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Anemia In Tribal Children: Epidemiological And Hematological Insights From A Multi School Screening Program In South India.

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Abstract

Background: Tribal populations inhabiting remote and underprivileged regions, are at risk of several health disparities, including a higher prevalence of anemia. The unique social, economic, and cultural factors influencing their lifestyle and access to healthcare services contribute to the increased burden of anemia in tribal children. Understanding the prevalence and hematological parameters of anemia among tribal children is crucial for designing strategies for effective intervention and improving health outcomes. This study highlights the magnitude of the problem and contributes to the existing data pool, paving the way for targeted interventions and policies. ***Material and Methods:*** A descriptive cross sectional epidemiological study was conducted across 1534 tribal children aged between 1 to 20 years attending Anganwadi centres and schools in Hukumpet from February 2023 to December 2023. Blood samples were collected in ethylenediaminetetraacetic acid (EDTA) anticoagulated vacutainer tubes and processed on the same day to measure various hematological parameters including Hemoglobin Electrophoresis. Age and gender focused analysis were performed using R statistical software version 4.3.0 and Python 3.10. Age specific WHO cutoffs were used to grade anemia. ***Results:*** Of the 1534 children evaluated, female to male ratio was 1.32:1 with 56.9% female and 43.1% male. Data showed anemia prevalence as 20.6% with 74.7% showing mild, 22.5% moderate and 2.8% severe anemia. Maximum prevalence in the younger 5-9 year age group was observed. ***Conclusion:*** This study suggests iron deficiency to be the leading cause of anemia among tribal children between 1-20 years in Hukumpet, Andhra Pradesh. The major finding of microcytic hypochromic erythrocytes with statistically significant positive correlation between mean corpuscular volume and hemoglobin adds further weight to the prevalence of anemia due to iron deficiency. The presence of microcytes in children with hemoglobinopathy variants as well as those with normal hemoglobin highlights an ideal target population for anemia prevention through iron supplementation. Adolescent females comprise a majority of the cohort, mandating targeted iron supplementation in the menstruating age group.

Keywords: Anemia prevalence, school children, haemoglobin levels, RBC indices, tribal health, school based nutrition programmes.

INTRODUCTION

Iron deficiency anemia (IDA), the most prevalent and treatable form of anemia globally, remains underdiagnosed in susceptible populations, causing developmental, cognitive and economic sequelae [1]. 39.8% of children 6-59 months in the world were affected by anemia in 2019 [2]. Dietary factors, physiological demands and endemic hookworm infection cause disproportionate burden in children from middle and low income countries [3]. In India, the National Family Health Survey-5 has reported the prevalence of anemia as 57% [4]. This underrepresents the anemia burden in tribal populations, who have a documented prevalence of 87% in preschool children, with a majority of these cases attributed to iron deficiency [5]. Hemoglobinopathies, which carry structural or functional hemoglobin aberrations, comprise the most prevalent monogenic disorders worldwide; approximately 20 million people and over 300,000 births are afflicted yearly by Sickle cell disease (SCD) [6]. Both IDA and hemoglobinopathies prevail in tribal populations, shaping a dual predicament to national public health.

Indian tribal populations (about 104 million-roughly 8.6% of the total population), face healthcare challenges resulting from geographic inaccessibility, economic constraints, undernutrition, and systemic neglect [7,8]. Therefore, IDA in tribal children surpasses their non tribal counterparts, yet remains undiscovered in community surveys and overlooked by preventive health programmes. Childhood iron deficiency affects longterm neurobiological development: by impaired myelination of the nervous system, reduced synthesis of neurotransmitters, and disruption of synaptic plasticity, culminating in diminished intelligence quotient (IQ), attention deficit, behavioral issues, and academic setbacks [9,10]. Longitudinal studies establish that children with iron deficiency have poorer academic outcomes, enter into employment at a disadvantage, and accumulate lower lifetime earnings, thus propagating intergenerational poverty [11]. India loses an estimated 24 billion USD yearly due to anemia-related causes. However, tribal population, despite considerable disease burden, remain outside the purview of the benefits of national anemia control programs [12].

