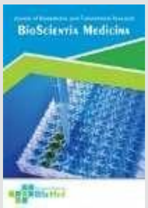
eISSN (Online): 2598-0580



Bioscientia Medicina: Journal of Biomedicine & Translational Research

Journal Homepage: www.bioscmed.com

Rapid Correction of Hypoalbuminemia and Promotion of Granulation Tissue in Pediatric Deep Partial-Thickness Burns via Targeted Immunonutrition: A Case Report

Ni Kadek Ari Puji Astiti[1]*, Christian[2]

[1]Medical Doctor, Famili Husada General Hospital, Gianyar, Indonesia

[2]Clinical Nutrition Specialized Doctor, Klungkung General Hospital, Klungkung, Indonesia

ARTICLE INFO

Keywords:

Curreri junior formula

Hypoalbuminemia

Immunonutrition

Pediatric burns

Wound healing

***Corresponding author:**

Ni Kadek Ari Puji Astiti

E-mail address:

aripuji324@gmail.com

All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/bsm.v10i3.1525

A B S T R A C T

Background: Pediatric burns induce a profound hypermetabolic and hypercatabolic state, distinct from adults due to limited physiological reserves and high growth demands. The rapid onset of negative nitrogen balance and hypoalbuminemia significantly impairs wound healing and immune function. **Case presentation:** We report the case of a 7-year-old male with pre-existing malnutrition (StrongKids Medium Risk) presenting with 23% total body surface area (TBSA) Grade IIB scald burns affecting the posterior humerus, lumbosacral, gluteus, and femoral regions. The patient exhibited acute hypoalbuminemia (2.3 g/dL) and anorexia due to pain. A specialized medical nutrition therapy (MNT) protocol was implemented using the Curreri Junior formula, targeting 2,500 kcal/day and 105 g protein/day. The intervention utilized a stepwise escalation of polymeric enteral nutrition enriched with immunonutrients (Glutamine, Zinc, and Vitamin C). Despite the severity of the injury, the patient demonstrated rapid nutritional rehabilitation. By day 5 of hospitalization, albumin levels normalized to 3.1 g/dL, and significant granulation tissue formation was observed. The patient achieved a weight gain of 0.4 kg during the acute phase, countering the expected catabolic weight loss. **Conclusion:** Early, aggressive, and calculated nutritional support incorporating specific immunonutrients can reverse the catabolic effects of thermal injury in pediatric patients. This case highlights the efficacy of the Curreri Junior formula combined with Glutamine and Zinc in accelerating wound closure and correcting biochemical markers in resource-limited settings.

1. Introduction

Thermal injuries represent one of the most devastating and neglected public health crises of the modern era. Despite advancements in preventative medicine and safety regulations, burn injuries continue to exact a heavy toll on global populations, with the World Health Organization (WHO) reporting approximately 180,000 deaths annually. This mortality figure, however, is merely the tip of the iceberg, masking the millions of non-fatal injuries that result in lifelong disability, disfigurement, and psychological trauma.[1] The epidemiology of burn injuries reveals a stark geopolitical and socioeconomic divide; a disproportionate majority of these incidents—nearly 95%—occur in low- and middle-income countries (LMICs), particularly within the Southeast Asian and African regions. In these resource-constrained settings, the intersection of rapid urbanization, overcrowding, and reliance on open-fire cooking or unsafe heating methods creates a hazardous environment where children are uniquely vulnerable.

In the pediatric population, the etiology of thermal injury differs significantly from adults. While adults frequently suffer from flame burns associated with occupational hazards, children, particularly toddlers and those under the age of ten, are predominantly victims of scald burns. The domestic environment, often perceived as a sanctuary, becomes the primary site of injury, where accidental spills of boiling water or hot food act as the mechanism of injury. This epidemiological trend is not merely statistical but carries profound clinical implications.[2] Scald burns in children, though often affecting a smaller total body surface area (TBSA) compared to catastrophic flame burns, possess a deceptive capacity for severity due to the distinct anatomical and physiological characteristics of the pediatric patient.

The management of pediatric burns presents a unique constellation of clinical challenges that distinguishes it from adult burn care. The axiom that children are not merely small adults is nowhere more relevant than in the pathophysiology of thermal trauma.[3] Children possess a significantly larger body surface area relative to their body mass compared to adults. This high surface-area-to-mass ratio predisposes the pediatric patient to rapid and profound fluid loss through evaporation, accelerating the onset of hypovolemic shock if resuscitation is not immediate and aggressive. Furthermore, this anatomical ratio makes children highly susceptible to hypothermia, which can induce coagulopathy and worsen metabolic acidosis, forming a lethal triad in the acute phase of injury.

Crucially, the integumentary system of a child differs structurally from that of an adult. Pediatric skin is markedly thinner, with a reduced thickness of the stratum corneum and dermis.[4] Consequently, a thermal exposure of a specific temperature and duration that might cause a superficial (first-degree) or superficial partial-thickness burn in an adult is often sufficient to cause a deep partial-thickness (Grade IIB) or full-thickness burn in a child. This reduced barrier function means that thermal energy penetrates deeper and faster, damaging the microvasculature and destroying the dermal matrix essential for spontaneous re-epithelialization. Additionally, children have limited physiologic reserves; their metabolic rate is naturally higher to support growth, leaving little buffer to absorb the immense stress of a thermal injury.

Following a severe burn—clinically defined in pediatrics as involving more than 10-20% TBSA—the body undergoes a radical systemic transformation. The immediate post-injury period, known as the ebb phase, is characterized by hypoperfusion and depressed metabolic activity.[5] However, within 24 to 48 hours, the patient transitions into the flow phase, a persistent hyperdynamic and hypermetabolic state described classically by Cuthbertson. This phase is driven by a massive surge in counter-regulatory stress hormones, including cortisol, catecholamines, and glucagon, alongside a torrential release of pro-inflammatory cytokines such as Interleukin-1 (IL-1), Interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α).

