DOI: https://doi.org/10.54393/pjhs.v7i5.3865

PAKISTAN JOURNAL OF HEALTH SCIENCES
(LAHORE)
https://thejas.com.pk/index.php/pjhs
ISSN (E): 2790-9352, (P): 2790-9344
Volume 7, Issue 05 (May 2026)

Original Article

OPEN ACCESS

Frequency of Iron Deficiency Anemia in Children Presenting with Febrile Seizures at Hayatabad Medical Complex, Peshawar: A Cross-Sectional Study

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ARTICLE INFO

Keywords:
Febrile Seizures, Iron Deficiency Anemia, Children, Ferritin, Transferrin Saturation

How to Cite:
Shah, M. U., Saleem, M., Hussain, A., Ahmad, A., Ali, W., & Khan, J. (2026). Frequency of Iron Deficiency Anemia in Children Presenting with Febrile Seizures at Hayatabad Medical Complex, Peshawar: A Cross-Sectional Study: Frequency of Iron Deficiency Anemia in Children Presenting with Febrile Seizures . Pakistan Journal of Health Sciences, 7(5), 98-103. https://doi.org/10.54393/pjhs.v7i5.3865

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Received Date: 21st January, 2026
1st Revision Received: 8th February, 2026
2nd Revision Received: 25th February, 2026
Acceptance Date: 9th March, 2026
Published Date: 31st May, 2026

ABSTRACT

Febrile seizures are the most common seizure disorder in early childhood. Iron deficiency anemia has been proposed as a potential risk factor due to iron's role in neuronal metabolism and neurotransmitter synthesis; however, evidence remains inconsistent, particularly in populations with a high burden of infection. **Objective:** To determine the frequency of iron deficiency anemia among children presenting with febrile seizures. **Methods:** This cross-sectional study was conducted in the Department of Pediatrics, Hayatabad Medical Complex, over six months. A total of 103 children aged 6–60 months presenting with simple febrile seizures were enrolled through consecutive sampling. Demographic and clinical data were recorded. Laboratory evaluation included complete blood count, serum ferritin, serum iron, total iron-binding capacity, transferrin saturation, and CRP. Anemia was defined as hemoglobin <11 g/dL, and iron deficiency anemia as anemia with serum ferritin <15 ng/mL. Data were analyzed using SPSS version 26.0. **Results:** The mean age was 35.47 ± 15.78 months. Anemia was present in 39 (37.9%) children, while iron deficiency anemia was identified in 6 (5.8%). Children with IDA had significantly lower hemoglobin and serum ferritin levels compared to non-IDA children (p=0.001). No significant associations were observed between IDA and demographic or clinical variables. **Conclusions:** Anemia was common among children with febrile seizures, whereas iron deficiency anemia was infrequent when defined strictly by low ferritin. Given the influence of inflammation on ferritin levels, reliance on ferritin alone may underestimate iron deficiency. Incorporating transferrin saturation and inflammatory markers may improve diagnostic accuracy.

INTRODUCTION

Febrile seizures are the most common seizure disorder of early childhood, affecting approximately 2–5% of children worldwide and typically occurring between 6 and 60 months of age [1]. They are associated with fever in the absence of central nervous system infection or underlying epilepsy [2]. Although generally benign, their recurrence and potential impact on neurodevelopment have prompted continued investigation into modifiable risk factors [3]. Iron deficiency anemia is the most prevalent nutritional deficiency globally, particularly in low- and middle-income countries [4]. Iron plays a critical role in neuronal energy metabolism, myelination, and neurotransmitter synthesis [3]. Deficiency may lower seizure threshold by altering dopaminergic and gamma-aminobutyric acid (GABA) pathways, providing a plausible biological link between iron deficiency and febrile seizures [5]. Several regional and international studies have reported higher frequencies of iron deficiency or iron deficiency anemia among children

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with febrile seizures compared to controls [6]. However, reported prevalence varies widely, largely due to differences in diagnostic criteria and laboratory thresholds [7]. Serum ferritin, the most commonly used marker of iron stores, is an acute-phase reactant and may be elevated during febrile or infectious illnesses, potentially masking underlying iron deficiency [8].

