During the first 10 days following the onset of cutaneous manifestations, total leukocyte and neutrophil counts remained within normal limits; however, on Day 10, there was an abrupt leukopenia with associated neutropenia that persisted for approximately three days before gradually recovering (Figure 4).

C-reactive protein levels were only marginally elevated during the initial period, but from Day 10 onward, a marked and sustained rise above 320 mg/L was observed for four consecutive days (Figure 5).

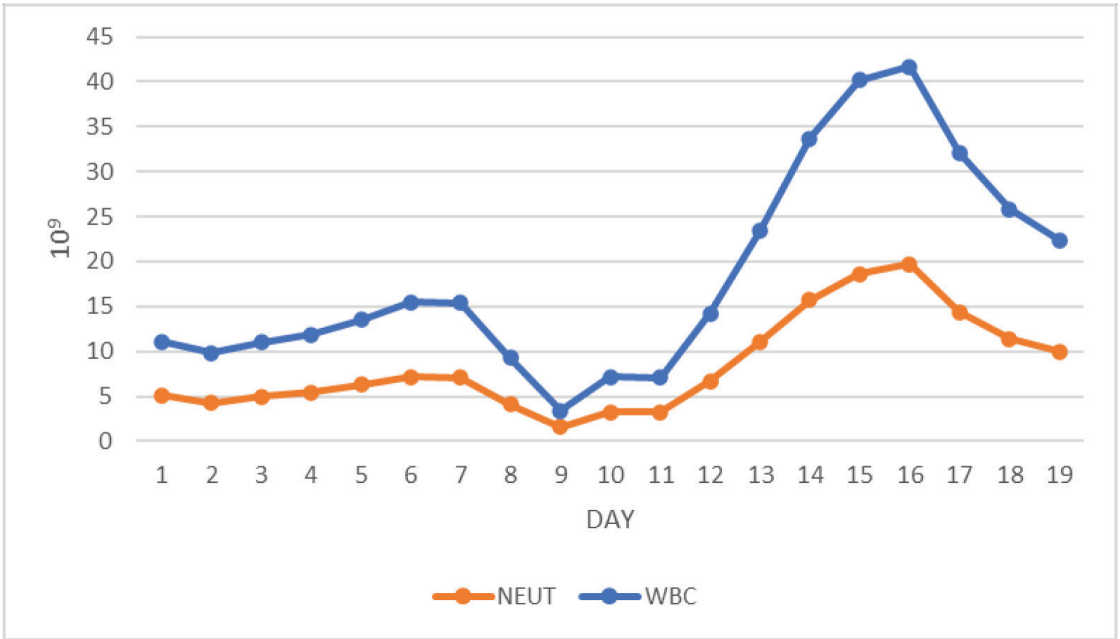


Figure 4. Sequential changes in WBC and neutrophil count during the timeline in toxic epidermal necrolysis.

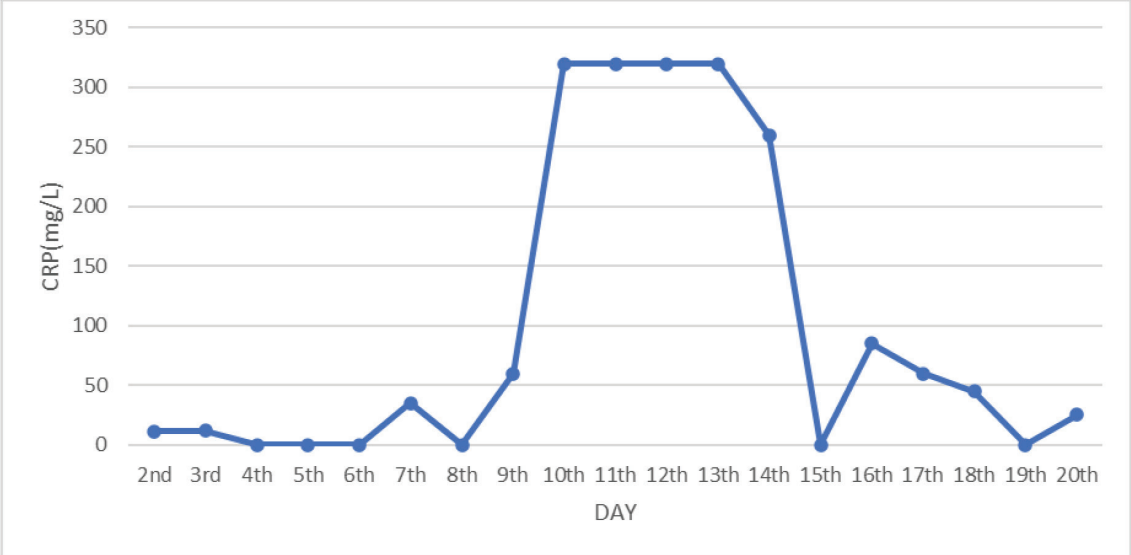


Figure 5. Sequential changes in C-reactive protein (CRP) levels.

