INSIGHTS-JOURNAL OF
LIFE AND SOCIAL SCIENCES

INSIGHTS-JLSS
Insights-Journal of Life and Social Sciences

CHILDREN WITH SEVERE ACUTE MALNUTRITION: AN EVALUATION OF THEIR HEMATOLOGICAL PARAMETERS: A STUDY CONDUCTED IN LARKANA

Original Article

Ishfaque Ahmed Mugheri[1]*, Delijan Mugheri[2], Mehar Ali Kazi[1], Nazia Faraz Shaikh[2], Faheem[1], Kashaf Faisal[3]
[1]Institute of Biochemistry, University of Sindh, Pakistan.
[2]Department of Pediatrics, Chandka Medical College, SMBBMU Larkana, Pakistan.
[3]Department of Nutrition and Food Sciences, University of Sindh, Pakistan.
Corresponding Author: Ishfaque Ahmed Mugheri, Institute of Biochemistry, University of Sindh, ishfaque339@gmail.com

Conflict of Interest: None
Grant Support & Financial Support: None
Acknowledgment: The authors sincerely acknowledge all participants and contributors for their valuable support in this study.

ABSTRACT

Background: Severe acute malnutrition (SAM) remains a critical public health issue, particularly in resource-limited settings, significantly impacting child morbidity and mortality. Malnutrition adversely affects hematopoiesis, leading to anemia, leukocyte abnormalities, and thrombocytopenia, which further compromise immune function and increase susceptibility to infections. Understanding the hematological alterations in children with SAM is essential for early detection, appropriate management, and improved clinical outcomes. This study aimed to assess the hematological profile of children with SAM and identify specific abnormalities associated with the condition.

Objective: To evaluate the hematological parameters of children with severe acute malnutrition and compare them with healthy controls.

Methods: This descriptive cross-sectional study was conducted over nine months, from January to September 2023, at the Nutrition Unit of PEDS Shaikh Zayed Hospital, Larkana. A total of 216 children aged 7 months to 5 years were included, with 108 diagnosed with SAM based on WHO criteria and 108 healthy controls. Hematological parameters, including red blood cell (RBC) count, hemoglobin (Hb), hematocrit (HCT), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), white blood cell (WBC) count, and platelet count, were analyzed using an automated hematology analyzer (XN-350). Socioeconomic and demographic data were collected through structured questionnaires.

Results: Among the 108 children with SAM, 56 (51.8%) were male, and 52 (48.1%) were female. The majority, 68 (49.2%), were aged 7–24 months. Rural residency was reported in 67 (62%) cases, and 74 (68.5%) belonged to low-income families. Normochromic normocytic anemia was the most prevalent type, affecting 42 (38.88%) children, followed by iron deficiency anemia in 28 (25.9%) and megaloblastic anemia in 2 (1.85%). Hemoglobin levels were significantly lower in SAM cases (8.13±1.09 g/dL) than in controls (9.80±1.21 g/dL) ($p=0.0005$). Hematocrit was also reduced in cases (25.07±3.34%) compared to controls (30.88±4.02%) ($p=0.0005$). RBC count was lower in cases (3.99±0.71 $\times 10^6/\mu L$) than controls (4.10±0.51 $\times 10^6/\mu L$) ($p=0.0003$). MCV, MCH, and MCHC were all significantly reduced in cases (60.45±4.93 fL, 18.75±2.15 pg, and 30.11±1.74 g/dL, respectively) compared to controls (70.35±6.14 fL, 24.39±2.99 pg, and 32.94±1.72 g/dL, respectively) ($p<0.05$). WBC count was lower in the SAM group (6.99±1.64 $\times 10^3/\mu L$) compared to controls (8.24±4.28 $\times 10^3/\mu L$) ($p=0.005$), and platelet count was also significantly reduced (262.36±85.22 $\times 10^3/\mu L$ vs. 302.11±128.15 $\times 10^3/\mu L$) ($p=0.002$).

