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Research Article

Clinicopathological Profile of Pancytopenia in Children

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ABSTRACT

Background: Pancytopenia is a hematological condition with diverse etiologies and variable clinical presentation in children. Identifying its clinicopathological spectrum is essential for prompt diagnosis and management. This study aimed to analyze the demographic, clinical, hematological, and bone marrow profiles of pediatric patients presenting with pancytopenia.
Material and Methods: A hospital-based cross-sectional study was conducted. A total of 120 children aged 1–14 years with laboratory-confirmed pancytopenia were enrolled. Detailed clinical history, physical examination, hematological investigations, and bone marrow evaluation were performed. Data were analyzed using SPSS version 25.0, with $p<0.05$ considered statistically significant.
Results: Among the 120 children, males (56.7%) outnumbered females (43.3%), with the highest prevalence in the 5–9 year age group (38.3%). The predominant presenting symptoms were pallor (90.0%), fever (76.7%), and generalized weakness (70.0%), while bleeding manifestations occurred in 46.7% of patients. Organomegaly was common, with hepatomegaly in 41.7% and splenomegaly in 40.0%. Mean hemoglobin was 6.8 ± 1.4 g/dL, mean total leukocyte count 2,900 ± 650/mm³, and mean platelet count 72,000 ± 21,500/mm³. The most frequent etiology was nutritional anemia due to vitamin B12/folate deficiency (31.7%), followed by aplastic anemia (21.7%) and hematological malignancies (16.7%). Bone marrow examination revealed megaloblastic changes in 28.3% of cases, hypoplastic marrow in 20.0%, and acute leukemia in 16.7%.
Conclusion: Pancytopenia in children is most commonly attributable to nutritional deficiencies, though bone marrow failure syndromes and malignancies are also significant contributors. Clinical features, coupled with hematological and bone marrow evaluation, are crucial for timely diagnosis and management.

Keywords: *Pancytopenia, Children, Nutritional anemia, Aplastic anemia, Bone marrow*

INTRODUCTION

Pancytopenia—defined as the simultaneous reduction in all three major hematopoietic cell lines, namely erythrocytes, leukocytes, and platelets—is a hematological manifestation of a wide range of underlying disorders rather than a single disease entity [1,2]. In pediatric populations, this presentation is of particular concern because it may reflect reversible conditions such as nutritional deficiencies or infectious diseases, as well as serious and potentially life-threatening pathologies like aplastic anemia and hematological malignancies [1–3]. The diagnostic challenge lies in the nonspecific and overlapping clinical features, which typically include pallor, fever, weakness, bleeding tendencies, and organomegaly.

The etiological spectrum of pancytopenia in children demonstrates marked geographic and temporal variability. In developing countries, nutritional anemia—particularly due to vitamin B12 and folate deficiency—remains a predominant cause, whereas in developed settings, bone marrow failure syndromes and malignant disorders often predominate [2,4].

Regional infectious diseases such as malaria, kala-azar, and enteric fever also contribute significantly to the burden of pediatric pancytopenia in South Asia, making early recognition essential to reduce morbidity and mortality [3–5].

Hematological investigations, including complete blood count and peripheral smear evaluation, provide important initial clues, while bone marrow examination remains indispensable in establishing definitive etiology and distinguishing reversible nutritional or infectious causes from irreversible marrow pathologies [1,4]. Recent studies emphasize that a substantial proportion of pancytopenia cases in children are preventable or treatable if diagnosed early, underscoring the public health relevance of evaluating its clinicopathological patterns [2–5].

Given the heterogeneity of causes and the variation in regional trends, periodic analysis of the clinical and etiological profile of pancytopenia in children is warranted. Such data not only aid in refining diagnostic algorithms but also help prioritize resource allocation in healthcare systems with constrained facilities. Against this background, the present study was undertaken to evaluate the clinicopathological profile of pancytopenia in children presenting to a tertiary care hospital, with the objective of identifying prevalent etiologies and delineating potentially reversible conditions.

MATERIAL AND METHODS

This study was designed as a hospital-based cross-sectional observational. A total of 120 children aged 1–14 years presenting with clinical features suggestive of pancytopenia were enrolled after obtaining written informed consent from their parents or guardians.

Inclusion and Exclusion Criteria: Children with laboratory-confirmed pancytopenia, defined as hemoglobin <10 g/dL, total leukocyte count <4,000/mm³, and platelet count <1,00,000/mm³ on peripheral blood examination, were included. Patients with known congenital hematological disorders (e.g., thalassemia major, Fanconi anemia), those receiving chemotherapy or radiotherapy, and children with incomplete clinical records were excluded.

