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Original Research Article

Can seizures cause glycemic disorder in children with epilepsy? a cross-sectional study

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ABSTRACT

Background: Aim of this study is to investigate the serum glucose level in children with epilepsy.
Methods: A cross-sectional study was conducted at Pediatric Centre of Hue Central Hospital between June 2017 and June 2019. A serum glucose level test was performed after the seizure.
Results: In total, 144 patients were included in the study. Of these, the most age group was 0 - <5 years (accounted for 59.7%), followed by group 5-10 years (24.3%) and 11-15 years (16%). Female was more likely than male (52.8% vs 47.2%). The most common type was generalized epilepsy (accounted for 72.2%), and the least was un-classified epilepsy (6.3%). The blood test investigated 19 (13.2%) of patients with hyperglycemia and 1 (1.4%) with hypoglycemia.
Conclusions: The present study highlights that seizure can cause glycemic disorder in children with epilepsy.

Keywords: Epilepsy, Seizure, Serum glucose level

INTRODUCTION

Epileptic seizures are events occurring in a close temporal relationship with an acute central nervous system (CNS) insult that may be metabolic, toxic, structural, infectious, or inflammatory in nature.[1] The prognosis after unprovoked seizures and acute symptomatic seizures differs with regard to the risk of seizure recurrence and mortality; therefore, it is essential to define metabolic conditions when acute symptomatic seizures might occur.[2,3]

Electrolyte abnormalities and other metabolic derangements have frequently been associated with epileptic seizures.[4] The International League Against Epilepsy (ILAE) has suggested cut-off values for different laboratory measures of metabolic disorders when seizures should be considered as acute symptomatic rather than unprovoked events.[3] The proposed cut-off values for metabolic derangement are arbitrary and based on case reports, and there are no systematic studies except for seizures due to hyponatremia.[5] Erroneous classification of a seizure as acute symptomatic may delay the diagnosis of an underlying progressive brain disorder, and it is essential that the cut-off values be subjected to systematic evaluation.

Glycemic disorder is frequently encountered in daily clinical practice.[5] Authors aim to investigate the serum glucose level in children with epilepsy in a cross-sectional study.

METHODS

Design and setting

The study design was a cross-sectional observational cohort study. The study was performed at Pediatric

Center of Hue Central Hospital. Serum glucose levels are analyzed as part of routine hospital procedures and stored in the database of the Biochemical Department. The study was approved by the central ethics committee of Hue Central Hospital, and the need for informed consent was waived.

Patient and data collection

Authors performed a search for all children diagnosed with epilepsy.

Inclusion criteria

- Children aged under 16 years who had experienced at least two seizures beyond 24 hours in concordance with ILAE classification in 2010; or children with a prior diagnosis of epilepsy.[6]

Exclusive criteria

- The seizure resulted from an acute situational etiology such as toxin, infection, or trauma. They had a chronic neurologic illness limiting their activities of daily living, such as cerebral palsy, mental retardation, and pervasive developmental disorder. Children were not excluded if it was determined that they had had a seizure that had not been previously recognized as such.

The medical records were carefully inspected for information on demographic and clinical variables including seizures or other disturbances of consciousness within 24 h of the sampling time.[7] The variables collected for the study included the following: age, gender, documented co-morbidities, seizure type and other abnormal hematologic-biochemical analyses when available (e.g., sodium, urea, creatinine, calcium, magnesium and, ammonia). The data were anonymized prior to analysis. Serum glucose level was classified according to the following three categories: normal range: 2.3-6.9mmol/l; hyperglycemia: ≥7mmol/l; hypoglycemia: ≤2,2mmol/l.

Statistical analysis

All calculations were performed using SPSS ver. 16 (SPSS Inc., Chicago, IL, USA). Results are presented as mean ± standard deviation for quantitative variables and percentages (%) for qualitative. The Pearson's Chi-square test was used for categorical variables. A p-value less than 0.05 was considered statistically significant.

RESULTS

In total, 144 patients were included in the study. Of these, the most age group was 0-<5 years (accounted for 59.7%), followed by group 5-10 years (24.3%) and 11-15 years (16%). The median age was 6.3 years (range from 1 month to 15 years). Female was more likely than male (52.8% vs 47.2%) (Table 1).

Table 1: Age and sex distribution.

