# Association of simple febrile seizure with iron deficiency anemia in children in a tertiary hospital in Gazipur, Bangladesh

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**Abstract**

**Background:** Simple febrile seizures represent the most frequent type of seizure disorder in early childhood, with multifactorial etiologies including nutritional and metabolic imbalances. Among these, iron deficiency anemia (IDA) has emerged as a possible modifiable risk factor, though evidence remains varied across populations and clinical settings.

**Aim of the study:** To investigate the association between iron deficiency anemia and simple febrile seizures among children presenting to a tertiary hospital in Gazipur, Bangladesh.

**Methods:** A case-control study was conducted over a one-year period, involving 80 children aged 6 months to 6 years. Fourty (40) children with a diagnosis of simple febrile seizure constituted the case group, while 40 age- and sex-matched children with febrile illness but no seizure history served as controls. Comprehensive hematological assessments, including hemoglobin, serum ferritin, mean corpuscular volume (MCV), and serum iron levels were performed. Statistical analyses were conducted using appropriate parametric and non-parametric tests, with a significance level set at $p < 0.05$.

**Result:** Children with simple febrile seizures exhibited significantly lower levels of hemoglobin (mean: 9.5 ± 1.0 g/dL), serum ferritin (10.6 ± 4.3 ng/mL), and MCV (66.0 ± 4.6 fL) compared to controls (11.0 ± 1.1 g/dL, 22.0 ± 5.8 ng/mL, and 77.0 ± 5.1 fL, respectively; $p < 0.001$ for all). The prevalence of iron deficiency anemia was markedly higher among cases than controls (72.5% vs. 25%), with an odds ratio of 7.91 (95% CI: 2.77-22.7).

**Conclusion:** Our study highlights a significant association between iron deficiency anemia and simple febrile seizures in children.

**Keywords:** Simple febrile seizure, iron deficiency anemia, hemoglobin, serum ferritin, case-control study, Bangladesh.

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## Introduction

Simple febrile seizures (SFS) represent the most common neurological disorder in children, characterized by convulsions occurring in the presence of fever in otherwise neurologically healthy children aged between 6 months and 6 years.[1] Globally, SFS are reported to affect approximately 2–5% of children, with the incidence varying across geographic regions and influenced by genetic and environmental factors.[2] While most SFS are benign and self-limiting, their abrupt onset often creates considerable anxiety among caregivers, and in some cases, may be associated with an increased risk of recurrent seizures or the development of epilepsy.[3] Multiple factors, such as genetic susceptibility, age, the severity and duration of fever, and the individual's underlying nutritional status, are thought to contribute to the occurrence of febrile seizures (SFS). Iron deficiency can influence brain function in critical ways, which may increase the likelihood of seizures occurring in response to febrile episodes.[4] Iron, as a critical micronutrient, plays a vital role in neurodevelopment, particularly in processes such as myelination, neurotransmitter synthesis, oxygen transport, and energy metabolism within the central nervous system.[5] The deficiency of iron during early childhood-a crucial period characterized by rapid brain development significantly impair several essential physiological and neurological functions and lower the seizure threshold and increase a child's susceptibility to convulsions, particularly during any febrile illnesses.[6] Several studies conducted in different parts of the world have examined the association between SFS and IDA, with findings suggesting that children with iron deficiency may be more susceptible to experiencing seizures during febrile illnesses compared to those with normal iron levels.[7]

However, the evidence remains inconclusive, as some studies have not demonstrated a statistically significant correlation, leading to an ongoing debate regarding the strength and consistency of this association.[8] In South Asian countries like Bangladesh, where both SFS and IDA remain prevalent public health concerns due to recurrent

childhood infections, limited access to healthcare, and widespread nutritional deficiencies, examining the relationship between these two conditions becomes especially relevant.[9] In the context of Bangladesh, IDA is one of the most common micronutrient deficiencies affecting young children, and febrile illnesses due to seasonal infections or poor sanitation are frequent clinical presentations in pediatric departments.[10] Despite this, very few studies have systematically explored the link between SFS and IDA, and data from local settings remain sparse.[11] Understanding whether IDA serves as an independent risk factor for SFS could have important implications for pediatric health policy, especially regarding early screening and nutritional interventions to prevent seizure occurrences.[12] The aim of this study is to evaluate the potential association between simple febrile seizures and iron deficiency anemia (IDA) in children, focusing on the impact of iron deficiency on the occurrence and severity of SFS.