Conclusion: Children with severe acute malnutrition exhibited significant hematological abnormalities, including anemia, leukocyte alterations, and thrombocytopenia. The high prevalence of normochromic normocytic anemia and iron deficiency anemia underscores the profound impact of malnutrition on hematopoiesis. These findings emphasize the need for early identification, targeted nutritional interventions, and routine hematological monitoring to improve health outcomes in malnourished children.

Keywords: Anemia, Blood Cell Count, Child Malnutrition, Hematology, Nutritional Deficiency, Severe Acute Malnutrition, Thrombocytopenia.

INTRODUCTION

Malnutrition, characterized by insufficient energy and micronutrient intake, remains a major global health challenge, significantly affecting children in low- and middle-income countries. It impairs essential bodily functions and daily activities, leading to severe complications across various organ systems. The World Health Organization (WHO) has highlighted malnutrition as a pressing concern, with Pakistan experiencing a particularly alarming prevalence. Approximately two out of every five children under five years suffer from malnutrition, making it one of the country's most critical public health issues. The Pakistan National Nutrition Survey reports that 31% of children are underweight, 24% are malnourished, 17% are wasted, and 33% suffer from iron deficiency. The relationship between malnutrition and hematological disorders is well established, as severe acute malnutrition (SAM) induces extensive alterations in the hematopoietic system, affecting all blood cell components (1-4). Anemia, a frequent consequence of SAM, is primarily driven by iron deficiency, which stems from inadequate dietary intake, reduced milk consumption, suboptimal infant and young child feeding (IYCF) practices, and parasitic infections (5,6). However, anemia in malnourished children is multifactorial, also linked to diminished protein intake, impaired absorption, erythropoietin insufficiency, and reduced red blood cell synthesis due to altered muscle-to-fat ratios (7-9).

Protein-energy malnutrition (PEM), a spectrum of disorders arising from insufficient calorie and protein intake, further exacerbates the burden of SAM. The WHO reported in 2009 that approximately 20 million children under five worldwide were affected by SAM, which contributes to nearly half of all child deaths in underdeveloped nations annually (10,11). Achieving the fourth Millennium Development Goal (MDG) has been a persistent challenge due to the high prevalence of SAM, which continues to pose a significant barrier to child health and survival (12). The National Family Health Survey-4 (NFHS-4) revealed that in India, 7.5% of children under five suffer from SAM, making it a severe public health crisis with profound consequences (13). This condition accounts for approximately 24.6 million disability-adjusted life years (DALYs) and nearly 600,000 deaths annually, emphasizing its critical impact on child morbidity and mortality (14). Beyond its systemic effects, SAM profoundly influences hematological parameters, resulting in characteristic alterations in red and white blood cells. Anemia, manifesting as macrocytic, microcytic hypochromic, or normochromic patterns, is a hallmark of PEM, reflecting the intricate interplay between malnutrition and impaired hematopoiesis (15,16). White blood cell abnormalities further illustrate the immunological consequences of SAM, often presenting as thymic atrophy and increased susceptibility to infections (18).

Given the profound implications of severe acute malnutrition on hematological parameters, there is a critical need to explore and quantify these alterations in affected children. Understanding the specific hematological changes associated with SAM can aid in early diagnosis, targeted intervention, and improved management strategies. This study aims to evaluate the hematological profiles of children with severe acute malnutrition, shedding light on the extent of hematopoietic dysfunction and its potential clinical repercussions. By systematically analyzing these parameters, the research seeks to contribute to the growing body of evidence guiding nutrition-related health policies and interventions.

METHODS

This case-control study was conducted over a period of nine months, from January 2023 to September 2023, in the pediatric department's nutrition ward at PEDS Shaikh Zayed Hospital, Larkana, and a private medical center. The study aimed to evaluate the hematological parameters of children with severe acute malnutrition (SAM) in comparison to healthy controls. Ethical approval was obtained from the institutional review board, and written informed consent was secured from the guardians of all participants before enrollment. The study adhered to the ethical principles outlined in the Declaration of Helsinki for research involving human subjects.

