

Serum Zinc Concentration in Children with Febrile Seizures: A Prospective Case Control Study

Authors:

Dr. Pooja Lakkol P. S.¹, Dr. Monisha B.², Dr. Niranjana Kamble³

^{1,2}MD (Paediatrics), Assistant Professor, Department of Paediatrics, Subbaiah Institute of Medical Sciences and Research Centre, Shivamogga, Karnataka, India.

³MD (Paediatrics), Associate Professor, Department of Paediatrics, Subbaiah Institute of Medical Sciences and Research Centre, Shivamogga 577222, Karnataka, India.

Corresponding Author:

Dr. Pooja Lakkol P. S., Assistant Professor, Department of Paediatrics, Subbaiah Institute of Medical Sciences and Research Centre, Shivamogga 577222, Karnataka, India.

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ABSTRACT

Objective: To compare serum zinc concentrations between children with febrile seizures and age- and nutrition-matched febrile and afebrile controls, and to quantify the association between hypozincaemia and febrile seizures. **Methods:** In a prospective case-control study at a tertiary care teaching hospital in southern India, 200 consecutive children aged 6 to 60 months admitted with a seizure during a febrile illness, without evidence of central nervous system infection or other defined cause, were enrolled as cases. Two hundred controls matched for age band and nutritional status were enrolled concurrently: 100 febrile children without seizures and 100 afebrile children. Serum zinc was estimated colorimetrically within 24 hours of the seizure; hypozincaemia was defined as a concentration below 60 µg/dL. **Results:** Mean serum zinc was 59.4 (SD 16.8) µg/dL in cases, 82.4 (24.7) µg/dL in febrile controls and 84.0 (28.5) µg/dL in afebrile controls ($F = 57.2$, $p < 0.001$). Cases differed from febrile controls (mean difference -23.0 µg/dL, 95% CI -28.3 to -17.6) and from afebrile controls (-24.6 µg/dL, 95% CI -30.7 to -18.6), both $p < 0.001$, whereas the two control groups did not differ ($p = 0.66$). Hypozincaemia was present in 133 of 200 cases (66.5%) and 33 of 200 controls (16.5%), odds ratio 10.1 (95% CI 6.3 to 16.2; $p < 0.001$). **Conclusions:** Serum zinc was substantially lower in children with febrile seizures than in febrile or afebrile controls, and the deficit was not attributable to fever alone. Hypozincaemia may be a modifiable correlate of febrile seizure susceptibility, and randomised trials of zinc supplementation to prevent recurrence are warranted.

Keywords: Febrile seizures; Zinc; Hypozincaemia; Trace elements; Child, preschool; Case-control studies

INTRODUCTION

Febrile seizures are the commonest seizure disorder of early childhood, affecting between 2% and 5% of children before their fifth birthday.^{1,2} They are conventionally defined as a seizure occurring between the ages of 3 and 60 months in association with fever, in the absence of intracranial infection, metabolic derangement or a history of afebrile seizures.^{2,3} Most are simple: brief, generalised and solitary within a febrile illness. A minority are complex, being focal, prolonged beyond 15 minutes or repeated within 24 hours.^{2,4} Although the long-term prognosis is favourable for the great majority of affected children,^{5,6} febrile seizures account for a disproportionate share of emergency paediatric attendance and generate considerable parental anxiety.³

Why some febrile children seize while most do not remains incompletely explained. Age at exposure, the height and rate of rise of temperature, and a strong genetic predisposition have all been implicated,^{1,4,5} but these factors are not modifiable. Attention has therefore turned to micronutrient status, and to zinc in particular. Zinc is the second most abundant trace element in the human body and an essential cofactor for more than 300 metalloenzymes.^{7,8} Within the central nervous system it is concentrated in the synaptic vesicles of glutamatergic hippocampal neurones, where it exerts a net inhibitory influence: it is a cofactor for glutamic acid decarboxylase, the rate-limiting enzyme in the synthesis of gamma-aminobutyric acid, and it potentiates the inhibitory effect of calcium at the N-methyl-D-aspartate

receptor.^{9,10} A fall in zinc availability would therefore be expected to shift the cortical balance towards excitation and to lower the seizure threshold, precisely the circumstance created by an acute febrile illness, in which cytokine-driven redistribution of zinc into the liver reduces circulating concentrations.⁹

