ORIGINAL ARTICLE

Hypoglycemic Seizure: Etiologies and Neurological Outcome in Two Differential Age of Children (Five Year Descriptive Study)

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ABSTRACT

Objectives: Hypoglycemia is a widespread pediatric emergency that can manifest in various ways. One of the most critical symptoms is the occurrence of seizures. Recognizing these episodes promptly is essential in managing the condition effectively. Physicians' apprehension, specifically pediatricians', can lead to early diagnosis and improve the prognosis by decreasing the neurologic aftermath. By presenting data and analysis on the prevalence of hypoglycemic seizures, triggers, and neurologic side effects, we plan to raise awareness of the issue. This study intends to demonstrate the leading causes of hypoglycemia and the major neurological sequels by assessing the prevalence of hypoglycemia in a children's hospital center to raise awareness of the condition.

Materials & Methods: This cross-sectional study was conducted with two parts: demographic data (age, gender), clinical presentations, and lab data (blood sugar) on seizure onset, cause of hypoglycemia, and comorbidities before admission. The second part was based on neurologic evaluation and sequela follow-up.

Results: Collectively, 79 pediatric patients were enrolled in the study, of which 51 were male (64.6%).44.3% had experienced a first episode of hypoglycemic seizures in neonacy, 24.05% in infancy, and 31.65% in childhood. In the neonatal period, poor intake, birth asphyxia, and IUGR are, respectively, the leading causes of hypoglycemic seizures. As for the infant-child group, the most common etiology was the following: diabetic patients treated with insulin, ketotic hypoglycemia, hyperinsulinism, and Glycogen Storage Disease (GSD), respectively. The most common sequelae were global psychomotor delay, isolated speech delay, and isolated motor delay, respectively.

Conclusion: This study reveals that we can help prevent hypoglycemia by maternal education on breast-feeding and avoiding early patient discharge. Regarding the post-neonatal group, parental education on the proper application of insulin can prevent probable seizure and other consequences.

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Introduction

Hypoglycemia is a common pediatric emergency that can present with diverse clinical presentations. Early diagnosis of hypoglycemia can help prevent permanent consequences such as seizures, which can lead to life-long brain damage and neurological impairment, specifically in infants. Although hypoglycemia needs prompt treatment, further evaluation should be performed to prevent the recurrence of the situation and minimize the risk of

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brain damage. Every episode of hypoglycemia can cause permanent damage to the developing brain. According to the American Academy of Pediatrics, children with serum or plasma glucose levels under 40 $^{mg}/_{dL}$ or blood glucose levels below $35^{mg}/_{dL}$ should be treated and assessed, for that matter, and a close follow-up should be considered in children with plasma glucose levels ranging from 40-50$^{mg}/_{dL}$ (1).

The effect of hypoglycemia on the brain depends on the duration and prolongation of hypoglycemia. In many references, the plasma glucose level that causes neuronal damage and changes in the neurophysiological threshold (2) is $47 mg/dl$ (2.5 mmol/L) more than three times on separate days (3-5).

Although these limits are commonly used as references, there are still debates over the normal limit. Some pediatricians believe the limit to be around $60^{mg}/_{dL}$ in order to prevent permanent brain damage in children (6).

Compared to adults, infants and children are prone to experience hypoglycemia much shorter after fasting due to lesser glycogen storage and lesser muscular mass (7, 8). Ketone bodies are significant energy sources for the brain, and free fatty acids cannot be used as a source of energy (9).

Etiologies of hypoglycemia can be divided into five categories: poor intake (gastroenteritis, malnutrition, intercurrent illness), increased metabolism (infection, burning, space-occupying tumors such as Wilms tumor or neuroblastoma), toxins and drugs (insulin, beta-blockers, ethanol, salicylates, oral hypoglycemic agents), endocrine and metabolic diseases (7). Each of the aforementioned conditions has alarming signs other than hypoglycemic seizure occurring before the seizure.

