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Case Report

Analysis of the nutritional approach and recovery in children with flame burns: case report and review of the literature

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Abstract: Pediatric burn injuries present unique challenges due to children's physiological vulnerabilities. This article provides a detailed analysis of the nutritional management of an 11-year-old patient with extensive burns affecting 60% of total body surface area. The patient received intensive care in a specialized pediatric burn unit, highlighting the fundamental role of nutrition in counteracting catabolic states and muscle loss frequently observed in these cases. Nutritional strategies-including gastrostomy infusion, albumin supplementation, and protein-enriched diets-were carefully implemented to optimize energy intake and promote wound healing. The article also reviews the metabolic and immunological responses of burn patients, emphasizing the importance of early nutritional support to mitigate hypermetabolism and enhance immune defense. Key elements of nutritional assessment, such as energy requirement estimation and macronutrient composition, are explored. Furthermore, the role of micronutrient supplementation in accelerating wound healing and reducing infectious complications is underscored. The article concludes by highlighting the evolving landscape of pediatric burn care, stressing the importance of interdisciplinary collaboration and the integration of advanced technologies to achieve precise nutritional interventions. This case study provides valuable insights into optimizing nutritional strategies for pediatric burn patients and contributes to the advancement of pediatric critical care.

Keywords: Burn, pediatric, nutrition, therapy, metabolism, hypermetabolism, treatment

Introduction

According to data collected by the World Health Organization (WHO), up to the date of this review, 8,640 cases of burn patients have been reported. Of these, 42% were children, with the highest incidence occurring between the first and fifth year of life [1].

Pediatric burn patients present greater vulnerability to metabolic and inflammatory compromise due to unique anatomical and physiological factors [2], such as limited energy reserves, impaired central regulation of neurotransmitter release (e.g., epinephrine), and a high metabolic rate resulting from a larger body surface area and thinner dermis [3]. These characteristics increase the susceptibility of pediatric patients to fluid and heat loss, hypothermia, and metabolic instability. Furthermore, their immunological immaturity may complicate the response to injury, favoring exaggerated inflammatory and catabolic states [4, 5].

From a diagnostic perspective, despite decades of experience in burn management, persistent variations remain in the care of children with burns, particularly regarding the evaluation and clinical assessment of these patients. This is especially evident in the continuous overestimation of total body surface area (TBSA) burned and burn depth, often followed by disproportionate fluid administration, leading to either excessive or inadequate resuscitation [6, 7].

Following extensive burns, the physiological response of the body can remain active for months, with a marked catabolic state and

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rapid loss of muscle mass and bone density. It has been observed that even after definitive burn treatment, pediatric patients may continue to experience muscle mass loss up to 9 months after the incident, while growth disturbances may persist for as long as two years.

Therefore, nutritional intervention and regulation of the metabolic response are critical for comprehensive management, recovery, and rehabilitation of children with severe burns [8].

Current principles for the treatment of pediatric burn patients emphasize early and accurate assessment of burn extent, weight and TBSA-adjusted fluid resuscitation, infection prevention, pain management and sedation, as well as timely surgical interventions such as escharotomy, grafting, and wound coverage. Nutritional support, thermoregulation, and rehabilitation are integral components of care, requiring an interdisciplinary approach involving pediatric intensivists, surgeons, nutritionists, and rehabilitation specialists [9].

These principles differ from those applied in adults primarily because of the need for stricter monitoring and dynamic adjustments in fluid and nutritional requirements across pediatric age groups, as well as a long-term focus on growth, development, and psychosocial recovery [10, 11].

In this article, we present the nutritional approach applied to an 11-year-old patient with burns covering 60% of total body surface area, requiring extensive and prolonged management in a pediatric intensive care and burn unit. Additionally, a comprehensive review of the existing literature on nutritional management in burn patients is provided.

Case report

We present the case of an 8-year-old male patient who sustained burns involving 60% of his total body surface area following the explosion of a gas tank at his home. After the incident, he was immediately transferred to a tertiary hospital for stabilization and initial management. Interventions included orotracheal intubation, initiation of vasopressor and inotropic support, debridement of the burn wounds, and subsequent application of petroleum jelly. The patient was later referred to our institution.

Admission to our burn unit occurred 16 hours after the event, where an immediate bronchoscopy confirmed airway burn injuries, leading to the performance of a tracheostomy. Additionally, compartment syndrome was identified in both hands, requiring urgent surgical intervention.

Due to the need for immediate surgical procedures, such as gastrostomy placement, the patient remained fasting for 12 hours after admission. Enteral nutrition was subsequently initiated via the gastrostomy tube, using a hypercaloric polymeric formula. At admission, a nutritional diagnosis of overweight was established based on pre-burn data. The nutritional prescription covered 110% of the total energy requirements, adjusted for critical condition and burn surface area, using the Schofield equation for energy expenditure, later modified with correction factors. In addition to enteral nutrition, 20% albumin was administered for three days.

On day 12 of enteral feeding via gastrostomy, malfunction of the tube balloon was detected, requiring replacement without complications, allowing enteral feeding to continue. In parallel, speech and swallowing rehabilitation was initiated. For approximately one month, the patient received enteral nutrition via gastrostomy, with a progressive reduction in volume as oral tolerance improved, until the gastrostomy was successfully closed.

