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TO STUDY SERUM ZINC LEVEL IN CHILDREN WITH FEBRILE SEIZURE.

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Abstract

Background: Febrile seizures are the most common cause of convulsions in children. However, the exact underlying etiology and the pathophysiological mechanisms are yet to be established. Various theories have been put forward regarding the role of trace elements as predisposing factors in causing the convulsions. Among them, Zinc is the most interesting trace element whose role in diarrhea and pneumonia is well proven.

Methods: Hospital based Prospective, Analytical, Case – Control study conducted on 100 patients were enrolled in the study out of which 50 were cases which were febrile convulsion patients and 50 were control who were age and weight matched children.

Results: Mean zinc level was 69.80±13.13mcg/dl and 80.76±10.24 mcg/dl in study and control groups respectively and this difference was found statistically highly significant ($p<0.001$).

Conclusion- This study establishes a definite relationship between zinc deficiency and febrile seizures thereby substantiating zinc as an important predisposing factor in febrile seizures.

Keywords: Zinc, Epilepsy, Deficiency

Introduction

Zn is as a co-factor of glutamate decarboxylase which is an enzyme needed for gamma-aminobutyric acid synthesis in the central nervous system and reduced CSF zinc levels have also been noted in febrile convulsions. Recent evidences indicate that zinc deficiency plays a significant role in febrile seizures. The following mechanisms can be postulated. Zinc increases storage capacity of glutamate or slows down the release rate of glutamate.[1]

Zinc increases the activity of pyridoxine needed for pyridoxine formation reciprocally pyridoxine increases the activity of glutamate decarboxylase which results in gamma-aminobutyric acid syntheses. Thus, decreased zinc levels lowers GABA synthesis which would precipitate seizures.[2]

Persistent and prolonged seizure activity cause cerebral edema, hypoxia, hyperthermia, hypoglycemia and vasomotor instability. respiratory depression may ensue from involvement of respiratory centre or from drugs used for seizure control. Vomiting and aspiration of secretions also increase morbidity. Hence treatment should be taken precedence over investigation of the cause. [3]

Zinc plays an important role in tissue or cell growth. This is related primarily to its function in the regulation of protein synthesis as well as synthesis and catabolism of nucleic acids. With respect to transcription, zinc appears to interact with nuclear proteins that bind to promoter sequences of specific genes, zinc forms a structural component of zinc fingers which recognize DNA base sequences during replication and transcription of DNA.[4]

Materials and Methods

Study design

Hospital based Prospective, Analytical, Case – Control study.

Study population

Infants and children aged between 6 months to 5 years.

Sample size

100 patients were enrolled in the study out of which 50 were cases which were febrile convulsion patients and 50 were control who were age and weight matched children.

Selection of control

The control group included the age and weight matched children suffering from a febrile illness without seizures, such as urinary tract infection, gastroenteritis and respiratory tract infection, coming to hospital.

Sampling Method

Convenience sampling

Inclusion Criteria

Children aged between 6 months to 5 years with simple/complex febrile seizures (seizure occurring in developmentally normal child in association with a febrile illnessin the absence of CNS infection or any other defined cause of seizures).

Exclusion Criteria

1. Children with previous history of established non febrile seizures
2. Neurological infections (meningitis, encephalitis)
3. Hereditary metabolic disorders
4. Developmental delay
5. Children with history of birth asphyxia
6. Persistent neurological deficits

Data Collection

Demographic data, seizure details, nature of febrile illness, complete developmental history, family history of epilepsy/febrile seizures, temperature at admission, general examination, Systemic examination and nutritional status were recorded (IAP weight for age classification was used to grade protein energy malnutrition) including the final diagnosis was recorded.

Serum Zinc detection from blood was carried out in a PerkinElmer A-Analyst 800 tool atomic absorption spectrophotometry after diluting the serum 5fold in 1/1000 priton.x.100 solution.

Data Analysis

Data was collected from eligible patients on a pre-structured pre-tested Proforma. For data analysis statistical software SPSS was used and data were analyzed with the help of frequencies, figures, proportions, measures of central tendency and appropriate statistical test.

Results

Table 1: Distribution of cases according to zinc (mcg/dl) level in both groups

Zinc (mcg/dl)	Groups				Total	
	Study		Control			
	No.	%	No.	%	No.	%
Abnormal (<64)	18	36.0	5	10.0	23	23.0
Normal (64-124)	32	64.0	45	90.0	77	77.0
Total	50	100	50	100	100	100
Mean	69.80		80.76			
SD	13.13		10.24			
t	4.623					
p	<0.001					

According to above table, in study group, 18(36%) patients had abnormal (<64 mcg/dl) range of zinc level while in control group only 5(10%) had abnormal level.

Mean zinc level was 69.80±13.13mcg/dl and 80.76±10.24 mcg/dl in study and control groups respectively and this difference was found statistically highly significant (p<0.001).

Discussion

Convulsions or seizures are one of the important pediatric health problems in developing and developed countries and febrile seizures are the most common seizure disorder in childhood, affecting 2% to 5% of children between the ages of 6 and 60 months[1]. It is generally believed that FS is an age-dependent response of the immature brain to fever. This postulation is supported by the fact that most (80-85%) febrile seizures occur between 6 months and 3 years of age, with the peak incidence at 18 months.

Zinc is a fundamental component of body enzymes that modulates CNS activities. CSF hypozinconemia activates N Methyl- D-aspartate receptors or disinhibits GABAergic action, thus resulting in febrile convulsion. In our study Mean zinc level was 69.80±13.13mcg/dl and 80.76±10.24mcg/dl in study and control groups respectively and this difference was found statistically highly significant (p<0.001). Similar findings were noted by others. Papierkowski et al found that the mean serum concentration of magnesium and zinc were significantly lower in the children with febrile convulsion. In another study by Tutuncuoglu s et al, researchers have shown that children with febrile convulsion had significantly higher plasma IL-1 beta and prostaglandin levels and lower serum zinc levels during the acute phase. They concluded that these changes may be responsible for the pathogenesis of febrile convulsion. In their study mean zinc levels were comparatively lower in FS group compared to FI group, but however it was statistically insignificant.

Conclusion

This study establishes a definite relationship between zinc deficiency and febrile seizures thereby substantiating zinc as an important predisposing factor in febrile seizures.

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