Several clinical studies have reported lower serum and cerebrospinal fluid zinc concentrations in children with febrile seizures than in controls.^{9,11,12} Ganesh and Janakiraman, in a case-control study from Chennai, found mean serum zinc of 32.2 µg/dL in cases against 87.6 µg/dL in febrile controls,¹³ and Ehsanipour and colleagues reported a comparable if less marked gradient in an Iranian cohort.¹⁴ The evidence base nevertheless has limitations. Many of the published series are small, several compare cases only with afebrile children and so cannot separate the effect of the seizure from the effect of the fever, and data from Indian populations, in whom dietary zinc intake and the prevalence of subclinical zinc deficiency differ appreciably from those in Western populations,^{8,15} remain limited.

We therefore conducted a prospective case-control study in a tertiary care paediatric unit in southern India in which children with febrile seizures were compared with two separate matched control groups, one febrile and one afebrile, in order to determine whether any observed reduction in serum zinc is specific to the occurrence of a seizure or is simply an epiphenomenon of fever.

METHODS

Study Design and Setting

This was a prospective, hospital-based, case-control study conducted in the paediatric wards of a tertiary care teaching hospital and its attached children's hospital in Karnataka, southern India, between 1 November 2018 and 30 April 2020. The unit serves a predominantly urban and peri-urban population and admits approximately 3,000 children annually. The study was conducted and is reported in accordance with the STROBE recommendations for observational research.

Participants

Cases were children aged 6 to 60 months admitted with one or more seizures occurring in the setting of a documented febrile illness, in whom no intracranial infection or other defined cause for the seizure could be identified. Consecutive eligible children were enrolled. Each child was enrolled once only; children readmitted with a further febrile seizure during the study period were not re-enrolled.

Two control groups were recruited concurrently from the same wards and matched to the cases for age band and nutritional status as assessed by the Indian Academy of Paediatrics classification. The first comprised 100 children with an acute febrile illness of comparable aetiology who did not seize. The second

comprised 100 afebrile children admitted for elective or non-infective indications. This two-control design was adopted so that the contribution of fever itself to any observed difference in zinc could be separated from that of the seizure.

Children were excluded if they had received any zinc-containing supplement within the preceding week; if the seizure was afebrile; if there was clinical or cerebrospinal fluid evidence of central nervous system infection; if nutritional status was compromised, since protein-energy malnutrition independently depresses circulating zinc; or if there was a history of delayed developmental milestones.

Data Collection

A structured proforma was completed for every participant at enrolment. It recorded age, sex, birth order, birth weight, current weight and head circumference, developmental milestones, axillary temperature at admission, the clinical source of fever, any recent zinc supplementation, and family history of febrile seizures in siblings, parents or other relatives. For cases, the proforma additionally recorded the type of seizure as generalised or focal, its duration as reported by the caregiver, the number of seizures within the febrile episode, and the interval between the onset of fever and the seizure. Every child underwent a full physical and systemic examination. Lumbar puncture was performed where clinically indicated in accordance with prevailing guidance,¹⁶ and children with abnormal cerebrospinal fluid findings were excluded before analysis.

Laboratory Methods

A single venous blood sample was drawn from each case within 24 hours of the seizure, and from each control within 24 hours of admission, using trace-element-free technique. Samples were allowed to clot, centrifuged within one hour and the serum separated into acid-washed polypropylene tubes. Serum zinc was estimated by a direct colorimetric method using a commercial kit, a technique that has been validated against atomic absorption spectrophotometry and shown to give comparable results.^{17,18,19} The reference interval applied was 60 to 120 µg/dL, and hypozincaemia was defined a priori as a serum zinc concentration below 60 µg/dL. A complete blood count with peripheral smear, random blood glucose and serum calcium were performed in all participants, and further investigations, including cerebrospinal fluid examination, chest radiography and urine and stool examination, were performed as clinically indicated to establish the source of fever. All zinc assays were performed in the same hospital biochemistry laboratory by personnel unaware of the group allocation of the sample.