Targeted interventions are based on mechanistic differences between IDA and hemoglobinopathy. IDA, characterised by diminished iron stores and impaired hemoglobin production, yields microcytic (mean corpuscular volume <80 femtoliters), hypochromic (mean corpuscular hemoglobin <27 picograms) erythrocytes. Anisocytosis indicates elevated red cell distribution width, and strong correlation between red blood cell (RBC) indices and hemoglobin concentration [13]. Sickle cell disease, a hemoglobinopathy-related anemia, presents with another distinctive pattern of erythrocyte morphology and RBC indices.
Pioneering research by Rao VR, et al demonstrated the prevalence of sickle cell trait between 10% to 33% in specific tribal groups across central India [14]. However, studies discriminating IDA from hemoglobinopathy, utilizing advanced hematological analysis with high-performance liquid chromatography (HPLC) confirmation and comprehensive RBC indices have remained limited [15,16].

The conceptual background of this study is based on the pathophysiological mechanisms behind childhood IDA: inadequate dietary intake in low-resource setting diminishes iron stores, compounded by elevated physiological demands during rapid growth and further iron losses through menstruation in adolescent females. Microcytic hypochromic anaemia, characterised by decrease in mean corpuscular volume and mean corpuscular haemoglobin and increase in red cell distribution width and erythrocyte count (signifying compensatory erythropoiesis), represents iron-restricted erythropoiesis brought on by progressive iron depletion. These hematological indices present a distinctive pattern for diagnosing iron deficiency anemia without performing serum iron studies. This study aims to deliver the evidence base necessary for diagnosing and planning of nutritional interventions and targeted iron supplementation within tribal areas.

The study is intended to determine the prevalence and severity of iron deficiency anemia (IDA) among tribal children aged 1–20 years in Andhra Pradesh using WHO-defined criteria and hematological indicators such as hemoglobin level, microcytosis, and hypochromia. It also aims to identify high-risk subgroups based on age, gender, and hematological parameters that may benefit from prioritized school- and community-based nutritional interventions.

MATERIALS AND METHODS

Study Design and Setting

A descriptive cross-sectional epidemiological study was performed among tribal children in Andhra Pradesh to ascertain the prevalence, severity, and hematological characteristics of IDA. The cross-sectional design achieved a snapshot prevalence of anemia across a defined population at a single point in time. Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for cross-sectional studies were followed, maintaining transparent documentation of study methodology, data collection, and analysis [17]. Government tribal schools and Anganwadi centers across Hukumpeta Mandal, Alluri Sitharama Raju (ASR) District, Andhra Pradesh, participated in the study conducted from February 2023 to December 2023. Hukumpeta Mandal was chosen due to its tribal population concentration (92%), administrative support for screening programs, and high prevalence of hemoglobinopathies and anaemia in government surveillance data. The rural landscape, insufficient healthcare infrastructure, and significant socioeconomic disadvantage typical of the state's tribal districts defined the physical backdrop.

Ethics and Regulatory Approval

Institutional Ethics Committee approval was obtained from Gayatri Vidya Parishad Institute of Health Care and Medical Technology, Visakhapatnam. The study adhered to the National Ethical Guidelines for Biomedical and Health Research, framed by the Indian Council of Medical Research (ICMR) which safeguard vulnerable groups including tribal population and children [18]. Written permission was obtained from the District Collector, ASR District, Paderu (File No. REV02-LMT/27/2023-LMS-CCLA, February 22, 2023, valid eighteen months). District authorities provided further amenities. Exhaustive participant information sheets in regional language were provided, and informed consent was taken from parents or local guardians.

Study Participants and Sampling

Tribal children aged 1 to 20 years attending Anganwadi centers and government-run tribal schools in Hukumpeta Mandal were selected. Inclusion criteria specified enrollment in partaking institutions, age 1–20 years, and parental/custodian consent for participation. Exclusion criteria included acute severe health condition on screening date, known hematological malignancy, or non consent of parent or guardian. Consecutive samples were obtained from all children meeting eligibility criteria. The final cohort comprised 1,534 participants.

Laboratory Equipment, Materials, and Quality Assurance

Blood was drawn by qualified technicians in sterile, ethylenediaminetetraacetic acid (EDTA)-anticoagulated vacutainer tubes and processed on the same day, minimizing artifactual hemolysis. Hemoglobin levels, hematocrit, mean corpuscular volume, mean corpuscular hemoglobin, red cell distribution width, erythrocyte count, leucocyte count, and platelet count were quantified by an automated hematology analyzer (XN-1000 Sysmex, Japan) calibrated as specified by manufacturer and validated by standardized quality control materials before commencing sample collection daily. Hemoglobin electrophoresis was done at the District Hospital, by high-performance liquid chromatography (HPLC).