A solitary febrile spike occurred on Day 1, while recurrent fever spikes between Days 10 and 17 correlated with infectious complications. Microbiological investigations revealed MRSA bacteremia beginning on Day 11 that persisted for several days, followed by Candida fungemia on Day 17, with blood cultures reverting to negative by Day 20. Wound and sputum cultures grew Pseudomonas aeruginosa on Day 12, persisting through Days 16–17, while urine cultures were positive for coliform organisms on Day 12 (Table 1).

Hepatic function demonstrated dual peaks of transaminase elevation on Days 11–12 and Day 18, with cholestasis evident from Day 10 and progressive bilirubin elevation that later resolved. (Figure 6) Electrolyte disturbances included mild hyponatremia and hyperkalemia, while most other baseline biochemical parameters remained within normal ranges. Collectively, Day 10 represented a critical turning point marked by hematologic suppression, inflammatory surge, hepatic dysfunction, and the onset of sequential infectious complications.

Table 1. Microorganisms isolated during the disease course

Day	Organism	Specimen
Day 11	MRSA (Methicillin-resistant Staphylococcus aureus)	Blood
Day 12	Escherichia coli (E. coli)	Urine
Day 12	Pseudomonas aeruginosa	Sputum
Day 12	Pseudomonas aeruginosa	Wounds
Day 16	Pseudomonas aeruginosa	Wounds
Day 17	Candida albicans	Blood
Day 20	Negative	All cultures

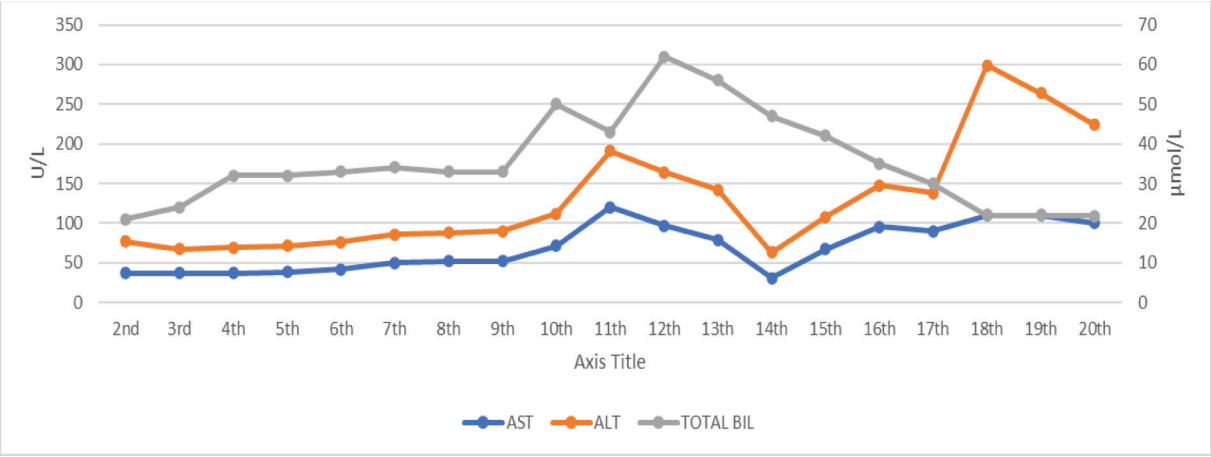


Figure 6. Sequential changes in transaminases and bilirubin profile.

Dual peaks in AST and ALT (U/L) are observed at Day 11 and Day 18, reflecting a systemic inflammatory response and potential drug-induced hepatotoxicity. Cholestasis, evidenced by elevated bilirubin (µmol/L), was prominent from Day 10 through Day 14.

Management: The patient was diagnosed with toxic epidermal necrolysis based on relevant clinical history and examination, and a multidisciplinary approach was adopted for management. Lamotrigine, identified as the offending agent, was promptly discontinued. Intravenous dexamethasone was administered at 0.5 mg/kg/day (30 mg) diluted in 500 ml of 5% dextrose and infused over three hours daily for three consecutive days. Adequate analgesia was provided, and urgent ophthalmology input was obtained. Topical therapy comprised combined antibiotic-steroid preparations applied to cutaneous and mucosal lesions, liquid paraffin for necrosed skin, and daily wound care initially with Vaseline, later supplemented with fusidic acid and 5% acetic acid. Oral mouthwashes containing antibacterial and antifungal agents were prescribed thrice daily, and nutritional support was optimized in view of swallowing difficulties. Psychiatric consultation was sought to ensure mental health stability during the disease course.