In Pakistan, data on iron deficiency anemia among children with febrile seizures are limited and inconsistent, despite a high burden of childhood anemia and infectious diseases. Local evidence incorporating inflammatory markers is therefore, essential to improve diagnostic accuracy and guide clinical screening. This study aimed to determine the frequency of iron deficiency anemia among children presenting with febrile seizures at a tertiary-care hospital in Peshawar.

METHODS

The study was a cross-sectional study carried out in the Department of Pediatrics, Hayatabad Medical Complex (HMC) in Peshawar, a teaching, a tertiary-care teaching hospital that serves a large pediatric population from both urban and peri-urban areas of Peshawar. A single-center design was adopted to ensure uniformity of clinical assessment, laboratory procedures, and data collection protocols [9], as well as feasibility within the defined study period. The study was conducted over six months, from 4 July 2024 to 4 January 2025. Consecutive enrollment of eligible children presenting to the Department of Pediatric Emergency or Pediatric Ward with febrile seizures was undertaken. Ethical approval for the study was obtained from the Research and Ethics Committee of the College of Physicians and Surgeons Pakistan (CPSP), Islamabad (Reference No. CPSP/REU/PED-2022-021-7261). The IRB ref no was 1823. Written informed consent was obtained from parents or legal guardians prior to enrollment. All study procedures were conducted in accordance with the principles outlined in the Declaration of Helsinki [10]. The sample size was calculated using the World Health Organization (WHO) sample size calculator [11] for prevalence studies, based on the formula: $n = Z^2 \times p(1 - p) / d^2$, assuming an expected prevalence of iron deficiency anemia of 30% [12], a 95% confidence level ($Z = 1.96$), and an absolute precision of 9%. This yielded a minimum required sample size of 100 participants. To compensate for potential incomplete laboratory data, an additional 3% was added, resulting in a final target sample size of 103 children, which was successfully achieved. A non-probability consecutive sampling technique was employed, whereby all eligible children presenting during the study period were approached for inclusion. Children aged 6-60 months presenting with simple febrile seizures, defined as generalized tonic-clonic seizures associated with fever (≥38°C), lasting less than 15 minutes, and not recurring within 24 hours, were included. Children with a prior diagnosis of epilepsy or other neurological disorders were excluded. Exclusion criteria comprised complex febrile seizures, known epilepsy, neurodevelopmental delay, central nervous system infections, metabolic disorders, chronic systemic illnesses, hemoglobinopathies, iron supplementation within the preceding three months, recent blood transfusion, acute blood loss, or incomplete laboratory data. Data were collected using a structured proforma specifically developed for this study [7], which included demographic variables (age, gender, weight), clinical variables (duration of fever, seizure duration, first or recurrent episode, family history of febrile seizures, and source of infection), and laboratory parameters. The proforma was pretested on 10 children before formal data collection to assess clarity, completeness, and feasibility, and minor modifications were made accordingly. Anthropometric measurements were obtained using standardized techniques, and nutritional status was assessed using weight-for-age criteria. Venous blood samples were collected under aseptic conditions for complete blood count, serum ferritin, serum iron, total iron-binding capacity (TIBC), transferrin saturation (TSAT), and C-reactive protein (CRP). All laboratory analyses were performed using calibrated automated analyzers at the central pathology laboratory of Hayatabad Medical Complex, with internal quality control measures in place. Upon presentation, children were stabilized according to standard pediatric protocols. Demographic and clinical data were recorded following consent. Body temperature was measured using a digital thermometer, and seizure duration was documented based on caregiver history and clinical records. Blood samples were obtained within 24 hours of admission to minimize temporal variability in iron indices related to acute illness. Anemia was defined according to WHO criteria as hemoglobin <11 g/dL. Iron deficiency anemia was defined as the presence of anemia in combination with serum ferritin <15 ng/mL. Given that serum ferritin is an acute-phase reactant, transferrin saturation was additionally measured to aid interpretation of iron status in the presence of inflammation, and CRP was used to assess inflammatory activity [13]. Anemia severity was categorized as mild, moderate, or severe based on standard hemoglobin thresholds [14].