Subsequently, the patient developed wound dehiscence at the gastrostomy closure site, requiring surgical intervention and suspension of oral feeding. In this context, parenteral nutrition was instituted for 12 days, followed by successful transition to oral feeding, ultimately achieving 100% of intake orally with a hypercaloric, high-protein diet.

During hospitalization, evaluations revealed a decrease in fat percentage through brachial circumference measurements, which prompted the inclusion of a protein supplement. Indirect calorimetry confirmed a eutrophic nutritional state, allowing objective adjustment of caloric intake according to energy needs and facilitat-

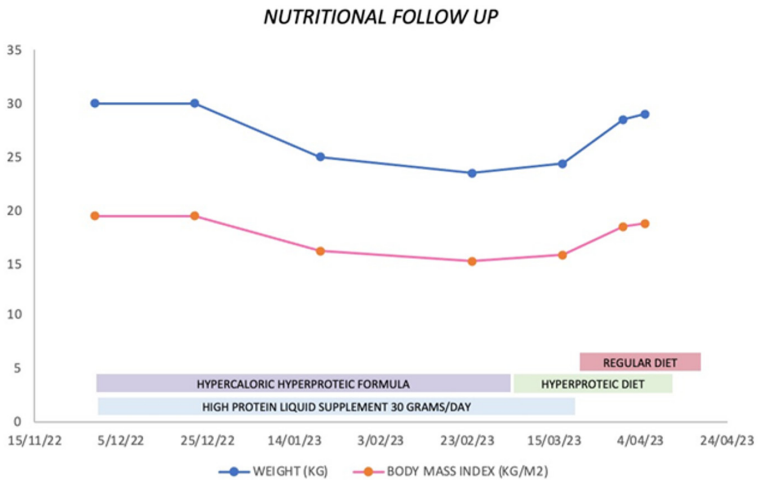

Figure 1. The image shows the patient's weight trend during hospitalization and its relationship to the nutritional strategy implemented, with a safe discharge weight achieved at the time of hospital release.

ing hospital discharge with a eutrophic nutritional classification **Figure 1**.

For the management of secondary hypermetabolic syndrome, the patient received treatment with beta-blockers and immunonutrition with glutamine for 10 days. In addition to meeting caloric requirements, vitamins, antioxidants, and minerals were administered from the start of enteral nutrition, a treatment that was maintained for 90 days after hospital discharge.

During hospitalization, infection markers were monitored, including leukocyte count, C-reactive protein (CRP), procalcitonin, and interleukin-6 (**Table 1**). These parameters showed transient elevations following surgical procedures, without correlation with sustained clinical sepsis. Infections were documented throughout the hospital stay including bloodstream infections and wound cultures requiring escalation to broad-spectrum antibiotics (**Appendix 1**).

These findings suggest that the combined strategy of early optimization of nutrition, immunonutrit, and strict infection prevention protocols may contribute to reducing the risk of septic complications.

Review

Metabolism in burned patients

The metabolic response in burn patients, especially when burns affect more than 15-20% of the total body surface, is characterized by being significantly more intense than the response observed in trauma patients. This phenomenon triggers systemic alterations, a heightened stress response, modifications in the immune response, and total body water redistribution [8].

A shift in lipid and glucose metabolism has been documented in these patients, marked by a decrease in insulin sensitivity [12]. The loss of circulating volume leads to increased production of counterregulatory hormones, resulting in a hypercatabolic state characterized by an elevation in the glucagon/insulin ratio [8, 12].

This hormonal response translates into a notable increase in epinephrine and norepinephrine levels, especially when the affected body surface exceeds 30-40%. Clinically, this manifests in symptoms such as tachycardia and hypertension, among others [4].

The hepatic glucagon shift has several consequences, including an increase in hemoglobin and plasma protein levels, a reduction in coagulation times, an enhancement in lipolysis, thermogenesis, and relaxation of the gastrointestinal smooth muscle. **Figure 2** graphically summarizes the metabolism in burn patients. It is crucial to note that this metabolic profile tends to vary between girls and boys, generally being less pronounced in the former [4, 8].

Metabolic and immunological response in burn patients

Hypothalamic stimulation increases the production of counterregulatory hormones, leading to lipolysis and proteolysis. This, in turn, enhances glucose production and muscle degradation for hepatic gluconeogenesis [4].

