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Original Research Article

ASSESSING IRON DEFICIENCY AS RISK FACTOR FOR FEBRILE SEIZURES IN CHILDREN BETWEEN SIX MONTHS AND FIVE YEARS OF AGE

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Abstract: *This case-control study was conducted from September 2023 to December 2023 at the Department of Pediatrics in Pak Emirates Military Hospital, Rawalpindi. The study aimed to determine the relationship between iron deficiency and the development of febrile seizures in children. A total of 150 patients aged between 1 and 6 years, with a history of febrile seizures and currently suffering from pyrexia, were included in the study. Children with a history of recent febrile seizures, structural/organic brain lesions, congenital metabolic/chromosomal anomalies, non-febrile seizures, delayed milestones, or those who were on medications that altered serum iron levels within the past six months were excluded. All participants were assessed for serum iron, ferritin, total iron binding capacity, magnesium, zinc, and calcium at enrollment. Patients were observed for the development of febrile seizures until they were afebrile for twenty-four continuous hours. Of the total sample, the mean age was 3.41 ± 1.51 years, and 83 (55.3%) were male. Patients with a transferrin saturation below 6.5% had a higher adjusted odds ratio (aOR) of 3.96 (CI 95% 1.29 – 12.18, p=0.016) for developing febrile seizures during a pyrexial illness. Similarly, patients with hypozincaemia, defined as a serum zinc level <65 µg/dL, had an aOR of 2.54 (CI 95% 1.13 – 5.67, p=0.023). The study concluded that low serum levels of iron were associated with a higher risk of developing febrile seizures in pyrexial children with a history of the disorder.*

Keywords: Febrile Seizures, Iron Deficiency, Risk

Introduction

Febrile seizures are the most common type of neurological disorder in children. They are convulsions associated with a fever greater than 100.4°F (38°C) without evidence of a central nervous system (CNS) infection (Xixis et al., 2022). They afflict approximately 2% to 5% of the global population aged between six months and five years and are a significant cause of concern for parents/guardians and caregivers (Leung et al., 2018). These seizures are usually benign and self-limiting but may be associated with an increased risk for the development of epilepsy in later life, especially if the seizures are complex (Jongruk et al., 2022). While the exact pathophysiology of febrile seizures is unclear, a number of factors and features are reported to be associated with their occurrences, such as a family history of febrile seizures, recent vaccinations, and the deficiency of certain minerals (Duffy et al., 2017; Shrestha et al., 2021).

Iron is an essential nutrient required for synthesizing hemoglobin and normal brain development and function (McCann et al., 2020). In Pakistan, its deficiency occurs in children at a frequency of between 40% and 70% and is associated with cognitive and behavioral problems in children (Habib et al., 2016; Pivina et al., 2019). In addition, low levels have been implicated in the development of febrile seizures, possibly also due to altered charge status resulting from deficiency across the neuronal membrane and the lack of the aforementioned developmental effects on the brain (Jang et al., 2019; Saghazadeh et al., 2015).

In this article, our objective was to determine the nature of the association between iron deficiency and the risk of febrile seizures in pediatric patients to establish the degree of association between deficiency and the occurrence of this disorder in Pakistani children. If a significant association between iron deficiency and an increased risk for the development of febrile seizures is found, it could have important implications for the prevention and management of this common neurological disorder in children. Furthermore, our findings could inform public health policies aimed at reducing the prevalence of iron deficiency and its associated health consequences in children, provide a basis for patient/attendant counseling, and serve as a foundation for future research.

Methodology

We conducted this case-control study from Sep 2023 to Dec 2023 in the Department of Paediatrics, Pak Emirates Military Hospital, Rawalpindi, on 150 pediatric patients who were currently febrile, selected via non-probability consecutive sampling. Informed consent was obtained from parents/guardians before including the child in the study. The WHO sample size calculator was used to calculate the sample size keeping a level of Significance (α) of 5%, power

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of the test (1-β) of 95%, an anticipated population proportion (P1) of 0.739, and an anticipated population proportion (P2) of 0.346, which were the proportion of febrile patients with and without seizures, respectively, who had iron deficiency anemia, from Ahmed et al. (Ahmed et al., 2019). The sample size per group came out to be 21, which we increased to 75.

All children between the ages of 1 and 6 years who had previously suffered from febrile seizures were included. Whereas all patients who had a history of febrile seizures within one month, had a structural/organic brain lesion, congenital metabolic/chromosomal anomalies, non-febrile seizures, delayed milestones, or were medications that altered serum iron levels within the past six months were excluded.

