

Correlation of Hematological Parameters with Severity of Iron Deficiency Anemia in Children Aged 6 Months to 12 Years**Rajeshkumar Narshangji Tervadiya¹, Smit Ashokkumar Patel², Shraddhaben Kanaiyalal Modi³**¹MBBS, DCH, Department of Paediatrics, GMERS Medical College and Hospital, Dharpur, Patan, Gujarat, India²MBBS, DCH, Department of Paediatrics, Civil Hospital, Mehsana, Gujarat, India³MBBS, DPB, Department of Microbiology, GMERS Medical College and Hospital, Dharpur, Patan, Gujarat, India

Received: 10-04-2026 / Revised: 20-05-2026 / Accepted: 28-05-2026

Corresponding author: Dr. Rajeshkumar Narshangji Tervadiya

Conflict of interest: Nil

Abstract**Background:** Iron deficiency anemia is the most prevalent nutritional anemia in children and can affect growth, immunity, cognition and development. Where iron studies are not available, hematological indices may be useful in determining the severity and for early diagnosis.**Methods:** This cross sectional study involved 210 children aged 6 months to 12 years who were diagnosed with IDA by hemoglobin, red cell indices, peripheral smear, serum ferritin and transferrin saturation. Children who had hemoglobinopathies, chronic kidney disease, acute infection, recent transfusion, or hematinic therapy were excluded. The hematological parameters were compared between the mild, moderate and severe anemia groups.**Results:** Mean age was 4.8 +/- 3.1 years; 116 children (55.2%) were male. The most common anemia was moderate (46.7%), followed by mild (31.9%) and severe (21.4%). Mean hemoglobin was 10.4 +/- 0.5 g/dL in mild, 8.3 +/- 0.8 g/dL in moderate, and 6.4 +/- 0.7 g/dL in severe anemia (p<0.001). As the severity increased, MCV, MCH, ferritin and transferrin saturation decreased, whereas RDW and platelet count increased. Hemoglobin correlated positively with ferritin (r=0.52, p<0.001) and MCV (r=0.61, p<0.001), and negatively with RDW (r=-0.58, p<0.001).**Conclusion:** Routine haematological parameters are significantly related to the severity of paediatric IDA and can be used as an aid to early grading and treatment decisions.**Keywords:** iron deficiency anemia, children, hemoglobin, MCV, RDW, ferritin, hematological parameters.**DOI:** 10.25258/ijcpr.18.6.3

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Iron deficiency anemia is the most common nutritional anemia in children globally and is a significant public health issue in LMICs. This is due to insufficient iron intake, low bioavailability, rapid growth, parasitic infestation, repeated infection and chronic blood loss.

Beyond pallor and fatigue, the effects include impaired neurodevelopment, behavioural changes, diminished immunity and poor school performance [1]. Infancy, toddlerhood, and school age are periods of rapid growth and iron needs, thus children ages 6 months to 12 years are a vulnerable group. Iron deficiency is caused by complementary feeding practices, excessive intake of cow milk, low intake of iron rich foods, and socioeconomic factors. Early detection is key, as developmental effects can occur even after hematological

correction [2]. Complete blood count parameters like hemoglobin, mean corpuscular volume, mean corpuscular hemoglobin, red cell distribution width, and platelet count are readily available and cheap. Iron deficiency usually gives rise to microcytic hypochromic anemia, which is characterized by a low MCV, a low MCH and a high RDW. Depleted iron stores can be confirmed by serum ferritin and transferrin saturation, which can be influenced by inflammation [3].

There are a few studies that have assessed hematological profiles in children with IDA. Mujib et al. described the characteristic changes in hemoglobin, MCV, MCH and RDW in pediatric population, highlighting the diagnostic utility of automated indices in resource-limited settings [4]. Current reviews still suggest that CBC, ferritin,

inflammatory markers and clinical context should be integrated [5].

The clinical importance of grading severity is that severe anemia may warrant immediate evaluation, treatment supervision and assessment for complications, while mild and moderate anemia may be treated with oral iron and dietary counselling. Hematological correlations can also assist the clinician in determining whether a child requires iron studies in the event of limited resources.

The purpose of this study was to determine the relationship between the severity of iron deficiency anemia and the haematological parameters in children 6 months to 12 years old. The aim of the study was to compare the red cell indices, RDW, platelet count, ferritin and transferrin saturation between severity groups and to determine which parameters correlated best with hemoglobin level.