In children, this hypermetabolic response is explosive and sustained. The resting energy expenditure (REE) can be elevated by up to 100% above basal levels, creating an energy deficit that is difficult to bridge.[6] The body, perceiving a catastrophic threat, reprioritizes substrate utilization. Glycogen stores are depleted rapidly. To maintain the glucose pool required by the brain and the healing wound (which utilizes glucose via anaerobic glycolysis), the body initiates massive gluconeogenesis. The primary substrates for this glucose production are amino acids derived from the breakdown of skeletal muscle.

This process of extensive proteolysis and lipolysis is the hallmark of the burn-induced catabolic state. In pediatric patients, who have smaller muscle mass reservoirs than adults, this muscle wasting is particularly deleterious. The breakdown of structural protein leads to a negative nitrogen balance, which, if left unchecked, results in severe malnutrition, immune dysfunction, and growth arrest. The liver shifts its protein synthesis machinery away from constitutive proteins like albumin and towards acute-phase reactants. Consequently, hypoalbuminemia develops rapidly, not only due to vascular leakage and losses through the burn wound but also due to this hepatic reprogramming. This hypoalbuminemia exacerbates edema, impairs gastrointestinal function, and compromises drug transport, creating a vicious cycle of delayed wound healing, graft failure, and increased susceptibility to sepsis.

The clinical picture is further complicated in LMICs by the prevalence of pre-existing malnutrition. Many pediatric patients in these regions present with sub-optimal nutritional status, characterized by stunting or wasting, prior to the injury. When a nutritionally compromised child sustains a burn, they lack the physiological buffer to withstand the hypercatabolic storm. The double hit of chronic malnutrition and acute burn-induced hypermetabolism significantly increases the risk of mortality and morbidity. The assessment of nutritional status in these patients is critical; tools like the STRONGkids screening are essential to identify risk early, as demonstrated in various clinical settings.[7] In such cases, standard nutritional support is insufficient; therapeutic, targeted intervention becomes a life-saving necessity. Historically, burn care focused heavily on fluid resuscitation (such as Parkland or Baxter formulas) and surgical closure, with nutrition relegated to a supportive role. However, modern burn care recognizes nutrition as a primary therapeutic modality. The goal is no longer just to prevent starvation but to modulate the metabolic response, blunt catabolism, and actively promote tissue repair.

Determining the precise caloric needs of a pediatric burn patient is complex. Underfeeding worsens catabolism, while overfeeding can lead to hyperglycemia, hepatic steatosis, and respiratory compromise. While various formulas exist, the Curreri Junior formula has established utility in estimating the high-energy requirements of children with significant burns, accounting for both basal needs and the stress factor of the injury.[8] Yet, macronutrients alone are often insufficient to reverse the deep metabolic derangements of a major burn. This realization has ushered in the era of immunonutrition—the use of specific nutrients in supranormal doses to modulate immune activity and healing. Three specific micronutrients have emerged as pivotal in this domain: Glutamine, Zinc, and Vitamin C. As the most abundant free amino acid in the body, glutamine becomes conditionally essential during catabolic states. It serves as the primary fuel source for enterocytes (intestinal cells) and lymphocytes. In burn patients, plasma glutamine levels plummet. Supplementation is hypothesized to maintain gut mucosal integrity, thereby preventing bacterial translocation—a primary source of sepsis in burns—and supporting lymphocyte proliferation. Zinc is a critical cofactor for over 300 metalloenzymes, including RNA and DNA polymerases required for cell division. Zinc is essential for the function of matrix metalloproteinases (MMPs), which facilitate the remodeling of the extracellular matrix during wound healing.[9] Zinc deficiency, common in burns due to exudative losses, halts re-epithelialization and impairs T-cell function. Ascorbic acid acts as an obligatory cofactor for the enzymes prolyl hydroxylase and lysyl hydroxylase. These enzymes are responsible for the cross-linking of collagen fibers, giving the new tissue its tensile strength. Furthermore, as a potent water-soluble anti-oxidant, Vitamin C helps neutralize the reactive oxygen species (ROS) generated during the intense inflammation of a burn injury.

Despite the theoretical benefits of these nutrients, the practical application of a combined, aggressive Medical Nutrition Therapy (MNT) protocol in pediatric patients with deep partial-thickness burns remains under-documented, particularly in the context of LMICs, where resource limitations often dictate care. Literature specifically addressing the synergistic effect of the Curreri Junior formula combined with a cocktail of Glutamine, Zinc, and Vitamin C in correcting acute hypoalbuminemia and accelerating granulation in malnourished children is limited. Most studies focus on isolated nutrients or adult populations, leaving a gap in evidence-based guidelines for comprehensive pediatric metabolic resuscitation. Furthermore, the nuances of treating Grade IIB or deep partial-thickness burns are critical.

These wounds sit on the precipice; with adequate nutritional and wound care, they can re-epithelialize without grafting. Conversely, with infection or malnutrition, they convert to full-thickness wounds requiring surgery. Therefore, the role of nutrition in tipping the scales toward spontaneous healing in this specific wound depth is of paramount clinical interest.[10]

This study aims to evaluate the clinical and biochemical efficacy of a targeted, high-intensity Medical Nutrition Therapy (MNT) protocol—specifically utilizing the Curreri Junior formula reinforced with Glutamine, Zinc, and Vitamin C supplementation—in a pediatric patient presenting with 23% Grade IIB scald burns and pre-existing malnutrition. The novelty of this case report lies in its detailed exposition of the metabolic resuscitation timeline. Unlike general case reviews, we provide a granular analysis of how specific nutritional interventions correlate with rapid biochemical correction (albumin normalization) and visible wound evolution (granulation tissue formation) within a resource-limited setting. By demonstrating that sophisticated metabolic management can be successfully implemented without advanced indirect calorimetry or isotopic studies, this report offers a replicable framework for clinicians in developing regions. We highlight the methodological approach to reversing catabolism, aiming to provide evidence that nutritional therapy is not merely supportive care, but a potent, active intervention that dictates the trajectory of survival and recovery in pediatric burns.

