NUTRITIONAL INTERVENTION IN A PATIENT WITH SEVERE TOXIC EPIDERMAL NECROLYSIS: A CASE REPORT

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A 70-year-old male with hypertension, gout, and stage 3a chronic kidney disease was admitted with fever, cough, and poor appetite. On day 2, he developed widespread bullae and mucosal erosions involving 70% of body surface area (BSA), leading to a diagnosis of severe toxic epidermal necrolysis (TEN). Multidisciplinary management in the intensive care unit included wound care, respiratory support, infection control, and early individualized nutritional therapy. Enteral feeding was initiated on ICU day 4, advancing to 1800 kcal/day (~30 kcal/kg/day) and 1.7 g/kg/day protein, including arginine, glutamine, vitamins C and E, and zinc, while monitoring renal and fluid-electrolyte status. Nitrogen balance was considered non-negative based on Nutrition Focused Physical Exam by the same experienced clinician, supported by stable weight from admission to discharge and rising serum albumin. Inflammatory markers decreased, lesions healed, and oral intake resumed. The patient was discharged in stable condition. This case highlights the feasibility and safety of early high-protein immunonutrition in TEN with chronic kidney disease, underscoring the importance of tailored nutrition as a core component of critical dermatologic care.

Keywords: Toxic epidermal necrolysis, nutritional therapy, immunonutrition, chronic kidney disease, critical illness.

I. INTRODUCTION

Toxic epidermal necrolysis (TEN) is a rare, severe dermatologic disorder characterized by massive keratinocyte apoptosis, leading to widespread epidermal detachment (> 30% body surface area [BSA]) and severe mucosal injury. Most cases are drug-induced, with allopurinol, sulfonamides, non-steroidal anti-inflammatory drugs (NSAIDs), and antiepileptics being common triggers. TEN has a high mortality rate (25 – 35%), especially among elderly patients or those with comorbidities such as chronic kidney disease (CKD) or sepsis.[1,2]

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From a pathophysiological perspective, TEN induces severe hypermetabolism, significant nitrogen loss through exudative wounds, marked fluid-electrolyte imbalance, and heightened susceptibility to sepsis. Management requires urgent and comprehensive care, including drug withdrawal, intensive support, infection control, wound care, respiratory support, and especially early nutritional intervention. In TEN, severe inflammation and systemic catabolism with protein and fluid losses make individualized nutrition support therapeutic, not merely supportive. Early nutrition with adequate energy delivery during the initial stress phase, gradually progressing to full requirements (20 – 35 kcal/kg/day) and protein (1.5 – 2.0 g/kg/day), along with immunonutrients such as arginine, glutamine, antioxidants (vitamins C

and E), zinc, and selenium, is recommended by ESPEN and ASPEN guidelines.[3-6] In patients with coexisting CKD, nutritional plans must be adapted to balance metabolic needs with renal function.

II. CASE PRESENTATION

Patient Information

A 70-year-old man with a history of hypertension, gout, and stage 3a chronic kidney disease was admitted to An Binh Hospital (Vietnam) on August 23, 2024, with fever, dry cough, and poor appetite. He was prescribed allopurinol, cilnidipine, candesartan, and bisoprolol. Pneumonia was diagnosed and managed with ampicillin/sulbactam, paracetamol, and fexofenadine. On day 2 of hospitalization, he developed widespread bullae and severe mucosal erosions involving the eyes and mouth. A diagnosis of severe TEN (70% BSA), was established, and the patient was transferred to the intensive care unit (ICU) on August 25.

Nutritional Assessment

On August 28, BMI was 24.03 kg/m² (60kg, 158cm). No recent weight loss or edema was observed. Based on Subjective Global Assessment (SGA), moderate malnutrition was diagnosed. Serum albumin declined from 36.4 to 25.81 g/L; CRP peaked at 80.3 mg/L; procalcitonin at 4.4 ng/mL; creatinine remained stable between 115 – 174 µmol/L.

