e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(9); 610-614

Original Research Article

Clinical and Etiological Profile of Anemia in Children with Severe Acute Malnutrition: A Case-Control Study

Parita Pancha[1], Hemant Jain[2]

[1,2]Department of Pediatrics, Geetanjali Medical College and Hospital, Udaipur, Rajasthan, India

Received: 01-06-2026 / Revised: 15-07-2026 / Accepted: 21-08-2026

Corresponding author: Dr. Hemant Jain

Conflict of interest: Nil

Abstract

Background: Anemia is a frequent and clinically important comorbidity in children with severe acute malnutrition (SAM), but its severity and etiological profile are multifactorial. This study compared the prevalence, severity, hematological characteristics, micronutrient-related parameters, and outcomes of anemia in children with SAM and age- and sex-matched non-SAM controls.

Methods: This hospital-based case-control study was conducted in the Department of Pediatrics, Geetanjali Medical College and Hospital, Udaipur, over 1.5 years. Children aged 6–59 months with SAM were enrolled consecutively, with age- and sex-matched children without SAM serving as controls. SAM was defined by weight-for-height below −3 SD, MUAC <11.5 cm, or bilateral pitting nutritional edema. Children with congenital malformations or congenital anemia and those without parental consent were excluded. Clinical and anthropometric assessments were performed, followed by complete blood count, peripheral blood film, reticulocyte count, serum vitamin B12, and iron profile evaluation.

Results: A total of 136 children were analyzed, 68 in each group. Anemia was present in 61 (89.7%) children with SAM compared with 6 (8.8%) controls ($p<0.01$). In the SAM group, 48 (70.6%) had moderate anemia, and 13 (19.1%) had severe anemia; No child in the SAM group had mild anemia. Mean hemoglobin was significantly lower in SAM than controls (8.42±1.58 vs 12.04±0.55 g/dL; $p<0.01$). SAM children had lower MCV, MCH, MCHC, RBC count, and reticulocyte count, with higher WBC and platelet counts. Mean vitamin B12 was lower in SAM (175.65±46.33 vs 304.46±36.27 pg/mL; $p<0.01$). Serum iron and ferritin were lower, and TIBC was higher in SAM. Mean hospital stay was significantly longer in SAM (17.38±1.46 vs 9.85±1.48 days; $p<0.01$). Mortality was 2.9% in SAM and 0% in controls ($p=0.15$).

Conclusion: SAM was strongly associated with a high prevalence and greater severity of anemia, altered erythrocyte indices, impaired reticulocyte response, and abnormalities in vitamin B12 and iron-related parameters. Routine anemia screening and comprehensive micronutrient and infection assessment should be integrated into the management of children with SAM.

Keywords: Severe Acute Malnutrition; Anemia; Children; Vitamin B12; Iron Profile; Reticulocyte Count; Micronutrient Deficiencies.

DOI: 10.25258/ijcpr.18.9.88

Introduction

Severe acute malnutrition (SAM) remains an important cause of childhood morbidity and mortality, particularly in low- and middle-income countries. In children aged 6–59 months, SAM is defined by severe wasting, a low mid-upper arm circumference, or bilateral nutritional edema. The condition is accompanied by metabolic, immune, and hematological disturbances and substantially increases susceptibility to infection and adverse outcomes. [1,2,3,4]

Anemia is one of the most frequent comorbidities in children with malnutrition. Its causes are not limited to iron deficiency and may include multiple micronutrient deficiencies, chronic inflammation, recurrent infection, impaired erythropoiesis, blood loss, and other systemic or hematological disorders. In SAM, inadequate nutrient intake and absorption may coexist with inflammatory pathways that alter iron utilization and suppress erythroid production. [5,6,7,8]

Previous studies have reported a high prevalence of anemia among children with SAM, but the reported magnitude and morphological pattern vary between populations. Some studies have identified predominantly microcytic anemia, whereas others have emphasized megaloblastic or mixed patterns

related to vitamin B12 and folate deficiency. Comparative studies including non-malnourished controls are relatively limited. [9,10,11,12,13] The present study was therefore undertaken to compare the prevalence and severity of anemia, hematological and biochemical characteristics, selected micronutrient-related parameters, and clinical outcomes between children with SAM and age- and sex-matched non-SAM controls.