2. Case Presentation

A 7-year-old male patient presented to the Emergency Department of a secondary referral hospital in Klungkung Regency, exhibiting signs of acute distress following a thermal trauma. The incident had occurred approximately 30 minutes prior to hospital admission in the domestic setting. According to the anamnesis provided by the guardians, the patient had been attempting to retrieve an object from an elevated surface when he accidentally destabilized a vessel containing boiling water. This resulted in a high-volume spill, precipitating an extensive scald injury predominantly affecting the posterior aspect of the body. Upon arrival at the triage station, the patient was alert but agitated, demonstrating a Glasgow Coma Scale (GCS) of 15. He was crying vigorously, a clinical indicator of patent airway and intact neurological sensation, albeit overwhelmed by nociception. The subjective pain assessment utilizing the Visual Analog Scale (VAS) indicated severe distress, with a score of 8/10.

The primary survey revealed a patent airway with no evidence of inhalational injury, stridor, or soot deposition in the oropharynx. Breathing was spontaneous and effective, with a respiratory rate of 18 breaths per minute and an oxygen saturation (SpO_2) of 98% on room air. Hemodynamic assessment indicated a compensated cardiovascular state. The heart rate was elevated at 112 beats per minute, a tachycardia consistent with the dual stimuli of severe pain and the incipient fluid shifts characteristic of the acute phase of burn shock. The blood pressure was recorded at 118/65 mmHg, and capillary refill time (CRT) was preserved at less than 2 seconds, suggesting that systemic perfusion remained adequate despite the developing localized hypovolemia. The patient's medical history was unremarkable, with no documented co-morbidities such as diabetes mellitus, autoimmune pathologies, or prior surgical interventions that would complicate wound healing. However, the dietary history obtained from the parents revealed a concerning pattern of nutritional inadequacy. The child was described as a selective eater, with a refusal to consume vegetables and fruits, and a dietary reliance on snacks with high sodium and refined carbohydrate content. This history raised an immediate red flag regarding potential micronutrient deficiencies and low somatic protein reserves.

A detailed dermatological examination was performed to assess the extent and depth of the thermal injury. The lesions presented as extensive, moist, erythematous plaques interspersed with large, tense bullae and areas of detached epidermis. The wound bed appeared pink-to-red and demonstrated blanching upon digital pressure, confirming the integrity of the deep dermal vascular plexus. These clinical features were diagnostic of deep partial-thickness (Grade IIB) burns. In these injuries, the thermal damage extends into the reticular dermis but spares the hair follicles and sweat glands, which are critical for re-epithelialization. The localized pain was intense, confirming that the free nerve endings were exposed and sensitized rather than destroyed, as would be seen in full-thickness injuries. The total body surface area (TBSA) was calculated utilizing the Lund and Browder chart, which offers superior accuracy for pediatric body proportions compared to the rule of nines (Figure 1). The scald pattern was consistent with a gravity-dependent flow of hot liquid down the posterior chain: (1) Posterior Right Humerus: 3%; (2) Lumbosacral Region: 5%; (3) Gluteus Maximus (Bilateral): 5%; (4) Posterior Right and Left Femoral: 10%. The cumulative assessment confirmed a 23% TBSA injury. This extent of injury is clinically significant, surpassing the 20% threshold that typically delineates a major pediatric burn capable of triggering a systemic inflammatory response syndrome (SIRS) and a hypermetabolic cascade.

Figure 1. Clinical figures of the patient on admission.

Given the critical role of substrate availability in burn survival, a comprehensive nutritional assessment was prioritized and conducted within two hours of admission by the Clinical Nutrition Specialist. The anthropometric data revealed a profound discordance between the patient's chronological age and his somatic growth; Current Weight: 16 kg; Height: 117 cm; Body Mass Index (BMI): 11.69 kg/m². To contextualize these parameters, a Z-score analysis was performed using the World Health Organization (WHO) Growth Standards. The results indicated severe chronic and acute malnutrition. The Weight-for-Age Z-score (WAZ) was -2.57 SD, classifying the patient as underweight. More alarmingly, the BMI-for-Age Z-score (BAZ) was -3.59 SD, indicative of severe wasting. This metric suggested a near-total depletion of adipose tissue and significant muscle atrophy prior to the injury. The Height-for-Age Z-score (HAZ) of -0.6 SD fell within the normal range, implying that the malnutrition was likely acute or sub-acute (wasting) rather than chronic stunting. The STRONGkids (Screening Tool for Risk on Nutritional Status and Growth) screening was administered, yielding a score of 2. This categorized the patient as medium risk, necessitating immediate and specialized nutritional intervention to prevent further deterioration during the catabolic flow phase of the burn injury.

The admission laboratory panel provided a window into the patient's acute physiological derangements. The hematological profile showed a Hemoglobin level of 16.3 g/dL and a Hematocrit of 49.8%. In a pediatric patient with documented malnutrition, these elevated values did not represent polycythemia but rather acute hemoconcentration. This phenomenon is a hallmark of the immediate post-burn period, driven by the widespread increase in capillary permeability (capillary leak syndrome) that allows plasma to extravasate into the interstitial space, leaving the cellular components of blood concentrated in the intravascular compartment. The leukocyte count was elevated at 17,270 /microL. Given the short time interval since the injury (30 minutes), this leukocytosis was interpreted not as a sign of infection, but as a stress leukocytosis resulting from the demargination of neutrophils in response to the massive surge of endogenous catecholamines and cortisol.

