Assessment of vitamin B12 status and its determinants in children with Severe acute malnutrition

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Abstract

Background: Severe acute malnutrition (SAM) remains a major global health problem, particularly in low- and middle-income countries, and is frequently associated with micronutrient deficiencies, including vitamin B12. Despite its clinical importance, vitamin B12 deficiency is often under-recognized in SAM.

Objective: To assess serum vitamin B12 status among children with SAM and to identify associated socio-demographic, clinical and haematological determinants.

Methods: This cross-sectional study was conducted at Bangladesh Shishu Hospital & Institute among 98 children aged 6–59 months with SAM. Data on socio-demographic characteristics, feeding practices and anthropometric measurement were collected. Serum vitamin B12 levels were measured using Chemiluminescence Immunoassay, with <203 pg/mL considered deficient. Associations were analyzed using Chi-square and Fisher's exact tests, and logistic regression was performed to identify independent predictors.

Results: Vitamin B12 deficiency was found in 12.2% of children. No significant association was observed with age, gender, or socioeconomic status. Predominant breastfeeding beyond six months (p=0.009) were significantly associated with deficiency. Among clinical and haematological factors, oedema (p=0.011), moderate-to-severe anaemia, and abnormal RBC morphology (both p<0.001) showed strong associations. Multivariate analysis identified prolonged breastfeeding (AOR 4.50), moderate-to-severe anaemia (AOR 22.40), and macrocytic/dimorphic RBC morphology (AOR 35.60) as independent predictors.

Conclusion: Vitamin B12 deficiency constitutes a significant micronutrient concern among children with severe acute malnutrition and is significantly associated with feeding patterns, and haematological alterations.

Keywords: Severe acute malnutrition, Vitamin B12 deficiency

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Introduction:

Severe acute malnutrition (SAM) remains a major global public health challenge, particularly in low- and middle-income countries, contributing significantly to child morbidity and mortality.[1] Globally, an estimated 13.7 million children under five years are affected by SAM, hindering progress toward Sustainable Development Goal 2 (Zero Hunger).[2] The burden is especially high in Asia, and in Bangladesh, 11% of under-five children are wasted, including 2% who are severely wasted.[3]

Micronutrient deficiencies commonly coexist with SAM and may worsen clinical outcomes. Among these, vitamin B12 deficiency is often overlooked in standard management protocols, which mainly emphasize macronutrient rehabilitation and selected micronutrients such as iron, folate, zinc, and vitamin A.[4,5] Vitamin B12 deficiency primarily results from inadequate intake of animal-source foods and is further exacerbated in SAM due to poor dietary intake and impaired absorption.[6] Although therapeutic diets like Ready-To-Use Therapeutic Food (RUTF), F-75, and F-100 may improve B12 status, they are often insufficient to fully correct the deficiency.[7]

Vitamin B12 deficiency has important haematological and neurological consequences, particularly affecting brain development during early life. Its deficiency affects multiple systems, ranging from mild fatigue to severe neurological impairment. It commonly causes bone marrow suppression involving all cell lines, most frequently presenting as megaloblastic anaemia. It can lead to neurodevelopmental delay, hypotonia, seizures, and long-term cognitive impairment if not treated promptly.[8] Despite this, deficiency is frequently underdiagnosed due to subtle clinical presentation.[9] In Bangladesh, although data among SAM children are lacking, vitamin B12 deficiency has been reported in 31% of infants and 20.2% of women of reproductive age, suggesting a high-risk population.[10]

Therefore, this study was undertaken to assess serum vitamin B12 status among children with SAM and to identify associated socio-demographic, clinical and haematological determinants with the aim of providing evidence to guide more comprehensive and targeted management strategies.

Materials and Methods:

This cross-sectional study was conducted in the Department of Paediatric Gastroenterology, Hepatology and Nutrition at Bangladesh Shishu Hospital & Institute from February 2024 to February 2025. A total of 98 children aged 6–59 months admitted with SAM were enrolled using purposive sampling. SAM was defined by any of the following WHO criteria: 1) Mid upper arm circumference (MUAC) <115 mm, 2) Weight-for-Length/Height Z-score <-3 SD, 3) Presence of bilateral pitting oedema. Children with a history of birth asphyxia, hypoxic-ischemic encephalopathy, prematurity, low birth weight, neonatal sepsis, significant neonatal hyperbilirubinaemia requiring admission, known neurological or genetic disorders, prior vitamin B12 supplementation or blood transfusion within the last 3 months, secondary malnutrition, or unstable clinical conditions were excluded.