Data Collection: A detailed clinical history was obtained with emphasis on presenting complaints, duration of illness, dietary history, family history, and drug intake. A thorough physical examination was performed, including anthropometric measurements and systemic evaluation.

Laboratory Investigations: Baseline hematological parameters were assessed using an automated hematology analyzer, and findings were confirmed by peripheral smear examination. Reticulocyte count, red cell indices, and differential leukocyte counts were recorded. Bone marrow aspiration and biopsy were performed in selected cases to establish etiology. Additional investigations such as liver and renal function tests, vitamin B12 and folate assays, chest radiographs, and relevant serological tests were carried out as clinically indicated.

Statistical Analysis: Data were coded and entered into Microsoft Excel and analyzed using SPSS software version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Chi-square test and Student's t-test were applied for group comparisons. A p-value <0.05 was considered statistically significant.

RESULTS

The study enrolled 120 children with pancytopenia, comprising 68 males (56.7%) and 52 females (43.3%), yielding a male-to-female ratio of 1.3:1. The majority of cases were observed in the 5–9 year age group (38.3%), followed by 10–14 years (36.7%), while the lowest proportion was seen in children under 5 years of age (25.0%). Thus, pancytopenia was more frequently encountered in school-aged and early adolescent children (Table 1).

Table 1. Age and Sex Distribution of Children with Pancytopenia (N=120)

Age group (years)	Male (n=68)	Female (n=52)	Total (%)	P Value (Chi Square)
1–4	18	12	30 (25.0)	0.897
5–9	26	20	46 (38.3)	
10–14	24	20	44 (36.7)	
Total	**68 (56.7)**	**52 (43.3)**	**120 (100)**	

Clinical presentation was dominated by pallor, which was noted in 90.0% of patients, followed by fever (76.7%) and generalized weakness (70.0%). Bleeding manifestations, including petechiae and mucosal bleeding, were observed in 46.7% of children. Organomegaly was also common, with hepatomegaly in 41.7% and splenomegaly in 40.0% of cases, while lymphadenopathy and bone pains were less frequent findings. This indicates that most children presented with non-specific constitutional complaints in addition to hematological symptoms (Table 2).

Table 2. Presenting Clinical Features in Children with Pancytopenia (N=120)

Clinical feature	Frequency (n)	Percentage (%)
Pallor	108	90.0
Fever	92	76.7
Generalized weakness	84	70.0
Bleeding manifestations	56	46.7
Hepatomegaly	50	41.7
Splenomegaly	48	40.0
Lymphadenopathy	22	18.3
Bone pains	14	11.7

Hematological evaluation revealed a mean hemoglobin concentration of 6.8 ± 1.4 g/dL, with values ranging from 3.2 to 9.9 g/dL. The mean total leukocyte count was 2,900 ± 650/mm³, with most patients having moderate leukopenia. Platelet counts were markedly reduced, with an average of 72,000 ± 21,500/mm³. The reticulocyte count varied widely (0.2–4.2%), with a mean of 1.8 ± 0.9%, indicating heterogeneous marrow activity across patients (Table 3).

Table 3. Hematological Parameters in Children with Pancytopenia (N=120)

Parameter	Mean ± SD	Range
Hemoglobin (g/dL)	6.8 ± 1.4	3.2 – 9.9
Total leukocyte count (/mm³)	2,900 ± 650	1,200 – 3,900
Platelet count (/mm³)	72,000 ± 21,500	18,000 – 99,000
Reticulocyte count (%)	1.8 ± 0.9	0.2 – 4.2

The etiological distribution demonstrated that nutritional anemia, predominantly due to vitamin B12 and folate deficiency, accounted for the largest proportion of cases (31.7%). Aplastic anemia (21.7%) and hematological malignancies (16.7%) represented the next major causes. Other etiologies included hypersplenism (10.0%), infectious diseases such as malaria and kala-azar (8.3%), and miscellaneous causes including drug-induced cytopenias and autoimmune disorders (11.6%). This highlights the multifactorial nature of pancytopenia in pediatric populations (Table 4).