Perhaps the most critical finding was the serum albumin level of 2.3 g/dL. This represents significant hypoalbuminemia. While burn injury causes an immediate drop in albumin due to vascular leakage, a level this low upon presentation, combined with the anthropometric evidence of wasting, strongly suggested a pre-existing state of nutritional compromise. The low albumin creates a dual challenge: it reduces the oncotic pressure, exacerbating tissue edema and third-spacing of fluids, and it signifies a lack of visceral protein reserves necessary for transport of therapeutics and wound healing. Electrolytes were relatively preserved (Sodium 136 mmol/L, Potassium 3.8 mmol/L, Chloride 101 mmol/L), indicating that while fluid shifts were occurring, renal regulation remained intact in the immediate period. In synthesis, this was a 7-year-old male with severe pre-existing protein-energy malnutrition (Severe Wasting, BAZ -3.59 SD) presenting with a major thermal injury (23% TBSA Deep Partial-Thickness). The patient entered the hospital in a state of compensated shock with evidence of significant hemoconcentration and acute-on-chronic hypoalbuminemia. This clinical picture identified him as a patient at exceptionally high risk for metabolic decompensation, wound conversion, and delayed healing, thereby mandating the aggressive, targeted nutritional strategy detailed in the subsequent therapeutic intervention.

The therapeutic management of this pediatric burn patient necessitated a synchronized, dual-modality approach integrating acute surgical care with metabolic resuscitation. Upon admission, the immediate priority was the restoration of intravascular volume to counter the systemic capillary leak syndrome characteristic of burns exceeding 20% TBSA. We initiated fluid resuscitation utilizing the Parkland Formula, calculated at 4 ml per kilogram of body weight per percentage of total body surface area burned (4 ml x 16 kg x 23% TBSA). Ringer's Lactate was selected as the crystalloid of choice due to its composition closely mirroring plasma electrolytes and its buffering capacity against metabolic acidosis. Concurrently, pain management was prioritized to attenuate the sympathetic surge driven by nociception, which can further exacerbate the hypermetabolic state. Analgesia was effectively managed with intravenous Paracetamol, avoiding the respiratory depression associated with high-dose opioids while providing adequate comfort for wound manipulation. Local wound care adhered to strict aseptic protocols, involving careful debridement of necrotic tissue and ruptured bullae, followed by the application of silver sulfadiazine. This topical antimicrobial agent was chosen for its broad-spectrum efficacy against gram-negative bacteria, thereby minimizing the risk of wound colonization and invasive sepsis.

Table 1. Summary of Clinical Findings, Physiological Status, and Nutritional Assessment on Admission

Category	Parameter	Finding/Value	Clinical Interpretation
DEMOGRAPHICS & INJURY PROFILE			
	Patient Profile	7-year-old Male	*Pediatric risk group*
	Mechanism	Scald (Hot Water)	*Domestic accident (30 mins prior)*
	TBSA (Lund & Browder)	**23%**	*Major Burn (>20%); High risk of SIRS*
	Burn Depth	**Grade IIB**	*Deep Partial-Thickness; Risk of conversion*
PRIMARY SURVEY & HEMODYNAMICS			
	Heart Rate	**112 bpm**	*Tachycardia (Pain/Inc. Hypovolemia)*
	Blood Pressure	118/65 mmHg	*Compensated Shock*
	SpO2	98% (Room Air)	*Adequate oxygenation; No inhalation injury*
	Pain Score (VAS)	**8 / 10**	*Severe distress; hypermetabolic trigger*
NUTRITIONAL STATUS (WHO STANDARDS)			
	BMI-for-Age (BAZ)	**-3.59 SD**	*Severe Wasting (Pre-existing Malnutrition)*
	Weight-for-Age (WAZ)	**-2.57 SD**	*Underweight*
	STRONGkids Score	**2 points**	*Medium Risk; Intervention Required*
BIOCHEMISTRY & LABS			
	Serum Albumin	**2.3 g/dL**	*Severe Hypoalbuminemia; Catabolic state*
	Hemoglobin	**16.3 g/dL**	*Hemoconcentration (Capillary Leak)*
	Leukocytes (WBC)	**17,270 /µL**	*Stress Leukocytosis (Demargination)*
	Hematocrit	**49.8%**	*Indicative of fluid shift/dehydration*

The cornerstone of the patient's recovery was a sophisticated medical nutrition therapy (MNT) protocol, designed to address the complex interplay between the acute thermal injury and the patient's chronic nutritional deficit. The nutritional diagnosis was established as Severe Protein-Energy Malnutrition precipitated by an acute hypercatabolic state and exacerbated by reduced oral intake due to pain (Table 2a). This diagnosis was clinically substantiated by anthropometric data revealing a BMI-for-age Z-score of -3.59 SD and a serum albumin level of 2.3 g/dL, indicating a critical depletion of both somatic and visceral protein reserves. Determining the precise caloric requirements for a pediatric burn patient is a delicate balance; underfeeding promotes autocannibalism of lean body mass, while overfeeding risks hepatic steatosis and respiratory compromise. We utilized the Curreri Junior formula to estimate energy needs. This equation is specifically validated for pediatric patients with burns exceeding 20% TBSA, as it uniquely accounts for the massive energy expenditure required for thermogenesis and tissue repair in a growing child.

The baseline calculation established a requirement derived from basal metabolic needs plus a burn-specific stress factor: Target Energy = (60 kcal x 16 kg) + (35 kcal x 23% TBSA) = 1,765 kcal. However, recognizing the patient's severe pre-existing malnutrition and the imperative need for catch-up growth alongside wound repair, we clinically adjusted the target upwards. The final caloric goal was set at 2,500 kcal/day. This equates to approximately 150 kcal/kg/day, a robust target intended to shift the patient from a catabolic to an anabolic state.

