EJMCM_Volume_7_Issue_8_Pages_4664-4668_1.pdf

by

Submission date: 26-Oct-2022 03:16PM (UTC+0700)
Submission ID: 1935778830
File name: EJMCM_Volume_7_Issue_8_Pages_4664-4668_1.pdf (192.95K)
Word count: 2626
Character count: 15443

European Journal of Molecular & Clinical Medicine
ISSN 2515-8260 Volume 07, Issue 08, 2020

Seizure Due To Electrolyte Imbalance In Pediatric

Nurul Hidayah[1], Huldani[2], Fauziah[3], M Naufal Risyad[3]

[1]*Department of Pediatrics, Faculty of Medicine, University of Lambung Mangkurat, Banjarmasin, South Kalimantan*
[2]*Department of Physiology, Faculty of Medicine, University of Lambung Mangkurat, Banjarmasin, South Kalimantan*
[3]*Doctor's Professional Education, Faculty of Medicine, Faculty of Medicine, Lambung Mangkurat University, Banjarmasin, South Kalimantan*

E-mail: [1]hidabella@yahoo.com

ABSTRACT
Seizures are symptoms that are temporary and sudden as a result of abnormal electrical activity in the brain. Seizures can be caused by various conditions, for example, epilepsy, fever, hypoglycemia, hypoxia, hypotension, brain tumors, meningitis, electrolyte imbalance, and drug overdose. Severe and acute electrolyte disturbances cause neurological deficits such as seizures, which may be the sole presenting symptom. Electrolytes are compounds in a solution that dissociate into particles with a positive (cation) or negative (anion) charge. Most of the metabolic processes require and are affected by electrolytes. Electrolyte imbalances are common in clinical practice and the diagnosis is confirmed by laboratory tests. Seizures are common in patients with hyponatremia, hypocalcemia, and hypomagnesia. Tonic-clonic seizures or other types can be found due to these electrolyte disturbances. Hypo or hyperkalemia can also cause seizures, but it is very rare. These electrolyte disturbances can trigger epileptic areas in the brain. Central nervous system disorders due to electrolyte imbalance are reversible. However, if this is not treated immediately it will cause permanent brain damage.

Key Words: electrolyte imbalance, pediatric, physiology, seizures.

1. INTRODUCTION

A seizure is a symptom that occurs temporarily and suddenly as a result of abnormal electrical activity in the brain. Disturbance of electrical activity that is limited to certain brain areas can cause partial seizures. However, if the disruption of electrical activity occurs in all areas of the brain, it causes generalized seizures. Seizures can be caused by various conditions, for example, epilepsy, fever, hypoglycemia, hypoxia, hypotension, brain tumors, meningitis, electrolyte imbalance, and drug overdose (Goldenberg, 2010).

The incidence of generalized seizures is 3-4% with a peak incidence in early life (neonates). Almost 30% of seizures are experienced by children. Seizures occur in 3-5% of children aged <5 years of age who have a fever. Seizures are also a symptom of epilepsy. The Incidence of epilepsy in developing countries is 139/100,000 compared to developed countries of 49/100,000 people (Martindale et al, 2011; Lee et al, 2012).

Electrolytes are compounds in a solution that dissociate into positively or negatively charged particles (ions). Positive ions are called cations and negative ions are called anions. Most of

the metabolic processes require and are affected by electrolytes. Abnormal electrolyte concentrations can cause many disturbances (Darwis et al., 2008; Matfin et al., 2009).
Electrolyte imbalances are common in clinical practice and the diagnosis is confirmed by laboratory tests. Electrolyte disorders can affect the nervous system. The severe and acute electrolyte disturbances are causing neurological deficits such as seizures. Seizures are common in patients with hyponatremia, hypocalcemia, and hypomagnesia. Tonic-clonic seizures and other types can be found due to these electrolyte disturbances (Castilla-Guerra et al., 2006; Beghi et al., 2010).
Central nervous system disorders due to electrolyte imbalance are reversible. However, if this is not treated immediately it will cause permanent brain damage (Nardone et al., 2016).

