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FREQUENCY OF IRON DEFICIENCY ANEMIA IN CHILDREN WITH FEBRILE FITS PRESENTING AT QAZI HUSSAIN AHMAD MEDICAL COMPLEX NOWSHERA

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Abstract

Background: To determine the frequency of iron deficiency anemia (IDA) in children aged 6 to 60 months presenting with febrile fits at Qazi Hussain Ahmad Medical Complex, Nowshera. Methods: This cross-sectional study was conducted at the Department of Pediatrics from February 6, 2025, to May 22, 2025. A total of 124 children of either gender, aged 6–60 months, presenting with febrile fits were enrolled using consecutive non-probability sampling. Iron deficiency anemia was defined as hemoglobin < 11 g/dL and serum ferritin < 30 ng/mL, accompanied by clinical symptoms. Data were analyzed using IBM SPSS version 21. Chi-square and Fisher's exact tests were applied, with $p \leq 0.05$ considered significant. Results: The mean age was 29.1 ± 15.2 months; 59.7% were aged 6–24 months. Male children constituted 54.8%. Overall frequency of IDA in children with febrile fits was 16.9% (21/124). IDA was significantly higher in children aged 6–24 months compared to those aged 25–60 months (23.0% vs. 8.0%; p=0.023), among lower socioeconomic status (20.7% vs. 6.3%; p=0.031), and in children of illiterate mothers (21.0% vs. 9.3%; p=0.048). No significant associations were found with gender, family history of fits, or area of residence. Conclusion: Iron deficiency anemia was found in approximately one in six children presenting with febrile fits. Younger age (6–24 months), lower socioeconomic status, and maternal illiteracy were significant associated factors. Routine screening for IDA in children with febrile seizures is recommended.

INTRODUCTION

Febrile seizures represent the most prevalent form of seizure in children, typically occurring alongside a fever. Children between the ages of six months and five years are typically affected, with the peak incidence occurring around 18 months. While febrile seizures can be quite distressing for parents and careers, it is important to note that most of these seizures are harmless and resolve on their own, typically not resulting in any long-term health problems or neurological damage. [1, 2] A febrile seizure is an abrupt, involuntary muscle contraction triggered by a swift rise in body temperature, typically resulting from a viral or bacterial infection. The exact cause of febrile seizures remains somewhat unclear; however, they are thought to arise from a mix of genetic and environmental influences. [3, 4] Distinguishing febrile seizures from other seizure types, like epilepsy, is crucial, as they generally do not reoccur after the fever has subsided. Febrile seizures fall into two categories: simple and complex. [3, 4] In 30% of children, there was a positive family history of febrile convulsions, and these convulsions occurred at an earlier age in this group. In 35% of cases, febrile convulsions were classified as complex, and among these, 29% of the children had a positive family history of convulsions. [5] Iron deficiency anaemia is a common condition in children that arises when the body lacks sufficient iron to generate the hemoglobin necessary for oxygen transport in the blood. This deficiency is recognised as the most prevalent nutritional issue among children globally, carrying substantial consequences for their growth, development, and overall well-being. [6] Iron is a vital mineral that significantly contributes to numerous bodily functions, especially in the formation of hemoglobin, the protein found in red blood cells that attaches to oxygen and transports it to tissues and organs. [7] The causes of iron deficiency anaemia in children are varied, typically arising from a mix of insufficient dietary intake, heightened iron needs, and challenges in iron absorption. Iron deficiency is especially common in young children due to their rapid growth and elevated iron needs, which rise during infancy and early childhood. [8, 9] According to a study, the frequency of iron deficiency anaemia in children with febrile fits was 17%. [10] Iron deficiency anemia in children is a common and significant health issue, particularly in developing regions, and its association with febrile fits is an area of growing concern. Due to the scarcity of literature on this subject locally, the goal of this study is to find out the frequency of iron deficiency anaemia in children with febrile fits at our health facility. Findings from this research could provide critical insights to our clinicians into whether addressing iron deficiency could serve as an adjunctive treatment for febrile seizures, contributing to better health outcomes and quality of life for affected children. This study aimed to determine the frequency of iron deficiency anaemia in children with febrile fits presenting at Qazi Hussain Ahmad medical complex Nowshera.

