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Prevalence and Clinical Profile of Pediatric Anemia in District Kupwara, Jammu and Kashmir, India

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Abstract

Background: Anemia is a widespread Pediatric health issue in India, especially in rural and high-burden regions like Jammu & Kashmir (J&K). This study aimed to determine the prevalence of Anemia in children from District Kupwara and analyse their clinical and haematological profiles.

Methods: A hospital-based cross-sectional study was conducted over one year (Jan 2024–Jan 2025) in the Department of Pathology (in collaboration with Pediatrics) at Government Medical College Handwara. A total of 20,000 children (0–18 years, 53% male, mean age 8.6±5.2 years) were evaluated. Complete blood counts, peripheral smear examinations, and relevant biochemical tests were performed. Anemia was defined and graded by World Health Organization criteria. Clinical features and etiological factors were recorded.

Results: The overall prevalence of anemia was 57%. The highest prevalence was in under-5 children (70%), and adolescent girls were the worst-affected adolescent subgroup (65% anemic). Most children presented with pallor, fatigue, poor feeding, irritability, or tachycardia. Mean hemoglobin was 9.2 g/dL. Severity-wise, 40% had mild anemia, 55% moderate, and 5% severe. Morphologically, 70% of cases were microcytic hypochromic (iron deficiency anemia predominant), 15% normocytic, 5% macrocytic, 5% dimorphic, and 5% hemolytic or other types. Notably, 22 children were diagnosed with thalassemia major/intermedia, 15 with hereditary spherocytosis or G6PD deficiency, and 8 with celiac disease-related anemia. Reactive thrombocytosis was observed in 25% of iron-deficiency cases (platelet count >500×10^9/L), which normalized after therapy. Treatment with iron supplements led to improvement in 88% of iron-deficiency anemia cases, with a mean hemoglobin rise of +2.8 g/dL after 3 months. Children with folate/B-12 deficiency showed dramatic hematologic response to vitamin therapy. There were no deaths attributable to anemia in the cohort, and all severely anemic children survived with appropriate treatment (iron supplementation and blood transfusions where needed).

Conclusions: Pediatric anemia is highly prevalent in Kupwara, J&K, particularly among young children and adolescent females. Nutritional iron deficiency is the leading cause, followed by much less frequent hemolytic and systemic causes. Early diagnosis and effective management of anemia yielded excellent outcomes in this study. However, the high prevalence underscores the need for strengthened public health measures – nutritional interventions, iron supplementation (e.g. Anemia Mukt Bharat program), deworming, and screening for hemoglobinopathies – to combat pediatric anemia in this region.

Keywords: Anemia, Microcytic Anemia, J&K, Iron Deficiency

Introduction

Anemia is a widespread public health problem affecting children globally. The World Health Organization estimates that approximately 40% of under-5 children in low- and middle-income countries are anemic[1]. Childhood anemia leads to significant morbidity – it impairs cognitive development, growth, and immune function, and increases the risk of infections[2]. The causes of pediatric anemia are multifactorial, including nutritional deficiencies (iron, folate, vitamin B_12), infections (malaria, helminthic infestations), hemoglobinopathies, and chronic diseases. Nutritional iron deficiency anemia (IDA) is the most common cause, especially in developing regions, often exacerbated by malnutrition and parasitic infections[3]

India bears a particularly high anemia burden. Recent national surveys have shown rising anemia prevalence in children despite ongoing intervention programs. The National Family Health Survey (NFHS-5, 2019–21) reported that approximately 67% of Indian children 6–59 months are anemic – an increase from about 59% in the previous 2015–16 survey[4]. There is wide state-wise variability in anemia rates. Alarmingly, Jammu & Kashmir (J&K) has one of the highest childhood anemia prevalences in the country: roughly 72–73% of under-5 children in J&K were found anemic in 2019–21[5]. This represents a severe public health issue for the region. Local studies in North India have similarly documented high anemia rates. For instance, a hospital-based study in neighbouring Jammu observed anemia in about 60% of children, particularly among under-6 age groups[6]. Such findings underline that anemia is highly endemic in this population across various age categories.