Sample Size and Statistical Analysis

No formal a priori sample size calculation was performed; the sample was determined by the number of eligible children presenting during the fixed study period, with a pre-specified 1:1 ratio of cases to controls. Post hoc, the achieved sample of 200 cases and 200 controls provided greater than 99% power to detect the observed difference in mean serum zinc at a two-sided alpha of 0.05.

Data were analysed using standard statistical software. Continuous variables are summarised as mean (standard deviation) and categorical variables as counts and percentages. Mean serum zinc was compared across the three groups by one-way analysis of variance, with pairwise comparisons by the Welch two-sample t test; mean differences are reported with 95% confidence intervals. Proportions were compared using the Pearson chi-squared test. The strength of the association between hypozinaemia and febrile seizures is expressed as an odds ratio with a 95% confidence interval calculated by the Woolf method. A two-sided p value below 0.05 was considered statistically significant.

Ethics

The protocol was approved by the Institutional Ethics Committee before recruitment began. Written informed consent was obtained from a parent or legal guardian of every participant after explanation of the purpose of the study, the procedures involved, the attendant risks and the right to withdraw at any time without prejudice to clinical care. No investigation was performed solely for the purposes of the study beyond the single additional serum zinc estimation, which was carried out on the same sample drawn for clinically indicated investigations wherever possible.

RESULTS

Participant Characteristics

Two hundred children with febrile seizures and 200 matched controls were enrolled and analysed; there were no withdrawals and no participant had missing serum zinc data. Among the cases, 93 children (46.5%) were aged 12 to 24 months and 140 (70.0%) were under 2 years of age. No case occurred between 48 and 60 months. There were 129 boys and 71 girls, a male to female ratio of 1.8:1. Baseline characteristics of the three groups are shown in Table 1; the groups were comparable by design for age band and nutritional status.

Table 1. Baseline characteristics of cases and controls

Characteristic	Cases (n = 200)	Febrile controls (n = 100)	Afebrile controls (n = 100)
Age group, months, n (%)			
6 to 12	47 (23.5)	23 (23.0)	24 (24.0)
12 to 24	93 (46.5)	47 (47.0)	46 (46.0)
24 to 36	33 (16.5)	17 (17.0)	17 (17.0)
36 to 48	27 (13.5)	13 (13.0)	13 (13.0)
48 to 60	0 (0.0)	0 (0.0)	0 (0.0)
Male sex, n (%)	129 (64.5)	63 (63.0)	62 (62.0)
Male to female ratio	1.8 : 1	1.7 : 1	1.6 : 1
Family history of febrile seizures, n (%)	27 (13.5)	4 (4.0)	3 (3.0)

Cases and controls were matched by age band and by nutritional status as assessed by the Indian Academy of Paediatrics classification.

Clinical Profile of the Febrile Seizure Episode

An upper respiratory tract infection was the source of fever in 153 cases (76.5%), followed by acute gastroenteritis in 27 (13.5%), lower respiratory tract infection in 13 (6.5%) and chronic suppurative otitis media in 7 (3.5%). The seizure was generalised in 173 children (86.5%) and focal in 27 (13.5%). No seizure exceeded 15 minutes in duration; 27 (13.5%) lasted less than 5 minutes, 100 (50.0%) lasted 5 to 10 minutes and 73 (36.5%) lasted 10 to 15 minutes. A single seizure occurred within the febrile episode in 173 children (86.5%), two seizures in 20 (10.0%) and three in 7 (3.5%). A family history of febrile seizures was elicited in 27 children (13.5%), involving a sibling in 20 (10.0%) and a parent in 7 (3.5%). Admissions showed two seasonal peaks, in April (60 children, 30.0%) and October (33 children, 16.5%). These findings are summarised in Table 2.