Nutrition Plan

From ICU Day 1 to Day 3, the patient was in the acute phase of critical illness and received exclusive parenteral nutrition with intravenous glucose and amino acid solution, providing approximately 25% of estimated energy requirements on Day 1, 50% on Day 2, and 75% on Day 3 (estimated needs 25 – 30 kcal/kg/day) according to ICU protocol for severe metabolic stress. Protein provision started at 0.8 – 1.0 g/kg/day on Day 1, increased to 1.2 g/kg/day on Day 2, and 1.5 g/kg/day on Day 3, consistent with recommendations for stage 3a CKD to avoid excessive renal load while meeting catabolic demands. Strict daily monitoring of fluid and electrolyte balance was performed due to 70% BSA skin loss, with adjustments based on input–output charts and serum electrolytes.

From August 28 (ICU Day 4), enteral nutrition via nasogastric tube was initiated using energy- and protein-dense formulas. Feeding was escalated to 100% of target energy (~1800 kcal/day) by Day 5. Protein intake was further increased to 1.7 g/kg/day (100 g/day) by Day 5, which was selected within the recommended range of 1.5 – 2.0 g/kg/day for severe TEN according to ESPEN and ASPEN guidelines, as renal function parameters (creatinine, urea) remained stable. The regimen included 10 g/day arginine and 10 g/day glutamine, along with vitamin C 500 mg/day, vitamin E 400 IU/day, and zinc 14 mg/day. No parenteral nutrition or albumin transfusion was required during the enteral phase.

Nitrogen balance was estimated from staged protein escalation, stable renal function, and preservation of lean tissue evaluated by Nutrition Focused Physical Exam (NFPE) performed by the same experienced clinical physician, assessing major muscle groups including temporal, sternocleidomastoid, deltoid, interosseous, quadriceps, and gastrocnemius muscles. This indicated a non-negative nitrogen balance despite extensive skin loss. Daily body weight monitoring was not feasible due to lack of ICU-compatible scale; weight was recorded at admission (60.0kg) and at the end of hospital treatment (58.5kg), showing only a 1.5kg decrease, which was within the expected range from fluid shifts and wound healing, without clinical signs of significant muscle wasting.

This careful nutritional strategy helped maintain muscle mass, support wound healing, and provide sufficient amino acids for immune and metabolic demands, while minimizing the risk of renal overload.

Daily fluid and electrolyte monitoring included all intravenous fluids, enteral nutrition, urine output, and wound exudate, with careful adjustments to maintain euvolemia and metabolic safety. Electrolytes such as sodium, potassium, and bicarbonate were regularly checked to prevent both dehydration and fluid overload, which are common complications in patients with extensive skin loss. The meticulous monitoring allowed for timely correction of imbalances, ensuring that metabolic stability was maintained throughout the acute and recovery phases.

The treatment process

On ICU day 1 (August 25), the patient presented with extensive epidermal necrolysis (BSA 70%), mucosal sloughing, corneal edema, conjunctival synechiae, and oropharyngeal ulcers. HFNC was required for respiratory failure. Blood cultures revealed *Pseudomonas aeruginosa*; sputum grew *Acinetobacter baumannii*.

Inflammation worsened during the first 2 weeks despite antibiotics: procalcitonin rose from 3.3 to 4.4 ng/mL; neutrophil percentage increased to 89.2%; albumin decreased to 25.8 g/L. Stable renal parameters (creatinine 115 – 122 μmol/L) indicated metabolic safety of the nutrition plan.