The magnitude of this metabolic response is linked to the extent of the burn and may persist for 9-12 months after the injury [4, 8, 12]. In addition to the triggered metabolic changes (gluconeogenesis, proteolysis, ureagenesis, nutrient sequestration, lipid metabolism alteration, etc.), the immune response in burn patients is also significantly affected, especially in

Table 1. Leukocyte count, C-reactive protein (CRP), procalcitonin, and interleukin-6

FECHA	WBC (/uL)	PCA (ng/mL)	PCR (mg/dL)	IL-6 (pg/mL)
29.11.2022	64.000	20.9	4.2	
30.11.2022	28.600		7.9	
01.12.2022	13.500	13.83		
04.12.2022	8.100	1.3		
06.12.2022	16.100		31.9	
10.12.2022	20.300		19.4	
12.12.2022	20.300	0.57	13.5	
14.12.2022	19.900	1.35		574.88
18.12.2022	18.300	0.36	16.2	
20.12.2022	18.400	0.20	72.2	
24.12.2022	13.000	0.16	14.6	6.22
01.01.2023	8.100	0.67	17.0	
08.01.2023	8.900	0.15	15.4	
11.01.2023	14.100	2.25		
13.01.2023	9.300	1.69		
15.01.2023	5.500		18.6	
16.01.2023	8.200	0.55	15.4	
17.01.2023	9.500	0.32		
19.01.2023	7.100	0.59	18.5	
22.01.2023	8.800	30.44	13.0	
23.01.2023	8.200	10.70	11.2	
24.01.2023		4.24		
26.01.2023	12.100	1.36		
04.02.2023	8.400	0.38	28.0	
11.02.2023	12.700	0.06		
25.02.2023	9.100	0.05		
27.02.2023	8.600	0.05	5.0	
15.03.2023	5.200			

burns involving more than 15-20% of the total body surface. This is characterized by an exponential production of cytokines such as IL-1β, IL-6, IL-8, IL-10, and TNF-α [2, 3] (**Figure 3**).

While this inflammatory and hypermetabolic response is crucial for survival, uncontrolled presentation can lead to harm. To halt it, it is essential to eliminate the initial stimulus. However, once inflammatory mediators are activated, any secondary stimulus can reactivate it, increasing the risk of infection and multiorgan failure [2, 8].

Therefore, strategies to mitigate this response focus on modifying altered metabolic pathways and stimulating the anabolic pathway using drugs such as growth hormone, insulin, and propranolol, in addition to providing adequate nutrition from the outset [4, 8].

Energetic requirements in burn patients

Patients suffering from severe burns undergo a prolonged hypermetabolic state, the severity of which is associated with the extent of the burn, endocrine and inflammatory factors, age, and gender [13].

Energy requirements are significantly higher than baseline conditions and vary over time, peaking in the initial weeks following the burn but gradually decreasing thereafter [4]. However, it has been observed that beyond 50-60% of the total burned body surface area, energy requirements reach a minimum [14].

Various equations have been developed to estimate these energy needs, based on body weight and the percentage of burned body surface area. Among these formulas are To-

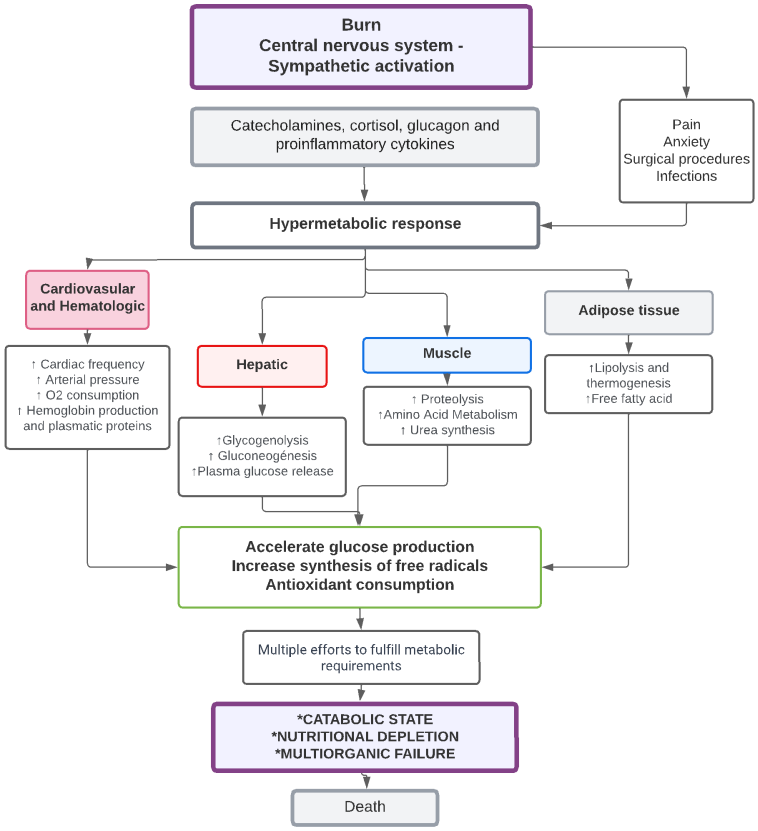

Figure 2. Metabolism in burned patients. This figure illustrates the metabolism in burned patients. Understanding this comprehensive metabolic response is crucial for tailored medical interventions and effective management strategies for burned patients. Authors' own work.

ronto, Schofield, Harris-Benedict, Mayes, WHO, and ASPEN [4, 14]. More accurate methods utilizing oxygen and carbon dioxide measurements have also been implemented to calculate energy expenditure [15]. Studies have revealed that these equations tend to overestimate caloric expenditure, potentially leading to issues such as hyperglycemia and pulmonary overload. Conversely, insufficient coverage of energy needs can result in malnutrition, weakened immune systems, prolonged dependence on mechanical ventilation, and delayed wound healing, increasing the risk of infection, morbidity, and mortality [14]. In this context, indirect calorimetry is considered the gold standard for measuring calorie expenditure, providing greater precision in assessing energy expenditure [4, 13, 14].

