Residência RP Pediátrica
Publicação Oficial da Sociedade Brasileira de Pediatria
ISSN-Online: 2236-6814

Submitted on: 06/04/2025
Approved on: 07/04/2025

CASE REPORT

Severe Malnutrition in Childhood: Different Etiologies, One Common Clinical Challenge

Iohana Falcão Rebouças Assayag[1], João Emanuel Farias Siqueira[1], Ana Teresa Parahyba Asfor[1], François Loiola Ponte Souza[1]

Keywords:
Protein-Energy Malnutrition, Diarrhea, Severe Combined Immunodeficiency, Cytomegalovirus Infections.

Abstract

Objective: To describe three cases of patients affected by severe protein-calorie malnutrition with distinct etiologies. **Case report:** The cases present severe malnutrition with different etiologies and treatments. The first patient, a 1-year-and-8-month-old female, was admitted with severe malnutrition, vomiting, dehydration, and severe anemia, requiring ICU admission, antibiotic therapy, vitamin supplementation, and an amino acid-based formula. Despite developing pseudomembranous colitis treated with metronidazole and vancomycin, she showed nutritional and motor recovery. The second patient, a 10-month-old male, initially hospitalized for gastroenteritis with bloody diarrhea, progressed to acidemia and hypogammaglobulinemia, and was diagnosed with SCID (undetectable TREC). He received an amino acid-based formula, antibiotics, intravenous immunoglobulin, and intensive nutritional support, with significant clinical improvement. The third patient, a previously healthy 5-year-old male, presented with hematemesis, fever, and pancytopenia, requiring ICU management for CMV-induced hemophagocytic syndrome. He was treated with immunoglobulin, corticosteroid therapy, ganciclovir, and nutritional support, showing progressive improvement, food tolerance, and weight gain. **Discussion:** Severe malnutrition in childhood is a public health problem with a significant impact on physical and cognitive development. Different etiologies can lead to this clinical condition, requiring a thorough diagnostic approach for appropriate management. The clinical evolution of the patients reinforces the need for a multidisciplinary and individualized assessment, addressing not only nutritional replacement but also the treatment of the underlying cause. The etiological differentiation of malnutrition is essential for adopting effective therapeutic strategies to achieve better clinical outcomes.

[1] Hospital Infantil Albert Sabin, Enfermaria de Pediatria Geral - Fortaleza - Ceará - Brazil.

Correspondence to:
Iohana Falcão Rebouças Assayag
Hospital Infantil Albert Sabin. Rua Tertuliano Sales, 544 - Vila União, Fortaleza - CE, CEP 60410-794. E-mail: iohanareboucas@gmail.com

Resid Pediatr. 2026;16(2).
DOI: https://doi.org/10.25060/residpediatr-2026-1493

Creative Commons. Attribution-NonCommercial-ShareAlike 4.0 International.

INTRODUCTION:

Severe childhood malnutrition is a public health problem[1] with lasting impacts on physical growth, neurodevelopment, and cognitive performance[2]. Nutritional deprivation during critical stages of brain development may affect processes such as myelination and synaptogenesis, leading to persistent cognitive deficits[1,3]. Thus, understanding the different clinical presentations of severe malnutrition and defining individualized therapeutic strategies are essential for improving guidelines for disease assessment and management.

This study contributes to the literature by presenting three clinical cases of severe protein-calorie malnutrition with distinct etiologies: feeding error, severe combined immunodeficiency (SCID), and chronic cytomegalovirus (CMV) infection associated with wasting syndrome.

By analyzing these cases, the study reinforces the complexity of the factors underlying severe malnutrition and the inherent challenges in its clinical management.

CASE DESCRIPTIONS:

The three patients were admitted with severe malnutrition. Despite clinical similarity, diagnostic investigation revealed distinct underlying etiologies for the nutritional impairment.

Case 1:

A female patient, aged one year and eight months, presented with an acute episode of vomiting and food refusal, associated with deterioration of general condition. On the third day of symptoms, she was taken to the emergency department due to increased drowsiness, where she was admitted with tachydyspnea, severe dehydration, hypoxemia, metabolic acidosis (bicarbonate 1.7), and severe anemia (Hb 1.1). After stabilization, she was admitted to the Intensive Care Unit and treated with ceftriaxone for five days, subsequently being discharged to the ward. On evaluation, she presented delayed neuropsychomotor development and severe malnutrition (Z-score -3.24), without edema, with hypotonia, thin hair, and a protruding abdomen.