Symptoms of hypoglycemia can be categorized into two groups: the first is neurogenic symptoms caused by falling of the glucose level, such as shaking, anxiety, palpitation, sweating, and the like. The second is neuroglycopenic symptoms (due to decreased availability of glucose in the CNS), including abnormal mentation, confusion, ataxia, headache, and, in severe form, seizure, coma, and death (10).

Researchers show that recurrent episodes of mild hypoglycemia lead to decreased brain awareness of hypoglycemia by a decline in autonomic and counter-regulatory responses, causing increased susception of hypoglycemic episodes (11) and also recurrence of hypoglycemia, specifically in the developing brain, can cause permanent but unmeasurable damage (12). Even moderate hypoglycemia can considerably increase low-frequency EEG activity and cause cognitive disturbances, while severe prolonged hypoglycemia can cause neuronal death and damage (13).

Although hypoglycemia can affect all age groups, it is more prevalent in neonates and infants. Duration of hypoglycemia is one of the most essential determinants of damage. Apart from duration, neurological defects are closely related to the underlying etiology of hypoglycemia, therefore rendering it much more challenging to estimate (9).

Acquaintance with hypoglycemia, its etiology, and its prevalence can prompt early diagnosis, mainly in pediatrics.

This study intends to demonstrate the leading causes of hypoglycemia and the major neurological sequels by assessing the prevalence of hypoglycemia in a children's hospital center in order to raise awareness of the condition.

Materials & Methods

This study is a descriptive-cross sectional study conducted on patients admitted to referral children's hospital in the years 2012-2017 who were diagnosed with hypoglycemia-provoked seizures. A questionnaire was designed consisting of two major parts: 1. Demographic information (age, gender), growth indexes, clinical findings (pigmentation of the skin, jaundice, hepatomegaly, cataract, micropenis, and the like), lab data (serum glucose level during the seizure, further lab data related to the suspected underlying cause), etiology of the hypoglycemia induces seizure (disease diagnosis based on ICD-10), comorbidities and history of seizure. The second part was completed on the follow-up consisting of age, the time gap between the onset of a hypoglycemic seizure and the present, further lab data, and the neurological sequel and neurologic evaluation (global psychomotor delay, isolated speech delay, isolated motor delay, and epilepsy). The informed consent form was obtained from all patient's parents or representatives. To optimize the conclusion, the neurological sequels were divided into two groups: Group 1 (milder symptom) consisted of isolated speech delay, isolated motor delay, and pharmaco-sensitive epilepsy. Group 2 (more severe symptoms) consisted of global psychomotor delay and pharmaco-resistant epilepsy. The neurological development upon follow-up was conducted by physical examination upon visit or via phone call. The gap between hypoglycemic seizure and the follow-up ranged between four months and six years. This study was approved by the National Committee for Ethics in Biomedical Research, Department of Pediatrics, Tehran University of Medical Sciences (9311165031).

Statistical analysis

Data was further analyzed by SPSS v22. Qualitative data was reported by prevalence (percentage), and quantitative data was reported by mean± standard deviation. Depending on the variable, t-test and chi2 tests were used to evaluate and compare the data assessed in two different periods. If the data distribution was not in accord with the normal distribution, the alternative assessment tests were Fisher, the exact test and Mann-Whitney. All the personal information gathered in this article was kept strictly confidential and was for the sole purpose of research.

Results

Seventy-nine patients were qualified to enter the research, of which 51(64.6%) were male and 28(35.4%) were female. The hypoglycemic seizure occurred in 35 (44.3%) of the patients in the neonatal period, 19 (24.05%) in infancy (the first year of life), and 25 (31.65%) in their childhood (after the first year of life). The mean blood sugar level in the neonatal group was estimated to be around 20.1±9.5 and 29.9±14.7 in the infant-child group. The blood sugar level range was from a minimum of eight to a maximum of 75 in a diabetic patient following the insulin injection (Figure 1).