Data Collection Procedures

Screening camps were conducted at designated centres on scheduled dates arranged with school authorities. Parents/custodians were notified through written advance announcement in local language prior to the screening, and trained investigators obtained informed consent from them on the screening day. Demographic data was entered digitally, including name, age, gender, date of birth, and institution. Five milliliters of blood were drawn and labeled into EDTA-anticoagulated collection tubes by technicians using aseptic precautions. Samples were sent on the same day to accredited laboratory using standard procedure. Reports were conveyed to participants within a fortnight, either through their school or parental contact.

Outcome Measures

The primary outcomes assessed were: (1) Anemia prevalence (hemoglobin <11 g/dL using WHO age-specific cutoffs) (2) Anemia severity (mild, moderate, severe) (3) Documenting microcytosis (MCV <80 fL, hypochromia defined as MCH <27 pg) as hematological markers of iron deficiency anemia.

Secondary outcome measures included: (1) Measurement and correlation of mean corpuscular volume with hemoglobin levels (2) Measurement and correlation of mean corpuscular hemoglobin with hemoglobin levels (3) Red cell distribution width (4) Prevalence of hemoglobinopathies detected through HPLC; (5) Age and gender based stratification of variances in iron deficiency markers (6) Determining the proportion of anemic children displaying microcytic hypochromic picture concomitant with iron deficiency.

Statistical Analysis

Descriptive statistical analysis to estimate prevalence, means, percentiles and standard deviations was performed by compiling hemoglobin levels, mean corpuscular volume, mean corpuscular hemoglobin, red cell distribution width, and age. Categorical variables like gender, anemia grading, and hemoglobinopathy status were expressed as frequencies and percentages. Association between mean corpuscular volume and hemoglobin level, and between other red blood cell indices and hemoglobin level was examined with Pearson correlation coefficients, to determine statistical significance ascertained at alpha equals 0.05. Age and gender-focused analysis illustrated anemia prevalence, grading, hematological indices, and iron deficiency markers among categories. Cross-tabulation assessed the correlation between hemoglobinopathy status and detection of microcytic hypochromic anemia. R statistical software version 4.3.0 and Python 3.10 were employed for statistical analysis. Bar graphs, stratified tables, and correlation plots were utilized for representing data and facilitating interpretation and identification of patterns of iron deficiency anemia.

RESULTS

Table 1. Demographic characteristics of the study population

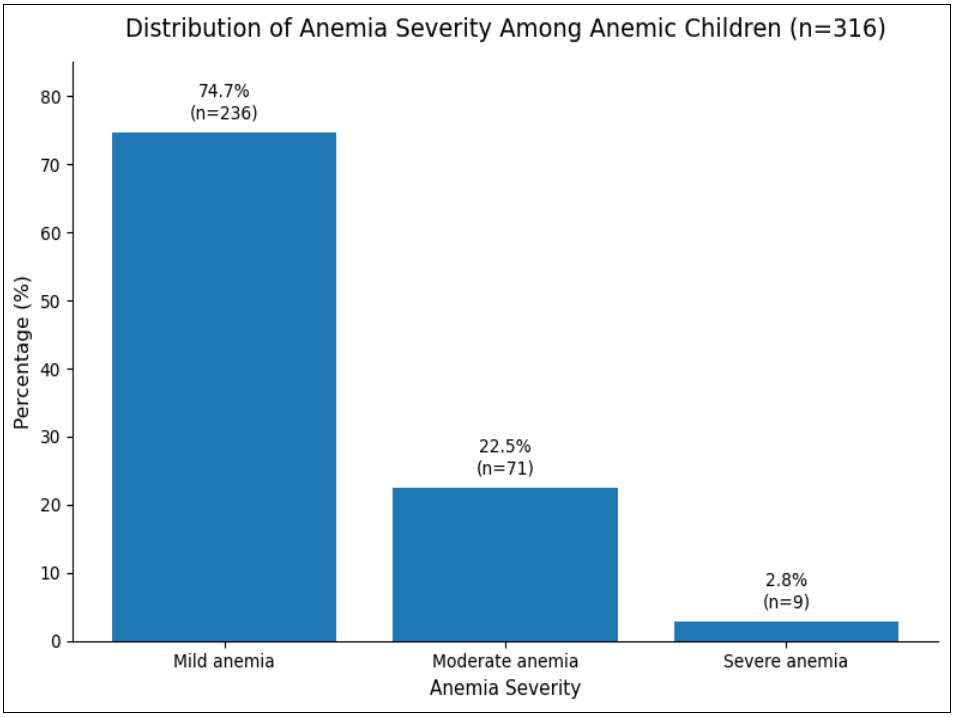
Characteristic	Number (n)	Percentage (%) / Mean ± SD
Male	662	43.1
Female	872	56.9
Age (years)	1–20	11.47 ± 3.95
Total participants	1,534	100.0

