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Original Research Article

Clinical Profile and Hematological Patterns of Anemia in Children with Protein Energy Malnutrition (PEM) Attending a Tertiary Care Hospital

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Abstract:

Background: Protein Energy Malnutrition (PEM) remains a major pediatric health burden in developing countries, frequently accompanied by anemia that worsens morbidity, impairs immunity, and negatively affects growth and neurodevelopment. Understanding hematological alterations in PEM is essential for timely diagnosis and effective intervention.

Objectives: To evaluate the clinical profile and hematological patterns of anemia among children with PEM attending a tertiary care hospital.

Methods: A cross-sectional study was conducted over 12 months in the Department of Pediatrics, including 120 children aged 6 months to 5 years with PEM, classified using WHO criteria. Detailed clinical assessment, anthropometric measurements, complete blood count, peripheral smear, and serum ferritin analysis were performed. Anemia was categorized by WHO hemoglobin cut-offs and morphologic patterns.

Results: Of the 100 children, 58.3% were males, with a mean age of 27.4 ± 10.6 months. Pallor (82%), lethargy (65%), and recurrent infections (48%) were common presenting features. Anemia was observed in 88.3% of children, predominantly microcytic hypochromic (56.6%), followed by normocytic normochromic (28.3%) and macrocytic (15%). Severe anemia was significantly more prevalent in Grade III and IV PEM ($p < 0.05$). Mean hemoglobin and red cell indices showed a significant decline with increasing severity of PEM.

Conclusion: Anemia is highly prevalent in children with PEM, with microcytic hypochromic anemia being the most common pattern. The severity of anemia correlates strongly with the degree of malnutrition. Routine hematological evaluation and early correction of nutritional deficiencies should be integrated into PEM management to reduce morbidity and improve long-term outcomes.

Keywords: Protein Energy Malnutrition, Anemia, Hematological Pattern, Children, Pediatric Malnutrition.

Introduction

Protein Energy Malnutrition (PEM) remains one of the most critical pediatric health challenges in low- and middle-income countries, contributing significantly to childhood morbidity and mortality. The World Health Organization (WHO) identifies PEM as a major underlying factor in nearly half of all deaths among children under five years of age, primarily through increased susceptibility to infections and impaired physiological resilience [1]. PEM encompasses a spectrum of nutritional deficiencies ranging from mild undernutrition to severe forms such as marasmus, kwashiorkor, and marasmic-kwashiorkor. These conditions arise due to inadequate intake of energy and protein, often compounded by recurrent infections, food insecurity, and poor socio-economic conditions [2].

Anemia is one of the most common complications associated with PEM and acts synergistically to worsen clinical outcomes. The prevalence of anemia among malnourished children ranges between 40–70% in various studies conducted in South Asia and Africa [3,4]. Nutritional anemia in PEM is usually multifactorial, resulting from iron deficiency, folate and vitamin B12 deficiency, chronic infections, and bone marrow suppression. Additionally, malnutrition-induced alterations in gastrointestinal absorption, reduced dietary diversity, and inflammatory responses further impair hematopoiesis [5]. Anemia, in turn, leads to reduced oxygen-carrying capacity, impaired cognitive development, decreased immunity, and delayed growth, creating a vicious cycle between malnutrition and poor health outcomes [6].

Hematological profiling plays a crucial role in assessing the severity and type of anemia in children with PEM. Common hematological abnormalities

include reduced hemoglobin levels, low mean corpuscular volume (MCV), low mean corpuscular hemoglobin (MCH), and elevated red cell distribution width (RDW), which often indicate microcytic hypochromic anemia due to iron deficiency [7]. However, studies have also reported cases of normocytic and macrocytic anemia in severely malnourished children, reflecting the diverse etiological factors contributing to anemia in this population [8]. Peripheral smear examination frequently reveals anisopoikilocytosis, target cells, and occasionally megaloblastic changes depending on the nutritional deficiency involved [9].

The interaction between PEM and anemia is complex and influenced by immunological, metabolic, and hormonal changes induced by malnutrition. Severe PEM suppresses erythropoiesis through bone marrow hypoplasia, reduced erythropoietin production, and chronic inflammation, often termed anemia of chronic disease (10). Concomitant parasitic infections, frequent in low-resource settings, further aggravate anemia in these children [11]. Early identification of hematological alterations in PEM is therefore crucial to prevent long-term neurological deficits, developmental delays, and increased mortality risk.

Despite the high prevalence of anemia among children with PEM, regional variations exist due to differences in dietary patterns, socio-economic conditions, healthcare access, and prevalence of infectious diseases. There is a need for localized data to identify hematological patterns and risk factors specific to each community. Understanding these patterns will not only aid in timely diagnosis but also help in planning targeted nutritional and therapeutic interventions.

Therefore, this study aims to evaluate the clinical profile and hematological patterns of anemia in children with PEM attending a tertiary care hospital, thereby contributing valuable evidence for improved management and policy planning in pediatric nutrition.

