e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(2); 556-562

Original Research Article

Clinical Profile and Hematological Parameters in Patients with Iron Deficiency Anemia

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Received: 12-12-2025 / Revised: 13-01-2026 / Accepted: 15-02-2026

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Conflict of interest: Nil

Abstract:

Background: Iron deficiency anemia (IDA) is the most prevalent nutritional deficiency disorder globally, affecting a disproportionately large segment of populations in developing countries. Despite its widespread recognition, comprehensive characterization of the clinical profile and hematological parameters across varying severities of IDA remains inadequately explored in many regional populations. This study aimed to evaluate the clinical presentations and hematological parameters in patients diagnosed with iron deficiency anemia and to assess their correlation with disease severity.

Methods: A hospital-based cross-sectional observational study was conducted at a tertiary care teaching hospital. A total of 320 patients diagnosed with IDA based on standard hematological and iron study criteria were enrolled. Demographic data, clinical symptoms, and comprehensive hematological parameters including complete blood count, peripheral blood smear morphology, serum iron, serum ferritin, total iron-binding capacity (TIBC), and transferrin saturation were systematically evaluated. Patients were categorized into mild, moderate, and severe anemia groups according to World Health Organization (WHO) criteria. Statistical analysis included descriptive statistics, chi-square tests, ANOVA, and Pearson correlation coefficients.

Results: The mean age of participants was 34.6 ± 12.8 years, with a female predominance (72.5%). The most common clinical presentations were generalized fatigue (89.4%), pallor (82.2%), and exertional dyspnea (54.1%). The mean hemoglobin was 8.2 ± 2.1 g/dL, mean corpuscular volume (MCV) was 68.4 ± 8.7 fL, mean serum ferritin was 6.8 ± 4.2 ng/mL, and mean TIBC was 428.6 ± 62.3 µg/dL. Significant progressive deterioration in hematological indices was observed across mild, moderate, and severe groups ($p < 0.001$). Serum ferritin demonstrated the strongest negative correlation with disease severity ($r = -0.72$, $p < 0.001$). Microcytic hypochromic morphology on peripheral smear was present in 74.1% of patients.

Conclusion: Iron deficiency anemia presents with a characteristic constellation of clinical symptoms and hematological abnormalities that progressively worsen with increasing disease severity. Serum ferritin remains the most reliable single marker for assessing iron depletion severity, while a comprehensive evaluation integrating clinical assessment with multiple hematological parameters is essential for accurate diagnosis and appropriate therapeutic stratification.

Keywords: Iron Deficiency Anemia; Hematological Parameters; Serum Ferritin; Clinical Profile; Microcytic Anemia; Red Blood Cell Indices; Nutritional Anemia.

DOI: 10.25258/ijcpr.18.2.87

Introduction

Iron deficiency anemia represents the most common nutritional deficiency disorder worldwide, affecting approximately 1.24 billion individuals globally and contributing significantly to the global burden of disease, particularly in low- and middle-income countries [1]. The World Health Organization estimates that anemia affects roughly 42% of children below 5 years of age, 40% of pregnant women, and 33% of non-pregnant women of reproductive age, with iron deficiency accounting for approximately 50% of all anemia cases [2]. The public health ramifications of IDA extend far beyond the hematological system, encompassing impaired cognitive development in children, reduced work productivity in adults, increased susceptibility to infections, adverse pregnancy outcomes, and elevated cardiovascular morbidity [3].

Iron is an essential micronutrient that serves as a critical component of hemoglobin, myoglobin, and numerous enzymatic systems involved in cellular respiration, DNA synthesis, and immune function [4]. Iron deficiency develops through a continuum of stages: initially, storage iron depletion (reflected by declining serum ferritin levels) occurs without functional consequences, followed by iron-deficient erythropoiesis (characterized by reduced transferrin saturation and elevated TIBC), and ultimately frank iron deficiency anemia with diminished hemoglobin synthesis and the hallmark morphological changes of microcytosis and hypochromia [5]. Understanding this progressive pathophysiology is essential for early detection and timely intervention.