The protein requirement was calculated with equal aggression to counteract the massive proteolysis driven by cortisol and cytokines. The formula utilized was: Target Protein = (3 g x 16 kg) + (1 g x 23% TBSA) = 71 g/day. Given the severity of the hypoalbuminemia (2.3 g/dL), this target was clinically adjusted to a range of 3.0 to 4.0 g/kg/day, totaling approximately 105 grams of protein daily. This high-protein strategy aimed to provide sufficient amino acid substrates for acute-phase protein synthesis and gluconeogenesis without depleting the patient's skeletal muscle reservoir.

To achieve these ambitious targets without inducing gastrointestinal intolerance or refeeding syndrome, a stepwise dietary strategy was implemented (Table 2b). The macronutrient distribution was calibrated to 55% carbohydrates, prioritizing complex sources to manage glycemic control; 20-25% protein of High Biological Value (HBV); and 20-25% fat, incorporating Medium Chain Triglycerides (MCTs) to facilitate direct absorption independent of bile salts. Crucially, the protocol integrated specific immunonutrients to modulate the inflammatory response and support cellular repair: (1) Glutamine: Supplemented at 0.3 g/kg/day via the specialized formula Neomune. Glutamine served as the primary fuel source for enterocytes, maintaining gut barrier integrity to prevent bacterial translocation, while simultaneously supporting lymphocyte proliferation during the acute stress phase; (2) Zinc: Administered as 20 mg elemental zinc every 12 hours. Zinc acted as an essential cofactor for metalloenzymes, including DNA polymerase and RNA polymerase, which are rate-limiting enzymes for cell division and re-epithelialization; (3) Vitamin C**:** Provided at 50 mg/day. Ascorbic acid was essential for the hydroxylation of proline and lysine residues in procollagen, a necessary step for the formation of stable, cross-linked collagen in the healing wound bed.

The nutritional rehabilitation followed a carefully monitored timeline to ensure physiological adaptation. On day 1, the intervention commenced cautiously at 1,800 kcal (75% of the ultimate target). The patient was started on a liquid diet supplemented with a polymeric enteral formula (Entrakid), administered as 200ml three times daily. This initial phase aimed to prime the gut and assess tolerance. Day 2, the caloric load was escalated to 2,100 kcal. The patient reported transient nausea, a common complication of high-volume enteral feeding and opioid/stress interactions. We responded by increasing the frequency of meals while decreasing the volume per serving (fractionated feeding). On this day, the immunonutrition formula (Neomune) was introduced to begin the specific modulation of the immune response. Day 3, the patient achieved the full caloric target of 2,500 kcal. Oral intake improved significantly as pain management stabilized. Clinical inspection of the wounds revealed a healthy wound bed with minimal slough, suggesting that the nutritional substrates were effectively reaching the peripheral tissues. Day 5, the patient demonstrated excellent tolerance to the high-calorie, high-protein regimen, consistently consuming 100% of the targeted intake.

By the fifth day of hospitalization, the aggressive nutritional intervention yielded quantifiable and remarkable clinical improvements. The most significant metabolic indicator of success was the rapid correction of serum albumin. Levels rose from a critical 2.3 g/dL on admission to 3.1 g/dL by discharge. This swift normalization—occurring over just five days—indicated a successful blunting of the catabolic response. It suggested that the provision of adequate protein and energy had allowed the liver to shift from prioritizing acute-phase reactants back to synthesizing constitutive proteins, thereby restoring oncotic pressure and reducing systemic edema. The impact of the Zinc and Vitamin C supplementation was visibly evident in the local wound status. Significant granulation tissue was observed at the wound edges (Figure 2). The wounds appeared clean, with healthy vascularization and no evidence of invasive infection or sepsis, validating the efficacy of the combined nutritional and antimicrobial strategy. Contrary to the typical weight loss observed in the acute phase of burns due to hypermetabolism, this patient demonstrated a weight gain from 16.0 kg to 16.4 kg. In the context of resolving edema (indicated by rising albumin), this weight gain likely represented a positive nitrogen balance and the preservation of lean body mass, rather than fluid retention alone. This outcome confirmed that the MNT protocol successfully met the immense metabolic demands of the injury while supporting the growth requirements of the pediatric patient.

Figure 2. Wound healing in patients.

Table 2a. Clinical Diagnosis and Acute Surgical Management (Phases I & II)

Phase / Category	Diagnosis / Action	Specific Findings & Protocol
PHASE I: DIAGNOSIS & ASSESSMENT		
Burn Profile	**Thermal Injury (Scald)**	• **Extent:** 23% TBSA (Lund & Browder) • **Depth:** Grade IIB (Deep Partial-Thickness) • **Site:** Posterior humerus, lumbosacral, gluteus, femoral.
Nutritional Risk	**Severe Malnutrition (PEM)**	• **Severe Wasting:** BMI/Age Z-Score -3.59 SD • **Underweight:** Weight/Age Z-Score -2.57 SD • **Screening:** STRONGkids Score 2 (Medium Risk)
PHASE II: ACUTE MEDICAL MANAGEMENT		
Resuscitation	**Fluid Replacement**	**Protocol:** Parkland Formula. **Dosage:** 4 ml x 16 kg x 23% TBSA using Ringer's Lactate. *Goal:* Restore intravascular volume & prevent burn shock.
Wound Care	**Debridement & Antimicrobial**	**Agent:** Silver Sulfadiazine (SSD). **Action:** Debridement of necrotic tissue and bullae. *Goal:* Prevent colonization (esp. *P. aeruginosa*).

