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Case Report

From fragility to recovery: a case of severe acute malnutrition in a 22-month-old

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ABSTRACT

Severe acute malnutrition (SAM) remains a major public health concern in many low-income and middle-income countries. It is characterized by markedly low weight-for-height (less than –3 z-scores according to World Health Organization (WHO) growth standards), evident severe wasting, or nutritional edema. Affected children have an estimated 9–11-fold greater risk of illness and death than those with adequate nutrition. This case report highlights the clinical presentation, diagnostic challenges, and management of a 22-month-old female patient diagnosed with SAM. The child presented with inadequate growth, recurrent infections, and severe nutritional deficits compounded by delayed weaning and improper feeding practices. Examination revealed hallmark signs of malnutrition, including stunting, muscle wasting, and dermatosis. Laboratory findings indicated anemia and leukocytosis, which further supported the diagnosis. The management adhered to the WHO's 10-step protocol for SAM, incorporating nutritional rehabilitation, micronutrient supplementation, and infection control, which led to gradual improvement. This case highlights the complex and varied causes of SAM, and stresses the need for timely management, improved community awareness regarding adequate nutrition, and coordinated care involving multiple health disciplines. Addressing the underlying causes of malnutrition is crucial for prevent recurrence and improve outcomes.

Keywords: Anthropometry, Edema, Severe acute malnutrition, Kwashiorkor, Marasmus

INTRODUCTION

Protein-energy malnutrition (PEM) represents a spectrum of disorders that develop due to the inadequate intake of both protein and calories in different proportions. The phrase "protein-calorie malnutrition" was first introduced by Jelliffe in 1959, and has since evolved into the term "acute malnutrition".[1] Olsen and colleagues described PEM as a state of nutritional insufficiency commonly observed in children from resource-limited settings. Severe acute malnutrition (SAM) is identified by markedly low weight-for-height (below –3 z-scores of World Health Organization (WHO) growth standards), presence of severe wasting, or nutritional edema.[2] Children with SAM are at 9–11 times greater risk of illness and death than their well-nourished counterparts.[3] The reported inpatient case fatality rates for SAM, even when managed according to WHO guidelines, vary between 3.4% and 35%.[4] Research has also indicated that children with moderate acute malnutrition can experience complications and mortality rates similar to those with severe disease.[5] This report describes the clinical features and management of a 22-month-old girl with SAM. This report emphasizes the importance of early diagnosis, adequate nutritional rehabilitation, and addressing the underlying etiologies.

CASE REPORT

A 22-month-old female child from West Godavari District of Andhra Pradesh, India, presented with a history of inadequate growth for 16 months, fever and vomiting for one month, and diarrhea for the past four days.

The child was brought to the Integrated Child Development Services (ICDS)-Anganwadi Centre in 2019, where her parents reported growth failure starting at six months of age and weight loss since she was 19 months age. Over the past month, they further noted she developed high-grade intermittent fever every 3–4 days, accompanied by 2–3 episodes of daily vomiting. Four days prior to presentation, the child experienced 2-3 episodes of diarrhea per day, including one episode of blood in stools and a cold with thick, white, mucoid nasal discharge for three days.

The past medical history revealed recurrent respiratory tract infections, although the child was apparently normal at birth, with no significant perinatal complications. Regarding feeding practices, Cerelac was introduced at 6 months of age but discontinued because of greasy stools, and breastfeeding was continued as the sole source of nutrition. Developmental history revealed delayed milestones with a developmental quotient (DQ) of 62.7, suggesting a nutritional impact on neurodevelopment.

Figure 1: Image depicts severe stunted growth accompanied by significant muscle wasting, prominent ribs, and reduced muscle mass of the upper limbs.

On examination, the child appeared irritable, undernourished, and exhibited an "old man" appearance. Pallor, generalized lymphadenopathy, and bilateral pedal edema were noted. Skin examination revealed flaky paint dermatosis with wrinkling and peeling of skin. There was severe muscle wasting, stunting, subcutaneous tissue emaciation, lusterless and depigmented hair that was easily pluckable, a baggy pant appearance, and prominence of ribs (Figures 1 and 3). The abdomen was distended with hepatomegaly, which was measured as a liver span of 7 cm. The child's anthropometric assessment revealed a weight of 4 kg (approximately 33% of the expected value for age), length of 66 cm, body mass index (BMI) of 9.2 kg/m^2, and mid–upper arm circumference (MUAC) of 8 cm (Figure 2). These findings are consistent with the WHO/IAP criteria for severe acute malnutrition, which include weight-for-height below –3 SD, MUAC less than 11.5 cm, or the presence of bilateral pedal edema. Systemic examinations of the cardiovascular, respiratory, and central nervous systems did not reveal any abnormalities. Laboratory evaluation indicated a hemoglobin level of 7.7 g/dl and leukocyte count of 19,100/mm^3. The other parameters were within normal limits. Anemia is consistent with nutritional deficiency, whereas leukocytosis suggests an associated infection, commonly seen in children with SAM.