Materials and Methods

This was a cross-sectional study in paediatrics outpatient and inpatient department of a tertiary care teaching hospital for one year. The children aged 6 months to 12 years who had been newly diagnosed with iron deficiency anemia were enrolled with parental consent. There were 210 children who met the inclusion criteria. Iron deficiency anemia was diagnosed by age adjusted hemoglobin cut-offs, microcytic hypochromic red cell morphology, low serum ferritin, and/or low transferrin saturation. Severity was defined as mild, moderate, and severe based on age-appropriate

hemoglobin levels, which were based on standard pediatric criteria. Children who had known hemoglobinopathy, a family history of thalassemia trait, disproportionate microcytosis, chronic kidney disease, malignancy, acute severe infection, inflammatory disease, recent blood transfusion, iron therapy within the last three months, or folate/vitamin B12 deficiency were excluded. The data collected were age, sex, socioeconomic status, dietary history, pica, worm infestation history, anthropometry, clinical sign and laboratory parameters. CBC was done with an automated analyser. Peripheral smear, reticulocyte count, serum ferritin, serum iron, total iron-binding capacity, and transferrin saturation were obtained when available.

The data were analyzed statistically using SPSS version 26. The data for continuous variables were presented as means \pm SDs and for categorical variables as frequencies and percentages. Hematological parameters were compared between the severity groups using ANOVA and post hoc testing. Pearson correlation was used to determine the relationships with hemoglobin and $p < 0.05$ was considered statistically significant.

Results

The study dataset was checked for completeness before analysis. All enrolled participants had complete clinical records and laboratory or imaging values required for the primary outcomes. Descriptive and inferential results are presented in the following tables.

Table 1: Demographic and clinical profile of children with IDA (n=210).

Variable	Category/Measure	Value
Age	Mean +/- SD	4.8 +/- 3.1 years
Age group	6-24 months	64 (30.5%)
Age group	2-5 years	83 (39.5%)
Age group	6-12 years	63 (30.0%)
Sex	Male	116 (55.2%)
Sex	Female	94 (44.8%)
Pica	Present	78 (37.1%)
Worm infestation history	Present	46 (21.9%)
Underweight	Present	72 (34.3%)

Table 1 shows that most children were below five years of age, and pica, underweight status, and worm infestation history were common.

Table 2: Hematological parameters according to anemia severity.

Parameter	Mild (n=67)	Moderate (n=98)	Severe (n=45)	p-value
Hemoglobin (g/dL)	10.4 +/- 0.5	8.3 +/- 0.8	6.4 +/- 0.7	<0.001
MCV (fL)	72.6 +/- 6.8	66.8 +/- 7.4	59.7 +/- 8.1	<0.001
MCH (pg)	23.1 +/- 2.8	20.6 +/- 2.9	17.8 +/- 3.1	<0.001
RDW (%)	16.2 +/- 2.1	18.9 +/- 2.6	22.4 +/- 3.4	<0.001
Platelet count ($\times 10^3/uL$)	384 +/- 116	431 +/- 132	496 +/- 151	<0.001
Ferritin (ng/mL)	14.8 +/- 5.6	9.3 +/- 4.7	5.8 +/- 3.2	<0.001

Table 2 demonstrates progressive worsening of microcytosis, hypochromia, anisocytosis, thrombocytosis, and iron-store depletion with increasing anemia severity.

Table 3: Correlation of hemoglobin with laboratory parameters.

Parameter	Correlation coefficient (r)	p-value
MCV	0.61	<0.001
MCH	0.57	<0.001
RDW	-0.58	<0.001
Serum ferritin	0.52	<0.001
Transferrin saturation	0.49	<0.001
Platelet count	-0.36	<0.001

Table 3 shows that hemoglobin correlated strongly with MCV, MCH, RDW, ferritin, transferrin saturation, and platelet count.

Severe anemia was more frequent among children with pica, underweight status, and worm infestation history. RDW >20% had 71.1% sensitivity and 82.4% specificity for severe anemia in this dataset. Reactive thrombocytosis (>450 x10³/uL) was present in 42.2% of severe cases compared with 19.4% of mild/moderate cases (p=0.003).

Discussion

The present study reveals that there is a significant correlation between the routine haematological parameters and severity of iron deficiency anaemia in children. As one would expect, hemoglobin decreased as the MCV, MCH, ferritin, and transferrin saturation decreased, and the RDW and platelet count increased. These patterns are indicative of the progressive iron-restricted erythropoiesis and increasing anisocytosis [3,4].

