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Original Article

Serum Sodium Levels and Probability of Recurrent Febrile Convulsions

M Kulandaivel

Professor and Head, Department of Paediatrics, Government Sivagangai Medical College and Hospital, Sivagangai, Tamil Nadu, India

Abstract

Introduction: Febrile seizures are seizures that occur between the age of 6 and 60 months with a temperature of 38°C or higher, that are not the result of the central nervous system infection or any metabolic imbalance, and that occur in the absence of a history of prior afebrile seizures. The mechanism of seizure activity is altered in hyponatremia, due to deficiency of sodium ion, more calcium ion influx, and generation of repetitive action potential which will cause repetitive seizure initiation.

Aim: To estimate the serum sodium levels in children with febrile convulsions and analyze the probability of recurrent febrile convulsions.

Materials and Methods: A prospective study which enrolled 190 children admitted with febrile seizures at Sivagangai Medical College Hospital. They were divided into five groups. Serum sodium levels were estimated. The probability of recurrent febrile convulsions and serum sodium was analyzed. The results obtained were statistically analyzed using Bartlett's test.

Results: A total of 53 of 190 children (29%) with febrile convulsion developed repeat convulsion. The mean sodium level in the group with repeat convulsion was 132.26 mmol/l which was significantly lower than the mean in the group without repeat convulsions. The increase in the probability of a repeat convulsion when the serum sodium level decreases are significant (P = 0.0025 [<0.05]).

Conclusion: When the serum sodium level decreases, the probability of recurrent febrile convulsion increases. This knowledge may be of practical value in deciding whether to admit the child or allow it to return home and in advising parents or caregivers of the risk of a repeat convulsion.

Key words: Sodium levels, Febrile, Convulsions

INTRODUCTION

Febrile convulsions are one of the most common problems encountered in clinical pediatric practice. About 4-5% of all children suffer from at least one febrile convulsion in early childhood. Febrile convulsions must be differentiated from the seizures occurring without fever or with another precipitating factor.

Febrile SEIZURES are seizures that occur between the age of 6 and 60 months with a temperature of 38°C or higher, that are not the result of the central nervous system infection or any metabolic imbalance, and that occur in the absence of a history of prior afebrile seizures.[1-8]

Febrile seizures do not increase the risk of death, mental retardation or cerebral palsy in these children. The only medical consequence of an initial febrile seizure is a greater chance of having further febrile seizures and a slight risk of later epilepsy (2% of 7 years of age). Nevertheless far and misunderstanding the consequences of a recurrence make parents and physicians anxious and drive them to use medication to treat this benign disorder.[9-15]

The mechanism by which febrile seizures predispose to later epilepsy is much less clear. Prolonged febrile convulsions in early infancy may precede a variety of different seizures but are particularly common in children who develop intractable seizures of temporal lobe origin. Prolonged febrile seizures in childhood are known to have

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Corresponding Author: M Kulandaivel, 45 A, Athisaya Nagar, Muthamil Nagar Extn., Thabal Thanthi Nagar, Maduari - 625 017, Tamil Nadu, India. Phone: +91-9443448268. E-mail: pmkulandai@gmail.com

adverse physiologic consequences, including increased cerebral metabolic demand and systemic changes such as hypoxia, hypoglycemia, and arterial hypotension.

Genetic factors appear to play a role when epilepsy develops after febrile seizures. Temporal lobe seizures are more likely to begin early but remit permanently if a first-degree relative had a febrile seizure. A single gene is held responsible because the siblings of the patients with temporal lobe and febrile seizures have a similar incidence of febrile seizures alone.

Collectively, children with complex febrile seizures can be said to have a small but identifiable risk for later epilepsy, based on genetic, developmental and acquired factors. If these children develop persistent temporal lobe seizures, they are likely to continue to experience seizures in later life.

Seizure activity can begin in a very discrete region of cortex and then spread to neighboring regions, there is a seizure initiation phase and a seizure propagation phase. Initiation phase is characterized by two concurrent events in an aggregate of neurons.

1. High-frequency bursts of action potentials and
2. Hypersynchronization.

The bursting activity is caused by a relatively long-lasting depolarization of the neuronal membrane due to the influx of extracellular calcium, which leads to the opening of voltage-dependent sodium channels which leads to an influx of sodium.

In the case of hyponatremia due to deficiency of sodium ion, more calcium ion influx, and generation of repetitive action potential which will cause repetitive seizure initiation.

Fever plays an important role in causing disturbances in fluid and electrolyte imbalance. Hyponatremia has been thought to enhance the susceptibility to seizures associated with febrile illness in childhood. Sodium levels are lower in those children with complicated convulsions in comparison with those having simple convulsions.[2] The sodium concentrations are lowest in children with repeated seizures compared with children having simple or other complicated types of febrile seizures such as focal seizures. Seizures lasting longer than 15 min during febrile convulsions have been studied serum potassium levels showed no significant differences between patient groups. However, calcium levels and osmolarity significantly lower than control groups. The electrolytic modification of overall hyponatremia is probably due to the syndrome of inappropriate antidiuretic hormone[3] may have a role in short-term relapses of febrile convulsions. Hyponatremia has been documented in some children with high fever, without seizures, it may be that hyponatremia in predisposed subjects lowers the threshold of neuromuscular excitability.[16-22]

MATERIALS AND METHODS

Over 1 year period from April 2016 to March 2017, a prospective study of 190 children admitted in the Paediatric ward, Sivagangai Medical College Hospital, Sivagangai, with febrile convulsions simple or complicated was done.

Inclusion Criteria

- Child with a generalized convulsion
- Rectal temperature is 101°F or
- Axillary temperature is 100°F
- Febrile children with recurrent seizure.