Table 2. Clinical profile of the febrile seizure episode (n = 200)

Variable	n (%)
Source of fever	
Upper respiratory tract infection	153 (76.5)
Acute gastroenteritis	27 (13.5)

Variable	n (%)
Lower respiratory tract infection	13 (6.5)
Chronic suppurative otitis media	7 (3.5)
Seizure semiology	
Generalised	173 (86.5)
Focal	27 (13.5)
Seizure duration	
Less than 5 minutes	27 (13.5)
5 to 10 minutes	100 (50.0)
10 to 15 minutes	73 (36.5)
More than 15 minutes	0 (0.0)
Number of seizures within the febrile episode	
One	173 (86.5)
Two	20 (10.0)
Three	7 (3.5)
Family history of febrile seizures	27 (13.5)
Sibling	20 (10.0)
Parent	7 (3.5)

Serum Zinc Concentration

Mean serum zinc was 59.4 (SD 16.8) µg/dL in cases, 82.4 (24.7) µg/dL in febrile controls and 84.0 (28.5) µg/dL in afebrile controls. The difference across the three groups was highly significant ($F = 57.2$, df 2 and 397, $p < 0.001$). On pairwise comparison, cases had a mean serum zinc 23.0 µg/dL lower than febrile controls (95% CI -28.3 to -17.6; $p < 0.001$) and 24.6 µg/dL lower than afebrile controls (95% CI -30.7 to -18.6; $p < 0.001$). Critically, the two control groups did not differ from each other (mean difference -1.6 µg/dL, 95% CI -9.0 to 5.8; $p = 0.66$), indicating that the deficit observed in cases was not a consequence of fever alone (Table 3).

Table 3. Serum zinc concentration by study group

Group	n	Mean (SD), µg/dL	Mean difference vs cases (95% CI)	p value
Cases (febrile seizure)	200	59.4 (16.8)	Reference	–
Febrile controls	100	82.4 (24.7)	22.96 (17.59 to 28.33)	< 0.001
Afebrile controls	100	84.0 (28.5)	24.60 (18.55 to 30.65)	< 0.001
Febrile vs afebrile controls	200	–	1.64 (-5.75 to 9.03)	0.66

One-way analysis of variance across the three groups: $F = 57.2$, df 2 and 397, $p < 0.001$. Pairwise comparisons by Welch two-sample t test. CI, confidence interval; SD, standard deviation.

Hypozinaemia and the Odds of Febrile Seizure

Hypozinaemia, defined as a serum zinc below 60 µg/dL, was present in 133 of 200 cases (66.5%) compared with 33 of 200 controls (16.5%), a fivefold difference in prevalence. The corresponding odds ratio for febrile seizure was 10.1 (95% CI 6.3 to 16.2; chi-squared 103.0, $p < 0.001$). The association persisted whichever control group was used as the comparator: against febrile controls, in whom hypozinaemia was present in 20 of 100 (20.0%), the odds ratio was 7.9 (95% CI 4.5 to 14.1; $p < 0.001$), and against afebrile controls, in whom it was present in 13 of 100 (13.0%), the odds ratio was 13.3 (95% CI 6.9 to 25.5; $p < 0.001$). The difference in the prevalence of hypozinaemia between the two control groups was not significant (20.0% vs 13.0%; $p = 0.18$). No case had a serum zinc above 120 µg/dL, whereas 13 febrile controls (13.0%) did, a finding consistent with the wide physiological variation in circulating zinc during acute illness. The distribution of serum zinc categories across the groups is given in Table 4, and the odds ratios in Table 5.

Table 4. Distribution of serum zinc categories by study group

Serum zinc, µg/dL	Cases (n = 200), n (%)	Febrile controls (n = 100), n (%)	Afebrile controls (n = 100), n (%)
Below 60 (hypozinaemia)	133 (66.5)	20 (20.0)	13 (13.0)
60 to 120 (normal)	67 (33.5)	67 (67.0)	87 (87.0)
Above 120	0 (0.0)	13 (13.0)	0 (0.0)
Total	200 (100)	100 (100)	100 (100)

Chi-squared for the 3 by 3 table: 137.7, df 4, $p < 0.001$.