2. DISCUSSION

There are two types of neurotransmitters, namely excitation neurotransmitters that facilitate depolarization of electric charge and inhibitory neurotransmitters (inhibitory of nerve electrical activity in the synapse) which cause hyperpolarization so that neuron cells are more stable and do not easily release the electricity. Excitation neurotransmitters are glutamate, aspartate, norepinephrine, and acetylcholine. And inhibitory neurotransmitters, for example, gamma-aminobutyric acid (GABA) and glycine. If the effect of both types releases electric charge and impulse transmission occurs. In a resting state, the neuron membrane has a certain electric potential and is polarizing. The action potential will trigger depolarization of the neuron membrane and all cells will release an electric charge (Haurer, 2013).
Various factors can change the function of the neuron membrane so that the membrane is easily passed by calcium and sodium ions from the extracellular to intracellular. Calcium influx will trigger membrane depolarization burst and release excessive, irregular, and controlled electric charge. The synchronous discharge of a large number of neurons is the basis of a seizure. A characteristic feature of epileptic seizures is that the attacks stop for a while as a result of the inhibition process. It is suspected that this inhibition is the influence of neurons around the epileptic site. There are also pre and post-synaptic inhibition systems that ensure that the neurons do not continuously discharge. Another condition that can cause an epileptic seizure to stop is the exhaustion of neurons due to the depletion of substances essential for brain function (Haurer, 2013).
Sodium is the predominant extracellular cation, combined with other anions which account for nearly all solutes osmotically in plasma and interstitial fluid. Sodium is the main determinant of ECF volume due to the relatively free movement of water throughout the fluid compartment. Only 10% of all sodium in the body is in intracellular space. The concentration gradient between intracellular and extracellular compartments (ratio 1:15) is maintained by ATPase and is very important for the function of excised tissue, including action potential and membrane potential (Sirait, 2019).
Hyponatremia is a condition where the serum sodium level is <135 mEq/L and severe hyponatremia if concentration is <125mEq/L. The etiologies of hyponatremia include depletion of circulating volume in cases of CHF, cirrhosis of the liver, diarrhea; ADH hormone disorders in adrenal insufficiency, hypothyroidism, pregnancy, surgery; polydipsia and drugs (thiazide diuretics, desmopressin, mannitol, sorbitol, carbamazepine, carbamazepine, and eslicarbazepine) (Nardone et al, 2016).
Clinical manifestations of hyponatremia affect the central nervous system, this condition causes cerebral edema which triggers symptoms of increased ICP such as decreased consciousness and seizures. Severe hyponatremia has a rapid onset, as are generalized tonic-clonic seizures at sodium levels <115 mEq/L. In a retrospective study, 70% of the causes of

seizures without fever in infants <6 months were hyponatremia. (Nardone et al, 2016; Bhardwaj, 2006).

Hyponatremia produces a nonspecific slow down of EEG waves. Severe hyponatremia causes delta wave activity, as well as several waves such as triphasic, high-voltage, and central high-voltage 6-Hz, causing paroxysmal delta waves. Hyponatremia due to polydipsia precipitates epileptic activity in the left frontal area, giving rise to status epilepticus (Nardone et al., 2016; Bhardwaj, 2006).

Hyponatremia categories are divided into three according to the status of fluids in the body, namely hypovolemia hyponatremia, euvolemia hyponatremia, and hypervolemia hyponatremia. The management needs to be adjusted to the category.

Research by Imaduddin et al showed that most cases of febrile seizures were accompanied by hyponatremia (80.4%) and there were no cases with hypernatremia. There was no significant difference in serum sodium values in cases of first seizure and repeated seizures. In another study conducted by Nickavar, it was found that the mean serum sodium value of children who suffered from febrile seizures was lower than children who did not have febrile seizures, but there was no significant difference in serum sodium values between first and recurring febrile seizures (Nickavar et al, 2009).

Research by Hawas et al, on the effect of electrolytes on the pathomechanism of febrile seizures, found that 150 children aged 6 - 60 months were divided into three groups. Group A 50 children with febrile seizures, group B 50 children with fever without a seizure, and group C 50 healthy children. The results of the study in the form of sodium and potassium levels were significantly lower in cases of febrile seizures compared to the control group (Hawas et al, 2018).

Research conducted by Zeng-Quan also showed a significant decrease in sodium values in patients with febrile seizures compared to the group without febrile seizures. This can be caused by mutations in genes that regulate sodium channels. This mutation results in a prolonged active process of the sodium ion channel gate. Activation of the door to the sodium ion channel results in the entry of sodium into the cell resulting in depolarization. Prolonged opening of the sodium ion channel door can increase the excitability of neuron cell membranes (Bahtera et al., 2009; Ganong, 2008).