After obtaining informed written consent, detailed information on sociodemographic characteristics, feeding practices, and medical history was collected using a semi structured proforma. Anthropometric measurements, including weight, length/height, and mid-upper arm circumference (MUAC), were recorded using standard techniques. Weight-for-height/length Z-score (WHZ), height-for-age Z-score (HAZ), and MUAC were assessed according to WHO Child Growth Standards.

The severity of anaemia was classified according to the age-specific WHO criteria.[11] For children aged 6–23 months, anaemia was categorized as mild (haemoglobin 95–104 g/L), moderate (70–94 g/L), and severe (<70 g/L). For children aged 24–59 months, anaemia was categorized as mild (haemoglobin 100–109 g/L), moderate (70–99 g/L), and severe (<70 g/L). Red cell morphology was classified according to cell size using mean corpuscular volume (MCV). Microcytic anaemia was defined as an MCV below the lower limit of normal for age (MCV < 70 + age in years, fL), while normocytic anaemia was defined as an MCV within the normal age-specific range. Macrocytic anaemia was defined as an MCV above the upper limit of normal for age (MCV > 84 + [age in years × 0.6], fL). Dimorphic anaemia was diagnosed when features of both microcytic and macrocytic anaemia were present.[12] Prior to initiation of treatment, serum vitamin B12 level was assessed using a Chemiluminescence Immunoassay Analyzer (IFlash 1800), with levels <203 pg/mL considered deficient.

Data were analysed using SPSS version 27.0. Continuous variables were presented as mean ± SD or median (IQR), and categorical variables as frequencies and percentages. Associations were assessed using Pearson's chi-square or Fisher's exact test. Significant variables in univariate analysis were entered into multivariate logistic regression to identify independent predictors of vitamin B12 deficiency. Odds ratios with 95% CIs were reported, with $p<0.05$ considered statistically significant.

Results:

A total of 98 children with severe acute malnutrition were included in the study. Among the study participants, majority (71.4%) were aged between 6-12 months. Males (64.3%) outnumbered females (35.7%). In terms of SES, most belonged to the upper-lower class (54.1%). The feeding practices showed that only 26.5% were exclusively breastfed during the first 6 months. Complementary feeding was introduced at the recommended 6 months in only 10.2% of cases. Predominant breastfeeding (BF) beyond 6 months was observed in 37.8% of infants (Table I).

Table I: Socio-demographic profile of the study participants (N=98)

Variables	No.	%
Age group (months)		
6-12	70	71.4
13-24	19	19.4
25-59	9	9.2
Gender		
Male	63	64.3
Female	35	35.7
Residence		
Rural	47	48.0
Urban	51	52.0
Socioeconomic status		
Lower middle	14	14.3
Upper lower	53	54.1
Lower	31	31.6
Feeding during 1st 6 months		
Exclusive breastfeeding	26	26.5
Mixed feeding	34	34.7
Formula feeding	38	38.8
Age of introducing complementary feeding		
at 6 months	10	10.2
<6 months	32	32.7
>6 months	56	57.1
Predominant breast feeding >6 months		
Yes	37	37.8
No	61	62.2

Vitamin B_{12} deficiency was present in 12.2% of participants, while the majority (87.8%) were not deficient. The mean B12 level was 319.68 pg/mL with a standard deviation of 80.45. The median value was 338.50 pg/mL, with levels ranging from a minimum of 102 pg/mL to a maximum of 413 pg/mL (Figure 1).