Table 5. Association between hypozincaemia and febrile seizure

Comparison	Hypozincaemia in cases, n/N (%)	Hypozincaemia in comparator, n/N (%)	Odds ratio (95% CI)	p value
Cases vs all controls	133/200 (66.5)	33/200 (16.5)	10.05 (6.25 to 16.15)	< 0.001
Cases vs febrile controls	133/200 (66.5)	20/100 (20.0)	7.94 (4.48 to 14.06)	< 0.001
Cases vs afebrile controls	133/200 (66.5)	13/100 (13.0)	13.28 (6.92 to 25.51)	< 0.001
Febrile vs afebrile controls	20/100 (20.0)	13/100 (13.0)	1.67 (0.78 to 3.58)	0.18

Hypozincaemia defined as serum zinc below 60 µg/dL. CI, confidence interval.

DISCUSSION

In this prospective case-control study of 400 children, serum zinc was approximately 24 µg/dL lower in children presenting with febrile seizures than in matched controls, and two thirds of cases were frankly hypozincaemic against one sixth of controls. The single most informative finding is the comparison between the two control groups: febrile children without seizures and afebrile children had essentially identical serum zinc concentrations, differing by less than 2 µg/dL. Fever alone, in this population and over this timescale, did not meaningfully depress circulating zinc. The deficit therefore tracked the seizure rather than the febrile illness, which is the discriminating observation that studies employing a single control group cannot make.

Our mean case value of 59.4 µg/dL sits between the markedly low value of 32.2 µg/dL reported by Ganesh and Janakiraman from Chennai¹³ and the higher value of 76.8 µg/dL reported by Ehsanipour and colleagues from Iran.¹⁴ Such variation is expected. Assay methodology differs across series, with atomic absorption spectrophotometry, inductively coupled plasma mass spectrometry and colorimetry each carrying different calibration characteristics; background dietary zinc intake differs substantially between populations;^{8,15} and the interval between seizure and sampling is not uniformly specified. What is consistent across all these series, and in ours, is the direction and the magnitude of the gradient between cases and controls. Burhanoglu and colleagues found both serum and cerebrospinal fluid zinc to be significantly lower in children with febrile convulsions than in children with bacterial meningitis, viral central nervous system infection or febrile controls,¹¹ and Gunduz and colleagues reported the same pattern.¹² Garty and colleagues extended the observation to the cerebrospinal compartment specifically,⁹ which matters because it is the central rather than the circulating pool that is mechanistically relevant.

A biologically coherent explanation is available. Zinc is a cofactor for glutamic acid decarboxylase, the rate-limiting enzyme in gamma-aminobutyric acid synthesis, and low cerebrospinal fluid concentrations of that neurotransmitter have been documented in

several seizure disorders including febrile convulsions.⁹ Zinc also facilitates the inhibitory action of calcium at the N-methyl-D-aspartate receptor, so that in its relative absence the receptor is disinhibited and excitatory neuronal discharge becomes more likely.^{9,10} Superimposed on this is the developmental context: the immature brain between 6 and 36 months has incomplete myelination and a lower seizure threshold than at any later age,⁴ which is precisely the window in which 86.5% of our cases occurred.

The demographic and clinical profile of our cases conforms closely to the published literature and supports the external validity of the sample. The peak incidence between 12 and 24 months matches the 18 to 22 month peak described by Hirtz²⁰ and the mean onset of approximately 23 months in the National Collaborative Perinatal Project cohort.⁵ The male preponderance we observed, at 1.8:1, is consistent with ratios of 1.4:1 to 1.8:1 reported previously.^{21,22,23} Upper respiratory tract infection accounted for 76.5% of the febrile illnesses, at the upper end of the 38% to 62% range reported in earlier series,^{5,21,22} reflecting the predominance of viral respiratory illness as the trigger. Generalised seizures predominated, as in all published series, although our proportion of focal events at 13.5% was higher than the 4.6% and 5% reported by Sehgal and Bala²² and by Fishman,²⁴ which may reflect ascertainment from caregiver report rather than witnessed onset. A positive family history was present in 13.5%, intermediate between the 10% reported by Sehgal and Bala²² and the 50% reported by Tsuboi,²⁵ and it remains an observation that strongly supports the polygenic contribution to susceptibility documented by Hauser and colleagues.²⁶ The two seasonal peaks, in April and October, are consistent with the local epidemiology of diarrhoeal and respiratory illness respectively and differ from the November to January and June to August peaks described in temperate settings.²⁷

