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Original Research Article

Role of Iron Deficiency in Febrile Seizures Among Children Aged 9 Months to 5 Years: A Case–Control Study

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Abstract

Background: Febrile seizures are the most common seizure disorder in children between 9 months and 5 years of age and represent a frequent cause of pediatric emergency visits. Although generally benign, they are associated with considerable parental anxiety and healthcare utilization. Iron deficiency has been proposed as a potentially modifiable risk factor for febrile seizures because of its role in brain development and neurotransmitter metabolism. This study was undertaken to evaluate the demographic characteristics and nutritional status of children with febrile seizures compared with febrile children without seizures as part of an investigation into iron deficiency as a possible risk factor.

Objectives: To compare the demographic characteristics and nutritional status of children with febrile seizures and age- and sex-matched febrile controls without seizures.

Materials and Methods: A hospital-based analytical case–control study was conducted in the Department of Paediatrics, Government Cuddalore Medical College and Hospital, Tamil Nadu. A total of 106 children aged 9 months to 5 years were enrolled, comprising 53 children with febrile seizures (cases) and 53 age- and sex-matched children with febrile illness without seizures (controls). Demographic characteristics and nutritional status were recorded using a structured proforma. Statistical analysis was performed using IBM SPSS version 26.0, with a p-value <0.05 considered statistically significant.

Results: Among the 106 participants, cases and controls each constituted 50% of the study population. The highest proportion of children belonged to the 13–24 months age group (37.7%). The mean age of children with febrile seizures was significantly lower than that of controls (24.8 ± 11.7 months vs. 31.6 ± 14.2 months; p = 0.008). A slight male predominance was observed in both groups, with males accounting for 59.4% of the total study population. Most children had normal weight for age (65.1%); however, undernutrition was more frequent among children with febrile seizures than among controls, with moderate underweight observed in 13.2% of cases compared with 5.7% of controls.

Conclusion: Febrile seizures occurred predominantly among younger children, particularly during the second year of life, and showed a slight male predominance. Undernutrition was more common among children with febrile seizures than among febrile controls.

Keywords: Febrile seizures; Iron deficiency; Children; Case–control study; Nutritional status; Pediatrics.
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Introduction

Febrile seizures are the most common seizure disorder encountered during early childhood, affecting approximately 2–5% of children between 9 months and 5 years of age worldwide.[1] They are defined as seizures occurring in association with fever (temperature ≥38°C) in the absence of central nervous system infection, metabolic disturbances, or a previous history of afebrile seizures.[2] Although febrile seizures are generally benign and self-limiting, they constitute one of the

leading causes of pediatric emergency visits and hospital admissions because of their sudden onset and alarming clinical presentation. The occurrence of febrile seizures generates considerable anxiety among parents and caregivers, often resulting in extensive diagnostic evaluation and increased healthcare utilization.[3]

The pathogenesis of febrile seizures is multifactorial and involves a complex interaction between genetic susceptibility, developmental immaturity of the central nervous system, inflammatory mediators, and environmental factors.[4] Fever acts as the immediate precipitating event by lowering the seizure threshold in susceptible children. However, not every child with fever develops seizures, indicating that additional biological factors may influence neuronal excitability.[5] Identification of such modifiable risk factors is essential because it offers opportunities for early intervention and prevention of seizure recurrence.

Iron is an essential micronutrient that plays a fundamental role in numerous physiological processes, including oxygen transport, cellular respiration, DNA synthesis, myelination, neurotransmitter metabolism, and normal brain development.[6] During infancy and early childhood, iron requirements increase substantially because of rapid somatic growth, expanding blood volume, and accelerated neurodevelopment. Consequently, children aged between 9 months and 5 years are particularly vulnerable to iron deficiency, especially in developing countries where nutritional inadequacy, recurrent infections, and socioeconomic challenges are common.[7] According to the World Health Organization (WHO), iron deficiency remains the most prevalent nutritional deficiency globally and is a leading cause of anemia among children under five years of age.[8]