## Methodology & Materials

This hospital-based case-control study was conducted at the Department of Pediatrics and Neonatology, Tairunnessa Memorial Medical College and Hospital (TMMC & H), a tertiary referral center in Gazipur, Bangladesh. The study spanned from March 2024 to February 2025. Participants were selected using purposive random sampling. Guardians of eligible children were approached, and written informed consent was obtained before enrollment. A total of 80 children were included in the study, with equal numbers in case and control groups.

Children aged 6months to 6 years presenting with fever (≥38°C), with or without seizures, were enrolled from both outpatient and inpatient departments. Participants were categorized into two groups:

- **Cases:** Children presenting with their first episode of febrile seizure, defined according to World Health Organization (WHO) criteria as seizures occurring in febrile children without evidence of intracranial infection, metabolic disturbance, or history of afebrile seizures.[13]
- **Controls:** Age- and sex-matched children presenting with fever (≥38°C) without any seizure activity.

### Inclusion Criteria

- Age between 6 months and 6 years
- Seizure occurring during a febrile episode (≥38°C)
- Generalized tonic-clonic seizures lasting <15 minutes
- No prior history of afebrile seizures
- Normal neurodevelopmental status
- No evidence of central nervous system infection or structural brain abnormalitie

### Exclusion Criteria

- Age <6 months or >6 years
- Focal seizures or seizures lasting >15 minutes
- Recurrent seizures in the same febrile episode
- Known neurological or developmental disorders
- History of epilepsy or previous afebrile seizures
- Evidence of meningitis, encephalitis, or other intracranial infections
- Metabolic or electrolyte imbalances (e.g., hypoglycemia, hyponatremia, hypocalcemia)
- Recent iron supplementation (within the last three months)

Where clinically indicated, additional diagnostic investigations such as lumbar puncture, CT brain, or serum studies (electrolytes, glucose, complete blood count) were performed to exclude alternative diagnoses.

### Data Collection and Assessment of Iron Deficiency Anemia

Children's demographic and background information-including age, sex, place of residence, family history of seizures, and history of iron supplementation-were collected through structured interviews conducted with parents or legal guardians. For participants in the case group, the type and classification of febrile seizure were retrieved from medical records based on clinical documentation at the time of presentation. Body temperature was measured in both the febrile seizure group and the control group (febrile children without convulsion) using a calibrated tympanic thermometer and recorded by attending physicians. Prior to blood sampling, it was confirmed that none of the participants had received iron supplementation in the three days preceding enrollment. A fasting venous blood sample (5 mL) was drawn from each child and sent to the hospital's central laboratory for analysis. Complete blood count (CBC) with differential, mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), serum iron, total iron-binding capacity (TIBC), and serum ferritin levels were assessed. Laboratory analyses were conducted using standardized instruments, including the 18-parameter Sysmex KX-21N hematology analyzer for CBC, the 717 Hitachi auto-analyzer for serum iron and TIBC (using chromatography), and the Genesis-Camna1 gamma counter for ferritin measurement via a radioimmunoassay (RIA) kit. All laboratory testing was performed by a technician blinded to the study objectives and participant group allocation. Interpretation of the laboratory results was conducted by a pediatrician who was also blinded to group assignments to prevent diagnostic bias. Diagnostic thresholds for iron deficiency anemia were established based on age-specific hematologic indices. For children aged 6 to 24 months, IDA was defined as hemoglobin (Hb) <10.5 g/dL, hematocrit (Hct) <33%, MCV <70 fL, MCH <23 pg, MCHC <30 g/dL, and red blood cell count (RBC) <3.7 × $10^6$/mm$^3$. For children aged 2 to 6 years, IDA was defined as Hb <11.5 g/dL, Hct <34%, MCV <75 fL, MCH <24 pg, MCHC <31 g/dL, and RBC <3.9 × $10^6$/mm$^3$. Serum ferritin level <12ng/ml was confirmatory for diagnosing IDA.[14] Peripheral blood smear findings, along with red cell indices (MCV, MCH, MCHC), were used to exclude other potential causes of anemia such as thalassemia or vitamin B12 deficiency.