In this assessment, the most common etiologies were poor intake, birth asphyxia, and IUGR/ SGA (intrauterine growth retardation/small for gestational age), respectively, in the neonatal group. However, in the infant-child group, the leading etiology was misuse of insulin in diabetic patients. Ketotic hypoglycemia and Glycogen Storage Disease (GSD) were the less common etiologies (Table 1).

Table 1: hypoglycemic seizure etiologies in neonates and infant-child group

Age	Etiology	frequency	Percent
Neonate	Transient Hyperinsulinism	1	2.9
	Hyperinsulinism	3	8.6
	Discordant twin	3	8.6
	Infant of diabetic mother	2	5.7
	Poor intake-Starvation	10	28.6
	Sepsis	1	2.9
	IUGR-SGA	5	14.3
	Birth Asphyxia	6	17.1
	Unknown	3	8.6
	Other Inborn error of metabolism	1	2.9
	Total	35	100.0
Infant-Child	Transient Hyperinsulinism	1	2.3
	Hyperinsulinism	6	13.6
	Ketotic Hypoglycemia	7	15.9
	Adrenal Insufficiency	5	11.4
	Poor intake-Starvation	3	6.8
	Sepsis	2	4.5
	DM-Insulin	10	22.7
	Unknown	1	2.3
	GSD	6	13.6
	Other Inborn errors of metabolism	2	4.5
	Infantile liver failure	1	2.3
	Total	44	100

In the infant-child group, the age of onset in the ketotic hypoglycemia patients was from ten months to six years of age (15.9% of the group). Seizure due to hypoglycemia in these patients was recurrent.

Six (13.6%) of the patients who had experienced seizures between one month and 40 months of life were diagnosed with GSD. However, due to financial reservations, only one patient's diagnosis of GSD was confirmed by genetic testing, and the rest were clinically diagnosed (based on liver biopsy and enzyme assay). 11.4% of the patients in this group were due to adrenal insufficiency. The age of onset in this group varies from two months to 36 months. Of these patients, one was diagnosed with pseudo-hypoaldosteronism and died in the follow-up, one was diagnosed with ACTH unresponsiveness, and another with congenital adrenal hypoplasia. Two cases were inborn errors of metabolism (4.5%). They were diagnosed with Glutaric aciduria and organic acidemia. One of the cases who had experienced recurrent seizures from 40 days of age and had been on anti-epileptic drugs was diagnosed with insulinoma at the

age of six months by PET scan and then underwent sub-total pancreatomy. Another case with the onset at two years and six months of age underwent genome sequencing with clinical suspicion of mitochondrial disease and was then diagnosed with Infantile Liver failure syndrome type 2.

In the neonatal group, six (17.1%) of the patients had a history of newborn ischemia and were admitted with hypoglycemic seizure.

Follow-up findings

Of the 79 patients enrolled in this study, 66 (83.5%) were available. Of these 66 patients, five cases (6.3%) were deceased. Despite the effort, 13 cases (16.5%) did not fully cooperate. Most cases were followed up after a 3-year period (4 months to 6 years)

Neurological sequels on the follow-up

The diagnosis of epilepsy is determined by the recurrence of unprovoked seizure more than once with an abnormal EEG in this study. Amongst neonates with hypoglycemic seizures follow-up, 11 (31.4%) were treated for epilepsy, whereas in the infant-child group follow-up, 30 (68.2%) were diagnosed with epilepsy (because of the recurrence of seizures in the later years). No statistically significant difference was found between the two groups with a P value of less than 0.05. Global psychomotor delay was reported in seven cases of the neonatal group (20%) and six of the infant-child group (13.8%). No statistical significance was observed between the two groups. Isolated motor delay was only reported in the infant-child group (2 patients, 4.5%). The prevalence of mild neurological sequels sorted by the underlying etiology in neonatal and infant-child groups is shown in two diagrams (Figure 1, 2). No statistically significant correlation was found between the level of blood sugar at the time of seizure and the prevalence of neurological impairments such as epilepsy or global psychomotor delay.