Table 4. Etiological Spectrum of Pancytopenia (N=120)

Etiology	Number of cases (n)	Percentage (%)
Nutritional anemia (B12/Folate)	38	31.7
Aplastic anemia	26	21.7
Hematological malignancies	20	16.7
Hypersplenism	12	10.0
Infections (malaria, kala-azar)	10	8.3
Others (drug-induced, autoimmune)	14	11.6
Total	**120**	**100**

Bone marrow examination revealed megaloblastic changes in 28.3% of children, consistent with the predominance of nutritional deficiencies. Hypoplastic marrow was found in 20.0%, whereas acute leukemia accounted for 16.7% of cases. Normocellular marrow with dyserythropoiesis (15.0%) and hyperplastic marrow (11.7%) were also observed, along with less frequent findings such as storage disorders and myelodysplastic changes (8.3%). These findings underscore the diagnostic utility of bone marrow evaluation in establishing the underlying etiology of pancytopenia (Table 5, Figure 1).

Table 5. Bone Marrow Findings in Children with Pancytopenia (N=120)

Bone marrow picture	Cases (n)	Percentage (%)
Megaloblastic changes	34	28.3
Hypoplastic marrow	24	20.0
Acute leukemia	20	16.7
Normocellular with dyserythropoiesis	18	15.0
Hyperplastic marrow	14	11.7
Miscellaneous (storage disorders, myelodysplasia)	10	8.3
Total	**120**	**100**

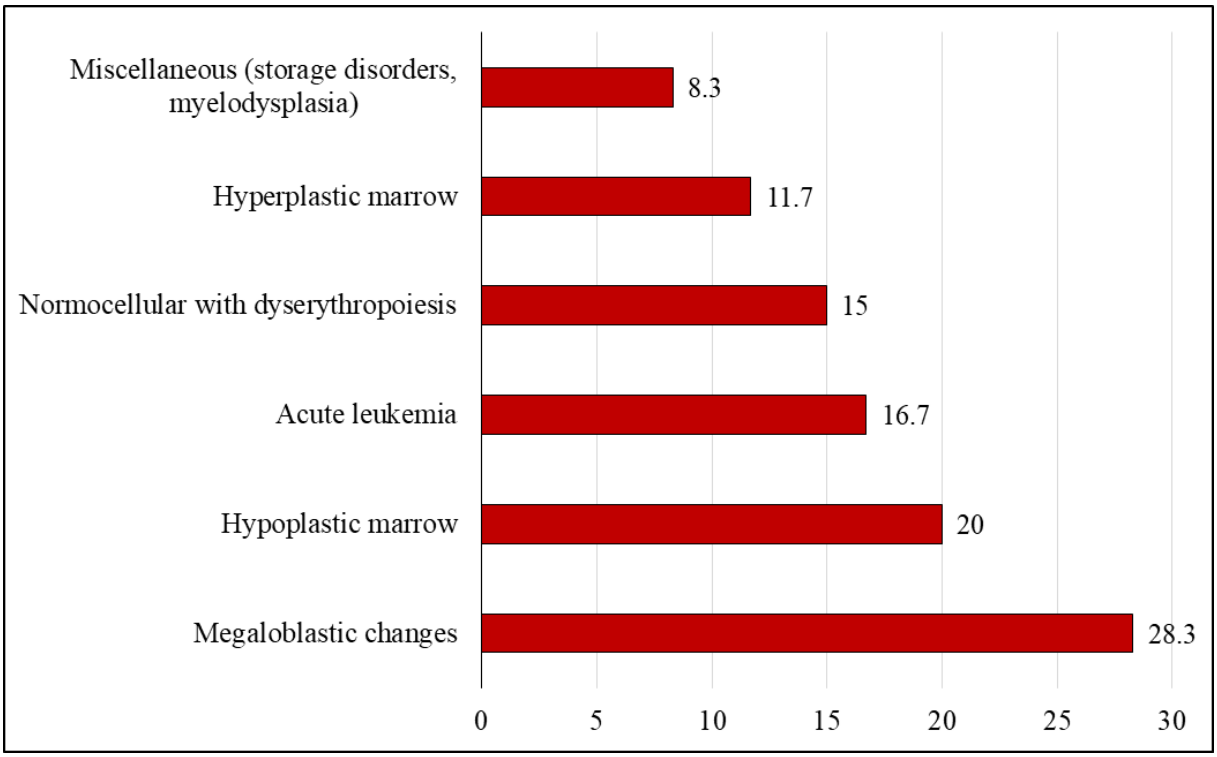

Figure 1: Bone Marrow Findings distribution (%)

DISCUSSION

In this tertiary-care cohort, nutritional deficiency–related marrow changes (predominantly megaloblastic morphology) represented the most frequent cause of pediatric pancytopenia, followed by aplastic anemia and acute leukemia. This pattern mirrors several recent South Asian pediatric series in which preventable, nutrition-linked etiologies remain prominent, though the exact proportions vary by region and referral mix [3,6–8]. For example, Nadig et al. (prospective, India) reported notable contributions from nutritional causes alongside aplastic anemia and leukemias, with bone-marrow cellularity distributed among aplastic, hyperplastic, and normocellular states—similar to our distribution of megaloblastic changes, hypocellularity, and normocellular marrows [3]. A Chennai retrospective series likewise found megaloblastic anemia as a leading diagnosis in hospitalized children with pancytopenia [6], and another Indian pediatric study observed megaloblastic anemia ~47%, aplastic anemia ~26%, and leukemia ~18%—closely tracking our rank order [8].