In the absence of this tool, the Schofield equation (**Table 2**) can be used for pediatric patients; however, it is important to note that this equation may underestimate requirements, necessitating rounding up the results [13]. Additionally, the inclusion of administered fluid volumes and the use of fat-soluble sedatives such as Propofol should be considered in the total energy calculation [4, 13].

Carbohydrates, proteins, and fats in burn patients

The metabolism of glucose is disrupted in burn patients due to the formation of glucose precursors, increased release of amino acids, and hepatic gluconeogenesis, leading to hyperglycemia and insulin resistance. Glucose levels >10 mmol/L are associated with delayed wound healing, infections, and multiple organ failure [8, 12].

Carbohydrates play a crucial role in retaining nitrogen in burn patients [4]. However, glucose doses >7 mg/kg/min do not convert into fat, so it is recommended to maintain carbohydrate delivery at 55-60%, not exceeding 5 mg/kg/min. Hypoglycemia also increases morbidity, so it is advised to maintain glucose at 100-150 mg/dL [12, 13] (**Table 3**).

Proteins

Optimizing protein production is fundamental in burn patients, correlating with skin regeneration and recovery rate [12]. A protein intake of 20-25% of total calories is suggested [4]. Protein intake of 2.5-4 g/kg is recommended for a caloric: nitrogen ratio of 80:1, adjusted according to renal function and fluid balance [4, 14].

Protein catabolism is not reversed by amino acids alone due to defects in their transport. Essential amino acids such as valine, leucine, and isoleucine are vital for tissue regeneration. Monitoring BUN, creatinine, and hydration status is essential in protein-rich diets to prevent

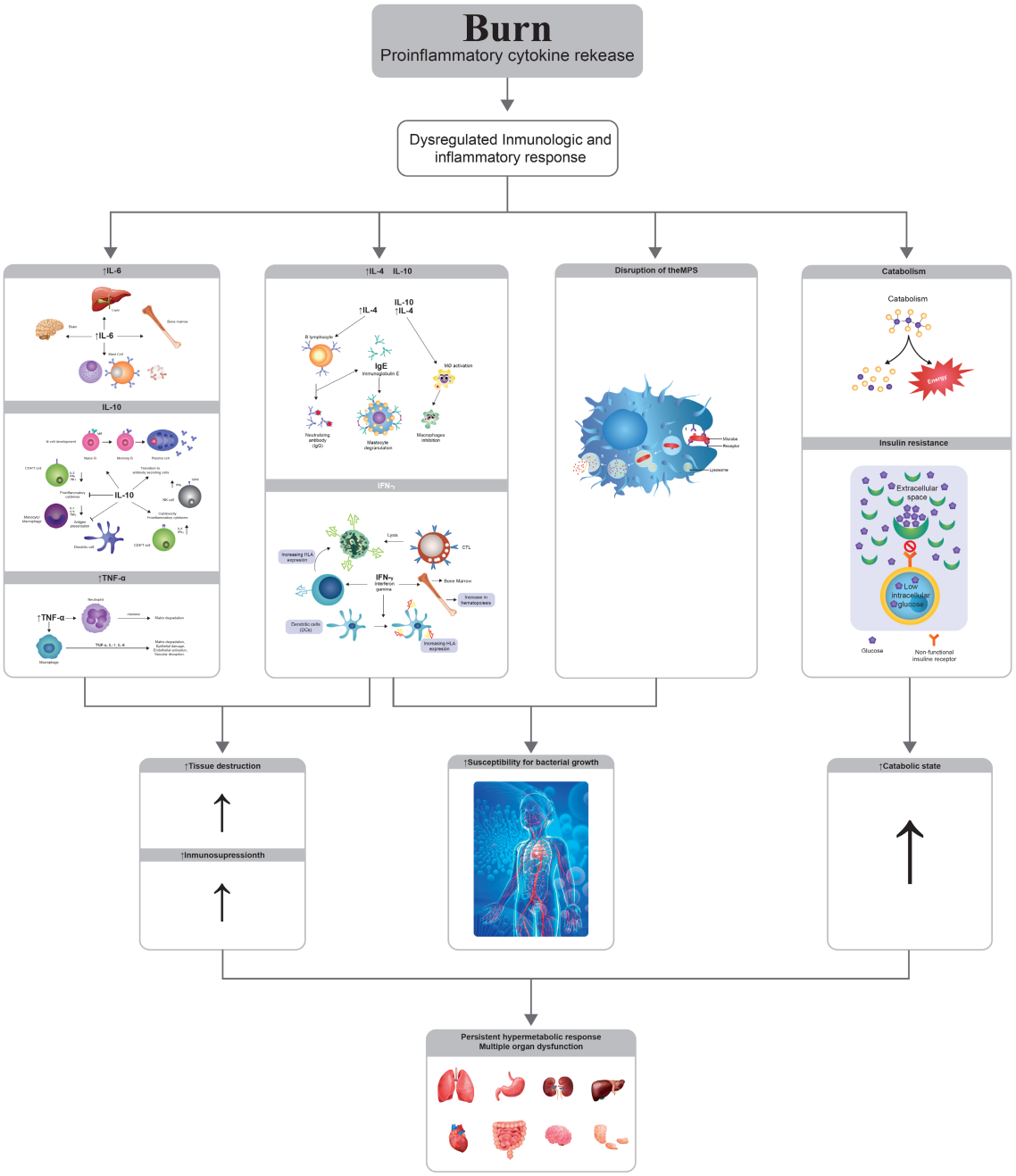

Figure 3. Immune response in burned patients. The figure illustrates the cascade of events in burn patients, where the initial activation in the hypothalamus triggers an inflammatory and hypermetabolic response. This response, characterized by the release of pro-inflammatory cytokines, predisposes the patient to infections and multiorgan failure.