Infectious complications were managed sequentially: methicillin-resistant *Staphylococcus aureus* bacteremia was treated with intravenous vancomycin for 14 days until cultures were sterile; septic shock with neutropenia and mild acute kidney injury required intensive care with fluid resuscitation but no inotropes; echocardiography and abdominal ultrasonography excluded infective endocarditis. Subsequent culture positivity for *Pseudomonas* (sputum) and coliforms-Escherichia coli (E. coli) (urine) necessitated intravenous meropenem in combination with vancomycin, which controlled fever spikes and reduced inflammatory markers. Oral fluconazole was commenced following candidemia and continued until blood cultures were negative. Neutropenia was managed conservatively as absolute counts remained above critical thresholds. With progressive reduction in inflammatory markers and healing of skin lesions, the patient was discharged and followed up regularly in the dermatology clinic.

Discussion

Toxic epidermal necrolysis (TEN) is a rare, life threatening mucocutaneous disorder characterized by widespread epidermal necrosis and detachment involving more than 30% of the body surface area, usually accompanied by severe mucosal involvement.[4] It represents the most severe end of the Stevens-Johnson syndrome (SJS) – TEN spectrum.[5] Clinically, patients present with fever, painful erythema, and rapid progression to full thickness epidermal sloughing, often complicated by sepsis and multi organ dysfunction.[3]

Toxic epidermal necrolysis is most frequently precipitated by adverse drug reactions, with antibiotics (such as sulfonamides, penicillins, cephalosporins, and fluoroquinolones), anticonvulsants (including lamotrigine, phenytoin, carbamazepine, and phenobarbital), non-steroidal anti-inflammatory drugs – particularly oxicam derivatives such as piroxicam – and allopurinol being the principal culprits.[4]

Patients with toxic epidermal necrolysis often face a cascade of systemic problems beyond the cutaneous detachment. The loss of epidermal integrity predisposes to severe infections and septicemia, which remain the leading threat to survival. Fluid imbalance and electrolyte shift from extensive skin loss can precipitate renal dysfunction. Hepatic stress may manifest as enzyme elevation or cholestatic changes, reflecting both inflammatory injury and drug toxicity.[6] Pancytopenia in patients with SJS/TEN starts with severe lymphopenia, which can be due to direct antigen reaction with the cells or antigens affecting bone marrow functions, and is the most common hematological abnormality seen in these patients. This can render the patient susceptible to many infections, the most commonly encountered of which are superficial bacterial skin infections, pneumonia, and sepsis.[7] Involvement of mucosal surfaces contributes to painful swallowing, nutritional deficits, and ocular damage, while long-term recovery is frequently complicated by scarring, pigmentary alterations, chronic discomfort, and psychological distress.

Between Day 10 and Day 17, our patient entered the most precarious stage of illness. During this interval, a sudden fall in white cell and neutrophil counts coincided with a steep rise in inflammatory markers and recurrent fever spikes, signaling the onset of bloodstream infection. At the same time, liver enzymes and bilirubin levels showed two distinct surges, reflecting both systemic inflammatory stress and possible drug-related hepatic injury.[7] This overlap

of neutropenia, uncontrolled inflammation, and hepatic dysfunction created a fragile physiological state that favored microbial invasion. The later appearance of Candida in blood cultures illustrates how opportunistic pathogens exploit weakened immunity once broad-spectrum antibiotics suppress bacterial flora.

This period represents the critical sepsis window in TEN, when close surveillance and timely escalation of intravenous antimicrobials are essential. Early institutions of vancomycin and meropenem were decisive in controlling bacterial sepsis, while subsequent antifungal therapy prevented progression to disseminated candidiasis. Recognizing this vulnerable phase is vital, as it emphasizes the need for dynamic antimicrobial strategies and continuous monitoring of hematological and hepatic parameters to avert multi-organ failure.

Conclusion

Lamotrigine was identified as the etiological agent for toxic epidermal necrolysis (TEN), with the most critical vulnerability occurring between Days 10–17, characterized by neutropenia, sepsis with multiple microorganisms, recurrent fever, and hepatic enzyme derangement. During this period, a sequential infectious spectrum emerged, progressing from MRSA bacteremia to Gram-negative coliforms and Pseudomonas, and ultimately candidemia, with neutropenia serving as the pivotal driver of pathogen shift and opportunistic susceptibility. Dual hepatic enzyme peaks and cholestasis reflected systemic inflammatory stress and possible drug-related hepatotoxicity. Timely initiation of intravenous antibiotics and antifungal therapy during the sepsis window was decisive in averting multiorgan failure, while early drug withdrawal, vigilant monitoring, and dynamic antimicrobial strategies tailored to evolving complications proved essential. Stabilization and recovery were achieved through multidisciplinary care involving dermatology, infectious disease, nephrology, hepatology, and psychiatry, underscoring the importance of integrated management in complex TEN cases.

Ethics statement

Written informed consent was obtained from the patients for publication of this case report and accompanying images.

Authors' contributions

AW and ND were responsible for patient management and clinical follow-up. SG and ASR provided clinical supervision, contributed to critical clinical decision-making, and guided the overall management of the patient. KA was responsible for patient management, manuscript writing, and conceptualization of the case series. All authors read and approved the final manuscript.

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