The etiology of IDA is multifactorial and varies considerably across populations. In developing countries, inadequate dietary iron intake, poor bioavailability due to phytate- and tannin-rich diets, chronic parasitic infections (particularly hookworm), and repeated pregnancies with inadequate supplementation are the predominant causative factors [6]. In developed nations, chronic blood loss from gastrointestinal pathology (peptic ulcer disease, colorectal malignancy, inflammatory bowel disease), menorrhagia, malabsorption syndromes (celiac disease, atrophic gastritis), and the widespread use of proton pump inhibitors and non-steroidal anti-inflammatory drugs contribute significantly to the disease burden [7].

The clinical manifestations of IDA are protean and often insidious in onset, ranging from mild fatigue and decreased exercise tolerance to severe symptoms including pica, restless leg syndrome, koilonychia, angular stomatitis, glossitis, and Plummer-Vinson syndrome in advanced cases [8]. The diagnosis of IDA relies upon a combination of clinical assessment and laboratory investigations, with serum ferritin widely regarded as the most specific biochemical marker for iron store depletion [9]. However, ferritin is an acute-phase reactant, and its interpretation may be confounded by concurrent inflammation, infection, liver disease, or malignancy, necessitating a comprehensive panel of iron studies for accurate diagnosis [10].

Recent epidemiological studies have highlighted persistent gaps in the understanding of IDA across different demographic groups. A large Indian population-based study by Khanduri et al. (2022) revealed that hematological parameters varied substantially across geographic regions and dietary patterns, suggesting the need for population-specific reference data [11]. Similarly, Pasricha et al. (2021) emphasized that many cases of IDA in developing countries remain undiagnosed or inadequately treated due to limited access to comprehensive laboratory testing and a reliance on hemoglobin alone for screening [12]. Furthermore, the correlation between specific hematological red cell indices and iron study parameters across varying severities of IDA has not been comprehensively evaluated in many clinical settings [13].

The aim of this study was to characterize the clinical profile, demographic features, and hematological parameters of patients presenting with iron deficiency anemia at a tertiary care hospital, and to assess the correlation between hematological indices and disease severity to identify the most reliable diagnostic parameters for clinical practice.

Materials and Methods

Study Design and Setting: This was a hospital-based cross-sectional observational study conducted in the Departments of Internal Medicine and Pathology at a tertiary care university teaching hospital.

Sample Size: Based on a previous study reporting a prevalence of microcytic hypochromic blood picture of approximately 70% among IDA patients, with a 5% margin of error and 95% confidence level, a minimum sample size of 292 patients was estimated. To account for potential incomplete data and dropout, 320 patients were enrolled.

Study Population and Eligibility Criteria

Inclusion Criteria: (1) Age ≥18 years; (2) Hemoglobin level below the WHO-defined threshold for anemia (male <13.0 g/dL; female <12.0 g/dL); (3) Laboratory confirmation of iron deficiency defined by at least two of the following: serum ferritin <15 ng/mL, transferrin saturation <16%, TIBC >400 µg/dL, or serum iron <60 µg/dL; and (4) Willingness to provide informed consent.

Exclusion Criteria: (1) Anemia attributable to chronic disease, hemolytic disorders, thalassemia trait (confirmed by hemoglobin electrophoresis), sideroblastic anemia, or combined nutritional deficiencies (concurrent vitamin B12 or folate deficiency); (2) Patients with active infection, sepsis, or acute inflammatory conditions (C-reactive protein >10 mg/L) at the time of enrollment, due to the confounding effect on ferritin levels; (3) Patients receiving iron supplementation therapy for more than two weeks prior to enrollment; (4) Chronic kidney disease (eGFR <30 mL/min/1.73 m^2); (5) Known hematological malignancies; (6) Pregnancy; and (7) Recent blood transfusion within the preceding four weeks.

Data Collection: A structured proforma was utilized to systematically collect data on demographic characteristics (age, sex, socioeconomic status, occupation, dietary habits), clinical history (duration and nature of symptoms, menstrual history in females, gastrointestinal symptoms, dietary iron intake), and findings on physical examination (pallor, koilonychia, glossitis,

angular stomatitis, tachycardia, pedal edema, hepatosplenomegaly).