Laboratory tests showed deficiencies of vitamin B12, vitamin D, and ferritin, and supplementation was initiated according to the severe malnutrition protocol. During hospitalization, she developed watery diarrhea, without blood or mucus. Parasitological tests, stool culture, and blood culture were negative, and anti-endomysial antibodies were non-reactive. She received zinc and probiotics, with partial improvement.

Subsequently, she developed fever, leukocytosis, tachycardia, tachypnea, and rectal prolapse with white and yellow plaques. Under the hypothesis of pseudomembranous colitis, oral metronidazole was initiated and later escalated to vancomycin. Tests for toxins A and B were positive, and colonoscopy was consistent with the hypothesis. Clinical improvement occurred after treatment. The patient maintained a diet with amino acid formula for two weeks, progressing to a regular diet, with weight gain and motor improvement.

Given the positive response to the malnutrition protocol, without other identifiable causes, severe malnutrition due to food deprivation was concluded.

Case 2:

A male patient, aged ten months, born at term, received mixed feeding until five months of age, followed by an exclusive diet with follow-up formula containing intact proteins and lactose. At six months, he developed vomiting and bloody diarrhea, without fever, progressing to dehydration requiring support in a ward bed. During the first hospitalization, non-IgE-mediated cow's milk protein allergy (CMPA) was suspected due to the presence of significant gastrointestinal symptoms after greater intake of infant formula, and an amino acid formula was introduced.

Despite the dietary change, the patient progressed with weight loss (1.7 kg in one month) and abdominal distension. At nine months, in the absence of new diarrheal episodes, the decision was made to resume conventional infant formula. However, upon reintroduction of formula milk, he presented clinical worsening with diarrhea, progressing to tachydyspnea and acidemia, requiring hospitalization and oral bicarbonate replacement.

Laboratory tests revealed severe hypogammaglobulinemia (IgA, IgM, and IgG below P3), and intravenous immunoglobulin was administered (800 mg/kg). Urine culture showed *Proteus mirabilis*, treated with ceftriaxone. The patient was transferred to a tertiary unit, where he was admitted with severe malnutrition (Z-score -3.12). Vitamin replacement and antibiotic therapy were initiated according to protocol. Subsequent tests identified *Pseudomonas aeruginosa* sensitive to meropenem. Immunoglobulins remained below the 3rd percentile, and immunophenotyping revealed reduced B lymphocytes and CD4 cells, raising the hypothesis of severe combined immunodeficiency (SCID). Undetectable TREC confirmed the diagnosis.

The patient received antibiotic therapy with meropenem and teicoplanin for 14 days, but presented clinical deterioration 24 hours after treatment discontinuation, requiring reintroduction of antimicrobial therapy, prophylaxis due to immunodeficiency, and intravenous immunoglobulin every two weeks according to immunology guidance. In parallel, caloric and nutritional intake remained optimized, promoting nutritional recovery during hospitalization.

Case 3:

A male patient, aged five years, previously healthy, with adequate weight and height development prior to the current illness. He presented with an acute episode of nausea, vomiting, watery diarrhea without blood or mucus, abdominal

pain, hyporexia, and fever, seeking emergency care one week after symptom onset due to the occurrence of hematemesis.

On initial evaluation, pancytopenia and severe dehydration were identified, and hospitalization was chosen for stabilization and etiological investigation. Due to clinical deterioration, the patient was transferred to the ICU eight days after admission, where he was diagnosed with hemophagocytic syndrome and started intravenous immunoglobulin, pulse therapy with corticosteroids, and ganciclovir due to positive cytomegalovirus (CMV) serology (IgM+).

During hospitalization, the patient continued to have persistent vomiting and weight loss, despite parenteral and enteral nutrition via nasoenteral tube. One month after ICU admission, the patient returned to the ward with severe malnutrition (Z-score -4.6), fever, and intolerance to oral feeding. Upper gastrointestinal endoscopy revealed Los Angeles grade D ulceration, and colonoscopy showed subepithelial hemorrhages, suggesting inflammatory enteropathy. He developed anemia requiring blood transfusion and underwent HIV serology at two different times during hospitalization, both with negative results. Brain magnetic resonance imaging showed signs of malnutrition, with enlargement of sulci and cisterns. Abdominal ultrasound revealed aortomesenteric compression causing segmental duodenal stenosis, suggesting superior mesenteric artery syndrome associated with the condition.