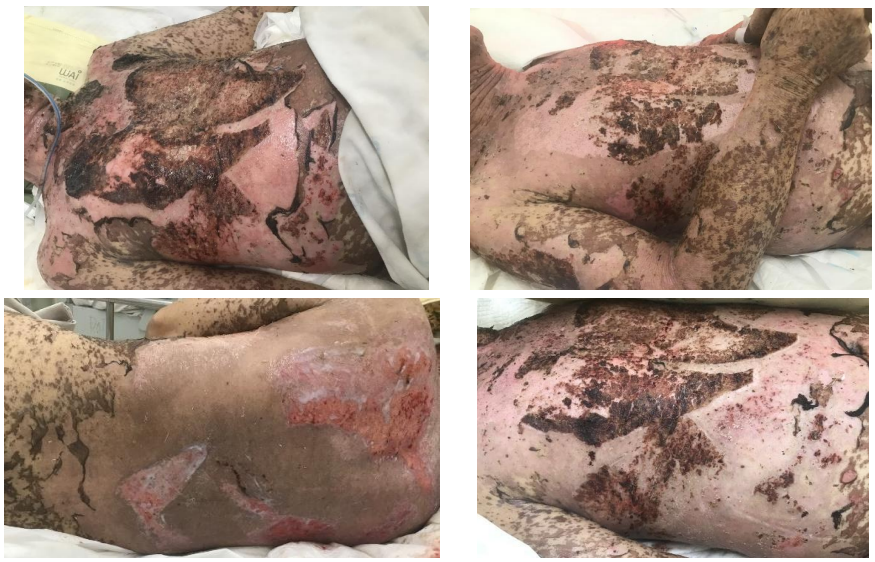
Between September 13 – 23, recovery became evident. CRP dropped from 80.3 to 19.5 mg/L; procalcitonin to 0.2 ng/mL; albumin increased to 30.3 g/L without exogenous albumin. Wound granulation improved; mucosa healed; HFNC was weaned off. Body weight decreased from 60.0 kg on ICU admission to 58.5 kg at discharge from the hospital; NFPE showed no appreciable reduction in muscle mass.

Enteral feeding was well tolerated; no gastrointestinal complication occurred. Clinical laboratory data are summarized in Table 1, showing trends in inflammation, nutrition, and renal markers. Wound improvement is shown in Figure 1.

Table 1. Clinical laboratory data during hospitalization

Date	Hgb (g/dL)	WBC ($\times 10^9$/L)	Neu (%)	Albumin (g/L)	CRP (mg/L)	PCT (ng/mL)	Cre (μmol/L)	Ure (mmol/L)
Aug 25	11.7	11.3	84.3	36.4	80.3	–	174.0	12.2
Aug 28	9.8	7.2	73.7	–	–	3.3	143.6	14.9
Aug 29	–	–	–	28.5	–	–	119.2	15.0
Sep 7	8.0	13.8	81.9	–	–	2.1	–	–
Sep 10	8.1	14.3	85.3	–	–	4.4	115.4	8.4
Sep 12	7.7	17.5	89.2	25.8	–	–	–	–
Sep 18	8.4	15.4	77.3	–	–	0.3	118.8	7.2
Sep 23	7.9	15.4	70.4	30.3	19.5	–	–	–
Sep 24	8.7	15.3	69.6	–	–	0.2	121.9	9.2

Hb: Hemoglobin; WBC: White blood cell count; Neu: Neutrophil percentage; CRP: C-reactive protein; PCT: Procalcitonin; "–" indicates data not available



Panel A. Initial stage **Panel B. After two weeks**

Figure 1. Clinical wound progression before and during treatment

Panel A: Initial stage with widespread epidermal detachment and mucosal ulceration. Panel B: After two weeks, re-epithelialization and granulation tissue formation were observed

III. DISCUSSION

Toxic epidermal necrolysis (TEN) is a life-threatening dermatologic emergency marked by extensive epidermal detachment, severe mucosal involvement, and systemic inflammation.[1,2] The metabolic response in TEN is characterized by hypercatabolism, protein loss, and immune dysregulation, particularly in patients with comorbidities such as chronic kidney disease (CKD).