Laboratory Investigations: Venous blood samples (8 mL) were collected from each participant following an overnight fast. The following investigations were performed:

- **Complete blood count (CBC):** Performed on an automated hematology analyzer (Sysmex XN-1000). Parameters recorded included hemoglobin (Hb), red blood cell count (RBC), hematocrit (Hct), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), red cell distribution width (RDW), white blood cell count (WBC), and platelet count.
- **Peripheral blood smear (PBS):** Prepared using Leishman staining and examined by an experienced hematopathologist for red cell morphology (microcytosis, hypochromia, anisocytosis, poikilocytosis, target cells, pencil cells).
- **Iron studies:** Serum iron and TIBC were measured by colorimetric assay on a Roche Cobas c501 analyzer. Serum ferritin was measured by electrochemiluminescence immunoassay (ECLIA) on a Roche Cobas e601 analyzer. Transferrin saturation was calculated as (serum iron / TIBC) × 100%.

Severity Classification: Anemia severity was classified according to WHO criteria: mild anemia (Hb 11.0–12.9 g/dL for males, 11.0–11.9 g/dL for females), moderate anemia (Hb 8.0–10.9 g/dL), and severe anemia (Hb <8.0 g/dL).

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using SPSS version 26.0 (IBM Corporation, Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation and compared using one-way ANOVA with post hoc Tukey's test for multiple group comparisons. Categorical variables were expressed as frequencies and percentages and compared using chi-square tests. Pearson correlation coefficients were calculated to assess the relationship between hematological parameters and anemia severity. A p-value <0.05 was considered statistically significant.

Results

Demographic and Clinical Characteristics: A total of 320 patients with confirmed IDA were enrolled. The mean age was 34.6 ± 12.8 years (range: 18–72 years), with a marked female predominance (n = 232, 72.5%). The largest age group was 21–40 years (52.5%). Among female participants, menorrhagia was identified as the most common etiological factor (38.8%), followed by inadequate dietary intake (26.3%). The majority of patients (62.8%) belonged to lower socioeconomic strata, and 58.4% reported predominantly vegetarian dietary habits. Regarding severity classification, 68 patients (21.3%) had mild anemia, 148 (46.3%) had moderate anemia, and 104 (32.5%) had severe anemia. Clinical findings and symptom frequencies are presented in Table 1. Generalized fatigue was the most prevalent symptom (89.4%), followed by pallor (82.2%), exertional dyspnea (54.1%), palpitations (41.6%), and dizziness (36.9%). Among specific iron deficiency signs, koilonychia was observed in 12.8%, glossitis in 18.4%, angular stomatitis in 15.3%, and pica in 8.4% of patients.

Table 1: Clinical Profile and Symptom Distribution of Study Participants (N = 320)

Variable	n (%) or Mean ± SD
Age (years), mean ± SD	34.6 ± 12.8
Female sex, n (%)	232 (72.5)
Vegetarian diet, n (%)	187 (58.4)
Lower socioeconomic status, n (%)	201 (62.8)
Severity classification	
Mild anemia, n (%)	68 (21.3)
Moderate anemia, n (%)	148 (46.3)
Severe anemia, n (%)	104 (32.5)
Symptoms	
Generalized fatigue, n (%)	286 (89.4)
Pallor, n (%)	263 (82.2)
Exertional dyspnea, n (%)	173 (54.1)
Palpitations, n (%)	133 (41.6)
Dizziness/lightheadedness, n (%)	118 (36.9)
Headache, n (%)	96 (30.0)
Anorexia, n (%)	84 (26.3)
Irritability/poor concentration, n (%)	72 (22.5)
Physical findings	
Koilonychia, n (%)	41 (12.8)
Glossitis, n (%)	59 (18.4)

Angular stomatitis, n (%)	49 (15.3)
Pica, n (%)	27 (8.4)
Tachycardia (>100/min), n (%)	86 (26.9)
Pedal edema, n (%)	34 (10.6)
Hepatomegaly, n (%)	12 (3.8)
Splenomegaly, n (%)	8 (2.5)