EEG was the most common paraclinical modality for assessing these patients over the past six years. Collectively, EEGs were performed on 60 out of 79 patients. Among these, 37 cases (46.8%) showed abnormal results, according to a report by one child neurologist. This study did not assess MRI findings.

Discussion

Hypoglycemic seizure in the infant-child group

In the infant-child group, most seizures were reported in diabetic patients treated with insulin (22.7%). In many recent pediatric articles, a concurrence has been reported between diabetes and epilepsy, which appears to be non-random (14, 15). In order to establish a valid non-random correlation between the two, we further investigated the cases. Physiopathology assessment articles show that although seizure etiology in these patients seems to be hypoglycemia caused by insulin misuse, GAD antibodies (specific auto-immune pathologic antibodies against CNS) also play a significant role (16, 17). Recurrent hypoglycemia episodes were shown to predispose to seizure in articles. In this study, 11% of the type 1 diabetic patients had an abnormal EEG, with the pattern most common in ones with a history of hypoglycemic seizures (18). In some studies, a correlation was found between hypoglycemia in diabetic patients and EEG abnormalities (19).

In a cohort study carried out in 2012, amongst 45851 patients diagnosed with type 1 DM, 700 were diagnosed with epilepsy, and half of these were treated for it. This study suggested an increase in the prevalence of seizure in diabetic patients (16).

The next leading cause of seizure amongst our patients was ketotic hypoglycemia, causing 15.9% of the cases. The age of onset was between ten months to six years of age. Seizure was mostly recurrent in these cases. In another study, twenty patients were introduced to ketotic hypoglycemia who had a history of hypoglycemic seizures in the first 36 hours of life, four of whom were non-identical twins (20).

In the present study, one case was reported to have had hypoglycemia-provoked seizure in neonacy. This patient was the youngest of the triplets.

Hyperinsulinism and GSD are the most common inborn errors of metabolism causing hypoglycemic seizures. Many studies were conducted on this matter. In 2013, a study was conducted on the length of treatment and neurological development of 17 patients with hyperinsulinemic hypoglycemia. Seizure due to hypoglycemia was the most common clinical manifestation reported in 13 (76.5%) of these cases. The most common neurological sequels were epilepsy, developmental delay, and psychomotor delay (21).

One of the studied cases had recurrent seizures from 40 days of age and had been treated for epilepsy. At six months of age, after further clinical investigation and a PET scan, he was diagnosed with insulinoma and underwent sub-total pancreatomy. A similar case was reported in a 9-year-old girl in another study emphasizing the importance of measuring blood sugar in infants and children presented with seizure (22).

Six (13.6%) of the patients who experienced seizures from one month to 40 months of age were diagnosed with GSD in this study. In 2012, another study reported 21 GSD patients (9 females and 12 males) with a mean age of hypoglycemia onset of 3.8 months (12)].