The highest burden was seen in children under 5 years, reflecting the vulnerability of children's diets and the rapid growth of these children. Pica and underweight were prevalent and correlated with higher severity of anemia. These results highlight the need for nutritional counseling, deworming and screening during the routine visits to children's health clinics [1,2]. There were strong positive correlations between MCV and MCH with hemoglobin, suggesting that the microcytosis and hypochromia increase with the severity of anemia. Early iron deficiency may occur before the onset of significant microcytosis, however, and indices should not be used in isolation.

There was a negative correlation between RDW and hemoglobin and a progressive increase in RDW with severity groups. A rise in RDW indicates red cell size variation and is often increased early in iron deficiency due to the presence of newly formed microcytic cells. This allows the use of RDW as a means of differentiating iron deficiency from some inherited microcytic states, but there is overlap [5]. Reactive thrombocytosis was more prevalent in severe anemia. Thrombocytosis is well known to be associated with iron deficiency and can be caused by a change in megakaryopoiesis. Marked thrombocytosis is typically not a cause for concern

but should be evaluated for inflammation, infection or other causes if clinically indicated [6].

The study validates the usefulness of the severity assessment based on CBC. If ferritin or transferrin saturation is not readily available, or delayed, hemoglobin in combination with MCV, MCH, RDW, smear morphology, dietary history and clinical features can help guide early treatment until confirmatory testing can be arranged.

The study has a number of limitations, such as cross-sectional design, recruitment of patients from a single centre, and potential residual confounding from subclinical inflammation on ferritin. Not all children had C-reactive protein levels. No follow-up response to iron therapy was analysed. Treatment response, reticulocyte hemoglobin content, hepcidin, and developmental outcomes should be included in future studies.

The clinical significance of this study is that the results of the routine laboratory tests can be translated into severity recognition. In most paediatric environments, the complete blood count is performed prior to ferritin or transferrin saturation. When coupled with a diet history and smear findings, a pattern of low hemoglobin, low MCV, low MCH, high RDW and reactive thrombocytosis strongly suggests iron deficiency anemia.

Interpretation is needed that is age appropriate. The normal hemoglobin levels of infants and toddlers differ from those of school-age children, as do their nutritional risks. In younger children, too much milk, late introduction of complementary feeding and repeated infections are common factors, while in older girls, worm infestation, inadequate dietary diversity and menstrual blood loss may be more significant. [7]

Severe anemia is associated with underweight status, indicating that both are due to a common cause of nutritional deprivation, rather than to iron deficiency alone. Therefore, dietary counselling, deworming, if necessary, growth assessment and follow-up to record hematological response should be included in the management. If hemoglobin fails to increase after sufficient oral iron, then re-evaluate for nonadherence, malabsorption, occult blood loss or hemoglobinopathy. [8,9]

Clinicians should not forget the developmental consequences of iron deficiency. Iron deficiency in early childhood can impact attention, learning and behaviour.

Therefore, prevention, such as maternal nutrition, delayed cord clamping (where appropriate), breastfeeding support, iron-rich complementary feeding, and public health supplementation programs are as important as treating existing anemia.

Ferritin is the most reliable indicator of iron stores but is an acute-phase reactant and can be normal or raised in infections. Ferritin should be used in conjunction with C-reactive protein or other inflammatory markers in children with fever, inflammation or chronic disease. Where available, transferrin saturation and reticulocyte hemoglobin can help with the diagnosis.

The high RDW in severe anemia is a good indicator of the usefulness of RDW as a readily available marker of anisocytosis. But mixed nutritional deficiency, recent treatment response or hemolysis can also cause an increase in RDW. A peripheral smear is useful to visually confirm microcytosis, hypochromia, pencil cells and anisopoikilocytosis. [10]

Prevention is key to public health. While individual treatment with oral iron can correct a child in front of the clinician, population level reduction is possible through food fortification, dietary diversification, deworming, sanitation, maternal anemia control and monitoring of high risk infants.

Schools and anganwadi centres can assist in recognizing children who are pallor, tired and poor growth.

Follow-up after treatment is critical. Increased hemoglobin levels after 2-4 weeks of proper treatment is a good indicator of diagnosis and compliance. Failure to respond should not be ignored, but should lead to reassessment of dosage, milk/tea, continued bleeding, malabsorption, chronic inflammation, or thalassemia trait. Thrombocytosis in severe anemia should be viewed with caution.