Children under the age of five who were diagnosed with malnutrition and met the World Health Organization (WHO) criteria for SAM, defined as a weight-for-height z-score (WHZ) less than -3, or moderate acute malnutrition (MAM), with WHZ between -3 and -2, were included. Additionally, children with a weight-for-age z-score (WAZ) or height-for-age z-score (HAZ) below -2 standard deviations were considered eligible if they were not already receiving treatment at the nutritional ward of CMC Larkana Hospital. Exclusion criteria included children with chronic conditions such as liver disorders, cancer, or renal failure, as well as those who had taken vitamin or iron supplements in the preceding four weeks or had received a blood transfusion in the past three months. Children with known HIV or tuberculosis infections were also excluded to eliminate confounding factors associated with these diseases. A total of 216 children between the ages of 7 months and 5 years were recruited for the study, with 108 children diagnosed with SAM according to WHO criteria comprising the case group, while an equal number of age-matched, healthy children with normal nutritional status and no known hematological or infectious conditions were selected as controls. Standardized definitions were employed for hematological parameters,

with anemia defined as hemoglobin (Hb) levels below 11 g/dL at an altitude of 100 feet above sea level. Thrombocytopenia was diagnosed when platelet counts were below 150,000/mm³, while thrombocytosis was defined as a platelet count exceeding 450,000/mm³. Leukopenia was categorized by a white blood cell (WBC) count below 4,000/mm³, whereas leukocytosis was characterized by a WBC count above 12,000/mm³.

Comprehensive data collection included anthropometric measurements, physical examinations, and a detailed medical history for each participant. A structured, pre-tested questionnaire was administered through face-to-face interviews with parents or guardians to obtain sociodemographic data, dietary intake patterns, breastfeeding status, complementary feeding practices, and parental characteristics. Trained clinical nurses from the nutritional rehabilitation center were responsible for data collection to ensure standardization and accuracy. Hematological analysis was conducted using venous blood samples collected under aseptic conditions. A total of 3 mL of venous blood was drawn from the superficial veins of the antecubital fossa using a sterile venipuncture technique. To prevent clotting, the blood sample was immediately transferred into an EDTA anticoagulated test tube and mixed thoroughly. Complete blood count (CBC) analysis was performed using the automated hematology analyzer Sysmex XN-350 to ensure precision in hematological parameter assessment.