Table 2b. Medical Nutrition Therapy Protocol and Clinical Outcomes (Phases III & IV)

Intervention Target	Strategy / Outcome	Methodology & Result Details
PHASE III: MEDICAL NUTRITION THERAPY (MNT)		
Energy Targets	**Curreri Junior Formula**	**Goal:** 2,500 kcal/day (~150 kcal/kg/day). *Adjustment:* Clinically increased to account for severe catch-up growth requirements beyond basal + stress needs.
Protein Targets	**High-Protein Load**	**Goal:** 3.0 - 4.0 g/kg/day (~105 g/day). *Function:* Substrate for acute-phase proteins and gluconeogenesis to prevent muscle wasting.
Immunonutrition	**Micronutrient cocktail**	• **Glutamine (0.3 g/kg):** Via Neomune formula (Gut/Immune support). • **Zinc (20mg/12h):** DNA polymerase cofactor for healing. • **Vitamin C (50mg/24h):** Collagen cross-linking.
PHASE IV: FOLLOW-UP & OUTCOMES (DAY 5-10)		
Biochemistry	**Albumin Correction**	Serum Albumin rose from 2.3 g/dL to 3.1 g/dL within 5 days.
Wound Status	**Granulation**	**Significant granulation tissue observed.** No invasive infection or sepsis.
Anthropometry	**Anabolism**	Weight gain: +0.4 kg (16.0 to 16.4 kg). Indicates positive nitrogen balance.

3. Discussion

The management of pediatric burns requires a nuanced understanding of the profound physiological alterations that occur post-injury. In the context of a developing child, a thermal injury is not merely a dermatological event but a systemic metabolic catastrophe.[11] This case demonstrates that aggressive, targeted nutrition—when delivered with precision regarding timing and composition—is not merely supportive care but a primary therapeutic intervention capable of altering the clinical trajectory. The thermal injury triggers an immediate and violent hypothalamic response, initiating what Cuthbertson classically described as the ebb and flow phases.[12] Following the initial ebb phase of shock, the patient transitions into the flow phase, a persistent hypermetabolic state driven by a neuroendocrine axis surge. The massive release of catecholamines (epinephrine, norepinephrine), glucagon, and cortisol creates a milieu of insulin resistance and intense catabolism.

In the pediatric population, this response is distinctively more hazardous than in adults. Children possess a significantly higher surface-area-to-mass ratio, leading to rapid evaporative heat loss.[13] To maintain thermal homeostasis, the pediatric metabolic rate can elevate by up to 100% above the resting baseline, creating an immense energy deficit. Furthermore, children have limited hepatic glycogen reserves. In a healthy child, these stores are depleted within 12 to 16 hours of fasting; in a burn-injured child, they may be exhausted in half that time. Once glycogen is depleted, the body aggressively shifts to proteolysis (muscle breakdown) to harvest amino acids—specifically alanine and glutamine—to serve as substrates for hepatic gluconeogenesis. This physiological autocannibalism is the body's desperate attempt to provide glucose to the brain and the obligate glycolytic wound bed. In our patient, this danger was compounded by severe pre-existing malnutrition, evidenced by a BMI-for-age Z-score of -3.59 SD. With virtually no adipose reserve to provide fatty acids and depleted somatic protein stores, this child was at imminent risk of respiratory muscle failure and immune collapse. Without the provision of exogenous high-protein substrates (3–4 g/kg/day), the patient would have inevitably spiraled into a negative nitrogen balance so severe that wound closure would become physiologically impossible.[14]

Determining the caloric target in pediatric burns is a subject of ongoing debate, with various equations such as the Galveston, Schofield, and World Health Organization formulas offering differing estimations.[15] However, this case validates the utility of the Curreri Junior formula in the context of severe burns (>20% TBSA) in low-resource settings where indirect calorimetry is unavailable. The Curreri Junior formula is specifically designed to account for the dual metabolic burdens unique to this demographic: the massive energy expenditure required for thermal regulation and tissue repair, and the non-negotiable energy cost of somatic growth. By targeting an aggressive 2,500 kcal/day (approximately 150 kcal/kg), we deliberately exceeded the standard measured resting energy expenditure (REE). This hyper-alimentation strategy was critical. The logic posits that in a severely wasted child (Z-score <-3 SD), meeting maintenance needs is insufficient; one must provide a catch-up surplus to arrest catabolism and drive anabolism. The clinical outcome supports this methodology. The patient achieved a weight gain of 0.4 kg over a 5-day acute period. In the typical course of burn injury, patients are expected to lose weight due to the catabolic consumption of lean body mass. Reversing this trend in the acute phase confirms that the caloric provision calculated by the Curreri Junior formula was sufficient to satisfy the hypermetabolic demand and spare the remaining muscle mass, effectively shifting the patient from a catabolic wasting state to an anabolic recovery state.

A defining aspect of this case was the transition from standard macronutrient support to immunonutrition—the use of specific nutrients in pharmacologic doses to modulate immune activity and wound healing.[16] The synergistic triad of Glutamine, Zinc, and Vitamin C proved pivotal. Under normal physiological conditions, glutamine is the most abundant free amino acid in the body. However, severe burns create a glutamine debt where consumption by the kidneys (for acid-base balance), the liver (for acute-phase proteins), and the wound (for fibroblast activity) outstrips skeletal muscle production. Glutamine is the primary fuel source for enterocytes and rapidly dividing lymphocytes. By supplementing 0.3 g/kg/day via the Neomune formula, we likely preserved the integrity of the intestinal mucosal barrier. This is critical in preventing gut-origin sepsis, a phenomenon where bacteria translocate across a compromised gut wall into the systemic circulation. Furthermore, glutamine promotes the synthesis of heat shock proteins (HSP), which protect cells from thermal and oxidative stress.[17]

The rapid appearance of granulation tissue in our patient correlates directly with aggressive Zinc supplementation. Zinc is a ubiquitous cofactor for over 300 metalloenzymes, including RNA polymerase and DNA polymerase, which are the rate-limiting enzymes for cell division.[18] In the proliferative phase of wound healing, cellular mitosis is widespread; without adequate zinc, this process halts. Additionally, zinc is essential for the function of Matrix Metalloproteinases (MMPs), the enzymes responsible for clearing devitalized tissue and remodeling the extracellular matrix. The zinc trap phenomenon, where the liver sequesters serum zinc during inflammation, often leads to functional deficiency in the wound bed. Our supplementation (20 mg/12h) overcame this sequestration, ensuring bioavailability at the tissue level.