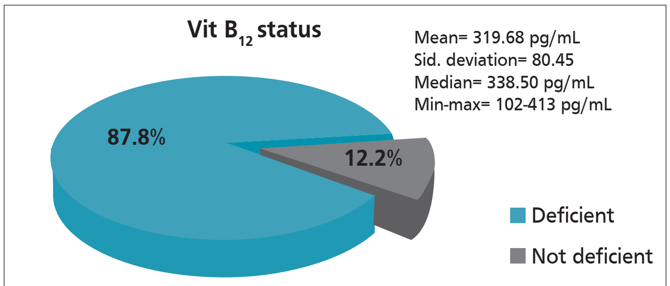

Figure 1: Status of serum vitamin B12 among children with SAM (N=98)

The association of vitamin B12 deficiency with socio-demographic factors indicated no significant association with age, gender or socio-economic status (Table II). But deficiency was more commonly (83.3%) observed in children age group 6-12 months compared to the not deficient group (69.8%). The association of vitamin B12 deficiency with feeding practices demonstrated that predominant breast feeding (BF) beyond six months was significantly associated with B12 deficiency (p=0.009).

Table II: Association of vitamin B12 deficiency with socio-demographic factors (N=98)

Variables	Vitamin-B12 Deficient (n=12)	Not deficient (n=86)	*p*-value
Age (months)			
6-12	10 (83.3)	60 (69.8)	
13-24	2(16.7)	17 (19.8)	0.454
25-59	0(0.0)	9 (10.4)	
Median (IQR)	8.5 (7.25-11.50)	10.0 (8.0-14.0)	0.212
Gender			
Male	6 (50.0)	57 (66.3)	0.339
Female	6 (50.0)	29 (33.7)	
Socioeconomic status			
Lower middle	0 (0.0)	14 (16.3)	
Upper lower	8 (66.7)	45 (52.3)	0.306
Lower	4 (33.3)	27 (31.4)	
Feeding during 1st 6m			
EBF	4 (33.3)	22 (25.6)	
Mixed feeding	5 (41.7)	29 (33.7)	0.577
Formula feeding	3 (25.0)	35 (40.7)	
Predominant BF >6m	9 (75.0)	28 (32.6)	0.009
Age of introducing complementary feeding			
at 6 months	1 (8.3)	9 (10.5)	
<6 months	3 (25.0)	29 (33.7)	0.775
>6 months	8 (66.7)	48 (55.8)	

The association of vitamin B12 deficiency with clinical and biochemical factors revealed no significant correlation with wasting, stunting or MUAC <115 mm. However, oedema was significantly associated (p=0.011). The severity of anaemia showed a strong association (p<0.001). RBC morphology was also significantly linked (p<0.001) with macrocytosis (50%) and dimorphic RBCs (33.3%) being more prevalent in B12-deficient children (Table III).

Table III: Association of vitamin B12 deficiency with clinical and biochemical factors (N=98)

Variables	Vitamin-B12 Deficient (n= 12)	Not deficient (n= 86)	*p*-value
Wasting			
Yes	10 (83.3)	59 (68.2)	0.50
No	2 (16.7)	27 (31.8)	
Stunting			
Yes	4 (33.3)	28 (33.0)	1.00
No	8 (66.7)	58 (67.0)	
MUAC <115 mm			
Yes	8 (66.7)	59 (68.6)	1.00
No	4 (33.3)	27 (31.4)	
Oedema			
Yes	9 (75)	30 (34.9)	0.011
No	3 (25)	56 (65.1)	
Anaemia			
No anaemia	0 (0.0)	17 (19.8)	
Mild	1 (8.3)	55 (64.0)	<0.001
Moderate	7 (58.3)	11 (12.8)	
Severe	4 (33.3)	3 (3.4)	
RBC morphology			
Normocytic	2 (16.7)	59 (68.6)	
Microcytic	0 (0.0)	23 (26.7)	<0.001
Macrocytic	6 (50.0)	2 (2.3)	
Dimorphic	4 (33.3)	2 (2.3)	

On univariate analysis, prolonged predominant breastfeeding beyond six months, presence of oedema, moderate-to-severe anaemia, and abnormal RBC morphology were significantly associated with vitamin B12 deficiency (Table IV). On multivariate logistic regression analysis, after adjustment, prolonged predominant breastfeeding beyond six months, moderate-to-severe anaemia, and macrocytic/dimorphic RBC morphology remained significant independent predictors of vitamin B12 deficiency. The strongest associations were observed with abnormal RBC morphology and anaemia, indicating a robust haematological correlation. Although oedema showed increased odds, it did not remain statistically significant after adjustment.