RESULTS

The demographic characteristics of the study participants revealed that among the total sample, 56 (51.8%) were male and 52 (48.1%) were female. Age distribution showed that the majority, 68 (49.2%), were within the age bracket of 7–24 months, followed by 17 (15.7%) aged 25–36 months, 9 (8.3%) within 37–48 months, and 14 (12.9%) aged 49–60 months. Regarding place of residence, 67 (62%) resided in rural areas, while 41 (38%) were from urban settings. Socioeconomic classification indicated that 74 (68.5%) children belonged to low-income families, 26 (24%) to middle-class families, and 8 (7.5%) to upper-class families. Parental education status demonstrated that 69 (63.8%) participants had parents with no formal education, 24 (23%) had parents who completed primary school, 8 (7.4%) had parents who completed secondary education, and only 7 (6.4%) had parents with a graduate-level education. Feeding practices among the participants' mothers showed that 39% exclusively breastfed their children, 9% relied solely on top feeding, while 52% practiced a combination of breastfeeding and top feeding. The assessment of anemia types revealed that normochromic normocytic anemia was the most prevalent, affecting 42 (38.88%) participants, followed by normocytic normochromic presentations in 36 (33.33%). Iron deficiency anemia was identified in 28 (25.9%) cases, while megaloblastic anemia was the least common, observed in only 2 (1.85%) participants. Comparative analysis of hematological parameters between the case and control groups demonstrated statistically significant differences across all measured indices ($p<0.05$). The mean hemoglobin level was notably lower in the case group (8.13±1.09 g/dL) compared to the control group (9.80±1.21 g/dL) ($p=0.0005$). Hematocrit (HCT) levels were also significantly lower in the case group (25.07±3.34%) than in the control group (30.88±4.02%) ($p=0.0005$). The red blood cell (RBC) count was lower in the malnourished children (3.99±0.71 $\times10^6$/L) compared to controls (4.10±0.51 $\times10^6$/L) ($p=0.0003$). Mean corpuscular volume (MCV) was significantly reduced in the case group (60.45±4.93 fL) versus controls (70.35±6.14 fL) ($p=0.0002$). Similarly, mean corpuscular hemoglobin (MCH) was lower in the case group (18.75±2.15 pg) than in controls (24.39±2.99 pg) ($p=0.0005$), while mean corpuscular hemoglobin concentration (MCHC) was also reduced in cases (30.11±1.74 g/dL) compared to controls (32.94±1.72 g/dL) ($p=0.0003$). White blood cell (WBC) counts were lower in malnourished children (6.99±1.64 $\times10^3$/µL) compared to healthy children (8.24±4.28 $\times10^3$/µL) ($p=0.005$). Platelet counts were also significantly lower in the case group (262.36±85.22 $\times10^3$/µL) compared to controls (302.11±128.15 $\times10^3$/µL) ($p=0.002$). The observed reductions in red blood cell indices, hemoglobin levels, and hematocrit indicate the profound hematological impact of severe acute malnutrition, supporting the association between malnutrition and anemia. The lower WBC and platelet counts in malnourished children suggest a possible immune compromise and altered hematopoiesis. The study findings highlight the critical need for targeted nutritional interventions and early hematological assessments in children with severe acute malnutrition to mitigate the risk of hematological abnormalities and associated complications.

Table 1: Socio-Demographic Characteristics of Patients

Variable	Category	Frequency	Percentages
Gender	Male	56	51.8
	Female	52	48.1
Age	<1	68	49.2
(Years)	1-2	17	15.7
	3-4	9	8.3
	5	14	12.9
Residency	Rural	67	62
	Urban	41	38
Family Socioeconomic	Poor Class Family	74	68.5
	Middle Class	26	24
	Upper-Class	8	7.5
Parental Education	Uneducated	69	63.8
	Primary School	24	23
	Secondary School	8	7.4
	Bachlor	7	6.4

The supplied information in table 1.0 show the several demographic characteristics as classified, including the following. Of the participants, 56 (51.8%) are male and 52 (48.1%) are female. In the age classification, 68 participants (49.2%) are within the age bracket of 7-24 months, 17 (15.7%) are within 25-36, 9 (8.3) fall within the 37-48 and 14 (12.9) are aged between 49 to 60 months. Concerning place of residence; 67 respondents which is 62% reside in the rural areas while 41 respondents that is 38% live in urban cities. In relation to their family's income 74 respondents 68.5 comes from low class income, 26 respondents 24% from the middle class and 8 respondents 7.5^ from the upper class. Finally, the level of parental education was as follows; the parents of 69 participants 63.8% of the participants have no education, 24 23% completed primary education, 8 (7.4%) completed secondary education, and 7 (6.4%) completed graduate level.