Age group (year)	n=144	Percentage
<5	86	59.7
5-10	35	24.3
11-15	23	16
Min - Max	1 month - 15 years	
Sex		
Male	68	47.2
Female	76	52.8

Table 2 shows the most common type was generalized epilepsy (accounted for 72.2%) and the least was un-classified epilepsy (6.3%).

Table 2: Classification of epilepsy according to ILAE 2010.

Seizure classification	n	%
Generalized epilepsy	104	72.2
Focal epilepsy	31	21.5
Un-classified epilepsy	9	6.3

The generalized epilepsy consist of these subtypes as shown in Table 3. Tonic-clonic seizure (58.7%), tonic seizure (21.2%), clonic seizure (14.4%), myoclonic seizure (0.9%), absence seizure (2.9%) and atonic seizure (1.9%).

Table 3: Subtype of generalized epilepsy.

Epilepsy subtype	n	%
Tonic-clonic seizure	61	58.7
Tonic seizure	22	21.2
Clonic seizure	15	14.4
Myoclonic seizure	1	0.9
Absence seizure	3	2.9
Atonic seizure	2	1.9

Among 144 patients, the epileptic discharge was found in 97 (67.4%) on EEG (Table 4). The blood test investigated 19 (13.2%) of patients with hyperglycemia (Table 5).

Table 4: EEG discharge.

EEG	n	%
Epileptic discharge	97	67.4
Normal	47	32.6

Table 5: Serum glucose level in children with epilepsy.

Glucose level	n	%
Hypoglycemia	2	1.4
Normal level	123	85.4
Hyperglycemia	19	13.2

DISCUSSION

This study is the first systematic attempt to evaluate the serum glucose level in children with epilepsy. Hyperglycaemia is a well-known phenomenon in electric convulsive therapy.[8] It has been studied in electrically shocked rats, and was found to be related to a rise in the level of catecholamines in the blood and urine of the treated animals. After adrenalectomy, the electric shock is followed by a decrease in the blood level, while vagotomy makes the hyperglycaemic response even more marked. It appears that the hyperglycemia during a convulsive episode is due to stress, is of short duration, and probably has no pathological significance.[9] It may be important to bear in mind because of its possible effects on the glucose level of the CSF. More studies should be undertaken to correlate the blood and CSF glucose levels in convulsive episodes.[10]

An acute neuroglycopenic symptom is a direct result of brain glucose deprivation and includes cognitive impairment, behavioral changes, psychomotor abnormalities, seizures and coma.[11]

Although it is recognized that hypoglycemia may cause neurological symptoms, only limited epidemiological data are available on the relationship between the incidence of seizures and hypoglycemia. In one study in an emergency department, 125 patients were identified with symptomatic hypoglycemia. The majority of the cases had impairment of consciousness, and only 9 cases with seizures were recognized. Of these, 3 had known epilepsy, and the remaining cases were related to alcoholism.[12] This is in line with our results, which indicate that coma is the dominating neurological symptom in hypoglycemia and seizures are rare manifestation.

From textbooks and general overviews, authors are taught that there are many causes of acute symptomatic seizure and electrolytic and metabolic disorders are believed to account for 9%.[5] How many of these cases that can be attributed to hypoglycemia are not known. From this study it is not possible to estimate the incidence of seizures induced by hypoglycemia. The risk of seizures at different blood glucose levels however, seems to be low.

This study has some limitations such as small sample size, the symptoms may also have been instigated or aggravated by co-morbidities and other metabolic disturbances. In addition, the study was performed in a single center, which may limit its generalizability. Importantly, the analysis could provide associations, although inference regarding causality cannot be drawn.

Not withstanding the limitations, in this study of a small number of patients with serum glyceric disorder, a notably low frequency of seizures was noticed. This indicates that the risk of seizures in association with low blood glucose levels seems to be low. This is a finding of potentially great clinical relevance, since seizures in the presence of hypoglycemia are often presumed to be acute symptomatic. Should our findings be validated, a more thorough work-up of patients presenting with seizures in the presence of hypoglycemia, especially at moderate levels, might be warranted. Future large prospective studies will hopefully be useful in predicting a more precise risk estimate for seizures related to hypoglycemia.

CONCLUSION

The present study highlights that seizure can cause serum glycemic disorder in children with epilepsy. Further studies are strongly recommended in the field with a larger sample size to ascertain conclusions drawn from this study.

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