Beyond its hematological functions, iron has a critical role in maintaining normal neuronal activity. It serves as a cofactor for enzymes involved in the synthesis of neurotransmitters such as dopamine, serotonin, and gamma-aminobutyric acid (GABA), all of which regulate neuronal excitability and synaptic transmission.[9] Iron deficiency may impair myelination, alter energy metabolism, reduce neurotransmitter synthesis, and disrupt normal neuronal signaling, thereby lowering the seizure threshold.[10] Experimental and clinical studies have demonstrated that inadequate iron stores during critical periods of brain development may result in persistent alterations in cognitive performance, behavioral development, and neurophysiological function.[11] These biological mechanisms provide a plausible explanation for the proposed association between iron deficiency and febrile seizures. The relationship between iron deficiency and febrile seizures has been investigated extensively over the past two decades, yet findings remain inconsistent. Several hospital-based case–control studies have reported significantly lower hemoglobin levels, serum ferritin concentrations, and other hematological indices among children with febrile seizures compared with febrile children without seizures.[12–15] A systematic review and meta-analysis further concluded that iron deficiency anemia is associated with an increased risk of febrile seizures, particularly in regions where nutritional deficiencies are highly prevalent.[16] Conversely, a limited number of studies have failed to demonstrate a significant association, suggesting that variations in study design, diagnostic criteria for iron deficiency, inflammatory status, ethnicity, and nutritional patterns may account for these discrepancies.[17] Consequently, the independent contribution of iron deficiency to febrile seizure susceptibility remains an area of ongoing investigation.

India continues to bear a substantial burden of childhood anemia and iron deficiency despite the implementation of national nutritional supplementation programs. According to the National Family Health Survey (NFHS-5), more than two-thirds of Indian children aged 6–59 months are anemic, highlighting the magnitude of this public health problem.[18] Simultaneously, febrile illnesses remain among the most common causes of pediatric hospital admissions. The coexistence of these two conditions raises an important clinical question regarding whether iron deficiency independently predisposes children to febrile seizures. Establishing such an association would have significant implications for routine pediatric practice, as early detection and correction of iron deficiency may represent a simple, safe, and cost-effective strategy to reduce seizure occurrence and recurrence.

Measurement of hematological parameters, including hemoglobin concentration, mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), serum ferritin, serum iron, and total iron-binding capacity (TIBC), provides a comprehensive assessment of iron status in children.[19] Comparison of these parameters between children presenting with febrile seizures and those with febrile illness without seizures can clarify whether iron deficiency functions as an independent risk factor rather than merely an associated finding secondary to acute illness. Furthermore, identifying iron deficiency during hospitalization offers an opportunity for timely nutritional intervention, caregiver counseling, and long-term follow-up.

In view of the high prevalence of both iron deficiency and febrile seizures among young

children, further evidence from well-designed hospital-based studies is warranted. Such studies are particularly relevant in resource-limited settings where nutritional deficiencies are common and preventive healthcare strategies are essential. Therefore, the present hospital-based case–control study was undertaken to evaluate whether iron deficiency is an independent risk factor for febrile seizures among children aged 9 months to 5 years by comparing hematological and biochemical markers of iron status between children with febrile seizures and age-matched febrile controls without seizures. The findings are expected to contribute to the existing evidence base and may support recommendations for routine iron status assessment in children presenting with febrile seizures.

Materials and Methods

Study Design: This hospital-based analytical case–control study was conducted to evaluate whether iron deficiency is an independent risk factor for febrile seizures among children aged 9 months to 5 years. A case–control design was considered appropriate because it enables efficient evaluation of the association between an exposure (iron deficiency) and an outcome (febrile seizures) by comparing children with febrile seizures (cases) to age- and sex-matched children with febrile illness without seizures (controls).

The study adhered to the principles of the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for observational research.

Study Setting: The study was carried out in the Department of Paediatrics, Government Cuddalore Medical College and Hospital, Chidambaram, Cuddalore District, Tamil Nadu, India. This tertiary care teaching hospital provides comprehensive pediatric services to both urban and rural populations and serves as a major referral center for childhood illnesses in the region. Children presenting with febrile illnesses and requiring inpatient management were recruited from the pediatric wards.

Study Duration: The study was conducted over a period of 18 months, during which eligible participants were enrolled consecutively after obtaining informed consent from their parents or legal guardians. The study duration was considered sufficient to achieve the required sample size while minimizing the influence of seasonal variations in the incidence of febrile illnesses and febrile seizures.

Study Population: The study population comprised children aged 9 months to 5 years admitted with febrile illness during the study period. The case group included children diagnosed with febrile seizures according to standard clinical criteria, whereas the control group consisted of age- and sex-matched children admitted with febrile illness but without any current or previous history of seizures. All participants underwent detailed clinical evaluation and laboratory investigations to determine their hematological and iron status.