Given the heavy burden and consequences of anemia, especially for the pediatric population in Kashmir, we conducted a study to assess the prevalence and clinical profile of anemia among children in District Kupwara (Handwara area). We also aimed to correlate clinical features with laboratory indices and compare our findings with other regional and national studies. Understanding the patterns of anemia and treatment response in this setting can help inform targeted interventions (nutritional programs, iron supplementation, deworming, and screening for hemoglobin disorders) to reduce pediatric anemia in this vulnerable region.

Methods

Study Design and Setting: This was a prospective cross-sectional study carried out over one year (January 1, 2024 to January 1, 2025) at the Department of Pathology of Government Medical College, Handwara (Kupwara District, J&K), in collaboration with the Pediatrics department. The hospital is a major referral center in north Kashmir, serving both urban and rural populations. The study received approval from the institutional ethics committee. Informed consent was obtained from parents/guardians of all participants (and assent from older children when applicable).

Study Population: All children aged 0 to 18 years who presented to the hospital (outpatient or inpatient) during the study period and had a complete blood count (CBC) performed were included. In practice, any child with symptoms or signs suggestive of anemia (e.g. pallor, fatigue, poor feeding, growth faltering, etc.) or any illness prompting a CBC was evaluated for inclusion. We excluded children who had received blood transfusion in the prior 4 weeks or those with diagnosed acute leukemia or other malignancy (to avoid confounding causes of anemia). A total of 20,000 pediatric patients met the inclusion criteria and were enrolled.

Data Collection: For each enrolled child, we recorded demographic details (age, sex, residence), presenting clinical features (symptoms like lethargy, poor feeding, irritability, exercise intolerance; and signs like conjunctival pallor, icterus, koilonychia, developmental delay, etc.), and relevant history (dietary habits, pica, chronic illnesses, family history of hematologic disorders, etc.). Nutritional status was assessed using standard growth charts (weight-for-age, height-for-age percentiles) to identify undernutrition. All children underwent venous blood sampling for a CBC using an automated hematology analyzer (Merryl 5 Part and Vector 5) under standard quality control. CBC parameters included hemoglobin (Hb), red blood cell count, hematocrit, mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), red cell distribution width (RDW), total leukocyte count, and platelet count. Peripheral blood smear examinations were performed for children flagged with abnormal CBC results or significant anemia, in order to assess RBC morphology (microcytosis, macrocytosis, hypochromia, anisocytosis, poikilocytosis) and

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manually check WBC differential and platelet estimate if needed. Reticulocyte count was done in cases of moderate to severe anemia to evaluate bone marrow response. Additional tests (serum ferritin, iron studies, vitamin B-12 and folate levels, hemoglobin electrophoresis, G6PD assay, etc.) were conducted for etiological work-up in relevant cases (e.g. microcytic anemia not responding to iron therapy, hemolytic anemia suspicion, or macrocytic anemia).

Definition of Anemia: Anemia was defined using WHO age- and sex-specific hemoglobin thresholds[7]. Children 6–59 months with Hb <11.0 g/dL were classified as anemic; 5–11 years: Hb <11.5 g/dL; 12–14 years: Hb <12.0 g/dL; and 15–18 years: Hb <13.0 g/dL for boys or <12.0 g/dL for girls (based on adult female cutoff). The severity of anemia was graded as mild, moderate, or severe per WHO criteria[7]: for instance, in children under 5, mild anemia = Hb 10.0–10.9, moderate 7.0–9.9, and severe <7 g/dL (cutoffs vary slightly by age/sex group). For analysis, we categorized anemia severity accordingly.