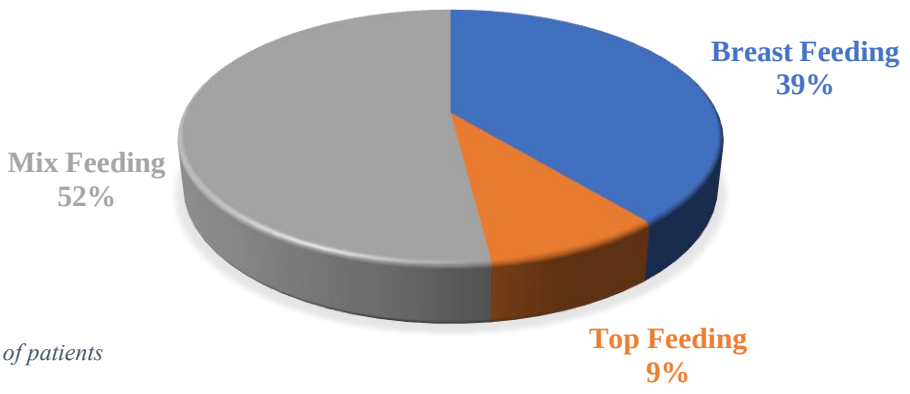

Figure 1 Feeding status of patients

Table 2 Shows the Complete Blood Count with type of Anemia

Type of Anemia	Frequency	Percentages
Normochromic Normocytic	36	33.33
Normochromic Normocytic Anemia	42	38.88
Iron Deficiency Anemia	28	25.9
Megaloblastic anemia	2	1.85

According to the distribution of anemia types in table 2.0, Normochromic Normocytic Anemia is the most prevalent kind, making up 38.88% of cases. Normochromic Normocytic anemia comes in second with 33.33%. With 25.9% of instances, iron deficiency anemia is the third most prevalent kind, but megaloblastic anemia is the least common, occurring in only 1.85% of people.

Table 3: Hematological Parameter in SAM and Control group

CBC parameter	**Case** **N= 108** **Mean ± SD**	**Control** **N= 108** **Mean ± SD**	**p-Value** **<0.05**
Hemoglobin (gm/dl)	8.13±1.09	9.80±1.21	0.0005
HCT (%)	25.07±3.34	30.88±4.02	0.0005
RBCs (10 10^6/L)	3.99±0.71	4.10± 0.51	0.0003
MCV (Fl)	60.45±4.93	70.35±6.14	0.0002
MCH (PG)	18.75±2.15	24.39±2.99	0.0005
MCHC (g/dl)	30.11±1.746	32.94±1.72	0.0003
WBC (10^3/UL)	6.99±1.64	8.24±4.28	0.005
Platelets (10^6/L)	262.36±85.22	302.11±128.15	0.002

The data presented in Table 3 compare Two groups, each with a sample size of 108. Several CBC (Complete Blood Count) parameters were compared between the two sets. For all the parameters, there were considerable differences between the two groups as p values

were less than 0.05. For instance, the mean hemoglobin values for the case group, 8.13±1.09, were lower than for the control group, 9.80±1.21. The P value here was 0.0005. Hematocrit (HCT) was lower in the case compared to the control group, the p-value is 0.0005. In the case group, RBC count stood at 3.99±0.71 while in the control group, it was 4.10±0.51 giving a p value of 0.0003. Mean corpuscular volume was significantly lesser in the case group (60.45±4.93) as compared to the control group (70.35,6.14) at a p-value of 0.0002. In comparison with the control group (24.39±2.99), MCH in the case group (18.75±2.15) was also reduced, where the p-value was 0.0005. In the case group, the mean corpuscular hemoglobin concentration (MCHC) was considerable lower mean value of 30.11±1.74 when compared with the control group 32.94±1.72. lastly, in the case group white blood cell count (6.99±1.64) was less as compared to the control group (8.24±4.28) with a p-value of 0.005. Platelet count was also lower in the case group (262.36±85.22) compared to the control.