Hematological Parameters Across Severity Groups: Hematological and iron study parameters stratified by anemia severity are presented in Table 2. Significant progressive deterioration was observed across all red cell indices and iron parameters from mild to severe groups. The mean hemoglobin levels were 11.4 ± 0.6 g/dL (mild), 9.2 ± 0.8 g/dL (moderate), and 5.8 ± 1.3 g/dL (severe) ($p < 0.001$). MCV decreased progressively from 76.8 ± 5.2 fL in the mild group to 67.4 ± 6.8 fL in the moderate group and 60.2 ± 8.4 fL in the severe group ($p < 0.001$). Similarly, MCH and MCHC demonstrated significant stepwise reductions. RDW increased significantly with worsening severity (14.8 ± 1.6% vs. 17.2 ± 2.3% vs. 21.4 ± 3.1%, $p < 0.001$). Serum ferritin declined from 11.2 ± 2.8 ng/mL in the mild group to 6.4 ± 3.1 ng/mL in the moderate group and 3.6 ± 2.4 ng/mL in the severe group ($p < 0.001$). TIBC increased progressively, and transferrin saturation decreased significantly across severity groups. Platelet count showed a trend toward reactive thrombocytosis in the severe group (342.6 ± 98.4 × 10^3/µL vs. 276.8 ± 72.1 × 10^3/µL in the mild group, $p < 0.001$).

Table 2: Hematological and Iron Study Parameters by Anemia Severity

Parameter	Mild (n = 68)	Moderate (n = 148)	Severe (n = 104)	p-value
Hemoglobin (g/dL)	11.4 ± 0.6	9.2 ± 0.8	5.8 ± 1.3	<0.001
RBC count (×10^6/µL)	4.3 ± 0.4	3.8 ± 0.5	3.1 ± 0.6	<0.001
Hematocrit (%)	35.2 ± 2.4	29.6 ± 3.1	21.4 ± 4.2	<0.001
MCV (fL)	76.8 ± 5.2	67.4 ± 6.8	60.2 ± 8.4	<0.001
MCH (pg)	25.8 ± 2.1	22.3 ± 2.6	18.6 ± 3.2	<0.001
MCHC (g/dL)	32.4 ± 1.3	30.1 ± 1.6	27.2 ± 2.4	<0.001
RDW (%)	14.8 ± 1.6	17.2 ± 2.3	21.4 ± 3.1	<0.001
Platelet count (×10^3/µL)	276.8 ± 72.1	308.4 ± 84.6	342.6 ± 98.4	<0.001
Serum iron (µg/dL)	48.2 ± 12.6	34.6 ± 10.8	22.4 ± 9.2	<0.001
Serum ferritin (ng/mL)	11.2 ± 2.8	6.4 ± 3.1	3.6 ± 2.4	<0.001
TIBC (µg/dL)	386.4 ± 42.8	432.6 ± 54.2	468.3 ± 68.7	<0.001
Transferrin saturation (%)	12.6 ± 3.4	8.2 ± 2.8	4.9 ± 2.1	<0.001

Peripheral Blood Smear Morphology and Correlation Analysis: Peripheral blood smear findings and correlation analysis are summarized in Table 3. Microcytic hypochromic morphology was the most common pattern, observed in 237 patients (74.1%), followed by dimorphic picture (12.5%), normocytic hypochromic pattern (8.4%), and normocytic normochromic picture (5.0%). The prevalence of microcytic hypochromic morphology increased significantly with disease severity: 47.1% in mild, 75.7% in moderate, and 89.4% in severe anemia ($p < 0.001$). Anisocytosis was documented in 68.4% and poikilocytosis in 42.5% of patients, with pencil cells and target cells observed in 28.4% and 18.1% of patients, respectively, predominantly in the severe group.