Inclusion criteria

Cases

- Children aged 9 months to 5 years presenting with at least one episode of febrile seizure.
- Children with fever (≥38°C) without evidence of central nervous system infection or previous afebrile seizures.

Controls

- Children aged 9 months to 5 years admitted with febrile illness without any history of seizures.
- Age- and sex-matched controls recruited during the same study period.

Exclusion Criteria: Children with acute central nervous system infections (meningitis or encephalitis), neurodevelopmental disorders, epilepsy or previous afebrile seizures, seizures secondary to electrolyte imbalance or drug toxicity, anemia due to causes other than iron deficiency, current iron supplementation, chronic systemic illnesses, or a family history of epilepsy were excluded from the study to minimize potential confounding factors.

Sample Size Calculation: The sample size was estimated using data reported by Sankar et al., which demonstrated a mean hemoglobin level of 10.99 ± 1.19 g/dL among children with febrile seizures and 11.36 ± 1.09 g/dL among febrile controls. Assuming a two-sided confidence level of 95%, a study power of 80%, and incorporating a 10% allowance for potential non-response, the minimum calculated sample size was 48 participants per group. After adjustment for possible attrition, the final sample size was rounded to 53 participants in each group, yielding a total study population of 106 children.

Sampling Technique: A consecutive sampling method was employed. All eligible children presenting to the pediatric ward during the study period were screened for inclusion. Consecutive recruitment continued until the predetermined sample size of 53 cases and 53 controls was achieved. Controls were selected during the same recruitment period and were frequency matched for age and sex to minimize selection bias and improve

comparability between the study groups. This sampling approach ensured representative recruitment of the target population while reducing the possibility of systematic selection bias.

Participant Recruitment: Eligible children admitted to the Department of Paediatrics with febrile illness during the study period were screened consecutively for enrolment. After confirming eligibility based on predefined inclusion and exclusion criteria, written informed consent was obtained from parents or legal guardians before recruitment. Children with febrile seizures constituted the case group, while age- and sex-matched children admitted with febrile illness without seizures served as controls. Recruitment was continued until the predetermined sample size was achieved.

Clinical Evaluation: A detailed clinical assessment was performed for all participants using a pre-tested structured case record form. Demographic characteristics, clinical history, duration and characteristics of fever, seizure type (simple or complex), seizure duration, previous history of febrile seizures, family history of seizures, medication history, and nutritional status were documented. Comprehensive physical and systemic examinations were performed by the treating pediatrician. Nutritional assessment was carried out according to the World Health Organization (WHO) weight-for-age classification.

Laboratory Methods: Venous blood samples were collected under strict aseptic precautions before initiation of iron therapy. Hematological parameters, including hemoglobin (Hb), mean corpuscular volume (MCV), and mean corpuscular hemoglobin (MCH), were measured using an automated hematologyanalyzer.

Iron status was evaluated by estimating serum ferritin, serum iron, and total iron-binding capacity (TIBC) using standard laboratory methods routinely employed in the hospital's central biochemistry laboratory. Lumbar puncture was performed only when clinically indicated to exclude central nervous system infection.

Operational Definitions: Febrile seizure was defined as a seizure occurring in children aged 9 months to 5 years in association with fever (≥38°C) without evidence of intracranial infection, metabolic disturbance, or prior afebrile seizures.

Iron deficiency was diagnosed using a combination of hematological indices and biochemical parameters, including hemoglobin concentration, serum ferritin, serum iron, MCV, MCH, and TIBC, interpreted according to age-appropriate reference values and standard laboratory criteria.

Study Variables: The primary outcome variable was the occurrence of febrile seizures. The primary exposure variable was iron deficiency. Secondary variables included age, sex, nutritional status, type of febrile illness, seizure characteristics, hemoglobin concentration, MCV, MCH, serum ferritin, serum iron, and TIBC. These variables were evaluated to determine their association with febrile seizures and to identify potential confounding factors.

Quality Assurance and Bias Control: To ensure methodological rigor, identical eligibility criteria and standardized clinical assessment protocols were applied to both study groups. Data were collected using a pre-tested structured proforma by trained investigators. Laboratory investigations were performed in the same institutional laboratory using standardized operating procedures and calibrated equipment to minimize measurement variability. Age- and sex-matching of controls, consecutive participant recruitment, uniform laboratory evaluation, and predefined diagnostic criteria were employed to reduce selection, measurement, and confounding biases. All collected data were reviewed for completeness and accuracy before statistical analysis.