## Statistical Analysis

Data were analyzed using SPSS version 23, for Windows XP. Descriptive statistics were used to summarize demographic and clinical characteristics. Categorical variables were compared using the Chi-square test or Fisher's exact test, and continuous variables were compared using the independent t-test or Mann–Whitney U test, based on data distribution. Multivariate logistic regression analysis was performed to calculate adjusted odds ratios (OR) with 95% confidence intervals (CI), assessing the independent association between iron deficiency anemia and simple febrile seizures. A *p*-value <0.05 was considered statistically significant.

## Result

The demographic characteristics of the study population (n=80) were divided equally in to two groups. One with simple febrile seizures (n=40) and another with fever without seizure (n=40) (Table I). The mean age was identical between groups at 2.0±0.8 years and 2.0±0.7 years respectively ($p$=0.35), showing no significant difference. Males constituted the majority in both groups-65% in the febrile seizure group and 60% in the control group ($p$=0.57). Birth weight was significantly lower in the seizure group (2.78±0.35 kg) compared to the control group (2.95±0.33 kg) ($p$=0.01). Nutritional status in infancy, birth status, mode of delivery, birth order, and place of residence did not differ significantly between groups, with all *p*-values >0.05. Previous seizure history was more common among cases (10%) compared to controls (2.5%), though not statistically significant ($p$=0.14). A significantly higher proportion of children in the seizure group had a family history of seizures (27.5% vs. 5.0%, $p$=0.002*) and recurrent infections (35% vs. 17.5%, $p$=0.03*). Diazepam was administered exclusively in the seizure group (65%, $p$<0.001). Complete immunization status and fever duration prior to seizure were comparable across groups ($p$=0.38 and $p$=0.42, respectively) (Table II). Children with febrile seizures had significantly lower levels of hemoglobin (9.5±1.0 vs. 11.1±1.1 g/dL), serum ferritin (10.6±4.3 vs. 22 ± 5.8 ng/mL), and serum iron (33.5±10.9 vs. 59.4±12.4 µg/dL), while their total iron binding capacity was notably higher (396±24.2 vs. 338±28.7 µg/dL), with all differences being statistically significant ($p$<0.001). MCV, MCH, MCHC, and RDW were also significantly altered in the seizure group, supporting iron deficiency anemia. Only RBC count showed a modest but significant difference (4.2±0.4 vs. 4.5±0.5 million/µL, $p$=0.02). No significant differences were observed in WBC and platelet counts (Table III). Our study showed that iron deficiency anemia (IDA) was significantly more prevalent among children with febrile seizures (72.5%) compared to controls (25%) with an odds ratio of 7.91 (95% CI: 2.77–22.7; $p$<0.001*) (Table IV). After adjustment, iron deficiency anemia remained a significant independent risk factor for febrile seizures (Adjusted OR: 4.76; 95% CI: 2.03–11.17; $p$<0.001*). Additionally, family history of seizures (Adjusted OR: 3.12; 95% CI: 1.09–8.91; $p$=0.034*) and recurrent infections (Adjusted OR: 2.13; 95% CI: 1.01–4.47; $p$=0.048*) were also identified as significant predictors, (Table V)

**Table I: Demographic characteristics of study population (N= 80)**

| Variables | Simple f ebrile Seizure (n=40) | | Fever without Seizure (n=40) | | *p*-value |
|---|---|---|---|---|---|
| | n | % | n | % | |
| **Age (years)** | | | | | |
| Mean±SD | 2.0 ± 0.8 | | 2.0 ± 0.7 | | 0.35 |
| **Gender** | | | | | |
| Male | 26 | 65 | 24 | 60.00 | 0.57 |
| Female | 14 | 35 | 16 | 40.00 | |
| **Birth Weight (kg)** | | | | | |
| Mean±SD | 2.78 ± 0.35 | | 2.95 ± 0.33 | | 0.01 * |
| **Infancy nutrition** | | | | | |
| Breast milk | 33 | 82.5 | 33 | 82.5 | 0.54 |
| Formula | 2 | 5.00 | 3 | 7.5 | |
| Both | 5 | 12.5 | 4 | 10.00 | |
| **Birth Status** | | | | | |
| Preterm | 6 | 15.00 | 1 | 2.50 | 0.21 |
| Term | 32 | 80.00 | 37 | 92.50 | |
| Post Term | 2 | 5.00 | 2 | 5.00 | |
| **Type of Delivery** | | | | | |
| Natural childbirth | 27 | 67.50 | 30 | 75.00 | 0.61 |
| Cesarean section | 13 | 32.50 | 10 | 25.00 | |
| **Birthday (Child Order)** | | | | | |
| First child | 18 | 45.00 | 17 | 42.50 | 0.52 |
| Second child | 14 | 35.00 | 15 | 37.50 | |
| Third child | 8 | 20.00 | 8 | 20.00 | |
| **Residence Place** | | | | | |
| Urban | 30 | 75.00 | 26 | 65.00 | 0.65 |
| Rural | 10 | 25.00 | 14 | 35.00 | |