Exclusion Criteria

- Children with signs of meningitis
- Children with gastroenteritis
- Children with inadequate fluid intake
- Children with developmental delay
- Children with neurologic disorders
- Children with a history of non-febrile seizures.

All children aged 6-60 months were divided into five age groups. Details of age, sex, family history of febrile seizures, previous history of febrile seizures, medications if any were recorded. Neurologic consultation was obtained for all. Children with the first episode of febrile seizure were subjected to lumbar puncture. Serum sodium levels were estimated by flame photometer in all 190 children by collecting 2 ml of venous blood within 30 min of admission. Hyponatremia was considered when serum sodium levels were <130 mEq/l. Serum sodium levels were also estimated in 63 normal children and 84 febrile children without seizures.

The sodium level so obtained was compared within four groups. The probability of recurrent febrile convulsions and serum sodium was analyzed. All children with febrile seizures were treated with rectal diazepam 0.3 mg/kg and acetaminophen 40 mg/kg in four divided doses. The control group received only acetaminophen in the same dosage as in study group.

The results were statistically analyzed using Bartlett's test.

An acute hyponatremia as seen in febrile seizure due to inappropriate secretion of antidiuretic hormone-serum sodium level falls rapidly in <24 h without the brain having any time to adapt to the electrolyte disturbance. In the brain when an osmotic gradient occurs within few hours, equilibrium may be restored by the movement of water molecules into cells causes cerebral edema, consequently patient develops convulsions.

RESULTS

There were 111 boys and 79 girls in the study group of 190 children with febrile convulsions. Between 6 and 12 months, there were 28 boys and 21 girls. Between 1 and 2 years there were 46 boys and 29 girls. Between 2 and 3 years there were 28 boys and 10 girls. Between 3 and 4 years there were 11 boys and 6 girls. Between 4 and 5 years there were 5 boys and 6 girls. The mean age of febrile seizure was 1.5 years. In this group mean serum sodium level was 135.2 mmol/l.

- Mean sodium levels in the first group (6 months - 1 year) were 134.85 mmol/l
- Mean sodium levels in the second group (1-2 years) were 134.38 mmol/l
- Mean sodium levels in the third group (2-3 years) were 135.26 mmol/l
- Mean sodium levels in the fourth group (3-4 years) were 135.29 mmol/l
- Mean sodium levels in the fifth group (4-5 years) were 136.18 mmol/l.

The mean sodium level in simple febrile seizures was 135.75 mmol/l and mean sodium level in complex seizures was 132.26 mmol/l.

The control group of 63 normal children had a mean serum sodium level of 140.29 mmol/l with a range of 136-145 mmol/l. No child had serum sodium levels<136 mmol/l in this group.

In the second group of 84 children with fever but without convulsions, the mean serum sodium level of 137.17 mmol/l with a range of 131-144 mmol/l. 11 of 84 children 13% had serum sodium level <135 mmol/l.

A total of 53 of 190 children (27%) with a febrile convulsion developed repeat convulsion. The mean serum sodium level in the group with repeat convulsions was 136.26 mmol/l which was significantly lower than the mean in the group without repeat convulsions. $P = 0.0025$ (<0.05) without repeat convulsions.

The least level of serum sodium recorded was 126 mmol/l, and in 2 of the 53 children of febrile convulsions, hyponatremia was documented. The remaining 51 with recurrent seizures had mean sodium levels of 132.50 mmol/l compared to the mean sodium of 135.75 mmol/l in 137 children with simple febrile seizures.

The relationship between the probability of a repeat convulsion and serum sodium level is significant. The risk approaches 90% in cases with serum sodium levels of 128 mmol/l and gradually decreases to <10% in cases with serum sodium levels of 136 mmol/l. The increase in the probability of a repeat convulsion when the serum sodium level decreases are significant ($P = 0.0025$ [<0.05]).

CONCLUSION

The mean serum sodium level of children with simple febrile convulsions, children with recurrent febrile convulsions, children without fever and convulsions and children with fever and without convulsions are derived from children attending pediatric casualty.

The mean serum sodium level of children with simple febrile convulsion is 135.75 mmol/l. The mean serum sodium level of children with recurrent febrile convulsions is 132.26 mmol/l. The mean serum sodium level of children without fever and convulsion is 140.29 mmol/l. The mean serum sodium level of children with fever without convulsions is 137.87 mmol/l.

The mean serum sodium level of children with simple febrile seizures and the mean serum sodium level of children with recurrent febrile convulsions obtained in our study are almost comparable to those of Hugen *et al*., Department of Pediatrics, Zuiderziekenbuis, Netherland.

In our study among the 53 recurrent febrile seizure children, 49 children (92.4%) had serum sodium level of <135 mmol/l, compared with 45 children (32.8%) of 137 children with simple febrile convulsion. This shows that when the serum sodium level decreases the probability of recurrent febrile convulsion is increased.

Febrile seizures may be extremely frightening events to parents and some may fear that their child is dying during the seizure. If the child through repeat convulsion, the parents are more worried about it.

Measurement of serum sodium is a valuable investigation in the child with febrile convulsion. The lower the serum sodium, higher the probability of a repeat convulsion. This knowledge may be of practical value in deciding whether to admit the child or allow it to return home and in advising parents or caregivers the risk of a repeat convulsion.

In addition, the hyponatremia in children with recurrent conclusion is due to inappropriate secretion of antidiuretic hormone. The inappropriate antidiuretic hormone secretion is best treated by fluid restriction. It seems reasonable, therefore that if a child is admitted to hospital after a febrile convulsion and is not dehydrated, and then an excessive fluid intake should be avoided. Such a policy, however, might be dangerous in a child treated at home.

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