After 40 days in the ward, the patient had remission of fever and reduction of vomiting, allowing progression to a pasty oral diet with good acceptance. After eight days without fever and two days without vomiting, he began to gain weight and showed progressive clinical improvement.

DISCUSSION:

In the study by Pawellek et al.[4] in 2007, 24.1% of patients admitted to the ward of Dr. von Hauner Children's Hospital presented malnutrition, with higher prevalence among those with multiple diagnoses (43.8%) and neurological involvement (40.0%). Infectious diseases (34.5%) and cystic fibrosis (33.3%) were important causes, while conditions such as cardiovascular, oncological, gastrointestinal, and other diseases contributed to a lesser extent.

In the first case, malnutrition resulting from low caloric and protein intake reinforces the importance of adequate nutritional support. Faced with an infectious episode, the patient developed severe complications due to previous malnutrition. The etiology was concluded based on clinical and laboratory characteristics, since the patient was admitted after three days of symptoms, presenting very low weight, hypotonia, thin hair, and deficiencies of vitamins D, B12, and iron, which improved after nutritional support. As occurred with the patient, interventions based on nutritional reeducation and dietary correction can reverse the effects of nutritional deficiency, provided there is continuous follow-up[4,5]. The study conducted by Calgaro et al.[6] in 2021 reinforces the possibility of reversing damage with adequate intervention. In this study, 197 undernourished children underwent the proposed nutritional rehabilitation treatment, and 89% presented anthropometric recovery. In addition, Machado et al.[7] highlight the importance of recognizing risk factors for refeeding syndrome, since identifying susceptible patients is crucial to prevent the morbidity and mortality associated with this disorder.

In contrast, the second case, related to SCID, demonstrates that immune failure may predispose to recurrent infections that generate chronic inflammation and impair intestinal absorption. Even with an apparently adequate diet, the presence of infectious and inflammatory processes interferes with nutritional homeostasis, promoting catabolism and deterioration of nutritional status[1].

The identification of SCID through laboratory tests guided therapy, which combined management of infections, immunoglobulin replacement, and intensive nutritional support[8,1]. Aluri et al.[9] described 57 pediatric cases of SCID with TREC below the reference value (<18 copies/µL). Of these cases, 60% of patients had impaired neuropsychomotor development due to recurrent infections. As reported by Scrimshaw,[1] infections directly affect an individual's nutritional status, which, according to Prado,[2] directly impairs the child's brain development and prospects for social advancement.

The third case illustrates the impact of chronic CMV infection, which may trigger a wasting syndrome due to a hypercatabolic state and systemic inflammation[10]. Viral infection induces metabolic alterations, increasing energy expenditure and impairing gastrointestinal integrity, in addition to anorexia, nausea, vomiting, and abdominal discomfort, which are common in superior mesenteric artery syndrome[11]. The therapeutic strategy involved antiviral therapy, control of the inflammatory response, and nutritional support.

Kovacs et al.[12] showed that children with congenital HIV have twice the risk of death and four times the risk of impaired neuromotor development when infected with CMV. Chevalier et al.[13] analyzed 67 cases of CMV-associated hemophagocytic syndrome, of which 70% occurred in immunosuppressed children. In the present case, the patient had negative HIV serology in two separate tests. In line with Refai et al., the need for CMV serology in patients admitted with persistent gastrointestinal abnormalities is emphasized[14].

Superior mesenteric artery syndrome results from reduction of the aortomesenteric angle, frequently associated with severe weight loss and depletion of mesenteric adipose tissue[11]. Shiu et al. demonstrated that intensive nutritional support can reverse symptoms and promote weight gain, as evidenced in the present case, reducing the need for surgical intervention[15].

The analysis of these cases shows that, despite the common clinical presentation, the underlying mechanisms are distinct. Successful treatment of severe malnutrition in

children depends not only on restoration of nutritional intake, but also on identification and appropriate management of the etiological causes involved.