METHODS

This cross-sectional study was conducted at the Department of Pediatrics, Qazi Hussain Ahmad Medical Complex, Nowshera, over a period of three and a half months from February 6, 2025, to May 22, 2025, following the approval of the synopsis from the College of Physicians and Surgeons Pakistan (CPSP) Ref No: CPSP/REU/PED-2023-305-7584 . AND Qazi Hussain Medical Complex Nowshera IRB Approval No. 12/ERB/NMC, dated 01-01-2025.The sample size was calculated using the WHO sample size calculator, assuming an expected frequency of iron deficiency anemia in children with febrile fits of 17%, as reported in the literature, with an absolute precision of 8% and a confidence level of 95%. The calculated sample size was 124. Consecutive non-probability sampling was employed to enroll participants. Children of either gender, aged between 06 and 60 months, presenting with febrile fits as defined in the operational definition were included in the study. Febrile fits were operationally defined as generalized tonic-clonic seizures lasting less than 5 minutes, without focal neurological symptoms, and without a history of previous afebrile seizures. Children with central nervous system infections, asthma, cardiac, renal, or liver disease, and those with hypoglycemia were excluded from the study.

The study commenced after obtaining formal approval from the hospital's ethical review board and the research unit of the CPSP Head office. All parents or guardians of eligible children were thoroughly informed about the aims, benefits, and the voluntary nature of the study, with assurance that participation posed no risk to their children. Written informed consent was obtained from each guardian prior to enrollment. Demographic and clinical information, including age, gender, weight, socioeconomic status, maternal education, maternal employment, area of residence, family history of fits, and infancy nutrition (exclusive breastfeeding, exclusive formula feeding, or

mixed feeding), was recorded on a predesigned proforma. In every registered child, iron deficiency anemia (IDA) was examined using laboratory techniques. Aseptic technique was used to take 2 mL of venous blood which was tested as hemoglobin (Hb) and serum ferritin levels. The operationally defined iron deficiency anemia was hemoglobin less than 11 g/dL and serum ferritin less than 30 ng/mL with clinical manifestation of fatigue, irritability, pale skin, and poor appetite. All clinical evaluation and laboratory interpretations were done under the direct supervision of a consultant pediatrician who had a minimum of five years of post-fellowship experience. To maintain the accuracy and consistency of the data, the researcher personally gathered all the information. IBM SPSS version 21 was used to analyze the data. The Shapiro-Wilk test was used to test the normality of numerical data (age, weight, serum hemoglobin and serum ferritin). Mean and standard deviation or median with interquartile range (IQR) were used to present numerical variables. Categorical variables, such as gender, iron deficiency anemia, family history of fits, infancy nutrition, socioeconomic status, maternal education, maternal employment, and area of residence were presented in the form of frequency and percentages. Age, weight, gender, family history of fits, infant nutrition, socioeconomic status, maternal education, maternal employment, and area of residence had been stratified to control the occurrence of effect modifiers. The Chi-square test was used to compare groups after stratification, and Fisher exact test was applied where the expected frequencies were below 5. The p-value less than 0.05 was taken to be statistically significant

RESULTS

This study was conducted over a period of six months at the Pediatrics Department of Qazi Hussain Ahmad Medical Complex, Nowshera. A total of 124 children, aged 6 to 60 months, presenting with febrile fits and meeting the inclusion criteria were enrolled. The mean age of the children was 29.1 ± 15.2 months. The majority of children, 74 (59.7%), were between 6 and 24 months of age. There was a slight male predominance, with 68 (54.8%) boys and 56 (45.2%) girls. The mean weight of the children was 11.8 ± 3.4 kg. Analysis of socioeconomic status revealed that most families belonged to a lower socioeconomic background (n=92, 74.2%). Regarding maternal education, 81 (65.3%) mothers were illiterate, while 43 (34.7%) had received some formal education. The majority of mothers were housewives (n=115, 92.7%). A positive family history of febrile fits in first-degree relatives was reported in 32 (25.8%) cases. In terms of infancy nutrition, 46 (37.1%) children were exclusively breastfed, 40 (32.3%) were exclusively formula-fed, and 38 (30.6%) had mixed feeding during the first six months of life. Most children resided in rural areas (n=85, 68.5%). The detailed demographic characteristics are presented in Table 1.