DISCUSSION

The findings of this study reaffirm the well-established relationship between severe acute malnutrition (SAM) and hematological abnormalities, particularly anemia. A significant proportion of children with SAM were anemic, with iron deficiency anemia comprising a notable percentage. These findings align with previous studies that have demonstrated a high prevalence of anemia among severely malnourished children, reinforcing the role of nutritional deficiencies in hematological impairment (18). The results were consistent with those reported by Thakur et al., who observed that the majority of severely malnourished children presented with anemia, with a considerable percentage experiencing severe forms of the condition (19). Similarly, Kumar et al. reported an even higher prevalence of anemia, with more than half of the children displaying moderate anemia and nearly a quarter suffering from severe anemia (20). The multifactorial etiology of anemia in malnourished children extends beyond iron deficiency, as micronutrient inadequacies, inflammation, and physiological adaptations to chronic undernutrition play contributory roles. The observed reductions in hemoglobin and hematocrit levels in the SAM group compared to controls were consistent with prior research, reinforcing the notion that SAM significantly impairs erythropoiesis (18,22,23). The decline in red blood cell indices, including mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC), further supports the hypothesis that chronic malnutrition alters erythrocyte morphology and function, potentially due to diminished lean body mass, reduced oxygen-carrying capacity, and shifts in plasma volume regulation (24,25). Additionally, micronutrient deficiencies such as copper, zinc, and iron are recognized contributors to these hematological alterations, further exacerbating the burden of anemia in this vulnerable population (26).

Leukocyte abnormalities were also evident in children with SAM, with significant variations observed in white blood cell (WBC) counts. Malnourished children frequently experience immune dysregulation, predisposing them to infections, which can manifest as leukocytosis. However, conflicting reports exist in the literature, with some studies documenting leukopenia and neutropenia as predominant findings in malnourished children, suggesting a spectrum of immune responses based on the severity and duration of malnutrition (27,28). The findings of this study indicated a higher mean WBC count in SAM cases compared to controls, which is suggestive of an inflammatory or infectious process, aligning with previous observations in similar clinical contexts. The hematopoietic system is highly sensitive to nutritional status, and the variations in WBC count among malnourished children highlight the complex interplay between immune suppression and compensatory mechanisms in response to recurrent infections. These findings emphasize the necessity for early detection and management of immune dysfunction in severely malnourished children to mitigate infection-related morbidity and mortality.

Platelet abnormalities were also observed, with mean platelet counts lower in malnourished children than in controls. Bone marrow suppression in malnourished children has been postulated as a contributing factor to thrombocytopenia, with megakaryocyte function being indirectly affected by diminished hematopoietic activity (25). Saka et al. and Uner et al. reported similar findings, highlighting the compromised platelet production in children with SAM (18,25). However, contrasting evidence exists, with studies such as that by Abdur Rehman et al. indicating higher platelet counts in SAM children, possibly due to variations in study populations, underlying inflammatory states, or differences in nutritional deficiencies among participants (29). These discrepancies underscore the need for further research to delineate the precise mechanisms by which malnutrition affects thrombopoiesis and platelet turnover. The strengths of this study lie in its rigorous methodology, well-defined inclusion criteria, and comprehensive hematological analysis, which collectively enhance the reliability of the findings. The case-control design provided a robust comparative framework, allowing for a clear distinction between malnourished and healthy children. Additionally, the use of an automated hematology analyzer ensured precision in measuring blood parameters, reducing the likelihood of observer bias. However, certain limitations must be acknowledged. The study did not assess micronutrient levels such as serum iron, folate, and vitamin B12, which could have provided further insights

into the etiology of hematological abnormalities in malnourished children. Moreover, inflammatory markers such as C-reactive protein (CRP) were not included, which could have helped differentiate anemia of chronic disease from pure iron deficiency anemia. The cross-sectional nature of the study also limits the ability to establish causality between malnutrition and hematological alterations, warranting longitudinal studies to explore these associations more comprehensively.

This study reinforces the significant impact of severe acute malnutrition on hematological parameters, particularly anemia, leukocyte alterations, and platelet abnormalities. The findings highlight the necessity for routine hematological assessments in malnourished children to facilitate early diagnosis and targeted interventions. Given the observed hematological impairments, integrated nutritional and medical management strategies should be prioritized to mitigate the long-term consequences of malnutrition on hematopoiesis and immune function. Future research should focus on elucidating the role of specific micronutrients in hematological abnormalities and exploring the potential benefits of targeted nutritional supplementation in reversing these deficits.