In this case, nutritional support was guided by ICU-phase physiology. During the acute phase (Days 1 – 3), exclusive parenteral nutrition with glucose and amino acids was initiated at a cautious progression (Day 1: 25% of target energy, Day 2: 50%, Day 3: 75%), in accordance with ESPEN and ASPEN recommendations to prevent overfeeding during stress-induced endogenous glucose production. Protein intake began at 0.8 – 1.0 g/kg/day on Day 1, gradually increasing to 1.2 g/kg/day on Day 2 and 1.5 g/kg/day on Day 3, respecting the safety thresholds for stage 3a CKD while addressing elevated nitrogen demands in TEN.[3-5]

Full energy (~1800 kcal/day, ~30 kcal/kg/day) and protein 1.7 g/kg/day targets were achieved by Day 5 via enteral nutrition, reflecting both gastrointestinal tolerance and stable renal function markers. Strict monitoring of fluid and electrolytes was essential due to 70% BSA skin loss, with adjustments guided by daily input–output charts and serum sodium, potassium, and bicarbonate levels, preventing both dehydration and fluid overload.

The patient demonstrated measurable improvement: serum albumin rose from 25.8 to 30.3 g/L without albumin infusion, inflammatory markers normalized, and mucocutaneous lesions progressively healed. Body weight decreased modestly (60.0 → 58.5kg) with minimal muscle mass loss,

suggesting preservation of lean tissue. In this case, direct daily weight monitoring and bedside body composition analysis were not feasible due to the lack of ICU-compatible equipment. Therefore, NFPE was employed as the most practical and reproducible bedside method for assessing muscle mass status.[6] The examination was performed by the same experienced clinical physician to minimize inter-observer variability. NFPE findings indicated no appreciable reduction in muscle mass, which was consistent with other objective clinical and biochemical indicators, including a rise in serum albumin, reduction in CRP and procalcitonin, and only minimal weight loss during hospitalization. This outcome likely reflects early protein optimization and inclusion of immunonutrients (arginine, glutamine, vitamins C and E, zinc), which modulate immune response and promote wound healing in severe burns and TEN.

Protein provision was carefully escalated-from 1.0 g/kg/day in the acute phase to 1.2 g/kg/day (Day 3), 1.5 g/kg/day (Day 4), and 1.7 g/kg/day from Day 5 onwards-balancing metabolic stress with CKD safety thresholds. Nitrogen balance was considered non-negative, as indicated by stable body weight, and rising serum albumin, while accounting for nitrogen losses from 70% BSA skin detachment. This approach aligns with protein requirements recommended for severe burns and critical dermatologic conditions.[3,7]

The reduction of CRP and procalcitonin suggests that immunonutrition may have contributed to attenuating systemic inflammation and supporting epithelial recovery.[4,5,8] Multidisciplinary collaboration among ICU, dermatology, nutrition, ophthalmology, and microbiology teams was integral to the favorable outcome. Ongoing nutritional reassessment was essential to optimize energy-protein delivery while maintaining renal safety.[9,10]

Nevertheless, questions remain regarding optimal dosing of immunonutrients-particularly arginine and glutamine-in TEN patients with CKD. Further research is warranted to determine whether higher doses can enhance immune recovery without compromising metabolic stability. Additionally, biomarkers such as prealbumin, interleukin-6 (IL-6), or lactate may have potential for monitoring nutritional response in patients with extensive epithelial injury.[11]

V. CONCLUSION

This case report underscores the critical role of early and individualized nutritional support in the treatment of severe TEN, particularly in patients with comorbidities such as chronic kidney disease. A targeted immunonutrition strategy not only helped control systemic inflammation and promote mucocutaneous healing but also contributed to clinical stabilization without the need for albumin replacement. Multidisciplinary collaboration and continuous nutritional reassessment were key to the favorable outcome. This case adds to the growing evidence that nutritional therapy should be considered a core component of TEN management protocols.

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Conflict of interest

The author declares no conflict of interest.

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