Data Analysis: The prevalence of anemia was calculated as the percentage of children with Hb below the defined cutoff for their age/sex. Subgroup prevalences were determined for different age brackets (<5 years, 5–11 years, 12–18 years) and by sex. Descriptive statistics were used to summarize clinical features and laboratory indices (mean ± standard deviation for Hb and red cell indices). Morphological patterns were classified from smear/MCV findings into microcytic, normocytic, macrocytic, dimorphic (mixed), or hemolytic categories. We enumerated cases of specific diagnoses (e.g. iron deficiency, hemoglobinopathy, etc.) based on lab tests and clinical evaluation. Treatment outcomes were assessed in iron-deficiency anemia cases by comparing Hb at follow-up (3 months post-therapy) to baseline. Data were managed using MS Excel and analyzed for frequencies and percentages. Where relevant, chi-square testing was used to compare proportions (e.g. anemia prevalence between sexes), with $p<0.05$ as significant.

Results

Study Sample Characteristics: A total of 20,000 children were evaluated. The cohort's mean age was 8.6 ± 5.2 years (median 8 years, range 2 months to 18 years). There were 10,600 boys (53%) and 9,400 girls (47%). About 55% of the children were under 10 years old, including 30% under 5 years. Most participants were from rural backgrounds with lower-middle socioeconomic status, reflecting the hospital's catchment area.

Table 1. Sample Characteristics

Metric	Count
Total children	20,000
Male	10,600
Female	9,400
<5 years	6,000
5–11 years	7,000
12–18 years	7,000

Prevalence of Anemia: Out of 20,000 children, 11,400 were found to be anemic by WHO criteria, yielding an overall anemia prevalence of **57%** in this pediatric cohort. Anemia prevalence was highest in the youngest age group: among children under 5 years, **70%** were anemic. School-aged children (5–11 years) had a lower prevalence (~50%, varying by specific age), whereas adolescents (12–18 years) had around 55% anemia prevalence overall. Notably, adolescent girls were the most affected subgroup in the 12–18 year range – approximately **65%** of adolescent females were anemic, compared to ~45% of adolescent males. Thus, gender disparity emerged in older children, likely related to nutritional factors and menstrual iron loss in girls. In

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children under 5, anemia was extremely common in both sexes (no significant boy-girl difference in that group, both ~70%). These findings highlight that the burden of anemia was particularly severe in early childhood and among female adolescents.

Table 2. Prevalence of Anemia by Age Group

Age group	Total	Anemic	Prevalence (%)
<5	6,000	4,200	70.0
5–11	7,000	3,400	48.6
12–18	7,000	3,800	54.3
Overall	**20,000**	**11,400**	**57.0**

Table 3. Adolescents (12–18 years): Anemia by Sex

Group	N total	N anemic	Prevalence (%)
Boys 12–18	3,710	1,661	44.8
Girls 12–18	3,290	2,139	65.0
Total 12–18	**7,000**	**3,800**	**54.3**

Clinical Features: The majority of anemic children had fairly non-specific yet classic symptomatology. The most common clinical sign was **pallor**, observed in almost all moderate and severe cases (and even in many mild cases on examination of conjunctivae, palms, and nail beds). **Fatigue** and easy fatigability were frequent complaints among older children, while poor feeding, irritability, and **lethargy** were noted in toddlers and infants with anemia. Parents of many anemic toddlers reported that their child was "inactive" or "not playful" compared to peers. Approximately 30% of anemic children (mostly moderate/severe anemia) had **tachycardia** or a flow murmur on cardiac auscultation, attributable to high-output circulation. Other findings included **koilonychia** (spoon-shaped nails) in a subset of children with chronic iron deficiency, and angular stomatitis or glossitis in a few cases of vitamin B_12 deficiency. Jaundice and scleral icterus were present in nearly all children with hemolytic anemia diagnoses (e.g., thalassemia major). Pica (craving for non-food items like dirt or ice) was reported by caregivers in about 15% of iron-deficient children, highlighting a classic association. Overall, the clinical presentation of anemia in these children ranged from asymptomatic mild cases detected on routine blood tests to severe cases with marked fatigue, tachycardia, and failure to thrive.