A total of 1,534 tribal children aged 1–20 years were included in the study, with a mean age of 11.47 ± 3.95 years. Females constituted a greater proportion of the study population, accounting for 872 (56.9%) participants, while 662 (43.1%) were

males. Thus, the female-to-male distribution was approximately 1.32:1. The study population represented a broad pediatric and adolescent age range. The relatively large sample provides an adequate basis for examining the burden and hematological characteristics of anemia among tribal children.

Table 2. Prevalence and severity of anemia among the study population

Anemia status/severity	**Number (n)**	**Percentage (%)**
Total study population	1,534	100.0
Anemic children	316	20.60
Non-anemic children	1,218	79.40
Severity among anemic children (n=316)		
Mild anemia	236	74.7
Moderate anemia	71	22.5
Severe anemia	9	2.8



The overall prevalence of anemia among the tribal children was 20.60%, with 316 of the 1,534 children identified as anemic. Among children with anemia, mild anemia was the predominant category and was observed in 236 (74.7%) cases. Moderate anemia was identified in 71 (22.5%) children, whereas severe anemia was relatively uncommon, affecting 9 (2.8%) children. Therefore, approximately three-fourths of the anemia burden consisted of mild cases. Nevertheless, the occurrence of moderate and severe anemia in nearly one-fourth of anemic children indicates a clinically important nutritional and public-health burden requiring early detection and intervention.

Table 3. Gender-wise distribution of anemia among tribal children

Gender	**Total n**	**Anemic n (%)**	**Non-anemic n (%)**	**χ^2 value**	**p value**
Male	662	91 (13.74)	571 (86.26)		
Female	872	225 (25.80)	647 (74.20)	**32.71**	**<0.001**
Total	1,534	316 (20.60)	1,218 (79.40)		

Anemia showed a marked gender disparity, with 225 of 872 females (25.80%) affected compared with 91 of 662 males (13.74%). The prevalence of anemia among females was approximately 1.32 times that observed among males. Chi-square analysis demonstrated that the association between gender and anemia was statistically significant (χ^2=32.71, p<0.001). These findings identify female children, particularly those approaching and entering adolescence, as an important high-risk subgroup. Increased physiological iron requirements during growth and menstrual iron loss among adolescent girls may contribute to this difference and support prioritization of female children for nutritional screening and intervention.

Table 4. Hematological profile of the study population according to anemia status

Hematological parameter	Overall cohort	Anemic children (n=316)	Non-anemic children (n=1,218)
Hemoglobin (g/dL)	—	<11.0*	≥11.0*
MCV (fL)	—	71.53 ± 7.28	77.41 ± 7.51
MCH (pg)	23.14 ± 2.99	20.31 ± 2.15	—
RBC count (million/µL)	5.47 ± 1.39	—	—
RDW	Raised	Raised	—
Microcytosis, MCV <80 fL	1,064 (69.36%)	274 (86.71%)	790 (64.86%)

The hematological profile demonstrated a predominantly microcytic and hypochromic pattern among anemic children. Mean MCV was substantially lower among anemic children (71.53 ± 7.28 fL) than among non-anemic children (77.41 ± 7.51 fL). Similarly, the mean MCH among anemic children was 20.31 ± 2.15 pg, indicating marked hypochromia. The overall mean MCH of the cohort was also low at 23.14 ± 2.99 pg. Raised RDW indicated variation in red-cell size or anisocytosis. Collectively, the high frequency of microcytosis and hypochromia provides hematological evidence of a substantial burden of iron-restricted erythropoiesis in this population.