Once enrolled, all participants underwent a clinical session with a history and examination and were documented for relevant demographic data. This was followed by a venous blood phlebotomy conducted by a trained and experienced pediatric phlebotomist. Samples were tested for serum iron and total iron binding capacity (TIBC) levels. Participants with a less than 6.5% transferrin saturation were labeled as suffering from iron deficiency. (Higgins et al., 2017) Additionally, serum magnesium, zinc, and calcium levels were also measured, and values of <0.62 mmol/L, <65 µg/dL, and <2.2 mmol/L, respectively, were considered to indicate deficiency of these cations (Narayanan and Scalici, 2015; Roizen et al., 2013; Vuralli et al., 2017). All participants remained under observation during the entire hospital stay for the development of febrile seizures; those who developed seizures were placed in the cases groups, while those who remained seizure-free were grouped as controls. Patients were only discharged after a fever-free period of twenty-four hours.

Data was analyzed using the Statistical Package for the Social Sciences (IBM SPSS Statistics for Windows version 26, IBM Corp; Armonk, USA). Mean and SD were calculated for quantitative variables: age, duration of febrile illness, number of previous episodes of febrile seizures, serum iron, TIBC, magnesium, zinc, and calcium levels. Qualitative variables like gender, family history of febrile seizures, whether febrile seizures developed or not, and whether the participant had iron, magnesium, zinc, or calcium deficiency were recorded in terms of frequency and percentage. Qualitative variables were compared across groups using the Chi-square test/Fischer exact test. In contrast, quantitative variables were compared using the independent samples t-test/non-parametric tests, where appropriate, and a p-value of ≤0.05 was considered significant. Odds ratios were also calculated to determine the odds of various variables, including iron deficiency with the development of febrile seizures, followed by logistic regression to calculate adjusted odds ratios (ORs).

Results

Our study was conducted on a sample of 150 patients, 75 cases and controls each, with a mean age of 3.41 ± 1.51 years at the time of the study, of whom 83 (55.3%) were male. The mean duration since the onset of the first febrile seizures was 20.33 ± 9.28 months, while the mean number of hot seizure episodes experienced by the study sample was 2.37 ± 1.32. A positive family history of febrile seizures in a first-degree relative was seen in 40 (26.7%) cases. Patient characteristics distributed according to the group are displayed in Table I.

The mean serum iron level was 79.09 ± 41.86 µg/dL, while the mean TIBC was 412.59 ± 43.67 µg/dL. This resulted in a mean transferrin saturation of 19.29 ± 10.36 %. A total of 23 (15.3%) patients were iron deficient. The mean zinc levels were 75.52 ± 16.93 µg/dL, and 46 (30.7%) patients were poor. The sample's mean serum magnesium and calcium levels were 0.85 ± 0.24 mmol/L and 2.19 ± 0.30 mmol/L, respectively. A total of 38 (25.3%) had hypomagnesemia, while 74 (49.3%) had hypocalcemia. The results displayed for the electrolyte studies according to the group are shown in Table II.

Table-I. Patient characteristics according to group (n=150)

Variable	Cases (n=75)	Controls (n=75)	*p-value*
Gender			
Male	42 (56.0%)	41 (54.7%)	0.870
Female	33 (44.0%)	34 (54.3%)	
Age (years)	3.15 ± 1.47	3.68 ± 1.52	0.03
Duration since Onset of Seizures (months)	18.61 ± 8.68	22.05 ± 9.60	0.023
Number of Episodes	2.32 ± 1.13	2.41 ± 1.50	0.667
Family History of Febrile Seizures	24 (32.0%)	16 (21.3%)	0.140

Table-II. Patient characteristics according to group (n=150)

Variable	Cases (n=75)	Controls (n-75)	*p-value*
Serum Iron Levels (µg/dL)	61.61 ± 38.73	96.56 ± 37.53	<0.001
Total Iron Binding Capacity (µg/dL)	409.77 ± 43.62	415.40 ± 43.82	0.432
Transferrin Saturation (%)	15.15 ± 9.61	23.44 ± 9.44	<0.001
Low Transferrin Saturation	18 (24.0%)	5 (6.7%)	0.003
Serum Zinc Levels (µg/dL)	69.19 ± 16.73	75.85 ± 16.57	0.015
Hypozincaemia Present?	29 (38.7%)	17 (22.7%)	0.034
Serum Magnesium Levels (mmol/L)	0.81 ± 0.24	0.89 ± 0.23	0.077
Hypomagnesaemia Present?	24 (32.0%)	14 (18.6%)	0.060
Serum Calcium Levels (mmol/L)	2.16 ± 0.31	2.21 ± 0.29	0.275
Hypocalcaemia Present?	39 (52.0%)	35 (46.7%)	0.514