CONCLUSION

This study highlights the profound hematological alterations observed in children with severe acute malnutrition, emphasizing the significant impact of malnutrition on blood cell production and overall hematopoietic function. The findings reinforce the high prevalence of anemia in malnourished children, along with notable reductions in platelet, white blood cell, and red blood cell counts. The frequent occurrence of pancytopenia and bicytopenia further underscores the severity of hematological compromise in this population. These results emphasize the critical need for early identification, targeted nutritional interventions, and comprehensive medical management to mitigate the adverse effects of malnutrition on hematopoiesis and overall child health. Addressing these hematological impairments through timely intervention could significantly improve clinical outcomes and reduce long-term complications associated with malnutrition.

AUTHOR CONTRIBUTIONS

Author	**Contribution**
Ishfaque Ahmed Mugheri*	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Delijan Mugheri	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Mehar Ali Kazi	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Nazia Faraz Shaikh	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Faheem	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Kashaf Faisal	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published

REFERENCES

1. Ahmed T, Hossain M, Mahfuz M, Choudhury N, Ahmed S. Severe acute malnutrition in Asia. Food Nutr Bull. 2014;35(2_suppl1):S14-26. doi:10.1177/15648265140352S103.

2. Ali Z, Saaka M, Adams AG, Kamwininaang SK, Abizari AR. The effect of maternal and child factors on stunting, wasting and underweight among preschool children in Northern Ghana. BMC Nutr. 2017;3:31. doi:10.1186/s40795-017-0154-2.

3. Development Initiatives. 2018 Global Nutrition Report: Shining a light to spur action on nutrition. Bristol, UK: Development Initiatives; 2018. Available from: https://globalnutritionreport.org/reports/global-nutrition-report-2018/. Accessed on: June 15, 2019.

4. Müller O, Krawinkel M. Malnutrition and health in developing countries. CMAJ. 2005;173(3):279-286. doi:10.1503/cmaj.050342.

5. Black RE, Victora CG, Walker SP, Bhutta ZA, Christian P, de Onis M, et al. Maternal and child undernutrition and overweight in low-income and middle-income countries. Lancet. 2013;382(9890):427-451. doi:10.1016/S0140-6736(13)60937-X.

6. Imdad A, Yakoob MY, Bhutta ZA. Impact of maternal education about complementary feeding and provision of complementary foods on child growth in developing countries. BMC Public Health. 2011;11(Suppl 3):S25. doi:10.1186/1471-2458-11-S3-S25.

7. Debnath DJ, Parulekar CV. Profile of under–five malnourished children admitted in a tertiary care teaching hospital in Pune, India. Int J Prev Med. 2014;5(7):882–886.

8. Devi RU, Krishnamurthy S, Bhat BV, Sahai A. Epidemiological and clinical profile of hospitalized children with moderate and severe acute malnutrition in South India. Indian J Pediatr. 2015;82(6):504-510. doi:10.1007/s12098-014-1641-5.

9. Yaikhomba T, Poswal L, Goyal S. Assessment of iron, folate and vitamin B12 status in severe acute malnutrition. Indian J Pediatr. 2015;82(6):511-514. doi:10.1007/s12098-014-1642-4.

10. World Health Organization, United Nations Children's Fund. WHO child growth standards and the identification of severe acute malnutrition in infants and children. Geneva: WHO; 2009. Available from: https://www.who.int/publications/i/item/9789241598163.

11. World Health Organization, World Food Programme, United Nations System Standing Committee on Nutrition, United Nations Children's Fund. Community-based management of severe acute malnutrition. Geneva: WHO; 2007. Available from: https://www.who.int/publications/i/item/978-92-806-4148-6.

12. United Nations. The Millennium Development Goals Report 2015. New York: United Nations; 2015. Available from: https://www.un.org/en/development/desa/publications/mdg-report-2015.html.