Table IV: Determinants of Vitamin B12 deficiency (N=98)

Variable	Crude OR (95% CI)	*p*-value	Adjusted OR (95% CI)	*p*-value
Predominant BF >6 m	6.22 (1.58–24.42)	0.009	4.50 (1.20–16.85)	0.03
Oedema	5.60 (1.38–22.68)	0.011	2.90 (0.78–10.74)	0.09
Moderate–severe anaemia	56.67 (6.42–500.12)	<0.001	22.40 (3.95–127.01)	<0.001
Macrocytic/dimorphic RBC	102.50 (15.30–686.90)	<0.001	35.60 (6.80–186.40)	<0.001

Discussion

This study evaluated serum vitamin B12 status and its determinants among children with severe acute malnutrition (SAM). The prevalence of vitamin B12 deficiency in the present study was 12.2%, with a mean serum level of 319.68±80.45 pg/mL. This prevalence is lower than that reported by Dutta et al., Verma et al., and Murthy et al., who documented higher frequencies of vitamin B12 deficiency among children with severe acute malnutrition.[4,14,15] However, it is comparable to findings by Bahalkani.[16] Such variations may be attributed to differences in dietary habits, particularly the lower prevalence of vegetarian diet in the study population, as well as variations in maternal nutritional status and regional feeding practices.

The majority of study participants were aged between 6–12 months, and although vitamin B12 deficiency was more frequently observed in this age group, the association was not statistically significant. This trend is consistent with previous studies done by Bahalkani and Dutta et al.[16,4] which have identified early infancy as a vulnerable period due to rapid growth demands and dependence on maternal vitamin B12 stores. The absence of statistical significance in this study may be due to the relatively small sample size.

Socioeconomic status did not show a statistically significant association with vitamin B12 deficiency. Nevertheless, a higher proportion of deficient children belonged to lower socioeconomic strata, suggesting that economic constraints may indirectly influence dietary quality and access to animal-source foods. Similar findings have been reported in previous studies[4,16] reinforcing the role of socioeconomic factors as underlying contributors.

Infant feeding practices were also significantly associated with vitamin B12 deficiency. Predominant breastfeeding beyond six months was identified as an independent predictor, even after adjustment. This may be explained by the low vitamin B12 content in breast milk of deficient mothers, particularly when complementary feeding is delayed or inadequate. These findings are consistent with previous reports by Goyal et al.,[6] emphasizing the importance of timely and appropriate complementary feeding.

Regarding clinical parameters, no significant association was observed between vitamin B12 deficiency and wasting, stunting, or MUAC <115 mm. This suggests that vitamin B12 deficiency may occur independently of the severity of anthropometric malnutrition. However, oedema showed a significant association on univariate analysis but did not retain significance after adjustment, indicating that its effect may be confounded by other clinical factors.

A prominent finding of this study was the strong association between vitamin B12 deficiency and haematological parameters. Moderate-to-severe anaemia and abnormal RBC morphology (macrocytic and dimorphic patterns) were identified as the strongest independent predictors in multivariate analysis, with markedly elevated odds ratios. These findings are in agreement with previous studies[17,18] which have highlighted the close relationship between vitamin B12 deficiency and haematological abnormalities. The pathophysiological basis lies in the role of vitamin B12 in DNA synthesis and erythropoiesis, where deficiency leads to impaired nuclear maturation, resulting in macrocytosis. The presence of dimorphic anaemia may further indicate coexisting micronutrient deficiencies such as iron deficiency.[10]

Overall, the findings from this study highlight that vitamin B12 deficiency in children with SAM is primarily influenced by infant feeding patterns, and haematological status, rather than by socio-demographic or anthropometric factors alone. The results underscore the multifactorial nature of vitamin B12 deficiency and the importance of addressing both maternal and child-related determinants.

Conclusion

Vitamin B12 deficiency was present in a notable proportion of children with severe acute malnutrition and was independently associated with prolonged predominant breastfeeding, moderate-to-severe anaemia, and abnormal RBC morphology.

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