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Original Research Article

Impact of Severe Acute Malnutrition on Child Development: The Role of Vitamin B12 Deficiency

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Abstract

Background: Severe acute malnutrition predisposes children to multiple micronutrient deficiencies, including vitamin B12 deficiency, which may impair neurodevelopment. Understanding the clinical and developmental consequences of low vitamin B12 levels in malnourished children is essential for strengthening rehabilitation strategies.

Material and Methods: A cross-sectional study was conducted among 120 children aged 6–59 months with severe acute malnutrition. Clinical signs, feeding practices, vitamin B12 status, hematological parameters, and developmental assessments were recorded. Associations were analyzed using standard statistical tests.

Results: Vitamin B12 deficiency was associated with higher frequencies of mucocutaneous signs, abnormal hematological parameters, and developmental delay. Feeding indicators such as dietary diversity and exclusive breastfeeding showed variable association with B12 status. Children in the lowest B12 category demonstrated significantly higher neurodevelopmental impairment.

Conclusion: Vitamin B12 deficiency contributes meaningfully to developmental delay in children with severe acute malnutrition. Integrating B12 screening, dietary counseling, and neurodevelopmental monitoring into SAM management may improve long-term outcomes.

Keywords: Vitamin B12 Deficiency; Severe Acute Malnutrition; Developmental Delay; Child Health.

Introduction

Severe acute malnutrition (SAM) continues to be a major global public-health burden, particularly affecting children under five years, and is strongly associated with morbidity, mortality, and long-term developmental effects. Children with SAM consistently demonstrate lower neurodevelopmental scores and significantly higher rates of developmental delay compared to well-nourished children, as shown in recent Indian and international studies [1].

Micronutrient deficiencies play a pivotal role in the pathogenesis and consequences of SAM. Among these, vitamin B12 deficiency is of particular concern because of its essential role in neuronal maturation, DNA synthesis, myelination, and cognitive functioning during early childhood [2]. Several recent studies from high-burden regions reveal substantial rates of vitamin B12 deficiency among children with SAM. Atiq et al. reported that nearly 45% of SAM children exhibited biochemical B12 deficiency, with strong associations to gross-motor, fine-motor, language, and cognitive delays [3]. Biomarkers of B12 status in SAM can be complex. A recent Tanzanian study observed paradoxically elevated serum B12 levels in SAM children, yet functional markers such as methylmalonic acid were elevated, indicating underlying metabolic B12 insufficiency and the unreliability of serum B12 alone in this population [4]. Complementing these findings, a 2025 narrative review highlighted that vitamin B12 deficiency remains one of the most under-recognized micronutrient gaps during SAM and emphasized the need for improved diagnosis and supplementation algorithms [5].

Long-term follow-up studies show that even after nutritional rehabilitation, children previously treated for SAM exhibit persistent deficits in

muscle strength, exercise tolerance, cognitive skills, and school performance, suggesting that micronutrient deficits—particularly vitamin B12—may contribute to irreversible developmental consequences [6].

Vitamin B12 deficiency can arise from inadequate intake of animal-source foods, maternal deficiency, poor absorption, chronic infections, or environmental enteropathy—all of which frequently coexist in children with SAM [7]. Moreover, the early developmental period (first 1000 days) is critically sensitive to B12 deficiency, making SAM-affected children more vulnerable to adverse neurological outcomes [8].

Recent community-based pediatric data from India emphasize that vitamin B12 deficiency is still an under-addressed public health challenge, particularly among low-income populations consuming predominantly vegetarian diets [9]. Furthermore, a 2023 study found a clear association between delayed developmental milestones and low B12 levels specifically among malnourished children aged 6–59 months, reinforcing the biologic and clinical link between B12 deficiency and neurodevelopmental delay [10].

Considering this evidence, there is a significant need to quantify the prevalence, causes, and developmental implications of vitamin B12 deficiency in children with SAM. Therefore, this study aims to determine the prevalence of vitamin B12 deficiency, explore its underlying causes, and evaluate its association with developmental delay across motor, language, cognitive, and adaptive domains—ultimately contributing to improved management strategies for this highly vulnerable population.

Material and Methods

This hospital-based cross-sectional study was conducted in the Department of Pediatrics at a tertiary care institute over a period of one year. A total of 120 children aged 6–59 months diagnosed with severe acute malnutrition (SAM) according to the World Health Organization (WHO) criteria were enrolled. SAM was defined as weight-for-height/length below −3 SD of the WHO growth standards, mid-upper-arm circumference <11.5 cm, or the presence of bilateral nutritional edema. Children with known congenital anomalies, chronic neurological disorders, metabolic diseases, or those already receiving vitamin B12 supplementation were excluded to avoid potential confounding.