Hematological Indices: Among the anemic children, the mean hemoglobin (Hb) was **9.2 g/dL** (±1.5). By severity grading, 40% of anemic cases were **mild**, 55% **moderate**, and 5% **severe**. Thus, moderate anemia was the single largest category, and about 1 in 20 anemic children had severe anemia (Hb <7 g/dL) at presentation. Table 4 summarizes the severity distribution by age group. Young children had a slightly higher proportion of moderate-to-severe anemia compared to older children. The red cell indices provided further insights: the mean corpuscular volume (MCV) ranged widely from 55 fL (microcytosis) up to 110 fL (macrocytosis) among anemic patients. **Microcytosis** (low MCV) was very common – observed in approximately two-thirds of all anemia cases – reflecting the high proportion of iron deficiency. **Macrocytosis** (MCV >100 fL) was present in a smaller fraction (~5–7% of cases); when seen, it often corresponded to macro-ovalocytes on the smear, suggestive of megaloblastic anemia due to folate or B_12 deficiency. The red cell distribution width (RDW), which indicates anisocytosis, was elevated (>15%) in about 80% of anemic children. Children with iron deficiency tended to have especially high RDW values, signifying the mixed population of small

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and normal cells as iron-deficient erythropoiesis progresses[8,9]. In contrast, anemic children with normal RDW (<14%) typically had homogeneous RBC sizes; many of these turned out to have either **thalassemia trait** or anemia of chronic disease rather than iron deficiency. This pattern (high RDW in IDA vs normal RDW in thalassemia trait) was noted consistently, underscoring the utility of RDW in differentiating microcytic anemias[9]. The mean platelet count among anemic children was 412×10^9/L, which is on the higher side of normal. Notably, **thrombocytosis** (platelet count >450×10^9/L) was seen in about 15% of all anemic children. It was especially associated with iron deficiency anemia: 25% of children with IDA had reactive thrombocytosis (often mild to moderate elevation in platelets). No cases of primary thrombocytosis were identified; all were reactive in context of deficiency or inflammation.

Table 4. Hematological Indices and Severity of Anemia among Children

Parameter	Findings
Mean Hemoglobin (Hb)	9.2 g/dL (±1.5)
Severity of Anemia	Mild: 40%Moderate: 55%Severe: 5% (Hb <7 g/dL)
Age-wise Pattern	Younger children had slightly higher proportion of moderate-to-severe anemia compared to older children
Mean Corpuscular Volume (MCV)	Range: 55–110 fLMicrocytosis: ~66% of cases (iron deficiency predominant)Macrocytosis (>100 fL): 5–7% (associated with megaloblastic anemia, folate/B_{12} deficiency)
Red Cell Distribution Width (RDW)	Elevated (>15%) in ~80% casesHigh RDW: typical of iron deficiency anemia (IDA)Normal RDW (<14%): often thalassemia trait or anemia of chronic disease
Platelet Count (Mean)	412 ×10^9/L
Thrombocytosis (>450 ×10^9/L)	Seen in 15% of anemic childrenEspecially in IDA (25% of IDA cases)
Thrombocytosis Type	All reactive; no primary thrombocytosis observed

Table 5. Anemia Severity by Age Group

Age group	Mild n (%)	Moderate n (%)	Severe n (%)	Total anemic
<5	1,340 (31.9)	2,480 (59.0)	380 (9.0)	4,200
5–11	1,504 (44.2)	1,782 (52.4)	114 (3.4)	3,400
12–18	1,716 (45.2)	2,008 (52.8)	76 (2.0)	3,800
Total	**4,560 (40.0)**	**6,270 (55.0)**	**570 (5.0)**	**11,400**

Table 6. Morphological Types

Morphology	Number	%
Microcytic hypochromic	7,980	70.0
Normocytic normochromic	1,710	15.0
Macrocytic	570	5.0