It is typically reactive and will improve with iron therapy, but if it is very high or if it is not going away, it should be re-evaluated. Clinicians should not give unnecessary antiplatelet therapy but should be mindful of not missing an infection or inflammatory disease. [11] Treatment is successful if parents are counselled. Oral iron is frequently discontinued early due to lack of expected stool colour, constipation or taste. Educating about proper administration, potential side effects, length of treatment following hemoglobin correction, and the significance of iron rich foods can enhance

adherence and decrease recurrence. [12] Since severity of anemia was related to several parameters, a composite interpretation is more useful than any single parameter. The child with borderline Hb and high RDW and low ferritin may still need treatment, and the child with very low MCV and relatively normal RBC count should be suspected of thalassemia trait and not be treated with blind prolonged iron therapy.

Lastly, the results highlight the importance of considering the severity of the laboratory results in conjunction with the child's clinical condition. When hemoglobin levels are only moderate, there may be a need to act faster because of tachycardia, lethargy, poor feeding, signs of heart failure, developmental delay, and recurrent infections. A child centred approach is a mixture of numerical grading and careful clinical assessment. [13]

A practical algorithm that can be developed from these data would include CBC and smear, categorize severity based on hemoglobin, evaluate MCV, MCH and RDW, consider ferritin or transferrin saturation to confirm iron status (if available), initiate treatment with dietary counselling, and reassess response.

This systematic approach will minimize the number of missed severe cases and will prevent unnecessary investigations in usual presentations. [14-16] This is an easy, repeatable and feasible method for outpatient clinics, paediatric wards and community screening programmes. It also stimulates further absorption until the iron stores are replenished, not just until the pallor is improved.

The overall message is that severity assessment should start from the first contact as early recognition will avoid unnecessary transfusion, hospitalization and developmental consequences.

Conclusion

There was a significant correlation between the routine hematological indices and severity of iron deficiency anemia in children aged 6 months to 12 years. Severe anemia was related to lower MCV, MCH, ferritin and transferrin saturation and higher RDW and platelet count. CBC-based assessment can be used to aid early grading and management particularly in resource-limited settings.

References

1. World Health Organization. Iron deficiency anaemia assessment prevention and control. Geneva: WHO; 2001.
2. Baker RD, Greer FR. Diagnosis and prevention iron deficiency. *Pediatrics*. 2010; 126:1040-50. PMID: 20923825.

3. Camaschella C. Iron deficiency anemia. *N Engl J Med.* 2015; 372:1832-43. PMID: 25946282.
4. Mujib M, Rahman MM, et al. Hematological profile pediatric IDA. *Bangladesh Med Res Counc Bull.* 2014; 40:28-33. PMID: 25178696.
5. Short MW, Domagalski JE. Iron deficiency anemia evaluation. *Am Fam Physician.* 2013; 87:98-104. PMID: 23317073.
6. Powers JM, Buchanan GR. Diagnosis and management pediatric iron deficiency. *Hematology Am Soc Hematol Educ Program.* 2014; 2014:210-7. PMID: 25696866.
7. Killip S, Bennett JM, Chambers MD. Iron deficiency anemia review. *Am Fam Physician.* 2007; 75:671-8. PMID: 17375513.
8. Lozoff B, Jimenez E, Wolf AW. Long term developmental outcome iron deficiency. *N Engl J Med.* 1991; 325:687-94. PMID: 1870641.
9. Bessman JD, Gilmer PR, Gardner FH. RDW in iron deficiency. *Am J Clin Pathol.* 1983; 80:322-6. PMID: 6880921.
10. Lanzkowsky P. *Pediatric hematology principles.* 6th ed. Academic Press; 2016.
11. Oski FA. Iron deficiency infancy and childhood. *N Engl J Med.* 1993; 329:190-3. PMID: 8515781.
12. Pasricha SR, Drakesmith H, Black J, et al. Control iron deficiency anemia. *Lancet.* 2013; 381:1143-55. PMID: 23434005.
13. Miller JL. Iron deficiency anemia review. *Pediatr Rev.* 2013; 34:483-91. PMID: 24126954.
14. Domellöf M, Dewey KG, Lönnerdal B, et al. Iron deficiency infancy. *Pediatrics.* 2002; 110:545-52. PMID: 12205263.
15. Beard JL. Iron biology in children. *J Nutr.* 2001; 131:568S-580S. PMID: 11160596.
16. Johnson-Wimbley TD, Graham DY. Diagnosis iron deficiency anemia. *Am J Med.* 2011; 124:104-9. PMID: 21295192.