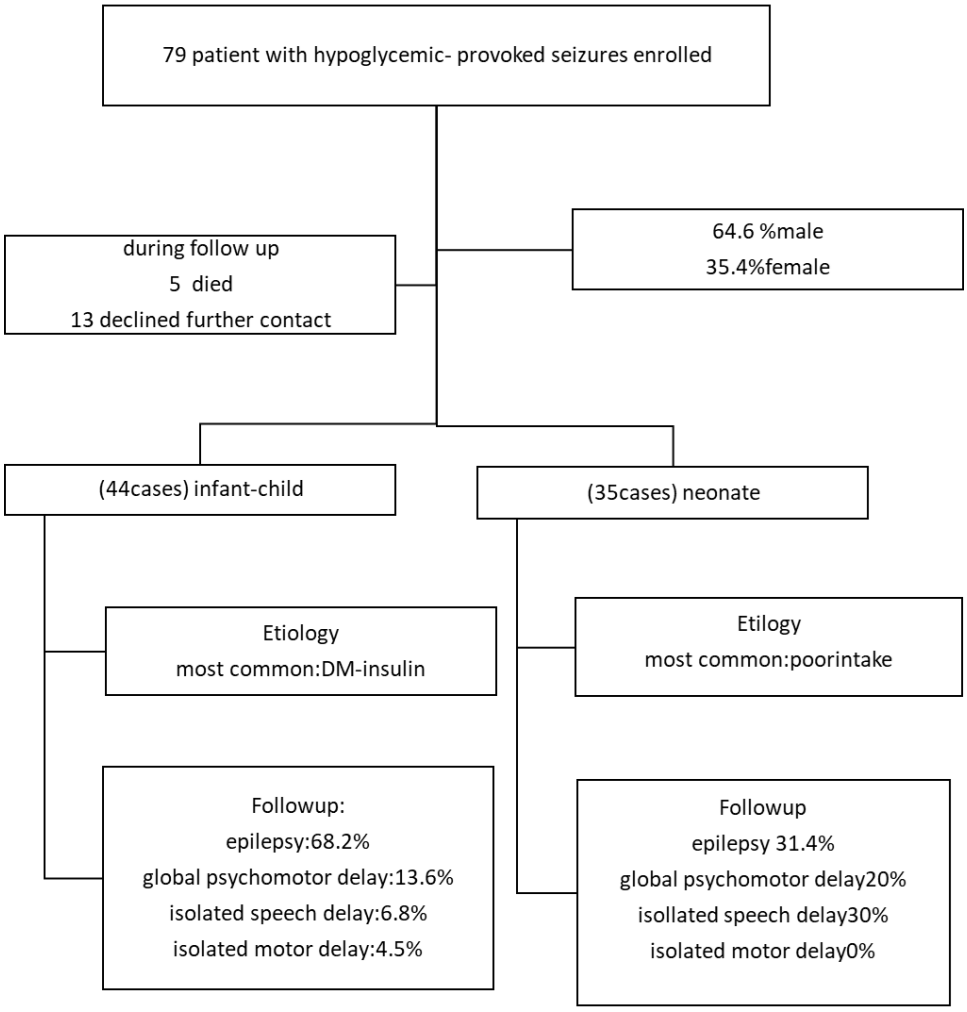

Figure 1: Flowchart of the research overally

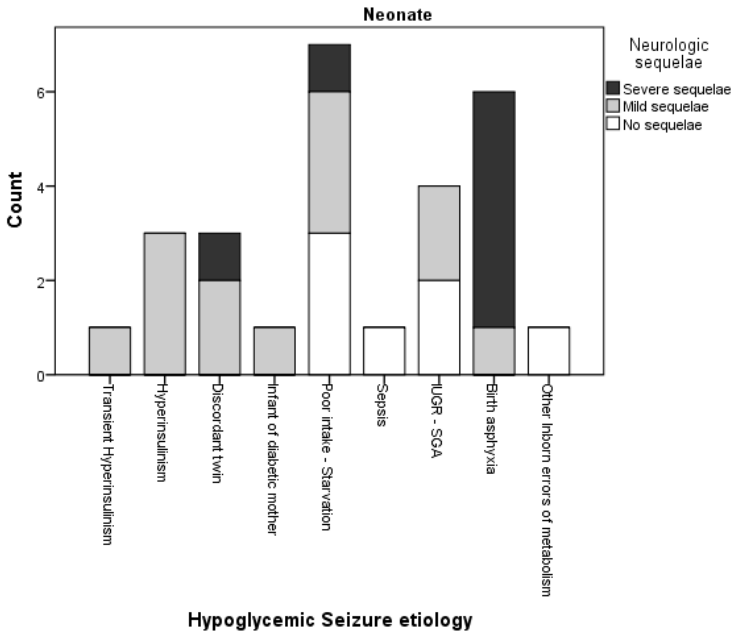

Figure 2: neurological sequelae in neonatal group sorting by the hypoglycemic etiology

11.4% of hypoglycemic seizures in this study were due to adrenal insufficiency, with an onset between two months and three years of age. Among them was a case of pseudo-hypoaldosteronism involving a deceased individual, a case of ACTH unresponsiveness, and a case of congenital adrenal hyperplasia. In research of 74 CAH patients, only 16 had episodes of hypoglycemia, mostly after the first year of life following an infection, and nine of these had hypoglycemic seizure (23).