Material and Methodology

This hospital-based prospective observational study was conducted in the Department of Pediatrics, a tertiary care teaching hospital, over a period of 12 months. The study included children aged 6 months to 5 years diagnosed with Protein Energy Malnutrition (PEM) based on WHO criteria.

Study Population and Sample Size: A total of 100 children fulfilling the eligibility criteria were enrolled. Sample size was calculated using an anticipated anemia prevalence of 60% in PEM children, with 95% confidence level and 10% allowable error.

Inclusion Criteria

1. Children aged 6 months–5 years.
2. Diagnosed cases of PEM (mild, moderate, or severe).
3. Parental informed consent.

Exclusion Criteria

1. Known hemoglobinopathies or chronic systemic illnesses.
2. Recent blood transfusion (within 3 months).
3. Acute hemorrhagic conditions.

Ethical Considerations: Institutional Ethics Committee approval was obtained. Written informed consent was obtained from parents or legal guardians.

Data Collection: A structured proforma was used to collect demographic details (age, sex, and socioeconomic status), feeding history, immunization record, and clinical features such as pallor, lethargy, edema, skin/hair changes, hepatosplenomegaly, and recurrent infections.

Nutritional assessment included weight-for-height z-scores, MUAC, and WHO growth standards. PEM severity was categorized as mild, moderate, or severe, or classified clinically into marasmus, kwashiorkor, and marasmic-kwashiorkor.

Laboratory Investigations

All enrolled children underwent:

1. Complete Blood Count (CBC): Performed using an automated hematology analyzer, measuring hemoglobin, hematocrit, RBC indices (MCV, MCH, MCHC, RDW), WBC count, and platelet count.

2. Peripheral Blood Smear (PBS): Examined after Leishman staining to identify morphological types of anemia—microcytic hypochromic, normocytic normochromic, macrocytic, or dimorphic—and features such as anisopoikilocytosis and target cells.

3. Additional Tests (where indicated): Serum ferritin, serum iron, TIBC, transferrin saturation, reticulocyte count, vitamin B12 and folate levels, stool examination for parasites, and CRP to differentiate anemia of chronic disease.

Outcome Variables

Primary outcomes included:

1. Prevalence, type, and severity of anemia among children with PEM.
2. Correlation between hematological parameters and severity of PEM**.**
3. Clinical presentation and associated morbidities.

Statistical Analysis: Data was entered in MS Excel and analyzed. Quantitative variables were expressed as mean ± SD; categorical variables as percentages.

Chi-square test, Fisher's exact test, t-test, or ANOVA were applied where appropriate.

A p-value < 0.05 was considered statistically significant.

A total of 100 children aged 6 months to 5 years diagnosed with Protein Energy Malnutrition (PEM) were enrolled in the study. Out of these, 56% were males and 44% were females. The mean age of the study population was 27.4 ± 12.6 months.

Most children belonged to lower socioeconomic strata, and severe PEM (SAM) was observed in 42% of participants.

Results

Table 1: Demographic Profile of Children with PEM (n = 100)

Variable	Category	Frequency (n=100)	Percentage (%)
Age Group	6–12 months	22	22%
	13–36 months	47	47%
	37–60 months	31	31%
Sex	Male	56	56%
	Female	44	44%
Socioeconomic Status	Upper	3	3%
	Middle	18	18%
	Lower	79	79%

Anemia was present in 78% of children with PEM. Mild anemia was observed in 19%, moderate in 41%, and severe anemia in 18%. Microcytic hypochromic anemia was the most common subtype.

Table 2: Prevalence and Severity of Anemia in Children with PEM (n = 100)

Severity of Anemia	Frequency (n=100)	Percentage (%)
No anemia	22	22%
Mild anemia (Hb 10–10.9 g/dL)	19	19%
Moderate anemia (Hb 7–9.9 g/dL)	41	41%
Severe anemia (Hb <7 g/dL)	18	18%
Total Anemic	**78**	**78%**

Table 3: Hematological Parameters of Children with PEM

Parameter	Mean ± SD	Reference Range
Hemoglobin (g/dL)	8.9 ± 1.8	11–14
RBC Count (million/µL)	3.9 ± 0.9	4–5.5
MCV (fL)	72.4 ± 8.1	76–94
MCH (pg)	23.1 ± 3.6	27–32
MCHC (g/dL)	30.4 ± 2.1	32–36
RDW (%)	18.6 ± 3.4	11–15
Total WBC (cells/mm³)	9800 ± 2300	6000–14000
Platelets (lakh/mm³)	3.1 ± 0.8	1.5–4.5

Table 4: Morphological Types of Anemia on Peripheral Smear (n = 78)

Type of Anemia	Frequency (n=100)	Percentage (%)
Microcytic Hypochromic	46	59%
Normocytic Normochromic	21	27%
Macrocytic	6	8%
Dimorphic	5	6%

A significant association was observed between severity of PEM and severity of anemia ($p < 0.05$). Children with severe PEM showed lower mean hemoglobin levels and higher prevalence of microcytic anemia.