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Table III shows the aORs for different variables for the development of febrile seizures. Our regression model showed that patients with a transferrin saturation below 6.5% had an aOR of 3.96 (CI 95% 1.29 – 12.18, *p*=0.016) for developing febrile seizures during a pyrexial illness. The other significant factor was hypozincaemia with an aOR of 2.54 (CI 95% 1.13 – 5.67, *p*=0.023).

Table-III. Adjusted odds ratios for different variables for the development of febrile seizures (n=150)

Variable	Odds Ratio	Adjusted Odds Ratio
Age	1.71 (CI 95% 0.87 – 3.40)	0.90 (CI 95% 0.40 – 2.03)
Male Gender	1.06 (CI 95% 0.55 – 2.01)	1.18 (CI 95% 0.56 – 2.47)
Duration since Onset of Seizures	0.69 (CI 95% 0.32 – 1.47)	1.07 (CI 95% 0.93 – 1.22)
Number of Episodes of Febrile Seizures	1.20 (CI 95% 0.61 – 2.38)	1.20 (CI 95% 0.91 – 1.60)
Family History of Febrile Seizures	1.74 (CI 95% 0.83 – 3.62)	1.65 (CI 95% 0.72 – 3.77)
Low Transferrin Saturation	4.42 (CI 95% 1.55 – 12.64)	3.96 (CI 95% 1.29 – 12.18)
Hypozincaemia	2.15 (CI 95% 1.05 – 4.39)	2.54 (CI 95% 1.13 – 5.67)
Hypomagnesaemia	2.05 (CI 95% 0.96 – 4.37)	2.19 (CI 95% 0.95 – 5.09)
Hypocalcaemia	1.24 (CI 95% 0.65 – 2.35)	1.55 (CI 95% 0.74 – 3.24)

Discussion

Our study revealed that a deficient iron status was associated with an increased risk for the development of febrile seizures in children suffering from fever. Furthermore, zinc deficiency also appears to be associated with increased risk. Patients may require regular evaluation of the status of both these elements, and supplementation may be beneficial in reducing the frequency of fits. However, this requires further study.

In our study, the sample mean age had an aOR of 0.90 (CI 95% 0.40 – 2.03, *p*=0.803) for the development of febrile seizures. Sharawat et al. also noted that age did not appear to be a risk factor for febrile seizures (*p*=0.60) but did report that the incidence of first febrile seizures tended to peak around sixteen months (Sharawat et al., 2016). Dreier et al. also reported that the peak time of onset of febrile seizures appeared to be at the median age of 16.7 months (IQR: 12.5-24.0), and the vast majority of their study sample, i.e., over 90.0%, had developed at least one episode of febrile seizures before age the age of three years (Dreier et al., 2019).

Males were slightly more common within the current study sample, i.e., 55.3%, and the male gender had an aOR of 1.18 (CI 95% 0.56 – 2.47, *p*=0.659). Kumar et al. also reported that any particular gender did not appear to be associated with an increased risk for the development of febrile seizures (aOR: 0.95, CI 95% 0.42 – 2.15, *p*=0.897),(Kumar et al., 2019) while the study conducted by Kumari et al. also came to the same conclusion (OR: 0.91, CI 95% 0.62 - 1.33)(Kumari et al., 2022).

The mean lifetime episodes of febrile seizures experienced was 2.37 ± 1.32 for our study sample, which did not appear to affect the risk of seizure development, aOR = 1.20 (CI 95% 0.91 – 1.60, *p*=0.203). While this aspect has not been previously studied in literature, to our knowledge, Dreier et al reported that recurrent febrile seizures occurred in 22.7% (CI 95%, 22.4 - 23.0) after the first episode of seizures, but the frequency increased to 35.6% (CI 95%, 34.9 - 36.3) after the second episode, and rose further to 43.5% (CI 95%, 42.3 - 44.7) after the third one, indicating that patients with repeated episodes had a higher frequency of recurrence(Dreier et al., 2019).