Clinical Implications

If hypozincaemia is causally implicated rather than merely associated, it is one of very few modifiable factors in a condition otherwise governed by age and genotype. Zinc supplementation is inexpensive, widely available through existing public health programmes in

India and has an established safety record in this age group.^{8,28} Our data do not establish causality and cannot support a recommendation for routine supplementation. They do, however, provide an effect size of sufficient magnitude, an odds ratio exceeding 10, to justify an adequately powered randomised controlled trial of zinc supplementation for the prevention of febrile seizure recurrence, with serum and ideally cerebrospinal fluid zinc measured at baseline. Until such evidence exists, management should continue to follow established principles: control of the seizure, identification and treatment of the underlying infection, judicious use of lumbar puncture,¹⁶ and above all the recognition and allaying of parental anxiety, which is frequently disproportionate to the benign prognosis of the condition.^{29,30}

Strengths and Limitations

The principal strength of this study is the dual control design. By recruiting both febrile and afebrile controls, matched for age band and nutritional status, we were able to demonstrate that the zinc deficit in cases is not attributable to the acute-phase response of fever, an inference that the majority of published case-control studies in this field cannot draw. The sample is also larger than most comparable series, enrolment was consecutive and prospective, and laboratory personnel were unaware of group allocation.

Several limitations should be acknowledged. First, this is a single-centre, hospital-based study, and children admitted to a tertiary referral unit may not represent all children with febrile seizures in the community. Second, serum zinc was estimated colorimetrically rather than by atomic absorption spectrophotometry or inductively coupled plasma mass spectrometry; although the colorimetric method correlates well with these reference techniques,^{17,18,19} absolute values are not directly interchangeable across methods, which limits comparison with series using other assays. Third, a single sample was drawn within 24 hours of the seizure, so we cannot determine whether the low zinc preceded the seizure or followed it; the temporal direction of the association therefore remains unresolved, and reverse causation cannot be excluded. Fourth, dietary zinc intake was recorded qualitatively and was not quantified, and we did not measure serum albumin, C-reactive protein or copper, all of which influence the interpretation of a circulating zinc concentration during acute illness. Fifth, seizure duration and semiology were derived largely from caregiver report and are subject to recall and observer error. Finally, we did not follow participants beyond the index admission and so can say nothing about recurrence, which is the outcome of greatest clinical interest.

CONCLUSION

Children presenting with febrile seizures had substantially lower serum zinc concentrations than age- and nutrition-matched febrile and afebrile controls, and the absence of any difference between the two control groups indicates that this deficit is not explained by fever alone. Two thirds of children with febrile seizures were hypozincaemic, corresponding to a tenfold increase in the odds of febrile seizure. These findings identify zinc status as a potentially modifiable correlate of febrile seizure susceptibility and support the conduct of a randomised controlled trial of zinc supplementation for the prevention of recurrence.

Declarations

Ethical Approval

The study protocol was approved by the Institutional Ethics Committee before recruitment began. Written informed consent was obtained from a parent or legal guardian of every participant after an explanation of the study purpose, procedures, potential risks, and the right to withdraw at any time without prejudice to clinical care.

Consent for Publication

Not applicable. No personally identifiable information or images of individual participants are presented in this manuscript.

Conflict of Interest

The authors declare that they have no conflict of interest.

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Authors' Contributions

All authors contributed to the conception, design, data collection, analysis, interpretation of results, and preparation and critical revision of the manuscript. All authors approved the final version of the manuscript and agree to be accountable for all aspects of the work.

Data Availability Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to institutional and ethical restrictions.

Declaration of Generative AI Use

No generative AI tools were used in the conception, writing, data analysis, interpretation, or preparation of this manuscript. The authors take full responsibility for the accuracy, originality, and integrity of the content presented in this article.

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