Aim and Objectives

To study the clinical and etiological profile of anemia in children with severe acute malnutrition.

1. To compare the prevalence of anemia among children with SAM and non-SAM controls.
2. To compare the clinico-etiological profile of anemia among children with SAM and non-SAM controls.
3. To compare outcomes, including mortality and duration of hospitalization, between the two groups.

Materials and Methods

Study Design and Setting: This hospital-based case-control study was conducted in the Department of Pediatrics, Geetanjali Medical College and Hospital, Udaipur, Rajasthan, over a period of 1.5 years.

The study included children aged 6–59 months with SAM and age- and sex-matched children without SAM as controls.

Sample Size and Sampling: The thesis sample-size calculation used a 95% confidence level, 80% power, 5% absolute error, and an assumed SAM prevalence of 2.2%, yielding a minimum sample size of 67. To allow balanced comparison, 68 children were enrolled in each group. Consecutive sampling was used.

Eligibility Criteria: SAM was defined using any of the following criteria: weight-for-height below −3 SD of the WHO median, MUAC <11.5 cm, or bilateral pitting nutritional edema. Age- and sex-matched children without SAM were included as controls. Children with congenital malformations, congenital anemia, or without parental consent were excluded.

Clinical and Laboratory Assessment: Clinical history, examination, and anthropometric assessment were performed using standard methods. Laboratory evaluation included complete blood count, peripheral blood film, reticulocyte count, serum vitamin B12, and iron profile. CBC parameters were measured using automated hematology analyzers documented in the thesis; vitamin B12 was estimated by electrochemiluminescence immunoassay and iron profile by colorimetric assay.

Ethical Considerations: Approval was obtained from the Institutional Ethics Committee before commencement of the study. Written informed consent was obtained from parents/guardians. Participant confidentiality was maintained.

Statistical Analysis: Data were analyzed using statistical software (the thesis records SPSS among the study tools). Categorical variables were expressed as numbers and percentages, and continuous variables as Mean±SD. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate.

Continuous variables were expressed as mean ± standard deviation and compared using the independent-samples t-test where applicable. A two-sided p-value <0.05 was considered statistically significant.

Before submission, the authors should insert the exact SPSS version and the exact continuous-variable test used in the original analysis, because these details are not explicitly stated in the thesis methods section.

Results

A total of 136 children aged 6–59 months were analyzed: 68 children with SAM and 68 age- and sex-matched non-SAM controls. The mean age was 33.12±15.37 months in the SAM group and 32.35±15.47 months in controls (p=0.78). Females constituted 52.9% and 54.4% of the SAM and control groups, respectively (p=0.80).

Table 1: Key clinical and demographic characteristics

Variable	SAM (n=68)	Non-SAM (n=68)	p value
Mean age, months	33.12±15.37	32.35±15.47	0.78
Female sex	36 (52.9%)	37 (54.4%)	0.80
Rural residence	54 (79.4%)	45 (66.2%)	0.01
Previous hospitalization	15 (22.1%)	7 (10.3%)	0.0014
Exclusive breastfeeding	25 (36.8%)	37 (54.4%)	0.03

Rural residence was more common among SAM children (79.4% vs 66.2%, p=0.01). Previous hospitalization was reported in 22.1% of SAM children compared with 10.3% of controls (p=0.0014). Exclusive breastfeeding was less frequent in SAM (36.8% vs 54.4%, p=0.03). Pallor, tiredness, failure to gain weight, vomiting, dizziness, and loose stools were more frequently reported among SAM children.