Table 1(a): ***Baseline Demographic (Qualitative variables) and Clinical Characteristics (n=124)***

Characteristic	Category	Frequency (n)	Percentage (%)
Age Group	6 – 24 months	74	59.7
	25 – 60 months	50	40.3
Gender	Male	68	54.8
	Female	56	45.2
Socioeconomic Status	Lower	92	74.2
	Middle	26	21.0
	Upper	6	4.8
Maternal Education	Illiterate	81	65.3
	Literate (Formal Education)	43	34.7
Maternal Employment	Housewife	115	92.7
	Employed	9	7.3
Area of Residence	Rural	85	68.5
	Urban	39	31.5
Infancy Nutrition	Exclusively Breastfed	46	37.1
	Exclusively Formula Fed	40	32.3
	Mixed Feeding	38	30.6
Family History of Fits	Positive	32	25.8
	Negative	92	74.2

Table 1(b): Baseline Demographic (Quantitative variables) and Clinical Characteristics (n=124)

Characteristic	Mean ± Standard Deviation
Age (months)	29.1 ± 15.2
Weight (kg)	11.8 ± 3.4
Hemoglobin (g/dL)	11.0 ± 1.4
Serum Ferritin (ng/mL)	45.3 ± 19.7

In this study, out of 124 children presenting with febrile fits, 21 (16.9%) were found to have iron deficiency anemia (IDA), defined as hemoglobin < 11 g/dL and serum ferritin < 30 ng/mL, accompanied by clinical symptoms such as pallor, fatigue, or poor appetite. The remaining 103 (83.1%) children did not meet the criteria for IDA (Figure 1).