Pearson correlation analysis demonstrated significant correlations between anemia severity (assessed by hemoglobin level) and all iron study parameters. Serum ferritin showed the strongest positive correlation with hemoglobin ($r = 0.72$, $p < 0.001$), while RDW demonstrated the strongest negative correlation ($r = -0.68$, $p < 0.001$). MCV ($r = 0.64$, $p < 0.001$), MCH ($r = 0.61$, $p < 0.001$), and transferrin saturation ($r = 0.58$, $p < 0.001$) were also significantly correlated with hemoglobin. TIBC showed a significant negative correlation with hemoglobin ($r = -0.54$, $p < 0.001$).

Table 3: Peripheral Blood Smear Morphology and Correlation of Hematological Parameters with Hemoglobin

PBS Morphology	Mild (n = 68)	Moderate (n = 148)	Severe (n = 104)	Total (%)	p-value
Microcytic hypochromic	32 (47.1)	112 (75.7)	93 (89.4)	237 (74.1)	<0.001
Normocytic hypochromic	14 (20.6)	11 (7.4)	2 (1.9)	27 (8.4)	<0.001
Normocytic normochromic	12 (17.6)	4 (2.7)	0 (0.0)	16 (5.0)	<0.001
Dimorphic picture	10 (14.7)	21 (14.2)	9 (8.7)	40 (12.5)	0.368
Anisocytosis	28 (41.2)	102 (68.9)	89 (85.6)	219 (68.4)	<0.001
Poikilocytosis	12 (17.6)	56 (37.8)	68 (65.4)	136 (42.5)	<0.001
Pencil cells	4 (5.9)	34 (23.0)	53 (51.0)	91 (28.4)	<0.001
Target cells	3 (4.4)	22 (14.9)	33 (31.7)	58 (18.1)	<0.001
Correlation with Hb	**r**	**p-value**			
MCV	0.64	<0.001			
MCH	0.61	<0.001			
MCHC	0.56	<0.001			
RDW	−0.68	<0.001			
Serum ferritin	0.72	<0.001			
Serum iron	0.59	<0.001			
TIBC	−0.54	<0.001			
Transferrin saturation	0.58	<0.001			

Discussion

This cross-sectional study provides a comprehensive characterization of the clinical profile and hematological parameters in 320 patients with confirmed iron deficiency anemia, demonstrating a consistent pattern of progressive clinical and laboratory deterioration across increasing severity grades. The findings confirm the established pathophysiological model of iron depletion and its sequential impact on erythropoiesis, while highlighting the diagnostic utility of individual hematological parameters in clinical practice.

The marked female predominance (72.5%) observed in our study is consistent with global epidemiological data. Kassebaum et al. (2014), in the Global Burden of Disease Study, reported that women of reproductive age bear a disproportionate burden of IDA, primarily attributable to menstrual blood loss, increased iron demands during pregnancy and lactation, and relatively lower dietary iron intake compared to males [14]. Similarly, the peak incidence in the 21–40 year age group and the strong association with lower socioeconomic status observed in our cohort mirrors findings from numerous population-based studies in South and Southeast Asia [15].

The clinical symptom profile observed in our study demonstrates the characteristic pattern of IDA, with fatigue (89.4%) and pallor (82.2%) being the most prevalent presenting complaints. These findings align with the observations of Camaschella (2015), who described a progressive constellation of symptoms beginning with nonspecific fatigue and diminished exercise tolerance, progressing to specific iron deficiency manifestations such as koilonychia, glossitis, and pica as stores become severely depleted [16]. The relatively low prevalence of specific iron deficiency signs such as koilonychia (12.8%) and pica (8.4%) likely reflects the fact that these findings typically manifest only in advanced, prolonged deficiency states and may be underreported due to patient unfamiliarity with these symptoms [17].

The progressive decline in red blood cell indices (MCV, MCH, MCHC) and serum iron parameters across severity groups confirms the well-established pathophysiological continuum of iron depletion. As iron stores become progressively exhausted, the supply of iron to erythroid precursors diminishes, resulting in impaired hemoglobin synthesis and the production of smaller, more poorly hemoglobinized red cells [18]. The concurrent progressive elevation of RDW from 14.8% in the mild group to 21.4% in the severe group reflects the increasing heterogeneity of the red cell population as iron-replete cells are gradually replaced by microcytic, iron-deficient cells—a phenomenon termed anisocytosis that serves as an early and sensitive indicator of evolving iron deficiency [19].

