

Calcium, Phosphorous and Magnesium in Epilepsy Cases

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Abstract: Prolonged or repeated seizures/epilepsy stimulates cerebral metabolism results in increase in cerebral consumption of oxygen, glucose and resulted into lactic acidosis, hypotension, hypoxia and impaired membrane regulation of ion fluxes and abnormal synaptic transmission where calcium, phosphorous and magnesium plays important role in stabilization, conduction of impulses etc. Magnesium plays protective role in prevention of hyper-excitability. Therefore calcium, phosphorous and magnesium status was assessed in epilepsy cases.

Keywords: calcium, epilepsy, magnesium, phosphorous, seizure.

1. Introduction

Epilepsy is characterized by sudden recurrent and transient disturbances of mental function or movements of the body resulting in metabolic changes in brain. Defective control of cation channels may cause unstable, readily depolarized neuron that implicate in some forms of epilepsy.

Trace elements like calcium, phosphorous and magnesium plays important role in many physiological functions, stabilization of membrane function. Calcium plays important role in the release of neurotransmitters in the brain and all preganglionic synapsis, postganglionic parasympathetic nerve endings, neuromuscular junction and adrenal medulla. Musculoskeletal symptoms are commonly resulted from phosphorous deficiency, diffuse weakness is severe in proximal muscle groups. Serum calcium levels are controlled in narrow range (Moe, 2008). Magnesium ion functions as a cofactor in almost in all cellular metabolism, thus impaired mimic of myriad of other disease processes. Painful muscle cramp may be early sign of magnesium deficiency. Magnesium is essential for regulation of muscle contraction (Grober et al., 2015).

2. Materials and Methods

A. Methodology

In all 50 control cases and 50 epilepsy cases blood samples were assessed for their calcium, phosphorous and magnesium status. The serum samples were subjected for calcium, phosphorous and magnesium.

Estimation of calcium was done as per Trindar (1956). Whereas, phosphorous was done as per Fiske and Subbarow (1925) and magnesium as per Neill and Neely, 1956.

The comparison of control and epilepsy status of calcium, phosphorous and magnesium was done by using paired t test (Snedecor and Cochran, 1980).

3. Results and Discussion

Serum calcium, phosphorous and magnesium levels were significantly decreased in epilepsy cases than control groups (Table 1).

Table 1
Comparison of (Mean \pm SE) of control and epilepsy group

Parameters	Control (n=50) Mean \pm SE	Epilepsy (n=50) Mean \pm SE
Calcium (mg/dl)	9.733 \pm 0.088	8.116 \pm 0.149**
Phosphorous (mg/dl)	3.956 \pm 0.076	3.458 \pm 0.055**
Magnesium (mg/dl)	2.403 \pm 0.715	2.197 \pm 0.063*

***= (p \leq 0.01)

The lowering of calcium levels might be triggering the conduction and precipitating seizures/epilepsy. Hypomagnesaemia along with hypocalcaemia might had precipitated the seizures/epilepsy. Mori et al., 1997 also reported hypocalcaemia. Gupta et al., 1994 also reported hypomagnesaemia in idiopathic epilepsy. Hypomagnesaemia might have resulted in neuromuscular hyper excitability in epilepsy cases (Galland, 1991). Therefore, assessment of calcium, phosphorous and magnesium is important in epilepsy cases.

References

- [1] Fiske, C. H. and Subbarow, Y. J. (1925) The colorimetric determination of phosphorus. *Biol. Chem.*, 66, 375-400.
- [2] Grober, U.; Schmidt, J.; Kisters, K. Magnesium in prevention and therapy. *Nutrients* 2015, 7, 8199-8226.
- [3] Gupta SK, Manhas AS, Gupta VK, Bhatt R. Serum magnesium levels in idiopathic epilepsy. *J Assoc Physicians India*. 1994 Jun;42(6):456-7.
- [4] Moe, Sharon M. Disorders Involving Calcium, Phosphorus, and Magnesium *PMC* 2008 July 28, Page 1-19.
- [5] Neill D.W and Neely, R.A. 1956. *J. Clin. Path.* (1956), 9, 162.
- [6] Snedecor G. W., and Cochran, W. G. *Statistical Methods*. USA: The Iowa State University Press, 1980. pp. 232-237.
- [7] Trinder, P.J. *Clin.* (1956) 9: 170.