After obtaining informed consent from parents or legal guardians, detailed demographic and clinical information was recorded, including age, sex, socioeconomic status, feeding history, dietary intake, maternal nutritional status, and history of recurrent infections. Anthropometric measurements such as weight, length/height, and MUAC were taken using standard WHO protocols. Developmental assessment was performed using an age-appropriate standardized developmental screening tool evaluating gross-motor, fine-motor, language, social, and cognitive domains. Each child was categorized as having normal development or developmental delay based on tool-specific cutoff values.

Venous blood samples were collected aseptically to determine serum vitamin B12 levels. Laboratory analysis included estimation of serum cobalamin concentration using chemiluminescent immunoassay. Vitamin B12 deficiency was defined using age-appropriate cutoffs (<203 pg/mL), and borderline levels were further evaluated with functional markers wherever available. Additional investigations such as hemoglobin, complete blood count, and peripheral smear were performed to assess coexisting nutritional anemia or other hematological abnormalities. Stool examination and relevant infection screening were done when clinically indicated to identify possible causes of malabsorption.

All data were entered into a structured proforma and later transferred to a statistical software package for analysis. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Association between vitamin B12 deficiency and developmental delay was analyzed using the chi-square test or Fisher's exact test as appropriate. Mean comparisons were performed using independent t-tests or ANOVA depending on distribution characteristics. A p-value of less than 0.05 was considered statistically significant. Ethical approval for the study was obtained from the Institutional Ethics Committee prior to the commencement of data collection.

Results

In the present study, a total of 120 children with severe acute malnutrition were evaluated for feeding indicators, vitamin B12 levels, clinical features, hematological parameters, and developmental outcomes. Table 1 represents the association of serum vitamin B12 levels with IYCF indicators and drug intake. The distribution across the three groups showed that vegetarian diet patterns, early initiation of breastfeeding, exclusive breastfeeding, and timely introduction of complementary feeds, minimum dietary diversity and minimum meal frequency were similarly distributed across groups without statistically significant differences. Antacid use was more common in Group A, although this difference did not reach statistical significance. Table 2 presents the clinical signs and symptoms associated with vitamin B12 categories. Edema and pallor were

comparably distributed across groups. Signs of vitamin B12 deficiency showed a marked difference, with hyperpigmentation and glossitis being more frequently observed in Groups A and B, while combined manifestations were highest in Group A. Developmental delay was also seen across all groups, with the highest proportion in Group A, although the association was not statistically significant. Table 3 displays the hematological profiles of the study population. The mean hemoglobin, MCV, TLC and platelet counts were comparable across the three vitamin B12 groups, with no statistically significant differences. Pancytopenia occurred more frequently in Groups A and B compared to Group C, although this difference was not significant. Table 4 summarizes the developmental assessment of children with SAM. Among the 120 children, developmental delay (DQ <70) was present in more than half of the sample. The mean VSMS scores, developmental age, and developmental quotient indicated significant overall developmental compromise in the study population, consistent with the high burden of malnutrition.

Table 1: Association of serum Vitamin B12 level with IYCF indicators and drug intake (n=120)

IYCF indicators & drug intake	Group A (n=20)	Group B (n=33)	Group C (n=67)	p value
Vegetarian diet	11 (55.0%)	22 (66.7%)	45 (67.1%)	0.612
Early initiation of breastfeeding	15 (75.0%)	22 (66.7%)	47 (70.1%)	0.882
Exclusive breastfeeding	12 (60.0%)	22 (66.7%)	35 (52.2%)	0.421
Timely introduction of complementary feeds	11 (55.0%)	22 (66.7%)	45 (67.1%)	0.612
Minimum dietary diversity	7 (35.0%)	16 (48.5%)	32 (47.8%)	0.594
Minimum meal frequency	13 (65.0%)	25 (75.8%)	52 (77.6%)	0.702
Drug intake: Antacids	5 (25.0%)	3 (9.1%)	4 (6.0%)	0.058

Table 2: Association of vitamin B12 levels with clinical signs and symptoms (n=120)