REFERÊNCIAS:

1. Scrimshaw NS, SanGiovanni JP. Synergism of nutrition, infection, and immunity: an overview. Am J Clin Nutr. 1997;66(2 Suppl):464S-477S. doi: http://www.doi.org/10.1093/ajcn/66.2.464S
2. Prado EL, Dewey KG. Nutrition and brain development in early life. Nutr Rev. 2014;72(4):267-284. doi: http://www.doi.org/10.1111/nure.12102
3. World Health Organization. WHO guideline on the prevention and management of wasting and nutritional oedema (acute malnutrition) in infants and children under 5 years. Geneva: World Health Organization; 1999.
4. Pawellek I, Dokoupil K, Koletzko B. Prevalence of malnutrition in paediatric hospital patients. Clin Nutr. 2008;27(1):72-76. doi: http://www.doi.org/10.1016/j.clnu.2007.11.001
5. Bhutta ZA, Das JK, Rizvi A, Gaffey MF, Walker N, Horton S, et al. Evidence-based interventions for improvement of maternal and child nutrition: what can be done and at what cost? Lancet. 2013;382(9890):452-477. doi: http://www.doi.org/10.1016/S0140-6736(13)60996-4
6. Calgaro S, Isidoris V, Girotto C, Chhaganlal K, Moiane J, Putoto G, et al. Children's nutritional rehabilitation program in Beira, Mozambique: a retrospective study. Am J Trop Med Hyg. 2021;105(6):1631-1637. doi: http://www.doi.org/10.4269/ajtmh.20-0297
7. Machado MEPF, Cardoso AL, Tumas R, Kawano C, Seo C. Síndrome de realimentação no desnutrido grave: os desafios nutricionais. Resid Pediatr. 2024;14(3):e1101. doi: http://www.doi.org/10.25060/residpediatr-2024.v14n3-1101
8. Notarangelo LD. Primary immunodeficiencies. J Allergy Clin Immunol. 2004;114(3):677-687. doi: http://www.doi.org/10.1016/j.jaci.2004.07.046
9. Aluri J, Desai M, Gupta M, Dalvi A, Terance A, Rosenzweig SD, et al. Clinical, immunological, and molecular findings in 57 patients with severe combined immunodeficiency (SCID) from India. Front Immunol. 2019;10:23. doi: http://www.doi.org/10.3389/fimmu.2019.00023
10. Zoller EE, Lykens JE, Terrell CE, Aliberti J, Filipovich AH, Henson PM, et al. Hemophagocytosis causes a consumptive anemia of inflammation. J Exp Med. 2011;208(6):1203-1214. doi: http://www.doi.org/10.1084/jem.20102538
11. Lee TH, Lee JS, Jo Y, Park KS, Cheon JH, Kim YS, et al. Superior mesenteric artery syndrome: where do we stand today? J Gastrointest Surg. 2012;16(12):2203-2211. doi: http://www.doi.org/10.1007/s11605-012-2049-5
12. Kovacs A, Schluchter M, Easley K, Demmler G, Shearer W, La Russa P, et al. Cytomegalovirus infection and HIV-1 disease progression in infants born to HIV-1-infected women. N Engl J Med. 1999;341(2):77-84. doi: http://www.doi.org/10.1056/NEJM199907083410203
13. Chevalier K, Schmidt J, Coppo P, Galicier L, Noël N, Lambotte O. Hemophagocytic lymphohistiocytosis associated with cytomegalovirus infection: 5 cases and a systematic review of the literature. Clin Infect Dis. 2023;76(2):351-358. doi: http://www.doi.org/10.1093/cid/ciac649
14. Refai Z, Nicholls S, Garg A. Infant death due to CMV enterocolitis. BMJ Case Rep. 2012;2012:bcr1120115080. doi: http://www.doi.org/10.1136/bcr.11.2011.5080
15. Shiu JR, Chao HC, Luo CC, Lai MW, Kong MS, Chen SY, et al. Clinical and nutritional outcomes in children with idiopathic superior mesenteric artery syndrome. J Pediatr Gastroenterol Nutr. 2010;51(2):177-182. doi: http://www.doi.org/10.1097/MPG.0b013e3181c7bdda