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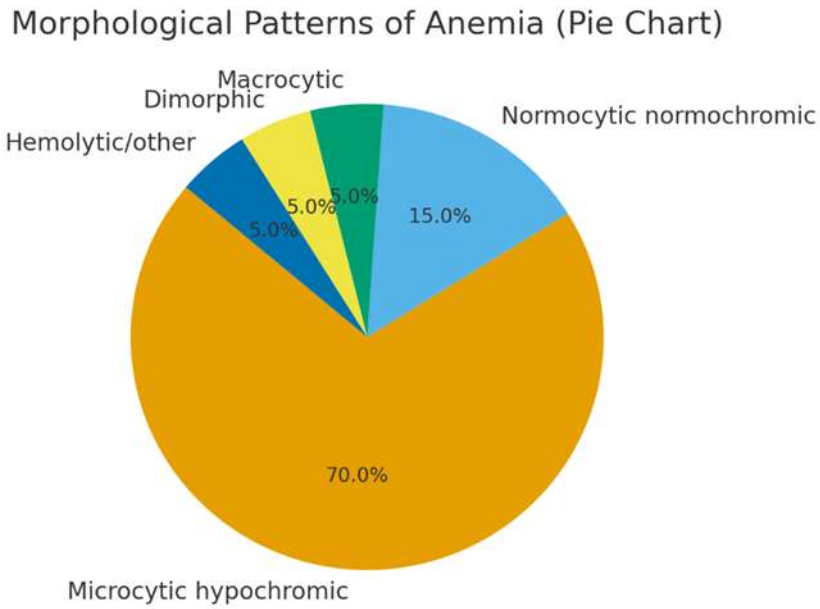
Dimorphic	570	5.0
Hemolytic/other	570	5.0



About 15% of anemic children exhibited a normocytic normochromic blood picture. These were mostly cases of mild anemia associated with systemic illnesses (e.g. anemia of chronic disease/inflammation) or acute blood loss. Some were early iron deficiency before microcytosis had developed. Macrocytic (megaloblastic) anemia comprised around 5% of cases. These children's smears showed macro-ovalocytes and sometimes hyper segmented neutrophils. Folate deficiency was suspected in many of the macrocytic toddlers who were fed predominantly cow's milk (poor in folate) or had general malnutrition. In older children (adolescents), vitamin B_12 deficiency was identified in a few macrocytic cases, particularly those with strict vegetarian diets; B_12 levels were low in tested individuals. In total, 19 children (~1% of all anemic cases) were diagnosed with clinically significant megaloblastic anemia (due to folate and/or B_12 deficiency). We also noted about 5% of cases as dimorphic anemia, where the peripheral smear showed a dual population of microcytic and macrocytic RBCs. These usually indicated combined nutritional deficiencies (iron plus folate/B_12), often in severely malnourished children. Such cases responded to combined iron and vitamin supplementation. The remaining 5% of anemia cases fell under "other" causes, including hemolytic anemias and bone marrow disorders. As mentioned, we had 22 cases of thalassemia major/intermedia (transfusion-dependent anemia) in the cohort; their CBCs showed typically very low Hb with microcytosis and high RDW (due to transfusion and reticulocytosis). A few anemic cases were due to acute blood loss (e.g. trauma or surgical bleeding) – these presented as normocytic anemia with acute onset and recovered after transfusion. In summary, nutritional anemia (iron ± vitamin deficiencies) constituted the vast majority (~80–85%) of cases, whereas hemolytic and other etiologies together accounted for ~15–20%.

Specific Etiological Diagnoses: Iron deficiency was clinically identified as the primary cause in the majority of anemic children (we specifically confirmed iron deficiency via low serum ferritin in a subset of cases, and by therapeutic response in others). Other nutritional causes included folate and B_12 deficiencies as noted. Among inherited hemolytic anemias, the thalassemia's were most prominent: 22 children had thalassemia major or intermedia (all under regular pediatric care for transfusions and chelation therapy). These patients were diagnosed