prerenal azotemia and dehydration. Plant proteins have distinct amino acid profiles from animal proteins, with soy being an exception [12]. Albumin supplementation is not routinely recommended for serum levels <2.5 g/dL [4] (**Table 3**).

Fats

Lipids are essential for neurological development and the transport of fat-soluble vitamins. Essential fatty acids are crucial for cell membranes and protein synthesis. Linoleic acid is

Table 2. Energy requirement equations in burns

Age Category	Equation	Requirement (kcal/day)
Adults	Toronto	-4343 + (10.5 × % TBSA) + (0.23 × caloric intake) + (0.84 × REE by Harris-Benedict "crude") + (114 × t°) - (4.5 × days after injury)
Girls 3-10 yrs	Schofield	(16.97 × weight in kg) + (1.618 × height in cm) + 371.2
Boys 3-10 yrs	Schofield	(19.6 × weight in kg) + (1.033 × height in cm) + 414.9
Girls 10-18 yrs	Schofield	(8.365 × weight in kg) + (4.65 × height in cm) + 200
Boys 10-18 yrs	Schofield	(16.25 × weight in kg) + (1.372 × height in cm) + 515.5

Abbreviations: TBSA, total body surface area; REE, resting energy expenditure; t°, body temperature (°C).

Table 3. Nutritional targets by age group in burn care

Age group	Total fluids (mL kg^{-1} d^{-1})	Total calories (kcal kg^{-1} d^{-1})	Amino acids (g kg^{-1} d^{-1})	Fat (g kg^{-1} d^{-1})
Infants	135-150	90-100	2-2.5	2-3
Children	60-80	70-100	1.5-2.0	1-2
Adults	30-40	40-45	0.8-1.0	0.5-1.0

Adapted from Chan MM, Chan GM. Nutritional therapy for burns in children and adults.

recommended at around 2-3% of total calories [4]. The use of other fatty acids such as omega-3 needs further studies [13]. Burn patients are sensitive to high lipid loads, associated with prolonged hospital stays and a higher risk of infection if diets contain >35% lipids. Low lipid quantities are suggested [13]. **Table 5** summarizes the carbohydrate, fat, and protein needs in burn patients and the caloric goal [14].

Early identification and nutritional management are essential in burn patients at risk of malnutrition [15]. Malnutrition in burn patients is associated with a decreased quality of life, increased morbidity, mortality, and healthcare costs. It occurs due to an increased metabolic demand, inadequate use of nutrients, and alterations in absorption [16].

The hypermetabolic response after a burn can triple within 48 hours, leading to increased production of proteins and lipids, insulin resistance, loss of body mass, and immune suppression. Early nutritional management improves survival rates and organ function [16].

Pre-existing malnutrition in burn patients exacerbates nutritional stress, but the literature on its impact on the hypermetabolic state is limited. Nutritional screening tools underestimate malnutrition in burns, mainly due to difficulties in assessing nutritional status upon admission [15, 16] (**Table 3**).

Vitamins, antioxidants, and minerals

In addition to energy requirements, vitamins, antioxidants, and minerals are crucial in patients with severe burns. These patients have higher micronutrient needs due to the hypermetabolic response, demands in wound healing, and losses in skin and exudative wounds. Oxidative stress, associated with thermal injury and increased inflammatory response, depletes endogenous micronutrient-dependent antioxidants [13].

In the acute phase, there is a loss of serum iron, zinc, and selenium, along with an increase in serum copper, ferritin, and ceruloplasmin. These micronutrient losses manifest in the first 7 days after the burn, amplifying abnormal metabolic response and catabolism [4].

Inadequate administration of micronutrients can lead to deficiency syndromes that clinically manifest around the first month, causing delayed wound healing and an increased risk of infectious complications. Pre-established parenteral and enteral solutions with multivitamins and trace elements do not meet the needs of burn patients [13].

Studies have shown that vitamins B, C, E, D, and thiamine normalize lactate and pyruvate metabolism, reducing oxidative stress and improving wound healing. For patients with a

TBSA of 20% or more, a high dose of vitamin C is recommended during the acute phase (0.66 mg/kg/hour for 24 hours), stabilizing the endothelium, and reducing capillary leakage by 30% [13].