Data were entered and analyzed using the Statistical Package for the Social Sciences (SPSS), version 26.0. Normality of continuous variables was assessed using the Shapiro–Wilk test. Variables with normal distribution were expressed as mean ± standard deviation, while non-normally distributed variables were summarized using median and interquartile range. Categorical variables were presented as frequencies and percentages. Associations

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between iron deficiency anemia and categorical variables were analyzed using the Chi-square test, with Fisher's Exact Test applied when expected cell counts were less than five. Comparisons of hematological and iron parameters between iron deficiency anemia and non-iron deficiency anemia groups were performed using the Mann–Whitney U test due to non-normal data distribution. A p-value <0.005 was considered statistically significant.

RESULTS

A total of 103 children aged 6–60 months presenting with simple febrile seizures were included. The mean age was 35.47 ± 15.78 months, with a female predominance (53.4%), giving a male-to-female ratio of 0.87:1. Most children belonged to the 25–60 months age group (71.8%). The mean body weight was 12.54 ± 3.01 kg, and more than half of the participants (54.4%) were classified as underweight based on weight-for-age criteria. Additionally, the mean temperature at presentation was 39.26 ± 0.46°C. Fever duration of ≥24 hours before seizure onset was observed in 80.6% of children. Seizure duration most commonly ranged between 5–10 minutes (46.6%), followed by 10–15 minutes (34.0%). Recurrent febrile seizures were reported in 51.5% of children, and a positive family history of febrile seizures was present in 50.5%. Upper respiratory tract infection and otitis media were the most frequent sources of infection (Table 1).

Table 1: Demographic Characteristics and Fever and Seizure Profile of Children with Febrile Seizures (n=103)

Variables	Category / Summary	n (%) / Mean ± SD
Demographic Characteristics		
Age (Months)	Mean ± SD	35.47 ± 15.78
	6–12 Months	11 (10.7%)
	13–24 Months	18 (17.5%)
	25–60 Months	74 (71.8%)
Gender	Male	48 (46.6%)
	Female	55 (53.4%)
Weight (kg)	Mean ± SD	12.54 ± 3.01
Nutritional Status	Underweight	56 (54.4%)
	Normal	47 (45.6%)
Fever and Seizure Profile		
Temperature at Presentation (°C)	Mean ± SD	39.12 ± 0.68
Duration of Fever	<24 Hours	20 (19.4%)
	≥24 Hours	83 (80.6%)
Seizure Duration (Minutes)	<5	20 (19.4%)
	5–10	48 (46.6%)
	10–15	35 (34.0%)
First Episode	Yes	50 (48.5%)
	No (recurrent)	53 (51.5%)
Family History of Febrile Seizures	Yes	52 (50.5%)
	No	51 (49.5%)
Source of Infection	URTI	18 (17.5%)
	Gastroenteritis	14 (13.6%)
	Otitis media	18 (17.5%)
	Pneumonia	17 (16.5%)
	UTI	16 (15.5%)
	Others	20 (19.4%)

The mean hemoglobin concentration was 11.32 ± 1.36 g/dL. Mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) were 73.50 ± 4.87 fL and 24.11 ± 2.37 pg, respectively, while red cell distribution width (RDW) was elevated (16.68 ± 2.03%). The mean serum ferritin level was 24.79 ± 11.28 ng/mL, the mean serum iron was 56.16 ± 16.90 µg/dL, and the mean transferrin saturation was 14.46 ± 4.84%. Raised C-reactive protein (CRP) levels were observed in 46.6% of children, indicating a high inflammatory burden in this febrile cohort. Given that serum ferritin is an acute-phase reactant, serum ferritin levels were compared between children with normal and raised CRP levels using the Mann–Whitney U test. Children with elevated CRP had slightly higher mean ranks for ferritin than those with normal CRP; however, this difference was not statistically significant (U = 1242.0, Z = −0.516, p=0.606), indicating that inflammation did not significantly influence ferritin levels in this cohort (Table 2).