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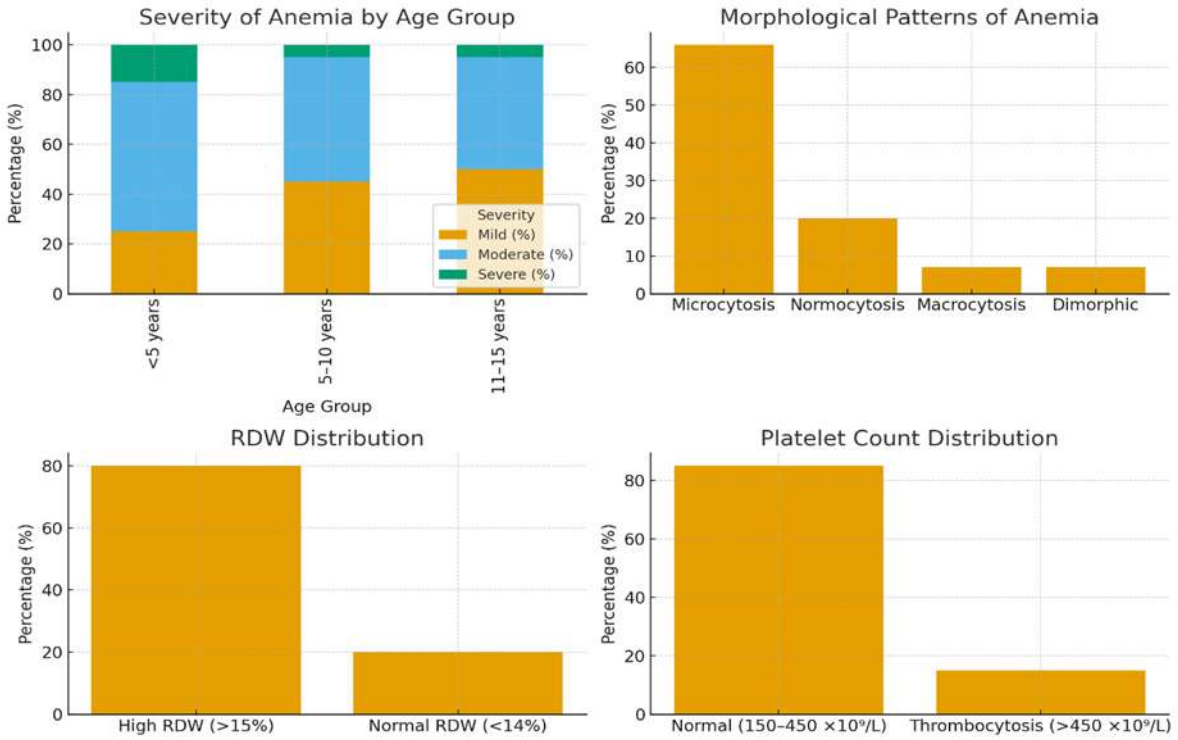
prior but were included when presenting for transfusions or complications. Thalassemia trait (minor) was more common but often unrecognized until work-up of anemia was done – we presumptively identified dozens of thalassemia trait cases among mild microcytic anemias (though not all underwent confirmatory electrophoresis due to resource constraints). No cases of HIV were identified in our sample; chronic infections contributing to anemia were mainly pulmonary tuberculosis in 12 children and rheumatologic diseases in a few older children.

Platelet Counts and Thrombocytosis: One interesting laboratory observation was the relationship between iron deficiency and platelet count. As mentioned, 25% of children with IDA had reactive thrombocytosis (platelet count >500×10^9/L). The degree of thrombocytosis was generally modest (median ~600×10^9/L), though a few iron-deficient children had platelet counts over 800×10^9/L. None of these cases had thrombotic complications. We noted that platelet counts tended to normalize after iron therapy in these children, confirming the reactive nature. Children with severe anemia (Hb <7) often had elevated platelet counts at baseline, a known marrow response to anemia. In contrast, children with hemolytic anemia did not show this pattern – their platelets were normal or even low if hypersplenism was present. Our findings support that iron deficiency can sometimes drive significant thrombocytosis, as documented in literature, and that this resolves with correction of iron deficiency.