Ethical Considerations: The study protocol was reviewed and approved by the Institutional Ethics Committee of Government Cuddalore Medical College and Hospital before participant enrolment. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki (2013 revision) and the Indian Council of Medical Research (ICMR) National Ethical Guidelines for Biomedical and Health Research involving Human Participants. Written informed consent was obtained from the parents or legal guardians of all enrolled children prior to participation. Participant confidentiality was ensured by assigning unique study identification numbers, and all clinical and laboratory data were anonymized before analysis. Participation was entirely voluntary, and parents were informed of their right to withdraw their child from the study at any stage without affecting the standard of medical care.

Data Management: Clinical and laboratory data were collected using a standardized pre-tested case record form and independently verified for completeness and accuracy before entry into a password-protected Microsoft Excel database. Double-checking of data entry was performed to minimize transcription errors. The final dataset was coded and exported to IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA) for statistical analysis. Missing or inconsistent data were reviewed against the original case records before database locking.

Statistical Analysis: Continuous variables were assessed for normality using the Shapiro–Wilk test and expressed as mean ± standard deviation (SD) or median (interquartile range), as appropriate. Categorical variables were summarized as frequencies and percentages. Between-group comparisons were performed using the independent-samples *t*-test or Mann–Whitney *U* test for continuous variables and the Chi-square test or Fisher's exact test for categorical variables. Crude odds ratios (ORs) with 95% confidence intervals (CIs) were calculated to evaluate the association between iron deficiency and febrile seizures.

Variables demonstrating clinical relevance or a univariable *P* value <0.20 were entered into a multivariable binary logistic regression model to identify independent predictors of febrile seizures after adjustment for potential confounders. Model adequacy was evaluated using the Hosmer–Lemeshow goodness-of-fit test, and multicollinearity was assessed using variance inflation factors. All statistical tests were two-tailed, and a *P* value <0.05 was considered statistically significant.

Results

A total of 106 children were enrolled in the present hospital-based case–control study. The study population comprised 53 children with febrile seizures (cases) and 53 age- and sex-matched children with febrile illness without seizures (controls), providing an equal distribution between the two study groups for comparative analysis.

Table 1: Distribution of Study Participants According to Study Group (N = 106)

Study Group	Frequency (n)	Percentage (%)
Cases (Children with febrile seizures)	53	50.0
Controls (Children with febrile illness without seizures)	53	50.0
Total	106	100.0

Table 1 shows the distribution of study participants according to the study group. A total of 106 children were included in the study, comprising 53 (50.0%) children with febrile seizures (cases) and 53 (50.0%) children with febrile illness without seizures (controls). Equal representation of participants in both groups ensured balanced comparison and strengthened the validity of the case–control design.

Table 2: Distribution of Study Participants According to Age Group (N = 106)

Age Group (Months)	Cases n (%)	Controls n (%)	Total n (%)
9–12	9 (17.0)	4 (7.5)	13 (12.3)
13–24	24 (45.3)	16 (30.2)	40 (37.7)
25–36	12 (22.6)	15 (28.3)	27 (25.5)
37–48	5 (9.4)	10 (18.9)	15 (14.2)
49–60	3 (5.7)	8 (15.1)	11 (10.4)
Total	53 (100.0)	53 (100.0)	106 (100.0)

Table 2 presents the age-wise distribution of the study participants. The largest proportion of children belonged to the 13–24 months age group (37.7%), followed by the 25–36 months age group (25.5%). Children aged 37–48 months accounted for 14.2%, whereas those aged 9–12 months and 49–60 months represented 12.3% and 10.4% of the study population, respectively. Among the cases, the highest proportion (45.3%) was observed in the 13–24 months age group, while among the controls, 30.2% belonged to the same age category. Overall, younger children, particularly those in the second year of life, constituted the majority of the study population.

Table 3: Distribution of Study Participants According to Sex (N = 106)

Sex	Cases n (%)	Controls n (%)	Total n (%)
Male	32 (60.4)	31 (58.5)	63 (59.4)
Female	21 (39.6)	22 (41.5)	43 (40.6)
Total	53 (100.0)	53 (100.0)	106 (100.0)

Table 3 illustrates the sex distribution of the study participants. Of the total 106 children, 63 (59.4%) were males and 43 (40.6%) were females. Among the cases, males accounted for 60.4% and females for 39.6%, whereas among the controls, males and females constituted 58.5% and 41.5%, respectively. A slight male predominance was observed in both study groups, with a comparable sex distribution between cases and controls.