13. International Institute for Population Sciences (IIPS) and ICF. National Family Health Survey (NFHS-4), 2015-16: India. Mumbai: IIPS; 2017. Available from: http://rchiips.org/nfhs/NFHS-4Reports/India.pdf.

14. Black RE, Allen LH, Bhutta ZA, Caulfield LE, de Onis M, Ezzati M, et al. Maternal and child undernutrition: global and regional exposures and health consequences. Lancet. 2008;371(9608):243-260. doi:10.1016/S0140-6736(07)61690-0.

15. Lukens JN. Iron metabolism and iron deficiency. In: Miller DR, Baehner RL, editors. Blood diseases of infancy and childhood. 7th ed. St. Louis: Mosby; 1995. p. 193-219.

16. Thakur N, Chandra J, Pemde H, Singh V. Anemia in severe acute malnutrition. Nutrition. 2014;30(4):440-2. doi:10.1016/j.nut.2013.10.017.

17. Kumar R, Singh J, Joshi K, Singh HP, Bijesh S. Comorbidities in hospitalized children with severe acute malnutrition. Indian Pediatr. 2014;51(2):125-7. doi:10.1007/s13312-014-0340-4.

18. Kraemer K, Zimmermann MB, editors. Nutritional anemia. Basel: Sight and Life Press; 2007.

19. Laditan AA, Tindimebwa G. The protein energy malnourished child in a Nigerian teaching hospital. J Trop Pediatr. 1983;29(2):61-4. doi:10.1093/tropej/29.2.61.

20. El-Nawawy S, Barakat T, Elwalily A, Deghady A, Hussein M. Evaluation of erythropoiesis in protein energy malnutrition. East Mediterr Health J. 2002;8(2-3):255-63.

21. Abidoye RO, Sikabofori. A study of prevalence of protein energy malnutrition among 0-5 years in rural Benue State, Nigeria. Nutr Health. 2000;13(4):235-47. doi:10.1177/026010600001300404.

22. Uner A, Caliskan U, Oner AF, Koc H, Kasap AF. Platelet functions in patients with protein-energy malnutrition. Clin Appl Thromb Hemost. 2001;7(4):286-8. doi:10.1177/107602960100700406.

23. Meffat ME, Longstaffe S, Besant J, Dureski C. Prevention of iron deficiency and psychomotor decline in high-risk infants through use of iron-fortified infant formula: a randomized clinical trial. J Pediatr. 1994;125(4):527-34. doi:10.1016/s0022-3476(05)83299-0.

24. Edozien JC, Khan MA, Waslien CL. Human protein deficiency: results of a Nigerian village study. J Nutr. 1976;106(3):312-28. doi:10.1093/jn/106.3.312.

25. Fondu P, Hariga-Muller C, Mozes N, Neve J, Van Steirteghem A, Mandelbaum IM. Protein-energy malnutrition and anemia in Kivu. Am J Clin Nutr. 1978;31(1):46-56. doi:10.1093/ajcn/31.1.46.

26. Uner A, Caliskan U, Oner AF, Koc H, Kasap AF. Platelet functions in patients with protein-energy malnutrition. Clin Appl Thromb Hemost. 2001;7(4):286-8. doi:10.1177/107602960100700406.

27. Mefatt ME, Longstaffe S, Besant J, Dureski C. Prevention of iron deficiency and psychomotor decline in high-risk infants through use of iron-fortified infant formula: a randomized clinical trial. J Pediatr. 1994;125(4):527-34. doi:10.1016/s0022-3476(05)83299-0.

28. Edozien JC, Khan MA, Waslien CL. Human protein deficiency: results of a Nigerian village study. J Nutr. 1976;106(3):312-28. doi:10.1093/jn/106.3.312.

29. Fondu P, Hariga-Muller C, Mozes N, Neve J, Van Steirteghem A, Mandelbaum IM. Protein-energy malnutrition and anemia in Kivu. Am J Clin Nutr. 1978;31(1):46-56. doi:10.1093/ajcn/31.1.46.