Table 5. Association of microcytosis with anemia status

Anemia status	Total n	Microcytosis n (%)	No microcytosis n (%)	χ^2 value	p value
Anemic	316	274 (86.71)	42 (13.29)		
Non-anemic	1,218	790 (64.86)	428 (35.14)	**55.34**	**<0.001**
Total	**1,534**	**1,064 (69.36)**	**470 (30.64)**		

Microcytosis was highly prevalent in the study population, occurring in 1,064 of 1,534 children (69.36%). Among anemic children, 274 (86.71%) demonstrated microcytosis compared with 790 (64.86%) of non-anemic children. The association between anemia and microcytosis was statistically significant (χ^2=55.34, p<0.001). Anemic children were approximately 3.53 times more likely to demonstrate microcytosis than non-anemic children based on the supplied data. Importantly, microcytosis was also observed in nearly two-thirds of non-anemic children, suggesting that abnormalities in erythrocyte indices may occur before overt reduction in hemoglobin concentration. This group may represent an important target for further evaluation of iron status and preventive nutritional intervention.

Table 6. Correlation of hematological indices with hemoglobin concentration

Hematological parameter correlated with Hb	Pearson correlation coefficient (r)	Strength/direction of correlation	p value
MCV	0.1932	Weak positive	<0.001
MCH	0.2553	Weak positive	<0.001
RBC count	0.8702	Strong positive	<0.001

Correlation analysis demonstrated statistically significant positive relationships between hemoglobin concentration and the evaluated red-cell indices. MCV showed a weak positive correlation with hemoglobin (r=0.1932, p<0.001), indicating that hemoglobin levels tended to increase modestly with increasing red-cell volume. MCH also showed a weak positive correlation with hemoglobin (r=0.2553, p<0.001). In contrast, RBC count demonstrated a strong positive correlation with hemoglobin concentration (r=0.8702, p<0.001). The statistically significant relationships support the usefulness of routinely available hematological indices in characterizing anemia. However, correlation alone does not establish the etiology of iron deficiency, and these indices should be interpreted together with the complete hematological profile.

Table 7. Hematological indices according to hemoglobinopathy status

Hemoglobinopathy status	n	Mean Hb (g/dL)	Mean MCV (fL)	Microcytosis n (%)
Normal hemoglobin	1,397	12.62	76.40 ± 7.77	962 (68.86)
Hemoglobinopathy trait/disease	137	12.15	74.21 ± 8.15	102 (74.45)
Total	**1,534**	—	—	**1,064 (69.36)**

Microcytosis was common among children with both normal and abnormal hemoglobin patterns. Among children with normal hemoglobin, 962 of 1,397 (68.86%) had an MCV below 80 fL, while microcytosis was observed in 102 of 137 (74.45%) children with a hemoglobinopathy. Mean MCV was slightly lower among children with hemoglobinopathy (74.21 ± 8.15 fL) compared with those with normal hemoglobin (76.40 ± 7.77 fL). Mean hemoglobin concentration was also modestly lower in the hemoglobinopathy group (12.15 versus 12.62 g/dL). The presence of a high proportion of

microcytosis even among children with normal hemoglobin patterns indicates that microcytosis cannot be attributed solely to hemoglobinopathy. Nutritional iron deficiency or other causes of microcytosis therefore require consideration in both groups.

Table 8. Prevalence and distribution of hemoglobinopathies among tribal children

Hemoglobin pattern	Number (n)	Prevalence (%)	Mean Hb (g/dL)	Anemic n (%)
Normal hemoglobin	1,397	91.06	12.62	290 (20.76)*
Sickle cell trait	131	8.55	12.29	25 (19.08)**
Sickle cell disease	6	0.39	9.64	4 (66.7)**
Any hemoglobinopathy	137	8.94	12.15*	26 (18.99)*
Total	1,534	100.0	—	316 (20.60)

*According to the reported hemoglobinopathy cross-tabulation.
**According to the separately reported sickle trait/disease results.

Hemoglobinopathies were detected in 137 of 1,534 children, giving an overall prevalence of 8.94%. Sickle cell trait was the predominant abnormality and was identified in 131 (8.55%) children, whereas homozygous sickle cell disease was found in 6 (0.39%) children. Trait carriers had a relatively preserved mean hemoglobin concentration of 12.29 g/dL, whereas children with sickle cell disease had a considerably lower mean hemoglobin of 9.64 g/dL. Anemia was reported in 19.08% of sickle cell trait carriers and 66.7% of children with sickle cell disease. These findings demonstrate that sickle cell trait constitutes the majority of detected hemoglobin variants, while overt sickle cell disease is associated with a substantially greater hematological impact.