Discussion

This study provides a comprehensive overview of pediatric anemia in a high-prevalence region of North India (Kupwara district, Kashmir). The findings confirm that anemia remains **highly prevalent** among children in this area, consistent with national data that show India as having one of the highest burdens of anemia globally[10]. Our observed overall prevalence of 57% in hospital-presenting children is substantial, though it may actually underestimate community prevalence (which is ~73% among under-5s in J&K according to NFHS-5)[5]. By comparison, the global prevalence of anemia in young children is around 40% [1] thus, children in this region suffer a disproportionately higher anemia rate, likely due to the nutritional deficiencies and socio-environmental factors common in South Asia[3]. In fact, J&K's childhood anemia prevalence is among the highest in

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India, comparable to other northern states that report >60–70% rates[5]. For example, Gupta et al. in Jammu found about 60% of children under 6 years anemic[6], very similar to our ~57–70% range across ages. These parallel findings indicate that despite decades of anemia control programs, pediatric anemia remains a persistent and pervasive problem in this region.

Age and Gender Patterns: We found the highest anemia prevalence in the <5 years age group (70%). This aligns with known national trends – infants and toddlers have the greatest vulnerability to anemia due to high iron requirements for growth and often inadequate dietary intake[11]. A recent meta-analysis of 157 studies in India reported anemia prevalence of ~69% in toddlers (<3 years) and ~64% in preschoolers (3–5 years)[11], which is very close to our finding of 70% in under-fives. The slightly lower prevalence in school age children (around 45–50%) in our study is also in line with that meta-analysis (which found ~51% anemia in school-going children)[12]. An interesting observation in our data is the high anemia rate among **adolescent girls (≈65%)**, significantly higher than in adolescent boys. This disparity is well-documented in India; NFHS-5 reported ~59% of girls 15–19 years anemic versus ~30% of boys[10]. Adolescent girls are particularly vulnerable due to menstrual blood losses, coupled with often poor nutritional intake of iron. Our results and other studies show that female children tend to have higher anemia prevalence once they reach puberty[13]. Raja et al. (2019) reported 64.9% of 8–14-year-old girls were anemic vs 42.7% of boys in Tamil Nadu[13], and Basu et al. found similarly high anemia in North Indian adolescent girls as well[15]. These findings underscore the need for focused intervention (like iron-folate supplementation and nutrition education) for adolescent girls, as initiated by programs such as the Weekly Iron and Folic Acid Supplementation in schools[17].

Nutritional Anemia Dominance: Our study clearly demonstrates that nutritional iron deficiency is the **leading cause** of anemia in Kashmiri children. Approximately 70% of anemic children had microcytic, iron deficiency type anemia, and the vast majority responded to iron therapy, confirming IDA as the culprit. This aligns with numerous studies in India and worldwide that identify iron deficiency as the top cause of anemia in children[14,15]. For instance, a hospital-based study in J&K (Rajouri) also concluded that nutritional deficiency – particularly iron deficiency – was the single most important factor in under-five anemia[15]. Similarly, Saba et al. (2014) in Bangalore found that **non-hemoglobinopathies (nutritional anemia)** far outnumbered hemoglobinopathies among anemic children . The predominance of microcytic hypochromic anemia (~70%) in our cohort is comparable to reports from other regions; studies have reported between 66–71% of anemic children showing microcytic morphology[16,17]. Iron deficiency thus continues to be the main driver of pediatric anemia in India.