Table 4: Comparison of Mean Age Between Cases and Controls (N = 106)

Variable	Cases (Mean ± SD)	Controls (Mean ± SD)	Mean Difference	*p*-value
Age (months)	24.8 ± 11.7	31.6 ± 14.2	9.5	0.008*

Table 4 compares the mean age of children between the case and control groups. The mean age of children with febrile seizures was 24.8 ± 11.7 months, whereas the mean age of children with febrile illness without seizures was 31.6 ± 14.2 months. The mean age difference between the two groups was 9.5 months, and this difference was statistically significant (p = 0.008). These findings indicate that children presenting with febrile seizures were significantly younger than children admitted with febrile illness without seizures.

Table 5: Distribution of Nutritional Status Among Cases and Controls (N = 106)

Nutritional Status	Cases n (%)	Controls n (%)	Total n (%)
Normal weight for age	30 (56.6)	39 (73.6)	69 (65.1)
Mild underweight	14 (26.4)	10 (18.9)	24 (22.6)
Moderate underweight	7 (13.2)	3 (5.7)	10 (9.4)
Severe underweight	2 (3.8)	1 (1.9)	3 (2.8)
Total	53 (100.0)	53 (100.0)	106 (100.0)

Table 5 presents the nutritional status of study participants based on the WHO weight-for-age classification. Overall, 69 (65.1%) children had normal weight for age, while 24 (22.6%) were mildly underweight, 10 (9.4%) were moderately underweight, and 3 (2.8%) were severely underweight. Among children with febrile seizures, 56.6% had normal nutritional status compared with 73.6% of controls.

Mild underweight was observed in 26.4% of cases and 18.9% of controls, whereas moderate underweight was reported in 13.2% and 5.7% of cases and controls, respectively. Severe underweight was identified in a small proportion of participants in both groups. Overall, undernutrition was more frequently observed among children with febrile seizures than among children with febrile illness without seizures.

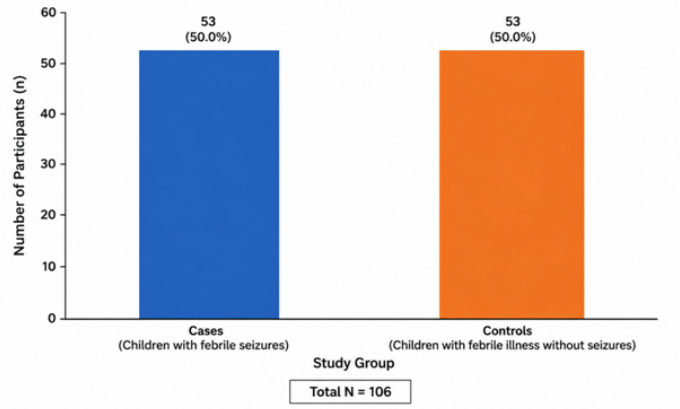

Figure 1: Distribution of study participants according to study group

Figure 1 illustrates the distribution of the study participants according to the study group. A total of 106 children were enrolled in the study, comprising 53 (50.0%) children with febrile seizures (cases) and 53 (50.0%) children with febrile illness without seizures (controls). Both groups contributed equally to the total study population, ensuring balanced allocation of participants and facilitating an unbiased comparison between cases and controls in the subsequent analyses.

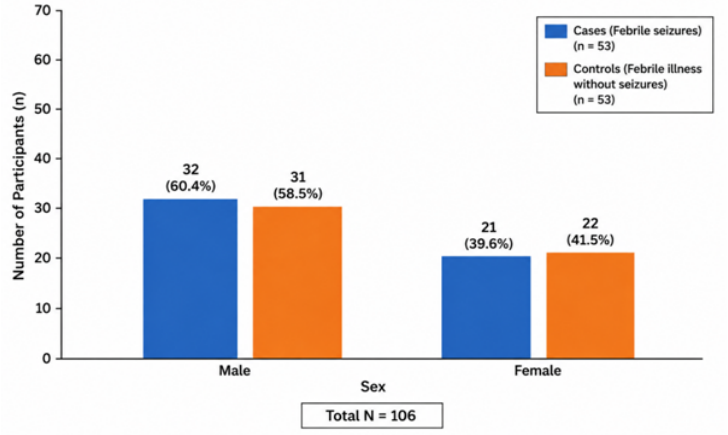

Figure 2: Distribution of study participants according to sex

Figure 2 illustrates the distribution of study participants according to sex. Of the total 106 children included in the study, 63 (59.4%) were males and 43 (40.6%) were females. Among the cases, 32 (60.4%) were male and 21 (39.6%) were female. Similarly, among the controls, 31 (58.5%) were male and 22 (41.5%) were female. A slight male predominance was observed in both study groups, with a comparable distribution of male and female participants between cases and controls, indicating successful age- and sex-matching of the study population.