Hypernatremia often follows seizure activity (especially generalized tonic-clonic seizures). This condition is characterized by serum sodium >145 mEq/L. The etiology of hypernatremia such as massive fluid loss (infant, elderly, diarrhea, nephrogenic central diabetes insipidus, mannitol), excess sodium on hypertonic fluid administration, water transfer out of cells (post convulsions, excessive physical exercise). During seizures, intracellular glycogen metabolizes lactic acid. Increased cellular osmolality, water moves outside the cell causing hypernatremia. A few minutes later there was dehydration of brain cells. Acute hyperosmolar hypernatremia will result in encephalopathy. Chronic hypernatremia is characterized by minimal neurological symptoms. Sodium levels> 180 mEq / L increase mortality, this incident is more common in adults than children. In infants, seizures due to hypernatremia rarely occur, except in conditions of excessive rehydration (Wang, 2010; Nass et al, 2017).

Calcium is the second most important intracellular messengers, playing a key role in muscle contraction, neuromuscular transmission, cell division and movement, and oxidative pathways. It also plays a role in the formation of bone structure (Sirait, 2019).

Decreased serum calcium causes neuromuscular hyperexcitability. Paresthesia, weakness, cramps, fasciculations, and tetany are some of the signs of peripheral neuromuscular. The latent tetany induced by Chvostek's sign (spasm of the facial muscles when pressing on the facial nerve) and Trousseau sign (carpal spasm after inflation of blood pressure to 20 mmHg

above systolic blood pressure for 3 minutes) are signs of hypocalcemia. CNS manifestations of hypocalcemia include depression, confusion, and seizures (Nardone et al, 2016).
Ion calcium plays a role in depolarization. A decreased serum calcium concentration can reduce the amount of depolarization needed to trigger changes in the conductance of sodium and potassium. This increases the excitability of nerve cells and muscle cells which can lead to seizure generation (Ganong, 2008).
Potassium is a cation that has a very large number in the body and is mostly intracellular. Approximately 98% of the potassium content is in intracellular fluid. Potassium functions in protein synthesis, muscle contraction, nerve conduction of hormone release, fluid transport, fetal development (Sirait, 2019).
To maintain the stability of intracellular potassium, an electrochemical balance is needed, which is a balance between the ability of negative charges in cells to bind potassium and the ability of chemical forces to push potassium out of cells (Sirait, 2019).
The concentration of potassium in the extracellular fluid reflects the balance between the intake of potassium through the ion pump process in the epithelium of the gastrointestinal mucosa and its excretion in urine. This equilibrium results in a rigid plasma level of potassium between 3.5–5 mEq/L. The excretion of potassium is regulated by the activity of the ion pump mechanism along the distal part of the nephron and collecting tube. When sodium reabsorption takes place in the renal tubules, there is an exchange with potassium in the peritubular tissue (Sirait, 2019).
Akbayram in his research found that the value of potassium in patients with febrile seizures was lower than in the control group. The opening of the potassium ion channel door resulted in hyperpolarization. This situation is an inhibitory mechanism that can inhibit action potential. The opening of the potassium ion channels causes potassium to leave the cell so that the concentration of potassium outside the cell will increase. The decrease in serum potassium value can reduce the inhibitory mechanism. Inhibition mechanisms that are not comparable to excitation mechanisms can trigger seizure seizures (Akbayram et al., 2012; Bahtera et al., 2009).
Magnesium has a variety of cellular actions, including modulation of ion channel activity and as an essential component of ATP production and hydrolysis. These are primarily intracellular anions, although they are predominantly located within organelles, bound to phospholipids, proteins, and nucleic acids.
Hypomagnesia is a condition in which magnesium levels <1.6 mEq/L, magnesium <0.8 mEq/L are classified as severe hypomagnesia. The etiology of hypomagnesia includes diarrhea, excessive use of laxatives, and drugs (diuretics, cyclosporine, aminoglycosides). Magnesium functions to maintain cell membrane stability and reacts with glutamate aspartate receptors, thereby activating neuronal depolarization. Hypomagnesia often manifests at levels <1.2 mEq/L, the symptoms that appear include neuromuscular irritability, CNS hyperexcitation, arrhythmias, seizures (tonic-clonic, often in neonates and adults with Mg levels <1 mEq/L) (Nardone et al., 2016; Gandhi et al, 2012).

3. CONCLUSION

Seizures have multifactorial etiologies, one of which is electrolyte balance disorders in the form of hyponatremia, hypocalcemia, and hypomagnesia. Hypo or hyperkalemia can also cause seizures, but it is very rare. Central nervous system disorders due to electrolyte imbalance are reversible. However, if this is not treated immediately it will cause permanent brain damage.