Vitamin C (ascorbic acid) acts as an obligatory cofactor for the enzymes prolyl hydroxylase and lysyl hydroxylase. These enzymes catalyze the hydroxylation of proline and lysine residues on procollagen chains, a biochemical step necessary for the formation of the triple-helix structure of collagen. Without this cross-linking, the new tissue is friable and weak. Furthermore, burns generate a massive surge of reactive oxygen species (ROS) that perpetuate tissue damage. As a potent water-soluble antioxidant, Vitamin C scavenges these free radicals, reducing capillary permeability and protecting the microvasculature. The clinical observation of healthy, non-edematous granulation tissue suggests that the Vitamin C supplementation successfully supported collagen architecture and reduced oxidative stress.

The biochemical trajectory of this patient—specifically the rise in serum albumin from 2.3 to 3.1 g/dL within 5 days —offers profound insight into the success of the intervention. Albumin is a negative acute-phase protein. During the active inflammatory phase, Interleukin-6 (IL-6) signals the liver to downregulate albumin synthesis in favor of positive acute-phase reactants like C-reactive protein (CRP) and fibrinogen.[19] Therefore, a persistent low albumin level reflects ongoing, uncontrolled systemic inflammation and capillary leak (third-spacing). The rapid normalization observed in this case is clinically significant. It suggests that the combined nutritional intervention and wound control successfully dampened the systemic inflammatory response syndrome (SIRS). The provision of adequate amino acids allowed the liver to escape the acute-phase lock and resume constitutive protein synthesis. This rise in albumin not only restores oncotic pressure, reducing edema, but also facilitates the transport of pharmacological agents and hormones essential for recovery (Figure 3).

This study is limited by its design as a single case report, which precludes broad statistical generalization. Additionally, due to setting constraints in a secondary referral hospital, advanced metabolic monitoring tools such as Indirect Calorimetry (the gold standard for energy expenditure) or Nitrogen Balance studies (via 24-hour Urine Urea Nitrogen) were not available. We relied on predictive equations and clinical surrogates (weight, albumin, wound status), which, while practical, lack the precision of isotopic metabolic studies. Future research in the Southeast Asian context should focus on randomized controlled trials (RCTs) comparing standard high-protein diets versus immunonutrition-enriched protocols. Specifically, investigating the cost-benefit analysis of early enteral immunonutrition in preventing sepsis and reducing the length of stay in pediatric burns would be of high value for global health guidelines.[20]

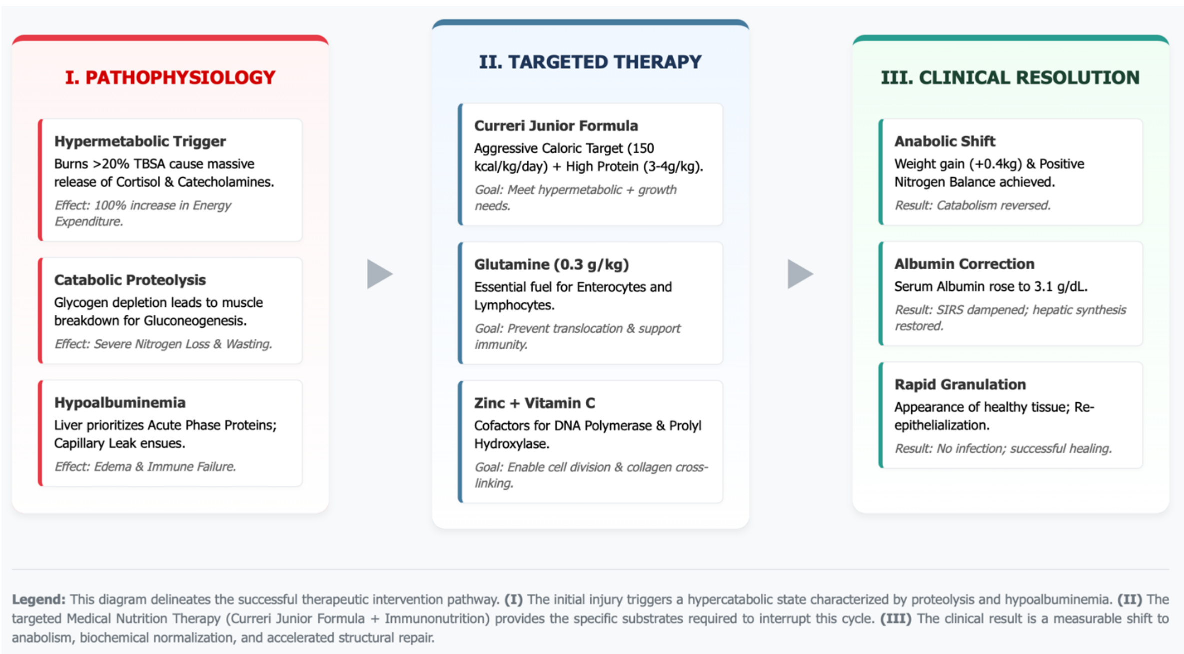


Figure 3. Metabolic resuscitation pathway.