Table 2: Hematological and Iron Parameters of Children with Febrile Seizures and Effect of Inflammation on Serum Ferritin Levels (n=103)

Parameters	Mean ± SD	Reference / Cut-off	n	Mean Rank	Mann–Whitney U	Z-value	p-value
Hematological and Iron Parameters							
Hemoglobin (g/dL)	11.32 ± 1.36	Anemia: <11	–	–	–	–	–
MCV (fL)	73.50 ± 4.87	Low if <75	–	–	–	–	–
MCH (pg)	24.11 ± 2.37	Low if <24	–	–	–	–	–
RDW (%)	16.68 ± 2.03	High if >14.5	–	–	–	–	–
Serum ferritin (ng/mL)	24.79 ± 11.28	Low if <15	–	–	–	–	–
Serum iron (µg/dL)	56.16 ± 16.90	Low if <50	–	–	–	–	–
TIBC (µg/dL)	395.40 ± 47.45	High if >360	–	–	–	–	–
Transferrin saturation (%)	14.46 ± 4.84	Low if <16	–	–	–	–	–
Effect of Inflammation on Serum Ferritin Levels							
CRP Status	Normal	55 (53.4%)	55	50.58	1242.0	−0.516	0.606
	Raised	48 (46.6%)	48	53.63			

DOI: https://doi.org/10.54393/pjhs.v7i5.3865

Anemia (hemoglobin <11 g/dL) was identified in 39 children (37.9%). Among these, 18 (17.5%) had mild anemia, and 21 (20.4%) had moderate anemia, while no cases of severe anemia were observed. Using the study's predefined criteria (anemia with serum ferritin <15 ng/mL), iron deficiency anemia was diagnosed in 6 children (5.8%) (Table 3).

Table 3: Frequency of Anemia and Iron Deficiency Anemia (n=103)

Variables	Category	n (%)
Anemia (WHO Cut-Off)	Yes	39 (37.9%)
	No	64 (62.1%)
Severity of Anemia	Mild	18 (17.5%)
	Moderate	21 (20.4%)
	Severe	0 (0.0%)
Iron Deficiency Anemia (IDA)	Yes	6 (5.8%)
	No	97 (94.2%)

The association between iron deficiency anemia and selected demographic and clinical variables was assessed. No statistically significant associations were found with age group, gender, nutritional status, seizure recurrence, family history of febrile seizures, or fever duration (all p>0.005). However, only six children fulfilled the criteria for iron deficiency anemia, resulting in very low statistical power for subgroup comparisons. Therefore, the absence of statistically significant associations should be interpreted cautiously and may reflect insufficient sample size rather than a true lack of relationship (Table 4).

Table 4: Association of Iron Deficiency Anemia with Selected Demographic and Clinical Factors (n=103)

Variables	Category	IDA Present, n (%)	IDA Absent, n (%)	χ^2 value	p-value
Age Group	6–12 months	1 (9.1%)	10 (90.9%)	1.444	0.486
	13–24 months	0 (0.0%)	18 (100.0%)		
	25–60 months	5 (6.8%)	69 (93.2%)		
Gender	Male	3 (6.3%)	45 (93.8%)	0.030	0.863
	Female	3 (5.5%)	52 (94.5%)		
Nutritional Status	Underweight	3 (5.4%)	53 (94.6%)	0.049	0.825
	Normal	3 (6.4%)	44 (93.6%)		
Seizure Episode	First episode	3 (6.0%)	47 (94.0%)	0.005	0.941
	Recurrent	3 (5.7%)	50 (94.3%)		
Family History	Yes	3 (5.8%)	49 (94.2%)	0.001	0.980
	No	3 (5.9%)	48 (94.1%)		
Fever Duration	<24 hours	2 (10.0%)	18 (90.0%)	0.789	0.375
	≥24 hours	4 (4.8%)	79 (95.2%)		

Chi-square test was applied; Fisher's Exact Test was used where expected cell counts were <5. Due to the small number of children with iron deficiency anemia (n = 6), subgroup comparisons are underpowered, and results should be interpreted cautiously. A p-value <0.005 was considered statistically significant

Normality testing using the Shapiro–Wilk test demonstrated that hemoglobin, MCV, MCH, RDW, serum ferritin, serum iron, TIBC, and transferrin saturation were not normally distributed (p<0.005); therefore, non-parametric tests were applied. Children with iron deficiency anemia had significantly lower hemoglobin and serum ferritin levels compared to non-IDA children (both p=0.001). No statistically significant differences were observed for MCV, MCH, RDW, serum iron, TIBC, or transferrin saturation (Table 5).