Micronutrients such as copper, selenium, and zinc are essential for improving immunity and wound healing in burn patients. Given their significant loss due to exudates, the duration of supplementation varies depending on the burned body surface area: 7-8 days for burns of 20-40%, 2 weeks for burns of 40-60%, and 30 days for burns greater than 60%. Early administration is associated with a decrease in lipid peroxidation, strengthening antioxidant defenses, improving immunity, reducing infectious complications, and decreasing ICU stay [13].

We include a **Table 4**, created by our team, which details specific nutritional contributions for pediatric burn patients. This table was prepared after an exhaustive literature review and customized according to the needs of our patients in the pediatric burn unit (**Table 4**).

Nutritional assessment

Nutritional assessment in burn patients is based on methods similar to those used in critical care patients, such as anthropometry and biochemical markers [14].

A relevant marker is prealbumin, reflecting protein synthesis, and levels below 10-15 mg/dL indicate early malnutrition. Factors like the inflammatory response, liver or kidney disease, and zinc or iron deficiency can influence albumin levels [4].

Nutritional risk in burns is determined by five main variables: admission weight, percentage of burned body surface area, metabolic rate (percentage of energy expenditure), time from burn to first surgical healing, and the presence of sepsis [14].

Body weight is the simplest indicator, but it is essential to continuously monitor patients for signs of malnutrition, such as significant weight loss, loss of more than 8% of body weight during hospitalization, lack of weight gain, and signs of muscle wasting [4].

Nutritional screening tools like the Screening Tool for Risk on Nutritional Status and Growth (STRONGKIDS), Pediatric Yorkhill Malnutrition Score (PYMS), and Screening Tool for the Assessment of Malnutrition in Pediatrics (STAMP) [15].

Have been developed and evaluated in burn patients. A multicenter study in a Korean referral center showed that burn patients classified as high-risk using these tools had worse clinical outcomes than those at moderate risk. However, the correlation between anthropometric measures and risk classification is still low due to the lack of specific standards for validating these measures [15].

Methods of nutritional support

Nutritional therapy (NT) plays a crucial role in the care of burn patients, supplying essential nutrients, fluids, and energy to maintain vital functions and homeostasis. Additionally, it contributes to the recovery of the immune system, minimizing protein catabolism and nitrogen loss [17, 18]. In cases of severe burns, early initiation of NT is essential to reduce nitrogen loss, decrease bacterial translocation, and improve immune function [17].

Adequate protein administration has been shown to have beneficial effects on immune function, reducing bacteremia and increasing survival [17, 19].

The evidence supports the use of enteral nutrition methods, although initiation should be individualized based on medical assessment, especially in patients requiring early nutritional support due to potential intolerance [18].

NT is recommended to be initiated within the first 12 hours of admission, preferably through enteral routes, due to advantages in reducing adverse responses, immunoglobulin production, and stress ulcer prevention. However, some studies suggest that enteral nutrition might be initiated after fluid resuscitation to avoid gastrointestinal dysfunction (within the first 24 hours). Administration can be done through a nasogastric tube, depending on tube location [17].

Research conducted in specialized burn centers in Iran and Colorado has compared early and late administration of enteral nutrition, demonstrating that the former is associated with shorter hospital stays and a reduction in patient mortality [20-22].

Table 4. Nutritional management protocol in burns: caloric and protein requirements, enteral support, and monitoring

Percentage of Total Body Surface area (%TBSA) Burn Injury	CALORIC NEEDS	PROTEIC NEEDS	TIME OF START ENTERAL SUPPORT	ENTERAL SUPPORT INITIATION AND ADVANCE	PROPRANO-LOL	MONITORING
10-20%	**0 to 6 months** 110-130 kcal*kg^{-1} × Factor (1.2-1.4)	**0 to 3 years** 2-3 g/kg/day **4 to 18 years** 1.5-2 g/kg/day	FIRST 4 to 6 HOURS	**<20 Kg** 1 mL/kg/hour every 2 hours **≥ 20 kg** 20 mL/hour every 2 hours	TBSA ≥ 40%	**Admission** Serum glucose each 6 hours × 24 hours, complete blood count, albumin
20-40%	**>6 months** 100-120 kcal*kg^{-1} × Factor (1.2-1.4)					**Daily** Electrolytes, BUN, creatinine, glucose, calcium, phosphorus, magnesium, % caloric goals and protein intake
	0 to 6 months 110-130 kcal*kg^{-1} × Factor (1.4-1.6)	**0 to 3 years** 3-4 g/kg/day **4 to 18 years** 2.5-3 g/kg/day				
>40%	**>6 months** 100-120 kcal*kg^{-1} × Factor (1.4-1.6)					**Weekly** Weight, indirect calorimetry (if it's available)
	<6 months 110-130 kcal*kg^{-1} × Factor (1.4-1.6)	**0 to 3 years** 4 g/kg/day **4 to 18 years** 3-4 g/kg/day				
Intubated*	**>6 months** 100-120 kcal*kg^{-1} × Factor (1.4-1.6)					**Every 2 weeks** 25-hydroxy vitamin D, CRP
	<6 months 110-130 kcal*kg^{-1} × Factor (0.85-0.9)					

Table 5. Summary of carbohydrates, fats, and proteins in burn patients

Carbohydrates	Fat	Protein
55-60% total caloric intake	<35% of non-protein calories (≈15%)	15-20% total caloric intake
Maximum glucose infusion rate: 5 mg/kg/min → 7 g/kg/day	Linoleic acid 2-3% of total calories	1.5-3 g/kg/day
Capillary blood glucose 100-150 mg	Omega 3	Albumin supplementation <2.5 g/dL serum

Goal: 20 - 25 kcal/kg/day.