**Table II: Clinical characteristics of study population**

| Variable | Simple Febrile Seizure (n=40) | | Fever without Seizure (n=40) | | *p*-value |
|---|---|---|---|---|---|
| | n | % | n | % | |
| **Family history of seizure** | | | | | |
| Yes | 11 | 27.5 | 2 | 5.00 | 0.002* |
| No | 29 | 72.5 | 38 | 95.00 | |
| **History of recurrent infection** | 14 | 35.00 | 7 | 17.50 | 0.03* |
| **Treated with diazepam** | 26 | 65.00 | 0 | 0.00 | 0 |
| **Immunization status complete** | 32 | 80.00 | 32 | 85.71 | 0.38 |
| **Duration of fever before seizure (hours)** | | | | | |
| Mean±SD | | 9.5 ± 2.7 | | 9.8 ± 2.8 | 0.42 |

**Table III: Hematological and Iron Profile Parameters of study population**

| Parameter | Simple febrile Seizure (mean ± SD) | Fever without Seizure(mean ± SD) | *p*-value |
|---|---|---|---|
| Hemoglobin (g/dL) | 9.5 ± 1.0 | 11.1 ± 1.1 | <0.001* |
| Serum Ferritin (ng/mL) | 10.6 ± 4.3 | 22 ± 5.8 | <0.001* |
| Serum Iron (µg/dL) | 33.5 ± 10.9 | 59.4 ± 12.4 | <0.001* |
| Total Iron Binding Capacity (µg/dL) | 396 ± 24.2 | 338 ± 28.7 | <0.001* |
| Hematocrit (%) | 30 ± 3.5 | 33 ± 3.6 | <0.001* |
| MCV (fL) | 66 ± 4.6 | 77 ± 5.1 | <0.001* |
| MCH (pg) | 20.5 ± 2.4 | 25 ± 2.6 | <0.001* |
| MCHC (g/dL) | 29.1 ± 1.2 | 31.5 ± 1.3 | <0.001* |
| RDW (%) | 18 ± 1.7 | 14.8 ± 1.2 | <0.001* |
| RBC (million/µL) | 4.2 ± 0.4 | 4.5 ± 0.5 | 0.02* |
| WBC (/cumm) | 9,200 ± 2,000 | 10,800 ± 1,900 | 0.23 |
| Platelet count (/cumm) | 308,000 ± 64,000 | 290000 ± 61,000 | 0.41 |

**Table IV: Prevalence of iron deficiency anemia among study population**

| IDA Status | Simple febrile Seizure (n=40), n (%) | Fever without Seizure (n=40), n (%) | Odds Ratio (95% CI) | *p*-value |
|---|---|---|---|---|
| IDA Present | 29 (72.50) | 10 (25.00) | 7.91 (2.77–22.7) | <0.001* |
| IDA Absent | 11 (27.50) | 30 (75.00) | | |

**Table V: Logistic Regression Analysis of Factors Associated with Simple febrile Seizure**

| Variable | Adjusted OR (95% CI) | *p*-value |
|---|---|---|
| Iron Deficiency Anemia (yes) | 4.76 (2.03–11.17) | <0.001* |
| Family history of seizures (yes) | 3.12 (1.09–8.91) | <0.034* |
| Recurrent infections (yes) | 2.13 (1.01–4.47) | <0.048* |