Figure 2: Photo shows severe wasting of the forearm and hand muscles with visible skin folds and loss of subcutaneous fat.

Figure 3: Clinical photograph of the patient lying supine, demonstrating severe wasting with visible ribs, dull and pale hair, loss of subcutaneous fat, and reduced muscle bulk of the extremities with abdominal distension.

Differential diagnoses included faulty feeding practices, malabsorption syndrome, immunodeficiency, and extrapulmonary tuberculosis. Malabsorption was considered due to a history of greasy stools but lacked consistent steatorrhea. Immunodeficiency was suspected due to recurrent infections; however, there were no opportunistic infections or severe sepsis. Extrapulmonary tuberculosis was considered, but not supported by history or systemic findings. Thus, a final diagnosis of SAM was made. The child exhibited a marasmic-Kwashiorkor phenotype, with features of marasmus (severe wasting, old man appearance, baggy pants sign) and kwashiorkor (bilateral pedal edema, flaky paint dermatosis, depigmented hair, hepatomegaly).

Nutritional rehabilitation was initiated using the WHO 10-step protocol for managing severe malnutrition. Stabilization includes the treatment of hypoglycemia,

hypothermia, and electrolyte imbalance. Nutritional rehabilitation was initiated with the F-75 therapeutic formula, followed by a gradual switch to F-100 once tolerated. In addition, the child received micronutrient supplementation including iron, folic acid, zinc, and vitamin A. Empirical antibiotics were administered to address potential infections. Regular follow-up was conducted to monitor the anthropometric and biochemical parameters. Despite the significant challenges, the child showed gradual improvement with multidisciplinary care involving pediatricians, nutritionists, and community health workers.

DISCUSSION

In India, SAM remains a pressing public health concern, with the National Family Health Survey (NFHS-5, 2019–21) reporting that approximately 17.3% of children below five years of age are affected.[6] The clinical presentation of SAM varies among marasmus, kwashiorkor, and marasmic-kwashiorkor, the latter being seen in this child. Key diagnostic markers include weight-for-height <-3 SD, MUAC <11.5 cm, and bilateral pedal edema, all of which were present in this case.[7]

This case underscores the multifactorial nature of severe malnutrition, exacerbated by delayed weaning, inadequate feeding practices, recurrent infections, and potential malabsorption. The child's clinical features, including stunting, severe wasting, and characteristic dermatoses, were consistent with Kwashiorkor-like malnutrition. Although India has experienced rapid economic growth over the last decade, this has not translated into substantial improvements in nutritional outcomes. Data indicate that the pace of decline in childhood stunting has slowed, falling by about half compared to the reduction achieved in the previous ten years, yet the country still accounts for the highest share of the world's undernourished population.[8] Field studies have consistently demonstrated a two-way association between malnutrition and infectious diseases.[9,10] Episodes of acute diarrhea and pneumonia are especially common in the first two-three years of life, a period when immune defenses are compromised.[10] Among children admitted with severe malnutrition, diarrhea was the most frequent presenting complication (68.4%), whereas sepsis was the leading complication during hospital stay (9%).[11] Pneumonia is prevalent among malnourished children and is often associated with fatal outcomes, particularly in children aged <24 months. Although acute respiratory infections (ARIs) may result from different bacterial pathogens, evidence consistently indicates that malnutrition greatly increases the risk of death, raising it by two-three times.[12] Consequently, pneumonia and malnutrition remain leading causes of death in children.[13]

Management of SAM requires stepwise care following the WHO's 10-step protocol, including stabilization, nutritional rehabilitation with therapeutic foods (F-75, F-100), micronutrient supplementation, and infection control. Early recognition and intervention are critical; as untreated SAM carries a mortality risk of up to 20–30% in resource-limited settings.[14]

From a public health perspective, strengthening government-led child nutrition and welfare initiatives such as the Integrated Child Development Programme, Anganwadi centers, and Poshan Abhiyaan are essential for the early identification and management of SAM. Regular growth monitoring, caregiver education on timely weaning and complementary feeding, and community-based management of malnutrition (CMAM) are vital strategies for reducing the disease burden.

Written informed consent was obtained from the patient's parents for the publication of this case report and the accompanying clinical details and images. Efforts were made to maintain anonymity.

CONCLUSION

This case highlights the classical marasmic-Kwashiorkor phenotype of SAM, arising from inadequate feeding practices and recurrent infections. Early recognition and stepwise management following the WHO protocols are essential for improving survival and long-term developmental outcomes. Beyond hospital care, strengthening community-based interventions and caregiver education are critical for preventing SAM at the population level. This case highlights the combined significance of effective clinical care and broader public health interventions while demonstrating how multidisciplinary collaboration among healthcare providers, nutritionists, and community workers can ensure both survival and improved long-term growth and development.

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