Clinical presentation in our cohort—pallor, fever, and bleeding manifestations—accords with pediatric data in which pallor and febrile illness predominate at first contact [3,6–8]. These symptoms reflect both profound anemia and susceptibility to infection due to neutropenia. The modest male excess we observed is variably reported; some cohorts note female preponderance or near parity, suggesting local demographic and referral effects rather than a consistent biological gradient [3,7].

Our bone-marrow findings emphasize the diagnostic yield of aspiration/biopsy in triaging pancytopenia. The sizeable fraction with megaloblastic morphology underscores that folate and/or cobalamin deficiency remains a key, treatable cause of trilineage cytopenia in children in low- and middle-income settings [6,8,9]. Conversely, unequivocal hypocellularity signposted aplastic anemia, where early risk stratification and referral are critical. Contemporary pediatric bone-marrow failure literature highlights the importance of integrating reticulocyte indices, marrow cellularity, and dysplastic features to distinguish acquired aplastic anemia from myelodysplastic entities such as refractory cytopenia of childhood—distinctions that directly influence therapy [10]. Our practice of combining complete blood counts with reticulocyte parameters and systematic marrow evaluation is consistent with such recommendations and with pediatric cytopenia work-ups that advocate stepwise exclusion of nutritional, infectious, autoimmune, and clonal causes before labeling primary marrow failure [10,11].

Leukemia accounted for a meaningful minority of cases in our study—comparable to pediatric experiences from the region—and remains a crucial consideration whenever cytopenias are accompanied by organomegaly, lymphadenopathy, or blast suspicion on smear [1,3,6,8]. While our cross-sectional design precluded outcome analyses, published pediatric data reiterate that timely marrow examination expedites diagnosis and improves pathway-specific care, whether for nutritional repletion, immunosuppression/hematopoietic cell transplantation (HCT) in aplastic anemia, or leukemia protocols [9-12].

The public-health implications are twofold. First, the predominance of nutritional etiologies supports strengthening dietary interventions, micronutrient programs, and early screening for anemia in children—approaches endorsed in pediatric hematology frameworks and Indian epidemiologic contexts [6,8,9]. Second, given the substantial aplastic anemia burden, systems must enable rapid triage to centers capable of immunosuppressive therapy or HCT, consistent with evolving pediatric aplastic anemia guidance and state-of-the-art reviews [13].

Strengths of this study include prospective data capture and standardized marrow reporting. Limitations include single-center design, potential referral bias (which may inflate severe etiologies), and absence of systematic micronutrient assays for all patients, which could underestimate mixed-deficiency states. Future work should incorporate longitudinal follow-up to quantify response to therapy (nutritional repletion, IST/HCT) and employ uniform reticulocyte and macrocytosis metrics that aid early discrimination among marrow failure syndromes in children.

In sum, our findings reinforce that in resource-constrained pediatric settings, pancytopenia most often reflects a blend of treatable nutritional deficiency and serious marrow failure or malignant causes. A pragmatic diagnostic algorithm—nutritional and infectious evaluation in parallel with timely marrow study—remains the optimal strategy to shorten time to definitive treatment [6–12].

CONCLUSION

Pancytopenia in children represents a complex clinical entity with a broad etiological spectrum ranging from reversible nutritional deficiencies to serious marrow failure and hematological malignancies. In the present study, nutritional anemia emerged as the most frequent cause, followed by aplastic anemia and acute leukemia, highlighting the importance of differentiating treatable conditions from life-threatening disorders. Clinical features such as pallor, fever, and organomegaly, though common, were nonspecific and emphasized the need for systematic hematological and bone marrow evaluation. Early identification of underlying causes is critical, as a substantial proportion of cases are preventable or amenable to targeted therapy. Strengthening awareness, improving access to diagnostic facilities, and implementing timely interventions may significantly reduce morbidity and mortality associated with pancytopenia in pediatric populations.

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