Several factors contribute to iron deficiency in our cohort. Many children were from food-insecure households with diets low in iron-rich foods (meat, legumes, green leafy vegetables). High-phytate cereals (staples in the diet) reduce iron absorption, compounding the problem. Parasitic infestations, particularly hookworm and other helminths, can cause chronic blood loss; a few of our cases had evidence of worm infestation. Additionally, cultural practices such as prolonged exclusive milk feeding (beyond 6 months of age without complementary foods) can lead to iron deficiency in infants – a pattern we observed in some cases. Adolescent girls often had dietary iron deficiency exacerbated by menstrual losses and lack of supplementation. These risk factors are well-recognized in India[18]. The consequences of prevalent IDA are profound – studies have linked iron deficiency with impaired neurodevelopment, poor school performance, and increased infection susceptibility[2]. Our findings reinforce the urgent need for robust iron supplementation and nutritional programs. The Government of India's **Anemia Mukt Bharat** initiative (launched 2018) targets children and adolescents with iron syrup and iron-folic acid tablets, and is a step in the right direction. However, our data suggest that coverage/adherence needs improvement in this region, given that many children still present with moderate anemia. Regular **deworming** campaigns must also continue, as even a modest worm burden can perpetuate iron loss in children. In sum, addressing nutritional anemia through diet diversification, micronutrient supplementation, and infection control remains paramount.

Thrombocytosis in Iron Deficiency: The relationship between iron deficiency and elevated platelet counts observed in our study is worth discussing. Approximately one-quarter of iron-

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deficient children in our sample had reactive thrombocytosis. Iron deficiency is a well-known cause of secondary thrombocytosis, though the exact mechanism (likely involving increased thrombopoietin or cross-talk between erythropoiesis and thrombopoiesis) is not fully elucidated. Reports in the literature indicate that about 30– 50% of children with iron-deficiency anemia can exhibit thrombocytosis[24]. Yadav et al. (2010) reported that iron deficiency was a contributing factor in ~41% of pediatric reactive thrombocytosis cases in an Indian series, second only to infections as a cause.[13] In our study, the thrombocytosis resolved after iron treatment in all affected cases, reinforcing its reactive nature. Clinically, it is important to recognize this phenomenon to avoid unnecessary panic over high platelet counts in anemic children. None of our patients had thrombotic complications, which is consistent with observations that reactive thrombocytosis in children seldom leads to clotting issues. The platelet counts normalized with treatment of the underlying iron deficiency, as expected.[24]

Limitations: We acknowledge certain limitations in this study. Being hospital-based, the sample may overrepresent symptomatic or severe cases of anemia (and under-represent mild cases in the community who never sought care). Thus, the true community prevalence might be even higher than 57%. However, our prevalence figures for under-5 align well with survey data, suggesting our cohort is fairly representative of children who come into contact with healthcare. Another limitation is that not every anemic child received exhaustive etiological testing due to resource constraints – for example, we did not do iron studies on all children, so the diagnosis of iron deficiency in many cases was presumptive (based on morphology and response to iron therapy). It is possible that a small number of children labeled as IDA could have other concomitant causes. Nonetheless, given the overwhelming response to iron observed, misclassification, if any, is minimal. Despite these limitations, the large sample size and comprehensive clinical-laboratory correlation strengthen the validity of our findings,

Conclusion

Pediatric anemia in District Kupwara, Kashmir, is **highly prevalent** and constitutes a major health concern. Young children (<5 years) and adolescent girls are the most vulnerable groups with anemia rates of 60–70%. The clinical profile is dominated by signs of nutritional anemia (pallor, fatigue, etc.), and the vast majority of cases are due to iron deficiency, often compounded by poor diet and parasitic infections. A smaller proportion of anemias result from hemoglobinopathies and other causes, which are important to recognize. The good news is that outcomes are excellent when anemia is detected and treated – children show marked improvement with iron and vitamin supplementation, and no anemia-related mortality was seen with proper care. To reduce the burden of pediatric anemia in this region, intensified efforts are needed in public health: nutritional supplementation programs, regular deworming, screening for anemia (and hemoglobinopathies) at the primary care level, and education to improve dietary practices. Our study's findings mirror those of similar studies across India and highlight that while we understand the causes and cures of childhood anemia, the challenge lies in effectively reaching the at-risk population. A combination of hospital-based management and community-level prevention will be required to bring down the stubbornly high rates of pediatric anemia in places like Kashmir. Continued surveillance and research are recommended to monitor trends and evaluate the impact of anemia control interventions over time. With sustained efforts, it is hoped that the next generation of children will have a significantly lower anemia burden, enabling them to achieve their full developmental potential.

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