In cases of diarrhea, a history of gastrointestinal problems, or severe nasogastric tube intolerance, parenteral nutrition (PN) is indicated and administered centrally to ensure the delivery of necessary calories and prevent burn-related catabolism. However, PN carries risks of catheter infections, septic thrombophlebitis, or endocarditis if used for an extended period [21]. In extreme situations where the patient cannot tolerate any enteral route, total parenteral nutrition (TPN) is used to meet essential fatty acid requirements [4]. **Table 4** suggests the total composition of TPN in infants and children.

Despite the controversy surrounding TPN due to its lack of physiology and association with complications in post-surgical critical patients, its advantage lies in being tolerated by critically ill patients. The combination of enteral and parenteral nutrition is considered safe and effective if it is administered appropriately according to clinical indicators. This practice has been shown to provide adequate caloric and protein intake, promoting wound healing, and reducing mortality [18].

Discussion

This clinical case highlights the critical importance of nutritional support in pediatric burn patients, with a direct impact on wound healing, infection prevention, and long-term recovery [23, 24]. The case demonstrates how early initiation and individualization of nutritional support can significantly influence clinical outcomes. The use of enteral nutrition as the first-line strategy, complemented with immunonutrition and micronutrient supplementation, allowed stabilization of metabolic demands, promoted healing, and minimized infectious complications [21, 25]. The temporary need for parenteral nutrition following surgical complications also underscores the importance of implementing flexible strategies adapted to the patient's tolerance and clinical condition [21, 26].

From a diagnostic perspective, close monitoring of nutritional status through anthropometry, indirect calorimetry, and biochemical markers provided objective parameters for adjusting therapy [10]. In pediatric practice, these tools are particularly relevant since traditional screening scales may underestimate the risk of malnutrition in this population [27].

From a therapeutic standpoint, this case reinforces the principle that the management of pediatric burn patients must be comprehensive: nutritional optimization should go hand in hand with timely surgical interventions, strict infection control, and early rehabilitation [2]. For clinicians, the key teaching from this case is that nutritional therapy should not be considered merely as supportive care but as a central component of treatment, with a direct impact on survival, functional recovery, and long-term growth [28, 29].

Managing hypermetabolism and hypercatabolism is crucial, requiring nutrition tailored to cover energy and protein requirements, minimize muscle loss, and strengthen the immune system [3]. Advanced technologies, such as real-time monitoring systems and telemedicine tools, have greatly enhanced the ability to deliver precise and personalized care [30].

In this framework, the optimization of protein synthesis constitutes a cornerstone of nutritional therapy in pediatric burns. Its importance lies in several aspects: promoting effective wound healing and graft integration, preserving lean body mass and preventing growth impairment, enhancing immune competence to reduce sepsis and infectious complications, and, particularly in children, supporting long-term growth and neurocognitive development [31].

These aspects emphasize why sustained protein optimization should remain a therapeutic goal not only during the acute phase but also throughout recovery and rehabilitation [10].

Although nutritional support is fundamental, by itself it cannot completely counteract the protein catabolism associated with burns. Therefore, pharmacological strategies have been explored, such as catecholamine blockade with propranolol, the administration of anabolic hormones such as growth hormone, testosterone, or oxandrolone, as well as the use of insulin and insulin-like growth factors [8, 12, 13, 19].

It is important to highlight the need for further research on high-protein diets, considering their potential adverse effects related to uremia. A systematic review indicated that this type of diet may significantly increase urea production and amino acid oxidation without providing clear benefits in protein synthesis. Therefore, higher-quality studies are needed to determine whether high-protein diets truly benefit the nutrition of burn patients [19].

Glutamine, a non-essential amino acid, has been shown to improve protein synthesis and wound healing when administered as a supplement [4].

It is also used in burn patients for its ability to modulate the immune response. However, glutamine supplementation has been little studied and shows variations in dosage, route of administration, and duration of treatment. Results regarding infectious complications, hospital stay, and mortality are heterogeneous. Currently, no standardized dose exists, although 0.3 g/kg/day for 5 to 10 days has been proposed. Administration during the acute phase improves healing, and doses of 30 grams, divided into 2-3 boluses, have been associated with better nitrogen balance. Nevertheless, there is no solid evidence to support arginine supplementation in burn patients [13].

Burn patients may present persistent biochemical and pathophysiological alterations for years after the initial injury. As patient care advances, experimental studies have explored the role of ghrelin, a hormone with orexigenic effects, in wound management. It is suggested that ghrelin participates in the inflammatory cascade of burns and could help prevent disease-associated cachexia. In addition to ghrelin, another hormone called asprosin has been identified, also with orexigenic effects and using signaling pathways similar to those of ghrelin [32]. These potential therapies open new perspectives in the treatment of burn patients.