Table 9. Association between anemia and hemoglobinopathy status

Hemoglobin status	Anemic n (%)	Non-anemic n (%)	Total	χ^2 value	p value
Normal hemoglobin	290 (20.76)	1,107 (79.24)	1,397		
Abnormal hemoglobin	26 (18.99)	111 (81.01)	137	**0.145**	**0.703**
Total	**316 (20.60)**	**1,218 (79.40)**	**1,534**		

Anemia was present in 20.76% of children with a normal hemoglobin pattern and in 18.99% of children with an abnormal hemoglobin pattern. The difference between the two groups was small and was not statistically significant (χ^2=0.145, p=0.703). Thus, in this dataset, the presence of a hemoglobinopathy as a combined category was not significantly associated with overall anemia status. This observation, together with the high prevalence of microcytosis in children with normal hemoglobin, supports consideration of nutritional and iron-related factors in the overall anemia burden. However, the markedly greater anemia frequency among the small subgroup with sickle cell disease should be interpreted separately from sickle cell trait. Etiological attribution specifically to iron deficiency would ideally require confirmation with iron-status investigations.

DISCUSSION

Results from this investigation reinforces iron deficiency as the leading cause of anemia in tribal children. Statistically significant positive correlation between mean corpuscular volume and hemoglobin concentration (r=0.1932, p<0.001), and exceptionally strong correlation between red blood cell count and hemoglobin (r=0.8702, p<0.001), confirms iron-restricted erythropoiesis as the pathophysiological mechanism of anemia in this cohort [19]. The 69.3% prevalence of microcytic erythrocytes (MCV <80 fL) in the population, rising to 86.71% in anemic children, denotes high prevalence of microcytosis which is established as the dominant hematological signature of the group [20]. This prevalence significantly exceeds anticipated prevalence in populations with balanced nutritional status, substantiating that iron deficiency is an extensive public health crisis in this tribal population.

The hematological analysis displayed features typical of iron deficiency anemia: microcytosis (mean MCV 76.2 fL), hypochromia (mean MCH 23.1 pg), increased red cell distribution width suggesting anisocytosis and elevated red blood cell count (5.47 million/µL) mirroring compensatory erythropoiesis to maintain oxygen-carrying capacity despite low hemoglobin levels [21]. Compensatory erythropoiesis remains inadequate in severe iron deficiency resulting in symptomatic anemia. The mean MCV of 71.53 fL in anemic children signifies severe microcytosis, considerably lower than normal (78–100 fL), confirming profound iron deficiency at the level of erythropoiesis [22].

Girls experienced 1.32 times higher anemia rates compared to boys, reflecting heightened vulnerability in adolescent females due to physiologic and socioeconomic factors. Unavoidable iron loss occurs through menstruation, with 15–30 mg iron lost for each menstrual cycle. This increases adolescent female iron requirements to 15 mg per day compared to 11 mg in adolescent males [2]. Physiologic iron deficit results in depleted iron stores and subsequently as IDA in populations

with restricted dietary iron intake, caused by extreme poverty, limited access to dietary sources, and reduced bioavailability of plant-based iron [23]. The revelation that 17.06% of adolescent girls (15–19 years) manifested anemia, surpassing younger age groups, delivers undeniable evidence that menarche and menstruation amplify risk of anemia in tribal populations.

This study demarcates between IDA and hemoglobinopathy-related anemia based on hematological findings. 301 out of 316 anemic children (95.3%) had normal hemoglobin patterns, whereas 15 (4.7%) showed hemoglobin variants (trait or disease). This tremendous majority reveals that nutritional factors constitute the dominating cause of anemia in this population. The comparable prevalence of anemia between children with normal hemoglobin (20.76%) and those with hemoglobinopathy (18.99%) indicates that hemoglobinopathy status does not independently increase anemia risk; rather, anemia in hemoglobinopathy carriers reflects parallel nutritional iron deficiency rather than disease-mediated erythrocyte destruction or hemoglobin dysfunction [24].