## Discussion

Simple febrile seizures(SFS) are the most common convulsive events occurring in young children and iron is particularly essential for maintaining the inhibitory tone of γ-aminobutyric acid (GABA), and its deficiency may lower the seizure threshold in children with febrile illnesses.[5] In our study, iron deficiency anemia (IDA) was significantly more prevalent among children with febrile seizures (72.5%) compared to those with fever but no seizures (25%). Notably, the adjusted odds ratio indicated that children with IDA were approximately 4.76 times more likely to experience febrile seizures ($p < 0.001$), even after controlling for potential confounders. This finding is consistent with a growing body of literature suggesting that iron deficiency may play a contributory role in the pathogenesis of febrile seizures.[15] A case-control study conducted in Bangladesh revealed that iron-deficiency anemia (IDA) was present in 45% of children with simple febrile seizures (SFS), compared to only 10% in the control group, yielding an odds ratio of 7.36. This indicates a significant association between IDA and the occurrence of SFS.[16] Our findings are also consistent with those of Fallah et al. (2009), who reported that children experiencing febrile seizures were more likely to have iron deficiency compared to febrile children without seizures.[17] Similarly, Pisacane et al. (1996) identified a markedly higher prevalence of iron deficiency among children with SFS and proposed a role for iron in modulating neuronal excitability and thermoregulation.[18] Further evidence is provided by Sadeghzadeh et al. (2012), who hypothesized that iron deficiency may reduce the seizure threshold by disrupting neurotransmitter metabolism and enzymatic activity within the central nervous system.[19] Billoo also demonstrated a significantly higher frequency of IDA in children with febrile seizures compared to those with other febrile illnesses.[20] A case-control study conducted in Kenya, along with a meta-analysis encompassing eight such studies, corroborated the association between iron deficiency and an elevated risk of FS in pediatric populations.[21]

In contrast to the findings of our study, several investigations have failed to establish a significant association between iron deficiency anemia (IDA) and febrile seizures (SFS). For instance, Kobrinsky et al. observed a lower prevalence of iron deficiency among children with febrile seizures, suggesting a potential protective role of iron deficiency against FS.[22] Similarly, Bidabadi et al. reported a slightly lower proportion of iron deficiency in the febrile seizure group (44%) compared to the control group (48%); however, the difference was not statistically significant, and thus, the hypothesized protective effect of iron deficiency could not be substantiated.[23] Moreover, iron plays a vital role in brain energy metabolism and neurotransmitter function, especially gamma-aminobutyric acid (GABA) metabolism, which is known to be involved in seizure regulation.[24] Consequently, iron deficiency may lower the seizure threshold by disrupting neurochemical balance, thereby increasing susceptibility to febrile seizures.[11] In our study, we observed significantly lower hemoglobin concentrations and serum iron levels in children with febrile seizures (SFS), while total iron-binding capacity (TIBC) was notably elevated. These hematologic findings are consistent with iron deficiency anemia and support the clinical diagnosis. Additionally, similar laboratory markers have been consistently reported in previous case-control studies investigating the relationship between iron status and SFS.[19] Furthermore, in our study, the mean serum ferritin concentration was significantly lower among children with febrile seizures, underscoring its value as a reliable marker of iron stores. However, it is crucial to recog

nize that ferritin also functions as an acute-phase reactant and may be elevated in the presence of infection or inflammation, potentially complicating its diagnostic interpretation.[22] To address this, we evaluated serum ferritin levels in conjunction with other iron parameters, such as serum iron and total iron-binding capacity, to ensure a more accurate assessment of iron status. A family history of seizures emerged as a significant predictor in our regression model (OR = 3.12, p = 0.034), echoing the findings of several earlier studies that emphasized the importance of genetic predisposition in febrile seizure susceptibility.[19]

## Limitations of the study

This study is hospital-based, case-control design may limit the generalizability of findings to the broader community. Moreover, while matching was performed for age and sex, unmeasured confounding variables such as nutritional status, socioeconomic factors, and underlying infections may have influenced the outcomes. Serum ferritin, although a reliable marker of iron stores, can be elevated during acute illness, potentially leading to misclassification of iron status. Finally, this cross-sectional assessment of iron parameters precludes establishing a temporal or causal relationship between iron deficiency and simple febrile seizures. Future longitudinal studies are needed to validate these findings.

## Conclusion

This study highlights a significant association between simple febrile seizures and iron deficiency anemia in children. Our findings suggest that children with simple febrile seizures are more likely to have lower iron status, including reduced serum ferritin, hemoglobin, and MCV levels, compared to age- and sex-matched controls. This relationship underscores the potential role of iron deficiency in the pathogenesis of simple febrile seizures, particularly in resource-limited settings like Bangladesh.

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