Table 2: Prevalence and severity of anemia

Anemia category	SAM (n=68)	Non-SAM (n=68)
Anemia present	61 (89.7%)	6 (8.8%)
Mild	0 (0%)	6 (8.8%)
Moderate	48 (70.6%)	0 (0%)
Severe	13 (19.1%)	0 (0%)
No anemia	7 (10.3%)	62 (91.2%)

Anemia was present in 89.7% of children with SAM compared with 8.8% of controls (χ^2=92.05, p<0.01). Moderate anemia was the predominant category in SAM (70.6%), followed by severe anemia (19.1%). No child with SAM had isolated mild anemia.

Table 3: Hematological profile

Parameter	SAM (Mean±SD)	Non-SAM (Mean±SD)	p value
Hemoglobin, g/dL	8.42±1.58	12.04±0.55	<0.01
MCV, fL	70.12±5.96	74.90±4.18	<0.01
MCH, pg	21.85±2.44	24.03±1.81	<0.01
MCHC, g/dL	30.30±1.06	31.53±0.95	<0.01
WBC, /mm³	16283.79±1120.17	12885.13±1905.15	<0.01
RBC, million/mm³	3.16±0.43	3.93±0.23	<0.01
Platelets, $\times 10^5/\mu L$	3.90±0.67	3.27±0.31	<0.01
Reticulocyte count, %	0.60±0.25	1.38±0.14	<0.01

Children with SAM had significantly lower hemoglobin, MCV, MCH, MCHC, RBC count, and reticulocyte count, along with higher WBC and platelet counts than controls. The reduced MCV, MCH, and MCHC suggest a predominantly microcytic-hypochromic pattern, while the lower reticulocyte count suggests a reduced erythropoietic response.

Table 4: Selected micronutrient and iron-profile parameters

Parameter	SAM (Mean±SD)	Non-SAM (Mean±SD)	p value
Vitamin B12, pg./mL	175.65±46.33	304.46±36.27	<0.01
Serum iron, µg/dL	34.09±9.08	49.75±8.56	<0.01
Serum ferritin, ng/mL	176.20±75.42	198.68±29.87	0.03
TIBC, µg/dL	401.34±33.24	374.74±27.94	<0.01

Vitamin B12 levels were substantially lower in SAM. Serum iron and ferritin were lower, and TIBC was higher in SAM, demonstrating significant alterations in iron-related parameters. Because ferritin is influenced by inflammation, these findings should be interpreted as an altered iron profile rather than proof of isolated iron-deficiency anemia.

Table 5: Clinical outcomes

Outcome	SAM (n=68)	Non-SAM (n=68)	p value
Mean hospital stay, days	17.38±1.46	9.85±1.48	<0.01
Mortality	2 (2.9%)	0	0.15

Mean hospital stay was significantly longer in children with SAM. Mortality was observed in two SAM children and none of the controls; the difference was not statistically significant.

Discussion

The principal finding of this study was the very high burden of anemia among children with SAM. Nearly nine of every ten SAM children were anemic, and most had moderate or severe anemia. This burden was substantially greater than that observed among matched controls. The finding is consistent with earlier hospital-based studies reporting anemia prevalence in the range of approximately 85–95% among children with SAM [9,10,11,12,13] The severity distribution is clinically important. In the present study, 70.6% of SAM children had moderate anemia and 19.1% had severe anemia, whereas controls had no moderate or severe anemia. Thakur et al. reported anemia in 90% of children with SAM, with severe anemia in 67.3%, while Dwivedi et al. reported anemia in 85% of SAM children. [9,10] Differences in severity across studies may reflect variation in case mix, referral patterns, infection burden, nutritional status, and study setting.

The hematological profile supports a major disturbance of erythropoiesis in SAM. Hemoglobin and RBC counts were lower, and MCV, MCH, and MCHC were reduced, indicating a predominantly microcytic-hypochromic pattern. Similar findings have been reported in other pediatric SAM cohorts. [12,13,14] However, anemia in SAM should not be

regarded as synonymous with iron deficiency because inflammation and other micronutrient deficiencies can coexist.

