ORIGINAL ARTICLE

ZINC DEFICIENCY AS A RISK FACTOR FOR FEBRILE SEIZURE

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Abstract
This study was conducted to determine the role of zinc deficiency as a risk factor for first febrile seizure in children. 75 children aged between 6 months to 6 years with first febrile seizure (cases) and 50 children with febrile illness but without convulsions (controls) were enrolled from the pediatric ward of a tertiary care hospital. The mean serum zinc levels in cases and controls were 81.84 and 90.38 mcg/dl respectively. This difference was statistically highly significant [p< 0.001]. Zinc deficiency could be a potential risk factor for febrile seizure in children.
Keywords: febrile seizure, serum zinc

Introduction

Febrile convulsion is a common cause of convulsion in childhood and about 4% of children in the age group of one to six years have at least one episode of febrile convulsion. (1) Many studies have investigated the etiology and natural history of febrile seizures and evaluated various management strategies, but very little information is available about zinc deficiency as a risk factor. Zinc modulates the activity of glutamic acid decarboxylase, the rate limiting enzyme in the synthesis of gamma-aminobutyric acid (GABA), which is a major inhibitory neurotransmitter (2). It complements the inhibitory effects of calcium on the excitatory N-methyl-d-aspartate receptors which become activated when a patient develops low levels of zinc and induce an epileptic discharge in children with high fever (3). Burhanoglu et al concluded zinc deprivation plays a role in the pathogenesis of febrile seizures (4). The findings were found to be similar to the study conducted by Ganesh et al (5). We performed this study to evaluate the association between zinc deficiency and febrile seizure.

Methods and Materials

This study was carried out in a tertiary hospital from Feb 2007 to Feb 2008. Seventy five consecutive children aged 6 months to 6 years, admitted to the pediatric ward with first episode of febrile seizure were enrolled as cases. Febrile seizure was defined as a seizure occurring in association with a febrile illness, in the absence of CNS infection or any other defined causes of seizure (6). Children with previous febrile/ afebrile seizures, neurological infections, developmental delay, or children taking zinc for therapeutic purposes and having abnormal zinc metabolism were excluded. Fifty age and sex matched children admitted with febrile illness but without seizures and without zinc supplements were enrolled as control. The Hospitals Institutional Ethics Committee approved the study and written informed consent was obtained from parents. After admission a detailed history and clinical evaluation was done in all patients. Baseline data including the age, sex, height/length and weight were recorded for all the children in a structured proforma. Serum zinc was measured by an atomic absorption spectro-photometer (model 1200, Varian Techtron).

All the statistical analyses were performed using statistical package for social sciences (SPSS Inc., Chicago, USA) version 11.0 for Microsoft Windows.

Results

Demographic data of both cases and controls were comparable and depicted in Table 1 except that lower respiratory tract infection (LRTI) and urinary tract infection (UTI) was more common in the control group.

The mean serum zinc levels in cases and controls were 81.84 13.23 mcg/dl and 90.38 6.88 mcg/dl respectively (p< 0.001).

Table 1: Demographic characteristics of both cases and controls

	Cases(%)	Controls(%)
Gender (males : females)	46:29	30:20
Age distribution		
0-12 months	31(41.3%)	22 (44.0%)
13-24 months	20 (26.7%)	12 (24.0%)
25-36 months	10(13.3%)	4(8.0%)
37-48 months	4(5.3%)	5(10.0%)
more than 48 months	10(13.3%)	7(14.0%)
Causes of fever		
LRTI	17(22%)	33 (66%)
Sepsis	1(1.3%)	2 (4%)
UTI	0	2 (4%)
AGE	22 (29%)	7 (14%)
PUO	35 (46%)	6 (12%)

LRTI - lower respiratory tract infection, UTI - urinary tract infection, AGE- acute gastroenteritis , PUO- Pyrexia of unknown origin

Discussion

The incidence of febrile seizures varies from 2-4% in western Europe and USA, 5-10% in India, 8.8% in Japan and 14% in Guam (7). In etiology of febrile convulsions, in addition to genetic factors, maternal disease, maternal smoking, abnormal pregnancy and birth history also take roles as predisposing factors (8, 9). Although its occurrence mechanisms are controversial, febrile convulsions are generally thought to be induced by metabolic changes during the rising-phase of body temperature (4). Zinc modulates the activity of glutamic acid decarboxylase, the rate limiting enzyme in the synthesis of gamma-aminobutyric acid (GABA), which is a major inhibitory neurotransmitter. The hypozincemia activates the NMDA receptor, one of the glutamate family of receptors, which may play an important role in the induction of epileptic discharge (2,3).

Ganesh et al (8) and Burhanoglu M (4) et al reported that mean serum zinc was significantly low in children with febrile seizure. Similarly in our patients with febrile seizures, we found that serum zinc levels were low. The study does have some limitations as it is done in small number of patients. Further studies in larger groups are necessary. It is also known that fever

and/or infections may cause a reduction in serum zinc concentrations (2) and whether that has affected the zinc levels in these patients is not known though serum zinc levels are known to be decreased in respiratory tract infections. However in our study though more patients with LRTI were in the control group, serum zinc levels were more decreased in the cases suggesting that LRTI may not have been the confounding factor.

Conclusion

Zinc deficiency could be a potential risk factor for febrile seizure in children.

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