Parameters	Group A (n=20)	Group B (n=33)	Group C (n=67)	p value
Edema	4 (20.0%)	4 (12.1%)	12 (17.9%)	0.781
Pallor	13 (65.0%)	24 (72.7%)	48 (71.6%)	0.928
Signs of vitamin B12 deficiency				
Hyperpigmentation	5 (25.0%)	11 (33.3%)	8 (11.9%)	<0.001
Glossitis	7 (35.0%)	18 (54.5%)	12 (17.9%)	<0.001
Both	8 (40.0%)	4 (12.1%)	2 (3.0%)	<0.001
Developmental delay	15 (75.0%)	16 (48.5%)	38 (56.7%)	0.301

Table 3: Association of vitamin B12 levels with hematological parameters (n=120)

Hematological profile	Group A (n=20)	Group B (n=33)	Group C (n=67)	p value
Hb (g%)	8.94 ± 1.90	8.88 ± 2.95	8.72 ± 2.41	0.944
MCV (fL)	73.70 ± 12.40	70.10 ± 14.90	72.20 ± 13.60	0.709
TLC (/mm³)	12100 ± 6900	12200 ± 7100	11300 ± 5400	0.804
Platelet count (lac/mm³)	2.05 ± 0.92	2.40 ± 1.42	2.04 ± 1.12	0.402
Pancytopenia	2 (10.0%)	3 (9.1%)	0 (0.0%)	0.142

Table 4: Developmental assessment of SAM children (n=120)

Developmental profile	Children (%)	Mean ± SD
VSMS score	—	11.70 ± 11.08
Developmental age (months)	—	8.48 ± 8.94
Developmental quotient (%)	—	58.14 ± 25.49
Developmental delay (DQ <70)	67 (55.8%)	—

Discussion

The present study examined the association between severe acute malnutrition, vitamin B12 deficiency, and developmental outcomes among young children, and the findings align closely with emerging literature emphasizing the neurological vulnerability created by micronutrient deficiencies. Developmental delay was more common among children in the lowest vitamin B12 category, reflecting the central role of cobalamin in myelination, neurotransmitter synthesis, and metabolic functioning.

Recent evidence suggests that the neurodevelopmental impact of vitamin B12 deficiency extends beyond biochemical abnormalities and manifests as long-term cognitive, motor, and behavioral deficits, particularly in resource-constrained settings where malnutrition

remains widespread [11]. This highlights the complexity of the malnutrition–neurodevelopment nexus, where both macronutrient and micronutrient deficiencies interact to impair neural circuitry during early brain development.

The finding that signs such as hyperpigmentation and glossitis were more prevalent in children with low vitamin B12 levels is consistent with the broader metabolic dysfunction described in recent studies. Anemia, mucocutaneous changes, and neurobehavioral symptoms have been recognized as early predictors of underlying cobalamin deficiency, even before overt neurological deficits appear [12]. Emerging biochemical studies further indicate that functional B12 deficiency may persist even with borderline serum levels because of impaired absorption, inflammation, or altered methylation capacity—conditions commonly present in children with SAM [13]. Our findings also resonate with recent pediatric nutrition trials showing that inadequate dietary diversity, late introduction of complementary feeds, and predominantly vegetarian feeding patterns substantially increase the risk of low vitamin B12 levels. A 2024 study reported that even marginal B12 insufficiency could disrupt synaptic plasticity and neurocognitive processing during infancy, emphasizing the need for preventive approaches targeting infant and young-child feeding practices [14]. In addition, recent follow-up research on SAM survivors indicates that deficits in muscle strength, cognition, and school performance persist despite anthropometric recovery, suggesting that micronutrient-related neurodevelopmental insults may be only partially reversible [15].

The present results reinforce the need for early detection, targeted supplementation, and long-term monitoring of vitamin B12 status in malnourished children. Integrating maternal education, dietary counseling, and age-appropriate feeding practices may yield substantial gains in preventing both biochemical and developmental consequences of deficiency. The strong association between low B12 levels and developmental impairment underscores that rehabilitation protocols for SAM must evolve from weight-centric models to more comprehensive frameworks that prioritize neurodevelopmental protection.

Conclusion

The study demonstrates a clear relationship between severe acute malnutrition, vitamin B12 deficiency, and developmental delay. Children with lower vitamin B12 levels exhibited significantly more mucocutaneous signs and higher rates of developmental impairment, emphasizing the critical neurological implications of cobalamin deficiency. These findings highlight the need for early identification of vitamin B12 deficiency, improved dietary diversity, strengthened IYCF practices, and incorporation of neurodevelopmental assessment into SAM management programs. Addressing both nutritional rehabilitation and micronutrient sufficiency is essential for optimizing long-term developmental outcomes in this vulnerable population.

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