The low reticulocyte counts in SAM compared with controls provide additional evidence of inadequate marrow response. This may reflect protein and micronutrient deficiency, inflammatory suppression of erythropoiesis, infection, and altered iron availability. The concomitant higher WBC and platelet counts may also indicate greater inflammatory or infectious burden in the SAM group.

Vitamin B12 was markedly lower in children with SAM. Shukla et al. similarly demonstrated significant abnormalities of vitamin B12 and iron status in children with SAM, supporting the concept that anemia in malnutrition is frequently multifactorial. [16] The present study did not include folate estimation; therefore, the relative contribution of folate deficiency cannot be determined.

The iron profile showed lower serum iron and ferritin with higher TIBC in SAM. Reduced serum iron with increased TIBC is compatible with iron-restricted erythropoiesis, but ferritin interpretation is difficult in the presence of inflammation because ferritin is an acute-phase reactant.

The findings therefore support disturbed iron metabolism rather than allowing a definitive classification of isolated iron-deficiency anemia. Children with SAM had a significantly longer hospital stay than controls.

This is consistent with the greater clinical and infectious burden associated with severe malnutrition and with previous literature showing prolonged hospitalization among malnourished children. The observed mortality difference did not reach statistical significance, likely in part because only two deaths occurred and the sample size was small.

The study also identified several characteristics associated with SAM, including rural residence, previous hospitalization, lower prevalence of exclusive breastfeeding, and a higher frequency of gastrointestinal and other clinical complaints. These observations reinforce the importance of early nutritional assessment, breastfeeding support, infection prevention, and comprehensive management of micronutrient deficiencies.

Strengths

- Age- and sex-matched control group enabled direct comparison with children without SAM.
- The study evaluated anemia using clinical, hematological, reticulocyte, vitamin B12, and iron-profile parameters rather than hemoglobin alone.
- Hospital outcomes, including duration of stay and mortality, were assessed.

Limitations

- This was a single-center hospital-based study with a relatively small sample size, limiting generalizability.
- The study was observational and therefore cannot establish causality between SAM and anemia.
- Folate levels and other micronutrients were not assessed, so the complete etiological spectrum of anemia could not be characterized.
- Inflammatory markers were not incorporated into the final etiological classification; therefore, ferritin should be interpreted cautiously.
- The exact statistical software version and the specific tests used for continuous variables are not explicitly documented in the thesis methods section and should be verified from the original statistical analysis before journal submission.

Conclusion

Severe acute malnutrition was strongly associated with a markedly higher prevalence and severity of anemia in children aged 6–59 months. SAM children demonstrated lower hemoglobin and red-cell indices, a reduced reticulocyte response, lower vitamin B12, and significant abnormalities in iron-related parameters. They also had a substantially longer hospital stay. These findings support routine anemia screening and comprehensive evaluation of micronutrient and infectious contributors in all children with SAM. Management should integrate nutritional rehabilitation with appropriate identification and correction of anemia-related deficiencies.

Declarations

Ethics Approval: The study was approved by the Institutional Ethics Committee of Geetanjali Medical College and Hospital, Udaipur.

Informed Consent: Written informed consent was obtained from the parents/guardians of all participants.

Data availability: Data are available from the corresponding author on reasonable request, subject to institutional and ethical restrictions.

Author contributions: Dr. Parita Pancha contributed to study conception, data collection, analysis, and manuscript preparation. Dr. Hemant Jain contributed to study supervision, interpretation of findings, and critical revision of the manuscript.

This should be retained only if it accurately reflects the actual contributions.