Finally, this case strengthened our ability to provide comprehensive and patient-centered pediatric care, supported by technological tools that allow for more precise and personalized attention. As part of this process, we were able to develop our specific table that synthesizes the main nutritional contributions-including macronutrients, micronutrients, and minerals-recommended for children with burns. Its construction was based on a rigorous review of the scientific literature and was adapted to the characteristics and needs of the pediatric patients treated in our burn unit.

Conclusion

This clinical case highlights the pivotal role of nutritional therapy in pediatric burn care. Early, individualized, and sustained nutritional support contributes to wound healing, infection control, preservation of lean body mass, and long-term recovery. For pediatric patients, nutritional optimization must be considered a central therapeutic strategy, integrated with surgical management, infection prevention, and rehabilitation. This experience reinforces that nutrition is not merely supportive but a cornerstone of treatment, shaping both immediate outcomes and long-term growth and development.

Disclosure of conflict of interest

None.

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Appendix 1. Report of isolates

Date	Sample/Site	Findings
29.11.2022	Urine culture	Negative after 5 days of incubation.
02.12.2022	Lower limb, quantitative tissue culture	Negative after 3 days of incubation.
02.12.2022	Upper limb, quantitative tissue culture	Pseudomonas putida 3.9×10^5 CFU. Susceptible to: Amikacin, Ceftazidime, Imipenem, Meropenem, Piperacillin-Tazobactam.
02.12.2022	Tracheal secretion culture	Negative after 72 hours.
05.12.2022	Blood cultures	Negative after 5 days of incubation.
07.12.2022	Left thigh eschar, quantitative tissue culture	Pseudomonas aeruginosa $>10\times10^5$ CFU. Susceptible to: Amikacin, Ceftazidime, Ceftazidime/Avibactam, Ceftolozane/Tazobactam, Ciprofloxacin, Imipenem, Meropenem. Resistant: Piperacillin-Tazobactam (MIC 32).
07.12.2022	Left thigh eschar, quantitative tissue culture	Pseudomonas aeruginosa $>10\times10^5$ CFU. Susceptible to: Amikacin, Ceftazidime, Ceftazidime/Avibactam, Ceftolozane/Tazobactam, Imipenem, Meropenem (I). Intermediate: Ciprofloxacin. Resistant: Piperacillin (MIC 32).
09.12.2022	Left thigh eschar, quantitative tissue culture	Pseudomonas aeruginosa 2.6×10^5 CFU. Susceptible to: Amikacin, Ceftazidime-Avibactam, Ceftolozane-Tazobactam, Ciprofloxacin, Imipenem, Ertapenem. Resistant: Ceftazidime. Candida albicans 1×10^3 CFU. Susceptible to: Amphotericin B, Caspofungin, Fluconazole, Flucytosine, Micafungin, Voriconazole.
14.12.2022	Back, quantitative tissue culture	Pseudomonas aeruginosa 1×10^7 CFU. Resistant: Ceftazidime-Avibactam, Cefepime. Intermediate: Ciprofloxacin. Susceptible: Meropenem. Candida albicans 1.5×10^4 CFU. Susceptible to Fluconazole.
14.12.2022	Blood cultures	Negative after 5 days of incubation.
23.12.2022	Thoracic necrotic devitalized tissue, quantitative tissue culture	10^3 CFU Candida parapsilosis.
26.12.2022	Left thigh, quantitative tissue culture	Negative after 48 hours.
01.01.2023	Sepsis FilmArray	Positive for Candida parapsilosis.
01.01.2023	Blood cultures	Positive at 44 hours for Candida parapsilosis.
11.01.2023	Left thigh necrotic edge, excised tissue culture	Klebsiella ESBL 10^5 CFU. Beta-lactamase production. Resistant to carbapenems (Meropenem MIC >16). Resistant to Ceftazidime-Avibactam. Susceptible to Amikacin. Resistant to Ciprofloxacin.
16.01.2023	Blood cultures/Fungal blood cultures	Negative.
16.01.2023	Urine culture	Negative.
16.01.2023	Gastrointestinal FilmArray	Positive for Clostridium difficile.
17.01.2023	CMV plasma viral load	Not detected.
17.01.2023	Respiratory FilmArray	Negative.
17.01.2023	Cytomegalovirus load	Negative.
18.01.2023	Pneumonia panel	Not detected.
19.01.2023	Distal third, inner thigh, punch biopsy	Negative after 72 hours.
19.01.2023	Mid-third, anterior thigh, punch biopsy	Negative after 72 hours.
19.01.2023	Bronchoalveolar lavage	Normal microbiota growth. CMV not detected. Mycobacterium tuberculosis not detected.
22.02.2023	Quantitative tissue culture	Pseudomonas aeruginosa 2.7×10^3 CFU. Carbapenem-resistant. Staphylococcus aureus 5.4×10^4 CFU. Methicillin-resistant.
08.02.2023	Left arm, posterior aspect, granulation tissue culture	Pseudomonas aeruginosa 2.2×10^5 CFU/g tissue. Resistant: Ciprofloxacin, Imipenem. Intermediate: Meropenem. Staphylococcus aureus 2.2×10^5 CFU. Methicillin-susceptible. Resistant to Clindamycin.
11.02.2023	Blood cultures	Negative after 72 hours of incubation.