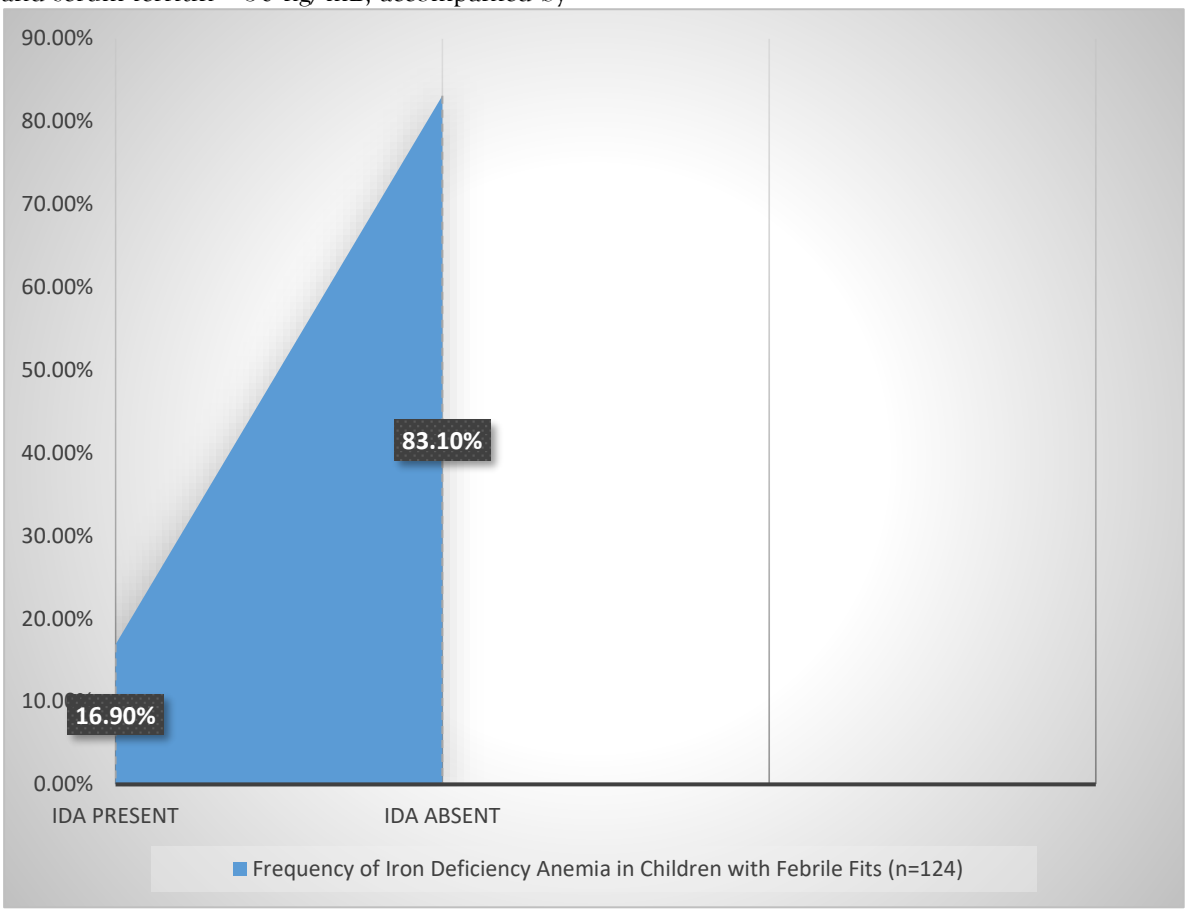

Figure 1: Frequency of Iron Deficiency Anemia in Children with Febrile Fits (n=124)

The frequency of IDA was analyzed across various subgroups to identify potential associations. For the purpose of statistical analysis to compare iron deficiency rates, the "Exclusively Formula Fed" and "Mixed Feeding" categories were combined into a single "Formula/Mixed Fed" group. The Chi-square test was applied for most categorical variables. However, due to a cell count of less than 5 in the "Middle/Upper" socioeconomic group with IDA, the Fisher's Exact test was applied to analyze the association between socioeconomic status and IDA. The results of the stratified analysis are presented in Table 2.

Table 2: ***Stratification of Iron Deficiency Anemia by Demographic and Clinical Variables (n=124)***

Variable	Category	Total (n)	IDA Present (n, %)	IDA Absent (n, %)	P-Value
Age	6 - 24 months	74	17 (23.0%)	57 (77.0%)	0.023
	25 - 60 months	50	4 (8.0%)	46 (92.0%)	
Gender	Male	68	11 (16.2%)	57 (83.8%)	0.813
	Female	56	10 (17.9%)	46 (82.1%)	
Socioeconomic Status	Lower	92	19 (20.7%)	73 (79.3%)	0.031

	Middle/Upper	32	2 (6.3%)	30 (93.8%)	
Maternal Education	Illiterate	81	17 (21.0%)	64 (79.0%)	0.048
	Literate	43	4 (9.3%)	39 (90.7%)	
Infancy Nutrition	Exclusively Breastfed	46	11 (23.9%)	35 (76.1%)	0.118
	Formula/Mixed Fed	78	10 (12.8%)	68 (87.2%)	
Family History of Fits	Positive	32	5 (15.6%)	27 (84.4%)	0.822
	Negative	92	16 (17.4%)	76 (82.6%)	
Area of Residence	Rural	85	16 (18.8%)	69 (81.2%)	0.399
	Urban	39	5 (12.8%)	34 (87.2%)	

A statistically significant association was found between iron deficiency anemia and younger age (6-24 months) (p=0.023), lower socioeconomic status (p=0.031), and illiterate mothers (p=0.048). There was a higher frequency of IDA in exclusively breastfed infants (23.9%) compared to those who were formula or mixed-fed (12.8%), although this association did not reach statistical significance (p=0.118). No significant association was found with gender, family history of fits, or area of residence.

The overall frequency of iron deficiency anemia (IDA) among children presenting with febrile fits was 16.9% (21/124). Stratified analysis demonstrated a statistically significant higher frequency of IDA in children aged 6-24 months compared to those aged 25-60 months (23.0% vs. 8.0%; p=0.023). Similarly, IDA was significantly more prevalent among children from lower socioeconomic backgrounds (20.7% vs. 6.3%; p=0.031) and those with illiterate mothers (21.0% vs. 9.3%; p=0.048). Although exclusively breastfed infants had a higher frequency of IDA (23.9%) compared to those who were formula or mixed-fed (12.8%), this association did not reach statistical significance (p=0.118). No significant associations were observed between IDA and gender, family history of febrile fits, or area of residence.