References

1. World Health Organization. Identification of severe acute malnutrition in children 6–59 months of age. Geneva: World Health Organization; 2023.
2. International Institute for Population Sciences (IIPS), ICF. National Family Health Survey (NFHS-5), 2019–21. Mumbai: IIPS; 2022.
3. Chama GC, Siame L, Kapoma C, Hamooya BM, Masenga SK. Severe acute malnutrition among children under the age of 5 years. PLoS One. 2024;19(8):e0309122.
4. Dipasquale V, Cucinotta U, Romano C. Acute malnutrition in children: pathophysiology, clinical effects and treatment. Nutrients. 2020;12(8):2413.
5. Venigalla WC, Nirmala C, Harshita C, Meghi SR. A study of the prevalence of anemia in children with severe acute malnutrition at a pediatric tertiary care hospital in South India. Cureus. 2024;16(8):e67657.
6. Takele WW, Baraki AG, Wolde HF, Desyibelew HD, Derseh BT, Dadi AF, et al. Anemia and contributing factors in severely malnourished infants and children aged 0–59 months admitted to treatment centers in the Amhara region, Ethiopia: a multicentre chart review study. Anemia. 2021;2021:6636043.
7. Mishra M, Raghav A, Tripathi P, Rao YK, Singh DD. Evaluation of micronutrients and pro-inflammatory cytokine levels in nutritiona lly deprived children: a tertiary care hospital-based study. Nutrients. 2023;15(23):4865.
8. Kulkarni B, Mamidi RS. Nutrition rehabilitation of children with severe acute malnutrition. Indian J Med Res. 2019;150(2):139-152.
9. Thakur N, Chandra J, Pemde H, Singh V. Anemia in severe acute malnutrition. Nutrition. 2014;30(4):440-442.
10. Dwivedi D, Singh V, Singh J, Sharma S. Study of anemia in children with severe acute malnutrition. J Nepal Paediatr Soc. 2017;37(3):250-253.
11. Rose N, Goel M, Patil R, Motwani B, Shrivastava J. Haemogram and iron profile in children suffering from severe acute malnutrition at a tertiary care centre, Bhopal: a cross-sectional study. J Clin Diagn Res. 2023;17(7).
12. Arya AK, Kumar P, Midha T, Singh M. Hematological profile of children with severe acute malnutrition: a tertiary care centre experience. Int J Contemp Pediatr. 2017;4(5):1577-1580.
13. Khan S, Rubab Z, Hussain S, Abbas A, Arshad R, Tareen BMK. Hematological profile of children with severe acute malnutrition at a tertiary care hospital in Multan. Isra Med J. 2020;12(1):12-16.
14. Getawa S, Getaneh Z, Melku M. Hematological abnormalities and associated factors among undernourished under-five children attending the University of Gondar Specialized Referral Hospital, Northwest Ethiopia. J Blood Med. 2020;11:465-474.
15. Shukla P, Pandey SK, Singh J, Bajaj N, Tripathi G, Dwivedi S. Clinico-etiopathogenesis of vitamin B12, folic acid and iron deficiency in children with severe acute malnutrition: a tertiary care hospital experience from Central India. Indian J Clin Biochem. 2024;39(2):221-225.
16. Khanna S, Kumar P, Sharma S, Chandra J, Sinha R. Nutritional and hematological profile of children with severe acute malnutrition rehabilitated with or without vitamin B12. Int J Community Med Public Health. 2022;9(2):882-886.
17. Patil RGP, Machakanur VL, Marol RN, Prakash MH, Naik MK. Anemia and hematological profile in children with moderate and severe acute malnutrition at a tertiary care center in Karnataka, India: a cross-sectional study. J Clin Diagn Res. 2026;20(4):SC33-SC36.
18. World Health Organization. Anaemia. Geneva: World Health Organization; 2025.
19. Jeevan J, Karun KM, Puranik A, Deepa C, Lakshmanan MK, Barvaliya M. Prevalence of anemia in India: a systematic review, meta-analysis and geospatial analysis. BMC Public Health. 2025;25(1):1270.
20. Topal A, Tolunay O. Effect of malnutrition on length of hospital stay in children. Turk Arch Pediatr. 2021;56(1):37-42.