In another 2010 study, the prevalence of hypoglycemia in CAH patients was estimated to be 10% (3 in the flame-up of the disease), and therefore, increased glucose intake plus the corticosteroid stress dose was emphasized to prevent hypoglycemia (24).

Other cases included inborn errors of metabolism, glutaric academia, and organic acidemia.

Hypoglycemic seizure in the neonatal group

When the neonate presents with a new onset of seizure, many critical etiologies should be considered and ruled out. Hypoglycemia is one of the leading causes at this age (25).

In 2014, a study showed that 5.9% of neonatal seizures were due to hypoglycemia (26). In this study, the most common etiology was poor oral intake, considering early discharge from the nursery, lack of appropriate instructions on breast-feeding and absence of breast-feeding assessment by mothers. In this study, all these cases are referred to the first week of life. In a case report study, the term, breast-fed neonate was referred to the third day of life with hypoglycemic seizure, believed to be because of early discharge from the hospital (27).

In the current study, six cases (17.1%) of birth asphyxia were admitted with hypoglycemic seizure.

Level of blood sugar during seizure and the neurological sequels

This research found no statistically significant correlation between the blood sugar level and neurological sequels. Meanwhile, a correlation was reported in Fong et al.'s 2014 research (28).

Side effects of hypoglycemia and seizure

A statistically significant correlation was not reported between the age of seizure onset and the prevalence of neurological sequels, which was also concluded in a study (29).

In another study of 34 patients with a history of neonatal seizure, 23 were diagnosed with epilepsy, of which five had refractory epilepsy. Developmental delay, learning and behavioral disabilities, attention-deficit/hyperactivity disorder, and autistic presentations were some of the reported neurological sequels (30).

Based on the present findings, severe neurological sequels in the neonatal period were mainly due to birth asphyxia and hyperinsulinism cases in the infant-child group. Although hypoglycemia has been proven to be the most common electrolyte abnormality seen in neonates with birth asphyxia (31), it appears that accounting for all the neurological sequels due to hypoglycemia is biased since asphyxia itself can cause several neurological damages.

Neurological sequels ranging from mild to severe were reported in all hyperinsulinism patients in our study. In a 2014 study in China, neurological sequels were reported in 50% of 27 hyperinsulinism patients (32).

Study limitation

Incomplete files and non-cooperation of parents in answering the questionnaire.

In Conclusion

A seizure is a symptom that can indicate an underlying issue. It may occur due to an electrolyte imbalance, such as low blood sugar (hypoglycemia). Early diagnosis and prompt treatment of this electrolyte imbalance are crucial for managing the condition early and preventing its sequels.

The present research aimed to clarify the etiologies of hypoglycemia, which can cause seizures, in order to draw the attention of medical doctors, specifically pediatricians. The first step is to measure the blood sugar during the seizure to avoid any delays. If the patient is proven to be hypoglycemic, the second step would be to consider all the aforementioned possible etiologies. In the neonatal group, poor feeding is the leading cause of hypoglycemia. Therefore, we can help prevent hypoglycemia by maternal education on breast-feeding and avoiding early patient discharge. Regarding the post-neonatal group, parental education on the proper application of insulin can prevent probable seizure and other consequences.

The researchers hope to extend their study, form a multi-centered study, and have more accurate follow-ups in the future.

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Authors' Contribution

Nahideh Khosroshahi designed the work, and approved the final version to be published. Maryam Hassani acquired and analyzed of the data. The patients were referred to our medical team by Kamyar Kamrani and Zahra Haghshenas. Nika Keshtkaran drafted the article and Simin Khayatzadeh Kakhki drafted the article and correspond the manuscript.

All authors gave final approval and agree to all aspects of work.

Conflict of Interest

The authors declared no conflict of interest.

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