Compellingly, 74.45% of children demonstrating hemoglobinopathy variants showed microcytic erythrocytes compared to 68.86% in those with normal hemoglobin, indicating that the microcytic pattern of iron deficiency anemia prevails in the entire cohort, regardless of genetic hemoglobin status. This conclusion could guide public health intervention strategies like iron supplementation and dietary fortification programs to form the primary prophylactic measure for anemia reduction, irrespective of hemoglobinopathy status.

The distribution of anemia severity (74.7% mild, 22.5% moderate, and only 2.8% severe cases) confers optimistic intervention potential. This distribution suggests that several anemia cases remain in early stages prompting nutritional intervention to suffice. Cognitive and developmental consequences, recognized even in sub-clinical iron deficiency, may be partly alleviated through early nutritional intervention before advancing to irrevocable neurobiological outcomes [25].

The discovery of microcytosis even in children without anemia (64.86%) elicits an observation: the demonstration of iron depletion (presenting as microcytosis) before manifesting anemia clinically. This population comprises the ideal target for preventive iron supplementation [26].

Study Limitations and Their Implications

Certain limitations warrant consideration.

1. The cross-sectional design precludes causal inference pertaining to temporal relationships between iron deficiency and anemia development. Longitudinal surveillance would facilitate observation of whether iron stores deplete to frank anemia or whether anemia remains stable.
2. Only hemoglobin and RBC indices were studied, without investigating biomarkers (serum ferritin and iron, transferrin saturation, soluble transferrin receptor), precluding definitive evidence that the principal anemia mechanism is iron deficiency. However, the predominant microcytic hypochromic pattern and sturdy correlation between RBC indices and hemoglobin provide ample evidence to substantiate iron deficiency as the cause.
3. Modifiable risk factors like dietary deficiencies, infections like endemic hookworm and malaria were not assessed.
4. The school-based population study excludes non-school-going children and those with severe illness preventing school attendance, therefore underrating anemia burden in the most susceptible subgroups.

Recommendations for Future Research

Future research could include:

1. Longitudinal cohort studies with at least 5 years surveillance for observing natural course of iron deficiency anemia and effectiveness of school-based iron supplementation programs.
2. Inclusion of iron studies (serum iron, ferritin) for confirmation of iron deficiency.
3. Complete dietary evaluation to verify adequacy of iron intake.
4. Screening for endemic parasites (hookworm, Ascaris, Trichuris) which comprise modifiable causes of anemia.
5. Cognitive and developmental assessment to determine intellectual aftermath of anemia.
6. Implementation studies to assess the impact of school-based nutritional interventions [27].

CONCLUSION

This detailed cross-sectional screening exercise involving 1,534 tribal children between 1–20 years in Andhra Pradesh has revealed epidemiological and hematological evidence indicating iron deficiency as the leading cause of anemia, with microcytic-hypochromic picture adding validity to targeted intervention. Although lower than other regional estimates, the overall prevalence of anemia affecting approximately 1 in 5 children (20.60%), embodies a significant public health challenge entailing immediate, coordinated nutritional intervention. The staggering presence of microcytes (69.36% of total cohort, 86.71% of anemic subjects), the statistically significant positive association between mean corpuscular volume

and hemoglobin concentration (r=0.1932, p<0.001) and significant correlation between red blood cell count and hemoglobin (r=0.8702) corroborate iron-restricted erythropoiesis as the mechanism of anemia.

The noteworthy female to male anemia ratio of 1.32:1, with adolescent girls exhibiting anemia rates of 17.06%, emphasizes their vulnerability during menstruating years and mandates gender-targeted iron supplementation strategies tackling both physiologic menstrual blood loss and socioeconomic pitfalls.

The parallel prevalence rates of microcytosis and anemia in children with normal hemoglobin (20.76%) and those with hemoglobinopathy variants (18.99%) suggests that iron deficiency impacts both populations similarly, validating nutritional rather than genetic mechanisms as the predominant driver of anemia burden. The proportion of children with mild anemia (74.7%) along with high prevalence of microcytosis even among non-anemic children (64.86%) signals ample prospects for prevention through iron supplementation before symptomatic anemia onset and permanent neurobiological consequences.

By further developing this epidemiological and hematological database, India's tribal health programs can transform from passive surveillance toward active, evidence-based prevention and management of iron deficiency anemia. This ultimately minimizes health inequities and enables tribal children to realize their full developmental, cognitive, and economic potential.

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