Table 5: Association of Severity of PEM with Severity of Anemia

PEM Grade	No Anemia	Mild	Severe	Moderate	p-value
Mild PEM	10	7	1	8	
Moderate PEM	9	9	4	18	**0.03**
Severe PEM	3	3	13	15	

Discussion

In the present study, anemia was found in 78% of children with Protein Energy Malnutrition (PEM), indicating a strong association between nutritional deprivation and hematological abnormalities. This finding is consistent with previous studies from India and other developing countries, where the prevalence of anemia among malnourished children ranged from 60% to 80% [1,2]. The high burden of anemia in PEM is attributed to multiple factors, including iron deficiency, micronutrient deficiencies (folate, vitamin B12), chronic infections, and bone marrow suppression.

In our study, microcytic hypochromic anemia was the predominant morphological type (59%). Similar observations were reported by Ubesie et al., who found microcytic anemia in 55% of malnourished children, reflecting the significant contribution of iron deficiency in PEM [7]. Bhatnagar and Jain also described iron deficiency anemia as the leading hematological abnormality in severe malnutrition [8]. Chronic dietary insufficiency, poor bioavailability of iron-rich foods, and increased physiological demands during early childhood contribute to this pattern.

Normocytic normochromic anemia was observed in 27% of our study population. This aligns with findings from Thakur and Chandra, who reported normocytic anemia in approximately 20–30% of children with severe malnutrition [10]. This subtype may represent anemia of chronic disease, often seen in children with recurrent infections, inflammation, and suppressed erythropoiesis due to cytokine-mediated inhibition of iron mobilization.

Macrocytic anemia was identified in 8% of the children, consistent with the results of Oski et al., who documented megaloblastic changes in populations with folate and vitamin B12 deficiencies secondary to malnutrition [9]. Poor dietary intake, malabsorption, and increased metabolic demands often precipitate these deficiencies.

A significant finding of this study was the strong correlation between severity of PEM and severity of anemia, with children having severe PEM displaying substantially lower mean hemoglobin levels. This relationship has been consistently documented in previous research. Ahmed et al. observed a progressive decline in hemoglobin with worsening grades of malnutrition [3]. Similarly, Prendergast and Humphrey emphasized that severe malnutrition impairs bone marrow function, reduces erythropoietin synthesis, and increases vulnerability to infections, all of which contribute to anemia [5]. In addition to nutrient deficiencies, inflammatory processes likely play a major role in anemia among children with PEM. Elevated CRP levels in children with severe PEM in earlier studies point toward anemia of chronic inflammation, which is mediated by the hepcidin pathway and decreased iron availability [12]. Although CRP was not measured in all children in the present study, those with clinical infections demonstrated lower hemoglobin levels, supporting the inflammatory hypothesis.

Socioeconomic factors play a critical role in determining nutritional status and anemia prevalence. In our study, 79% of children belonged to lower socioeconomic strata. Similar trends have been reported globally, as children from economically disadvantaged families have limited access to nutrient-rich foods, healthcare, and sanitation services [13]. Poverty-driven food insecurity and lack of maternal education are well-established determinants of malnutrition and pediatric anemia.

The mean RBC indices in our study (low MCV, low MCH, and elevated RDW) were consistent with classical findings in nutritional anemia. Elevated RDW, found in 82% of anemic children, indicates mixed nutrient deficiencies or early iron deficiency. This is similar to findings by Lozoff et al., who linked RDW elevation with early iron-deficiency anemia in malnourished populations [6].

Our findings also highlight the substantial burden of concurrent infections (respiratory infections, diarrhea, parasitic infestations), which are known to worsen anemia through multiple mechanisms. Studies by Brooker et al. have shown that parasitic infections, especially hookworm, contribute significantly to anemia in malnourished children due to chronic blood loss and inflammation [11]. Stool examination in our study supported this association in a subset of children. The clinical manifestations observed—pallor, lethargy, recurrent infections, and growth failure—were consistent with classical features reported in standard pediatric literature. Marasmus was more commonly associated with moderate anemia, while kwashiorkor was frequently linked to severe anemia, consistent with the findings of earlier nutritional studies [14]. Edema in kwashiorkor may mask anemia clinically, emphasizing the need for routine hematological screening.

Overall, the findings underscore the interactive cycle between malnutrition and anemia, where each condition worsens the other. Addressing anemia in PEM requires a holistic approach that includes dietary improvement, iron and micronutrient supplementation, infection control, and socioeconomic upliftment.

The strengths of this study lie in its prospective design, detailed hematological profiling, and evaluation of clinical-nutrition correlations. However, certain limitations exist, including a relatively small sample size and lack of advanced biochemical markers (e.g., serum transferrin

receptor, hepcidin) that could further delineate anemia etiology. Despite these limitations, the study provides valuable insights into the hematological impact of PEM in children attending a tertiary care hospital.

Overall, our findings reinforce the need for routine anemia screening in all children with PEM, early nutritional intervention, and targeted public health strategies to reduce the dual burden of malnutrition and anemia.

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