4. Conclusion

This case report validates the critical importance of early, aggressive medical nutrition therapy (MNT) in the management of pediatric burns, particularly in the context of pre-existing malnutrition. The findings challenge the outdated paradigm of nutrition as supportive care and elevate it to the status of a primary therapeutic modality, equal in importance to fluid resuscitation and surgical debridement. The utilization of the Curreri Junior formula proved highly effective in establishing a caloric target (approximately 150 kcal/kg/day) that met the dual demands of hypermetabolism and catch-up growth. This metabolic resuscitation, when combined with a high-protein intake (>3 g/kg/day), successfully arrested the catabolic spiral of muscle wasting that threatens the survival of malnourished children. Furthermore, the targeted supplementation of Glutamine, Zinc, and Vitamin C demonstrated a clear clinical benefit. These immunonutrients acted synergistically to restore gut barrier integrity, fuel the immune response, and provide the enzymatic cofactors necessary for rapid collagen synthesis and re-epithelialization. The rapid correction of severe hypoalbuminemia (from 2.3 g/dL to 3.1 g/dL in 5 days) and the accelerated appearance of healthy granulation tissue serve as objective validators of this protocol. These outcomes underscore a vital lesson for clinicians in resource-limited settings: in patients with the double burden of burn injury and malnutrition, the wait and see approach to nutrition is detrimental. Immediate enteral feeding, calculated with precision and enriched with specific micronutrients, is essential to blunt the hypermetabolic response, prevent immune failure, and ensure optimal functional recovery. This case provides a replicable framework for metabolic management that can significantly improve morbidity and mortality in the vulnerable pediatric burn population.

5. References

1. Yang HT, Yim H, Cho YS, Kim D, Hur J, Kim JH, et al. Serum transthyretin level is associated with clinical severity rather than nutrition status in massively burned patients. JPEN J Parenter Enteral Nutr. 2014; 38(8): 966–72.
2. Hammad SM, Naser IA, Taleb MH, Abutair AS. Dietary intake and Biochemical Indicators and their association with wound healing process among adult burned patients in the Gaza Strip. Curr Res Nutr Food Sci J. 2019; 7(1): 169–81.
3. Yang Y-J, Liu M-M, Zhang Y, Wang ZE, Dan-Wu, Fan S-J, et al. Effectiveness and mechanism study of glutamine on alleviating hypermetabolism in burned rats. Nutrition. 2020; 79–80(110934): 110934.
4. Ma Z, Zhang Y, Zhang Q, Wu B. Modified Nutrition Risk in Critically ill is an effective nutrition risk screening tool in severely burned patients, compared with Nutrition Risk Screening 2002. Front Nutr. 2022; 9: 1007885.
5. Linden MA, Teixeira TL de M, Freitas RGB de ON, Brandão MÂB, Nogueira RJN. Arginine and glutamine supplementation on transthyretin levels in severely burned patients: a systematic review. Nutrition. 2022; 101(111657): 111657.
6. Hurtado-Torres GF. Are minor burned patients really hypermetabolic? JPEN J Parenter Enteral Nutr. 2024; 48(7): 776.
7. Perniciaro G, Bonifazio L, Angelomè C, Ferrante M, Corsaro E, Vaccaro G. Clinical outcomes of burned patients receiving early enteral immunonutrition: a retrospective analysis. Clin Nutr Open Sci. 2025.
8. Zhang Y, Dou Y, Yin Z, Gao M, Wu B, Zhang Q. Initiation of adaptive feeding within 24 h after burn injury improves nutritional therapy for severely burned patients. Front Nutr. 2024; 11: 1342682.
9. Rugg C, Ströhle M, Schmid S, Kreutziger J. The link between hypermetabolism and hypernatremia in severely burned patients.

Nutrients. 2020; 12(3): 774.
10. Aktaç Ş, Başoğlu İA, Cebeci A, Oguz E, Ömeroğlu B. Determination of energy and nutrient intakes in pediatric burned patients. Clin Exp Health Sci. 2021; 11(4): 689–94.
11. Kamal MM, Aziz F, Islam MR, Ahsan M, Islam SN. Effect of immunonutritional status, healthcare factors, and lifestyle on acute respiratory infections among under-5 children in Bangladesh. SAGE Open Med. 2020; 8: 2050312120940533.
12. Pannirselvam M, Zakaria Z, Wong MP-K, Abdul Satar MHS, Jusoh NS, Zakaria AD, et al. Effects of preoperative immunonutrition vs standard oral nutrition in patients undergoing colorectal surgery: a randomized controlled trial. World J Gastrointest Surg. 2025; 17(9): 108127.
13. Ohashi W, Yoneda K, Kanda D, Ueda K, Ito H, Takegawa R, et al. Wound management strategy for burn injuries during pelvic external fixation: a case report on the practical use of negative pressure wound therapy. J Burn Care Res. 2025; (iraf231).
14. Crawley WT, Pulikkottil B, Busch M. Negative pressure wound therapy in the setting of acute abdominal evisceration secondary to major thoracoabdominal burns. J Surg Case Rep. 2025; 2025(4): rjaf203.
15. Yudiasti DA, Saputro ID. Management of pediatric minor burn using hydroactive wound dressing: a case report. Int J Sci Adv. 2025; 6(5).
16. Verma M. Pediatric burn wound complicated with herpes simplex virus infection. Ind J Burn. 2021; 29(1): 87–9.
17. Miller C, Edwards I, Osterbauer M, Crane S, Kesey J, Griswold J. The impact of emotion-focused therapy interventions on wound care anxiety in a pediatric patient with severe burns: a case report. Burns Open. 2025; 12(100422): 100422.
18. Liu Y, Zhang J. Management of pediatric deep partial-thickness burn wounds. Zhonghua Shao Shang Za Zhi. 2021; 37(8): 797–800.
19. Wang J, Yang B, Zhang X-H, Liu S-H, Wu W. The effectiveness of silver-containing hydrofiber dressing compared with topical silver sulfadiazine cream in pediatric patients with deep partial-thickness burns: a retrospective review. Wound Manag Prev. 2022; 68(3): 29–36.
20. Liu H, Wang S, Li D, Li D, Zhang Y, Li X, et al. Characteristics and significance of blister fluid cell-free mitochondrial DNA in pediatric small area intermediate-depth burn wounds. Burns. 2025; 52(1): 107780.