The finding that serum ferritin demonstrated the strongest correlation with hemoglobin level (r = 0.72) among all iron study parameters reinforces its position as the single most useful laboratory test for the assessment of iron stores. This observation is supported by the meta-analysis of Stable markers of iron status conducted by Garcia-Casal et al. (2018), who confirmed the superior diagnostic accuracy of serum ferritin for detecting iron deficiency compared to other individual parameters [20]. However, the well-known limitation of ferritin as an acute-phase reactant must be recognized, and in clinical scenarios involving concurrent

inflammation, additional markers such as soluble transferrin receptor or the ferritin index may provide incremental diagnostic value [21].

The observation of reactive thrombocytosis in the severe anemia group (mean platelet count 342.6 ± 98.4 × 10^3/μL) is a recognized phenomenon in IDA. The mechanism involves the structural homology between thrombopoietin and erythropoietin, whereby elevated erythropoietin levels in severe anemia may cross-stimulate megakaryopoiesis, resulting in increased platelet production [22]. This finding has clinical significance, as it may mimic myeloproliferative disorders and lead to unnecessary hematological investigations if the underlying IDA is not recognized.

The predominance of microcytic hypochromic morphology on peripheral blood smear (74.1% overall, increasing to 89.4% in severe IDA) underscores the continued diagnostic value of morphological examination in an era of automated hematology analyzers. However, the presence of normocytic normochromic (5.0%) and dimorphic (12.5%) patterns highlights that IDA does not always present with the classic morphological picture, particularly in early disease stages or when complicated by concurrent nutritional deficiencies or chronic disease [23]. This morphological heterogeneity emphasizes the necessity of integrating clinical, hematological, and biochemical data for definitive diagnosis.

Our findings regarding the association between vegetarian dietary habits and IDA (58.4% of patients reported predominantly vegetarian diets) are noteworthy and consistent with established nutritional science. Non-heme iron, which constitutes the predominant form in plant-based diets, has substantially lower bioavailability (2–20%) compared to heme iron from animal sources (15–35%), and its absorption is further inhibited by dietary phytates, tannins, and calcium [24]. This association carries important implications for dietary counseling and public health strategies aimed at iron deficiency prevention.

This study has several limitations warranting acknowledgment. First, the cross-sectional design precludes establishment of temporal relationships and does not permit evaluation of treatment outcomes. Second, the hospital-based sampling may introduce selection bias toward more symptomatic or severe cases, potentially limiting generalizability to the broader community. Third, soluble transferrin receptor levels and hepcidin measurements, which represent more recently validated biomarkers of iron status, were not assessed due to resource constraints. Fourth, detailed dietary iron intake quantification using validated food frequency questionnaires was not performed. Finally, the exclusion of patients with concurrent inflammation, while necessary to ensure accurate ferritin interpretation, may limit the applicability of findings to populations with high infectious disease burdens where the distinction between IDA and anemia of chronic disease is clinically challenging [25].

Conclusion

This study provides a comprehensive characterization of the clinical profile and hematological parameters in patients with iron deficiency anemia across a spectrum of disease severity. The findings confirm that IDA presents with a progressive pattern of clinical symptomatology and laboratory abnormalities, with generalized fatigue, pallor, and exertional dyspnea being the most prevalent clinical manifestations, and microcytic hypochromic morphology representing the predominant peripheral smear pattern. All hematological indices and iron study parameters demonstrated significant progressive deterioration from mild to severe anemia, with serum ferritin emerging as the single most reliable marker correlating with disease severity. Red cell distribution width serves as a valuable and readily available early indicator of evolving iron deficiency. The high prevalence of IDA among young women of lower socioeconomic status with vegetarian dietary habits underscores the critical need for targeted nutritional education, dietary diversification strategies, and proactive screening programs. A comprehensive diagnostic approach integrating clinical assessment, automated red cell indices, peripheral smear morphology, and iron biochemistry is recommended for accurate diagnosis and severity stratification to guide individualized therapeutic interventions.

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