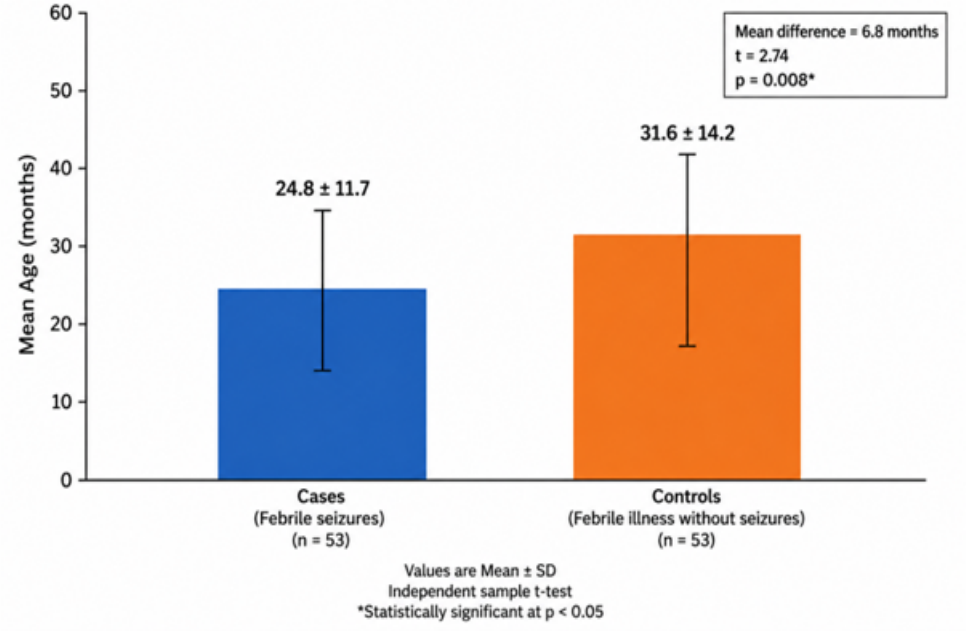

Figure 3: Comparison of mean age between cases and controls

Figure 3 compares the mean age of children in the case and control groups. The mean age of children with febrile seizures (cases) was 24.8 ± 11.7 months, whereas the mean age of children with febrile illness without seizures (controls) was 31.6 ± 14.2 months. The mean age difference between the two groups was 9.5 months, with children in the case group being significantly younger than those in the control group. Statistical analysis using the independent-samples *t*-test demonstrated that this difference was statistically significant ($p = 0.008$), indicating that febrile seizures occurred more frequently among younger children in the study population.

Discussion

The present hospital-based case–control study included 106 children aged 9 months to 5 years, comprising 53 children with febrile seizures and 53 age- and sex-matched febrile controls without seizures. Equal representation of cases and controls strengthened the internal validity of the study and minimized selection bias. Case–control studies remain the preferred design for evaluating risk factors associated with febrile seizures because they allow comparison of exposure variables among children presenting with similar febrile illnesses while differing in seizure occurrence.[20]

The age distribution observed in the present study demonstrated that febrile seizures occurred predominantly among younger children, with the highest proportion (45.3%) belonging to the 13–24 months age group. Furthermore, the mean age of children with febrile seizures (24.8 ± 11.7 months) was significantly lower than that of controls (31.6 ± 14.2 months), and this difference reached statistical significance ($p = 0.008$). These findings are consistent with the established epidemiology of febrile seizures, which typically peak between 12 and 24 months of age because of developmental immaturity of the central nervous system and increased neuronal excitability during early childhood.[21]