Discussion

This cross-sectional study enrolled 124 children aged 6 to 60 months presenting with febrile fits. The study found that 16.9% of children with febrile seizures had concurrent iron deficiency anemia, with statistically significant associations observed for younger age (6-24 months), lower socioeconomic status, and illiterate mothers. The frequency of IDA (16.9%) observed in our study aligns closely with the findings of Sarfaraz et al., who reported a 17% frequency of IDA in children with febrile seizures in their Pakistani cohort [10]. This consistency across different Pakistani populations strengthens the evidence that IDA is a relevant comorbidity in approximately one-sixth of children presenting with febrile seizures. However, our findings are considerably lower than those reported in other regional studies. Ahmad et al. from Lahore found a mean hemoglobin of 10.1 ± 1.5 g/dL in children with febrile fits, with their study substantiating IDA as a significant risk factor, though they did not report a specific frequency percentage [11]. More strikingly, Anwar et al. from Quetta reported a 46.5% prevalence of IDA in children with febrile seizures compared to 28% in controls, yielding an odds ratio of 2.23 (95% CI: 1.30–3.82) [12]. This substantially higher frequency may reflect geographic and socioeconomic variations within Pakistan, as Quetta and Balochistan face unique nutritional challenges. Similarly, a study from Ayub Teaching Hospital, Abbottabad, reported that 66.4% of children with febrile seizures had iron deficiency, though their mean hemoglobin (8 ± 1.46 g/dL) and serum ferritin (21 ± 2.87 ng/mL) indicated more severe deficiency states [13]. The variation across Pakistani studies highlights the need for region-specific data and targeted interventions.

The significant association between younger age (6-24 months) and IDA (23.0% vs. 8.0% in older children, p=0.023) observed in our study is biologically plausible and clinically important. This age group corresponds to a period of rapid brain growth and increased iron requirements, as extensively documented in the literature. Lozoff et al. emphasized that the first 1,000 days of life represent a critical window for iron acquisition, during which iron demands are higher than at any other life stage, and deficiency during this period can lead to irreversible neurodevelopmental impairments [14]. Iron plays a vital role in myelination, neurotransmitter synthesis, and hippocampal development, processes that are particularly active in the first two years of life [15]. The vulnerability of this age group to both febrile seizures and iron deficiency creates a potential intersection that clinicians must recognize. Our finding that exclusively breastfed infants had a higher frequency of IDA (23.9%) compared to formula or mixed-fed infants (12.8%), though not reaching statistical significance (p=0.118), warrants discussion. Breast

milk, while optimal for infant nutrition, has relatively low iron content. The World Health Organization recommends iron supplementation for breastfed infants after 6 months of age when iron stores become depleted [16]. In our cohort, 46 (37.1%) infants were exclusively breastfed, and among these, nearly one-quarter had IDA. This suggests that delayed introduction of iron-rich complementary foods may be contributing to deficiency states. Baker et al. emphasized that timely introduction of iron-fortified complementary foods at 4-6 months is crucial for preventing iron deficiency [17]. The lack of statistical significance in our study may reflect the relatively small sample size in subgroup analyses and warrants further investigation with larger cohorts.

The significant association between lower socioeconomic status and IDA (20.7% vs. 6.3% in middle/upper class, p=0.031) aligns with findings from Muttath et al., who identified low socioeconomic status as a significant predictor of recurrent febrile seizures (OR=5.87, 95% CI: 3.32–10.38) [18]. Similarly, maternal illiteracy was significantly associated with IDA in our study (21.0% vs. 9.3% in literate mothers, p=0.048). These associations reflect the complex interplay between social determinants of health and nutritional outcomes. Families with limited resources may struggle to afford iron-rich foods such as meat, poultry, and commercially available iron-fortified cereals. Additionally, maternal education influences feeding practices, healthcare-seeking behaviors, and compliance with preventive recommendations. Khan et al. documented higher rates of malnutrition and micronutrient deficiencies in lower socioeconomic strata and among children of uneducated mothers in their multicenter Pakistani study [19]. The pathophysiological link between iron deficiency and febrile seizures merits discussion. Iron is a cofactor for tyrosine hydroxylase, the rate-limiting enzyme in catecholamine synthesis, and for tryptophan hydroxylase, involved in serotonin production [12]. Disruption of neurotransmitter systems may lower the seizure threshold. Additionally, iron deficiency affects cytochrome c oxidase activity in the brain, potentially impairing energy metabolism in neurons [14]. Animal models have demonstrated that iron-deficient rats exhibit altered hippocampal electrophysiology and increased susceptibility to chemically induced seizures. The observation by McCann and Ames that iron deficiency might paradoxically raise the seizure threshold has not been consistently replicated, and the preponderance of evidence now supports iron deficiency as a risk factor for febrile seizures [20].