Table 5: Comparison of Hematological and Iron Parameters Between IDA and Non-IDA Groups (n=103)

Parameters	IDA Present (n=6) Median (IQR)	IDA Absent (n=97) Median (IQR)	Mann–Whitney U	p-value
Hemoglobin (g/dL)	9.40 (0.78)	11.60 (1.80)	58.5	0.001
MCV (fL)	76.25 (8.88)	73.20 (8.20)	214.5	0.281
MCH (pg)	24.15 (4.70)	24.00 (4.20)	279.0	0.866
RDW (%)	18.75 (3.25)	16.70 (3.85)	189.5	0.153
Serum Ferritin (ng/mL)	11.40 (5.95)	25.30 (18.80)	65.0	0.001
Serum Iron (µg/dL)	64.15 (31.63)	56.10 (28.80)	253.0	0.593
TIBC (µg/dL)	398.40 (90.95)	395.90 (84.70)	271.5	0.784
Transferrin Saturation (%)	16.90 (10.93)	14.10 (7.70)	263.5	0.699

Mann–Whitney U (non-parametric). Values are presented as Median (IQR); p<0.005 is considered significant

DISCUSSION

Febrile seizures remain the most common seizure disorder in early childhood, and the potential contribution of iron deficiency to seizure susceptibility continues to generate debate. In the present study, anemia was identified in 37.9% of children presenting with simple febrile seizures, whereas iron deficiency anemia (IDA), defined strictly as hemoglobin <11 g/dL with serum ferritin <15 ng/mL, was observed in only 5.8% of cases. These findings suggest that while anemia is common in febrile seizure patients, only a small proportion meet stringent biochemical criteria for IDA when ferritin-based definitions are applied during acute illness. Several recent international studies have reported higher frequencies of iron deficiency or IDA among children with febrile seizures than observed in our cohort. A systematic review and meta-analysis by Sulviani *et al.* concluded that iron deficiency and anemia were significantly associated with increased susceptibility to febrile seizures [15]. Similarly, Bakkannavar *et al.* reported consistent associations across multiple datasets, highlighting iron deficiency as a modifiable risk factor [14]. Case-control studies from India and Bangladesh have also demonstrated higher IDA prevalence among febrile seizure cases compared with controls [6, 16]. In contrast to these reports, our observed IDA frequency (5.8%) is substantially lower. Within Pakistan, Awais *et al.* and Nargis *et al.* documented higher proportions of iron deficiency anemia among children with febrile seizures [17, 18]. Hussain *et al.* [12]. Similarly, a stronger association between IDA and febrile seizures in a case-control design. The discrepancy between our findings and these regional reports may be