In the current study, a positive family history of febrile seizures was not associated with an increased risk of febrile seizures, aOR = 1.65 (CI 95% 0.72 – 3.77, *p*=0.232), despite it being more common in cases. Conversely, Tosun et al. reported a positive family history was indeed associated with an increased risk for the occurrence of febrile seizures (aOR: 1.93, CI 95% 1.12 – 3.33, *p*=0.018)(Tosun et al., 2010). Sharawat et al. also noted that a positive family history was associated with the development of febrile seizures (*p*=0.05). We believe that the results of our study may have occurred due to a reporting bias, wherein the attendants of the study participants were sometimes vague and unclear about the presence of family history or were hesitant to share such details due to the existence of social stigmas associated with epileptiform disorders(Kanemura et al., 2016).

Patients who were iron deficient had an aOR of 3.96 (CI 95% 1.29 – 12.18, *p*=0.016) for the development of febrile seizures during pyrexial episodes. Kumari et al. also reported that iron deficiency was associated significantly with the development of febrile seizures (OR: 5.79 (CI 95% 3.56 - 9.38, *p*=0.001)(Kumari et al., 2022). Ahmed et al. also noted that serum ferritin levels were significantly lower in patients with febrile seizures than those without (*p*=0.001)(Ahmed et al., 2019).

In our study, the mean zinc levels had an aOR of 2.54 (CI 95% 1.13 – 5.67, *p*=0.023) for the development of febrile seizures. Previously, Saghazadeh et al. have reported that zinc is significantly lower in patients with febrile seizures when compared to normal controls (*p*=0.018), a finding that was concurred with by Namakin et al., (*p*<0.001) (Namakin et al., 2016).

The current study determined hypomagnesemia was not associated with an increased risk of febrile seizures, aOR= 2.19 (CI 95% 0.95 – 5.09, *p*=0.067). Khosroshahi et al. also reported that serum magnesium levels had no relationship with seizure activity (*p*=0.87). Conversely, Baek et al. reported that low serum magnesium levels were significantly associated with the development of febrile seizures (OR: 22.12, CI 95% 9.23 - 53.02, *p*<0.001). We believe this difference arose due to how serum magnesium was measured: while our study looked at total serum magnesium levels, the latter study measured ionized magnesium, which is electro-physiologically active and, as mentioned earlier, the deficiency in trace elements precipitating seizures may be associated with charge and not function (Jang et al., 2019; Saghazadeh et al., 2015).

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The current study sample did not demonstrate an increase in risk for the development of febrile seizures with serum calcium levels, aOR: 1.55 (CI 95% 0.74 – 3.24, *p*=0.247), which was in keeping with existing conclusions on the subject such as by Shajari et al. who reported that there was no difference in serum calcium levels between children suffering from febrile seizures and those who were not, (*p*=0.564) (Shajari et al., 2021).

Iron deficiency is widespread in the pediatric population in Pakistan, and children are advised routine supplements and fortified food regularly, sometimes even without the direct knowledge of the parents, which could not be accounted for in our study and may have resulted in some degree of confounding within our results. The same is true for other trace elements evaluated in this study: the Pakistani population is also deficient in vitamin D, and modern diets lack magnesium which may have altered our outcomes, the deficiencies having a chance of being incidental. Additionally, this was a small, single-center study, the results of which require further evaluation of larger, more diverse populations before concrete conclusions can be drawn.

Conclusion

Low serum levels of iron are associated with a substantially higher risk for the development of febrile seizure in children who are currently suffering from pyrexia and have a positive history of suffering from similar fits. Correcting iron deficiency may be associated with reducing this risk, but this requires further research before such interventions can be practiced routinely in febrile children. However, regular testing of serum iron status in such children may still be practiced as it will give the treating clinician useful information regarding prognosis. It sensitizes them to remain vigilant for the development of febrile seizures in iron-deficient patients.

Declarations

Data Availability statement
All data generated or analyzed during the study are included in the manuscript.
Ethics approval and consent to participate
Approved by the department Concerned.
Consent for publication
Approved
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Conflict of interest

The authors declared absence of conflict of interest.

Author Contribution

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Coordination of collaborative efforts.
Conception of Study, Development of Research Methodology Design, Study Design,, Review of manuscript, final approval of manuscript
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Conception of Study, Development of Research Methodology Design, Study Design,, Review of manuscript, final approval of manuscript
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Data acquisition, analysis.

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