Our finding that 16.9% of children with febrile seizures had IDA has important clinical implications. Current WHO guidelines recommend intermittent iron supplementation in populations where anemia prevalence exceeds 20% [16]. In our study population, the overall anemia prevalence (combining IDA and other causes) was likely higher than 20%, suggesting that population-level interventions may be warranted. For individual children presenting with febrile seizures, routine screening for IDA through hemoglobin and ferritin measurements could identify a modifiable risk factor. Treatment of IDA with appropriate iron supplementation (3-6 mg/kg/day of elemental iron) for at least 3 months, continuing for 6-8 weeks after hemoglobin normalization to replenish stores, represents a simple and cost-effective intervention [17].

The long-term consequences of untreated IDA extend beyond seizure risk. Lozoff et al.'s longitudinal studies demonstrated that early-life iron deficiency leads to persistent cognitive, motor, and behavioral deficits that may be irreversible despite subsequent iron repletion [14]. Children with a history of early childhood iron deficiency showed poorer outcomes across multiple neurodevelopmental domains including cognition, motor function, verbal abilities, and behavior [15]. These findings underscore the importance of primary prevention through adequate maternal nutrition during pregnancy, promotion of breastfeeding with timely complementary feeding, and routine screening as recommended by pediatric guidelines [17].

Our study had several limitations. First, the cross-sectional design precludes establishing causality between IDA and febrile seizures; we can only report association. Second, the single-center setting and consecutive sampling may limit generalizability to other populations. Third, we did not assess other micronutrient deficiencies (zinc, magnesium) that might also influence seizure susceptibility. Fourth, we relied on a single ferritin measurement without acute phase reactants, potentially misclassifying some children with infection-related ferritin elevation. Fifth, the sample size, while adequate for estimating overall frequency, limited the power of subgroup analyses, particularly for variables like infancy nutrition where clinically meaningful differences did not reach statistical significance. Sixth, we did not differentiate

between simple and complex febrile seizures, which may have different associations with iron status [21].

The strengths of our study include its prospective design, clear operational definitions, and comprehensive assessment of potential effect modifiers including socioeconomic status, maternal education, and infant feeding practices. The use of both hemoglobin and ferritin measurements with clinical symptom assessment provided a robust definition of IDA. The study duration of 3.5 months (February to May 2025) captured a winter-to-spring transition period when respiratory infections and febrile illnesses are common, making the findings representative of typical clinical practice. Furthermore, our study adds to the limited local literature on this topic and provides baseline data for future interventional studies.

Future research directions should include larger multicenter studies across diverse Pakistani populations to better define regional variations in the IDA-febrile seizure association. Prospective cohort studies following iron-deficient children over time could help establish whether iron supplementation reduces febrile seizure recurrence. Randomized controlled trials of iron supplementation in at-risk populations would provide the highest level of evidence for causality. Additionally, studies examining the dose-response relationship between severity of iron deficiency and seizure characteristics would refine our understanding. The role of other micronutrients and the potential benefits of multiple micronutrient supplementation also warrant investigation [22].

In conclusion, this study demonstrates that iron deficiency anemia is present in approximately 17% of children presenting with febrile seizures in our population, with younger age, lower socioeconomic status, and maternal illiteracy as significant associated factors. These findings highlight the need for routine screening for IDA in children with febrile seizures and targeted nutritional interventions in at-risk populations. Given the potentially modifiable nature of iron deficiency and its implications for both seizure susceptibility and neurodevelopment, addressing this common nutritional deficiency should be a priority in pediatric practice.

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