REFERENCES

[1]. Goldenberg, M.M. 2010. Overview of Drugs Used for epilepsy and Seizures. P & T. p36:7.
[2]. Martindale JL, Goldstein JN, Pallin DJ. 2011. Emergency department seizure epidemiology. Emerg Med Clin North Am. 29:15‑27.
[3]. Lee JH, Kim JH. 2012. Comparison of serum zinc levels measured by inductively coupled plasma mass spectrometry in preschool children with febrile and afebrile seizures. Ann Lab Med. 32:190‑3
[4]. Darwis D, Moenajat Y, Nur B.M, Madjid A.S, Siregar P, Aniwidyaningsih W, et al. 2008. Fisiologi Keseimbangan Air dan Elektrolit dalam Gangguan Keseimbangan Air-Elektrolit dan Asam-Basa, Fisiologi, Patofisiologi, Diagnosis dan Tatalaksana. Ed 2. Jakarta: Universitas Indonesia. p29-114.
[5]. Matfin G. and Porth C.M. 2009. Disorders of Fluid and Electrolyte Balance In: Pathophysiology Concepts of Altered Health States, 8th Edition. McGraw Hill Companies USA. p761-803
[6]. Castilla-Guerra L, del Carmen Fernández-Moreno M, LÓpez-Chozas JM, Fernández-Bolaños R. 2006. Electrolytes disturbances and seizures. Epilepsia. 47:1990-1998.
[7]. Beghi E, Carpio A, Forsgren L, Hesdorffer DC, Malmgren K, Sander JW, et al. 2010. Recommendation for a definition of acute symptomatic sei¬zure. Epilepsia. 51:671-675.
[8]. Nardone R, Brigo F, Trinka E. 2016. Acute symptomatic seizure caused by electrolyte disturbances. J Clin Neurol. 12(1):21-33.
[9]. Haurer Stephen L. 2013. Harrison"s Neurology In clinical Medicine. USA: Mc Graw Hill Education.
[10]. Sirait R.H. 2019. Bahan Kuliah Fisiologi Cairan Tubuh dan Elektrolit. Jakarta: Fakultas Kedokteran Universitas Kristen Indonesia.
[11]. Bhardwaj A. 2006. Neurological impact of vasopressin dysregulation and hyponatremia. Ann Neurol. 59:229-236
[12]. Nickavar, Azar., Hosein Hasanpour, dan Kambiz Sotoudeh, 2009. Validity of Serum Sodium and Calcium Screening in Children with Febrile Convulsion. Acta Medica Iranica. 47 (3): 229-231
[13]. Hawas AF, Al-Shalah HH, Al-Jothary AH. 2018. The impact of electrolytes in pathogenesis of simple febrile convulsions. Med J Babylon. 15:12-5.
[14]. Bahtera, Tjipta., Susilo Wibowo, AG Soemantri Hardjojuwono. 2009. Faktor Genetik Sebagai Risiko Kejang Demam Berulang. Sari Pediatri. 10: 378-384
[15]. Ganong, W. F. 2008. Buku Ajar Fisiologi Kedokteran Edisi 22. Alih bahasa Brahm U. Pendit. EGC: 41-72
[16]. Wang NE. 2010. Management of electrolyte emergency. Emergency Medicine Review Board. (8):3.
[17]. Nass R, Sassen R, Elger C, Surges R. 2017 The role of postictal laboratory blood analyses in the diagnosis and prognosis of seizures. British Epilepsy Association. 47:51–65.
[18]. Akbayram, Cemek M, Büyükben A, Aymelek F, Karaman S, Yilmaz F, et al. 2012. Major and Minor Bio-element Status in Children with Febrile Seizure. [Abstrac]. Bratisl Lek Listy. 113(7): 421-3
[19]. Gandhi NY, Sharif WK, Chadha S, Shakher J. 2012. A patient on long-term proton pump inhibitors develops sudden seizures and encephalopa¬thy: an unusual presentation of hypomagnesemia. Case Rep Gastro¬intest Med 2012. 632721

EJMCM_Volume_7_Issue_8_Pages_4664-4668_1.pdf

ORIGINALITY REPORT

11%	7%	6%	3%
SIMILARITY INDEX	INTERNET SOURCES	PUBLICATIONS	STUDENT PAPERS

MATCH ALL SOURCES (ONLY SELECTED SOURCE PRINTED)

4%

★ aneskey.com

Internet Source

Exclude quotes	On	Exclude matches	< 2%
Exclude bibliography	On		