Several investigators have reported similar observations. Berg et al. demonstrated that the majority of first febrile seizures occur before two years of age, with incidence gradually declining thereafter.[22] Likewise, Chung reported that approximately 60–70% of febrile seizures develop during the second year of life, reflecting the age-dependent susceptibility of the immature brain to fever-induced neuronal excitation.[5] Offringa and Moyer also emphasized that younger age at presentation is one of the strongest epidemiological characteristics of febrile seizures and contributes to the higher risk of recurrence.[23] The findings of the present study therefore corroborate previous literature and reinforce the importance of close surveillance of febrile illnesses during infancy and early toddlerhood. The biological explanation for

this age predilection is multifactorial. During the first two years of life, rapid brain growth, incomplete myelination, immature inhibitory neurotransmitter pathways, and enhanced excitatory synaptic activity collectively reduce the seizure threshold. Fever-associated inflammatory cytokines such as interleukin-1β further increase neuronal excitability, thereby facilitating seizure occurrence in genetically susceptible children.[24] Consequently, younger children remain particularly vulnerable to febrile seizures even in response to relatively common childhood infections.

A slight male predominance was observed in the present study, with males accounting for 60.4% of cases and 58.5% of controls. Although the distribution between cases and controls was comparable, the overall predominance of boys is consistent with findings reported in previous epidemiological studies. Steering Committee on Quality Improvement and Management reported that febrile seizures occur slightly more frequently in boys than girls, although the underlying mechanisms remain incompletely understood.[1]

Similar observations have been reported by Patel and Vidaurre, who found a male-to-female ratio ranging from 1.2:1 to 1.5:1 among children with febrile seizures.[2] Jang et al. likewise observed a predominance of male participants in their prospective case–control study evaluating iron deficiency and febrile seizures.[14] The reasons for this modest sex difference remain speculative but may involve genetic susceptibility, sex-related neurodevelopmental differences, and variations in immune responses during early childhood.[25] Nevertheless, sex alone has not consistently emerged as an independent predictor of febrile seizures after adjustment for other risk factors.

Nutritional assessment in the present study revealed that undernutrition was more common among children with febrile seizures than among febrile controls. While 73.6% of controls had normal weight-for-age, only 56.6% of cases were nutritionally normal. Conversely, mild and moderate undernutrition were observed more frequently among cases than controls. Although statistical significance was not assessed for this comparison, the trend suggests that poorer nutritional status may coexist with febrile seizures. These observations are biologically plausible because nutritional deficiencies, particularly iron deficiency, frequently accompany undernutrition in developing countries. Chronic malnutrition may adversely affect brain maturation, neurotransmitter synthesis, immune competence, and neuronal metabolism, thereby lowering the seizure threshold.[26] The World Health Organization has consistently emphasized that undernutrition remains one of the major contributors to childhood morbidity, impaired neurodevelopment, and micronutrient deficiencies worldwide.[27] Several investigators have also demonstrated an association between poor nutritional status and febrile seizures. Kumari et al. reported that children with febrile seizures had significantly lower nutritional indices and a higher prevalence of iron deficiency compared with febrile controls.[12] Papageorgiou et al. similarly suggested that nutritional deficiencies may indirectly contribute to seizure susceptibility through reduced iron stores and altered neuronal function.[13] However, nutritional status alone is unlikely to be the sole determinant of febrile seizures because multiple interacting factors—including genetic predisposition, fever characteristics, inflammatory response, and micronutrient deficiencies—collectively influence seizure occurrence.[24]

The balanced distribution of participants between the study groups and comparable sex composition reduced potential confounding arising from demographic variables. The significant difference in mean age observed between cases and controls is clinically important because younger age itself has been identified as an established risk factor for febrile seizures and should therefore be considered when interpreting subsequent analyses involving hematological and biochemical parameters.[23]

Overall, the demographic findings of the present study closely parallel previously published literature describing febrile seizures as predominantly affecting boys during the second year of life. The greater frequency of undernutrition among children with febrile seizures further supports the hypothesis that nutritional factors, including iron deficiency, may contribute to seizure susceptibility and warrants detailed evaluation of iron status parameters, which constitute the principal objective of the present investigation.

Conclusion

The present hospital-based case–control study evaluated the demographic and nutritional characteristics of children with febrile seizures and age- and sex-matched febrile controls. Febrile seizures were observed predominantly in younger children, particularly those between 13 and 24 months of age, and the mean age of affected children was significantly lower than that of controls. A slight male predominance was noted in both study groups, consistent with the known epidemiological pattern of febrile seizures.

Although most children had normal nutritional status, undernutrition was more frequently observed among children with febrile seizures than among febrile controls.

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