DOI: https://doi.org/10.54393/pjhs.v7i5.3865

explained by differences in study design and diagnostic criteria. Many prior studies relied primarily on hemoglobin levels or ferritin alone without incorporating inflammatory markers. In our study, CRP was measured to evaluate inflammatory burden, and ferritin interpretation was considered in this context. Given that 46.6% of children had elevated CRP, ferritin values may have been influenced by acute-phase responses, potentially masking iron deficiency at the individual level. Ferritin is a well-recognized acute-phase reactant, and its elevation during infection can obscure underlying iron deficiency, as described by Lee [5] and Weiss *et al.* [13]. Although ferritin levels did not differ significantly between CRP strata in our cohort (p=0.606), this does not exclude misclassification at the individual level. Acute inflammation may maintain ferritin within normal range despite depleted iron stores. Therefore, reliance on ferritin alone may underestimate the true prevalence of iron deficiency in febrile children. This study's finding of a mean transferrin saturation (TSAT) of 14.46%, below commonly accepted normal thresholds, further supports the possibility of functional or early iron deficiency not captured by strict ferritin criteria. The high prevalence of underweight children (54.4%) in our cohort also reflects underlying nutritional vulnerability. However, the coexistence of undernutrition and low ferritin-defined IDA suggests that anemia in this population may be multifactorial, including contributions from inflammation, recurrent infections, and other micronutrient deficiencies. Camaschella emphasizes that iron deficiency exists along a biological continuum, and early or functional deficiency may precede overt ferritin decline. This may partly explain why overall anemia was common (37.9%) while strict IDA frequency remained low [19]. Importantly, no statistically significant associations were observed between IDA and age, gender, nutritional status, seizure recurrence, or fever duration. However, only six children fulfilled IDA criteria, substantially limiting statistical power. The absence of association should therefore be interpreted cautiously. Studies with larger IDA subgroups, such as those by Mandal *et al.* and Jadhav *et al.* were able to detect stronger relationships, likely due to greater case numbers and comparative control groups [6, 8]. An important clinical implication arises from the study by Sharawat et al. which demonstrated reduced recurrence of febrile seizures with prophylactic iron supplementation [20]. Even if strict ferritin-defined IDA is infrequent, early identification and correction of iron deficiency may still influence seizure recurrence risk. Therefore, broader diagnostic strategies incorporating TSAT and inflammation-adjusted ferritin thresholds may provide a more clinically relevant assessment. Overall, our findings contribute to the ongoing debate by suggesting that strict ferritin-based definitions during acute febrile illness may underestimate iron deficiency prevalence. The variability across studies likely reflects differences in biomarker selection, timing of sampling, inflammatory burden, and regional nutritional patterns.

This study has several limitations. First, it was conducted at a single tertiary-care center, which may limit generalizability to other populations with different socioeconomic and nutritional profiles. Second, the cross-sectional design precludes causal inference between iron deficiency and febrile seizures. Third, the small number of IDA cases (n=6) reduced statistical power for subgroup analyses. Fourth, iron indices were measured during acute febrile illness, when inflammatory responses may influence ferritin interpretation despite CRP assessment. Finally, advanced biomarkers such as soluble transferrin receptor or post-recovery reassessment of iron status were not available. Future multicenter studies with larger sample sizes should employ composite definitions of iron deficiency incorporating ferritin, transferrin saturation, and inflammation-adjusted thresholds. Longitudinal designs evaluating iron status after recovery from acute illness would improve diagnostic accuracy. Randomized trials assessing whether iron supplementation reduces recurrence of febrile seizures in iron-deficient children are also warranted. Incorporating soluble transferrin receptor measurement may further refine differentiation between anemia of inflammation and true iron deficiency in febrile populations.

CONCLUSIONS

Anemia was common among children presenting with febrile seizures; however, iron deficiency anemia was infrequent when defined strictly by low ferritin in the presence of anemia. Despite the low prevalence of IDA, affected children demonstrated significantly lower hemoglobin and ferritin levels. Given that ferritin is an acute-phase reactant and may be elevated during infection, reliance on ferritin alone may underestimate true iron deficiency. Incorporating inflammation-adjusted diagnostic criteria and transferrin saturation into routine assessment may improve the detection of iron deficiency in febrile seizure patients. Further studies using standardized and composite definitions of iron deficiency are warranted to clarify its true prevalence and clinical relevance in this population.

Authors' Contribution

Conceptualization: MUS
Methodology: MUS, JK
Formal analysis: MS, WA
Writing and Drafting: MUS, MS, AH, AA, WA, JK
Review and Editing: MUS, MS, AH, AA, WA, JK

All authors approved the final manuscript and take responsibility for the integrity of the work

DOI: https://doi.org/10.54393/pjhs.v7i5.3865

Conflicts of Interest

All the authors declare no conflict of interest.

Source of Funding

The author received no financial support for the research, authorship and/or publication of this article.

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