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## **The role of magnesium in protein-energy malnutrition**

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With 2 tables

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Low levels of mental performance has been documented among children in several countries where malnutrition is prevalent (1). Studies on mental performance of children suffering from kwashiorkor during the period of rehabilitation have shown that during recovery from malnutrition developmental quotients improved in most cases (2, 3). In addition, other studies have demonstrated that inadequacy in the peripheral nervous system also occurs (4, 5). At the same time, changes in magnesium (Mg) levels of blood serum and cerebrospinal fluid (CSF) have been reported in children suffering from other neurological disorders not relating to protein-energy malnutrition (PEM) (6) and muscle dystrophy (7).

These reports stimulated us to explore further variations in the levels of Mg in blood serum and CSF among children suffering from PEM for a better understanding of the nature of the neurological abnormalities detected in PEM.

In this respect, blood sera and CSF levels of Mg have been studied in thirteen cases suffering from PEM and compared with ten normal cases serving as a control group.

### **Materials and methods**

The materials of the present study are fasting blood samples and CSF from thirteen cases suffering from kwashiorkor of the mild-moderated severity according to *Jelliffe* classification (8). Their ages range between nine and thirty-six months. Ten cases of normal children within the same age range and socioeconomic status were also included severing as a control group. Sera were separated immediately. Samples of both blood sera and CSF containing any haemolysis were excluded.

Mg determinations were performed on tungstic acid filtrate using titan yellow in alkaline solution as described by *Neill and Neely* (9). The analyses were carried out on the same day of sampling. Data obtained were statistically analysed using the *Student t* test.

### **Results and discussion**

The results of the present work are presented in the 2 tables.

The method used in our study for Mg determination in serum revealed results in agreement with those reported in literature for normal children (10, 11). In PEM cases, serum Mg levels are statistically and significantly

Table 1. Mg levels in blood serum and CSF of normal children (M. Eq. per litre).

	Serum	CSF
A. N.	2.05	2.02
N. H.	1.84	2.41
A. O.	1.90	2.35
L. M.	2.38	2.54
Z. I.	1.57	2.51
S. M.	2.30	2.00
E. H.	1.70	2.70
B. E.	2.64	2.37
M. H.	1.70	2.55
S. S.	2.71	2.65
Mean	2.08	2.41
SE $\pm$	0.13	0.08

lower if compared with their normal controls, a finding which also agreed with that reported by *Sandstead* (12). This hypomagnesemia may be either due to low Mg intake or to some abnormalities in Mg metabolism in PEM cases. The low Mg intake among children suffering from PEM cannot be ignored since their animal protein intake is markedly low. It is worth mentioning here that serum Mg levels are significantly low in cases of gastroenteritis (13), a state which is usually accompanying PEM.

Diarrhea which is one of the main symptoms of PEM plays a major role in producing alterations in Mg balance shifting it to the negative side. Intestinal malabsorption in our cases may be another factor for such

Table 2. Mg levels in blood serum and CSF of PEM children (M. Eq. per litre).

	Serum	CSF
A. S.	1.57	1.72
B. I.	1.48	1.64
S. K.	1.84	1.84
A. H.	1.23	1.64
S. A.	1.43	1.84
S. M.	1.31	1.47
M. M.	1.47	1.47
Z. M.	1.46	1.64
H. K.	1.14	1.31
S. R.	1.64	2.13
F. L.	1.39	1.89
M. M.	1.64	1.89
M. G.	1.64	1.97
Mean	1.48	1.73
SE $\pm$	0.05	0.06
P <	0.01	0.01

hypomagnesemia. Although very limited informations are available concerning Mg metabolism in the alimentary tract, the major manifestations of Mg deficiency are related to cellular deficits.

Mg which is the second abundant intracellular ion, is involved in a wide variety of physiological and biochemical functions. Its essentiality lies in its extensive role as an enzyme cofactor for several enzyme systems. Parallel to this fact and due to its deficiency in blood serum and tissues of PEM children, most of tissue and blood serum enzymes containing Mg as a cofactor are reduced in these cases, especially those of intracellular activities. Among these are the glycolytic enzymes responsible for glucose absorption and utilization. In a previous study (14), glucose absorption and utilization was found to be impaired in these cases. Salem (15) reported decreased activities of several glycolytic enzymes in the tissues of PEM cases on human and experimental animals.

Because of the relative inaccessibility of the brain tissues, the concentrations of certain elements and metabolites in the CSF may, however, provide an index for the metabolism of these substances in the brain tissues. At the same time informations on the levels of Mg in the CSF are still scanty in the literature available. This encouraged us to study its level in these cases suffering from PEM.

Mg levels in the CSF of PEM children showed a statistically significant reduction when compared with normals. Such decreased Mg levels may throw some light on derangements in brain metabolism in these cases. Brown (16) reported a significant decrease in the level of Mg in CSF in cases suffering from neurological disorders and he established that Mg is required for the proper neuromuscular conduction. Mg interacts with insulin, glucose and galactose and hence its deficiency will hinder the transport of carbohydrate and amino acids to the brain (17).

Cravioto and De Licardie (18) in attempting to define a causal linkage between insufficient dietary intake and subnormal mental functions, postulated that nutrient deficiency affects the intellect by directly modifying the growth and biochemical maturation of the brain. Mg deficiencies in CSF of children suffering from PEM will add another support for the above postulation and can reasonably explain clearly the mental performance in these cases on biochemical basis.

#### Summary

The present study was aiming to explore further the variations in the levels of Mg in both serum and CSF of PEM children, hoping a better understanding for the nature of the neurological abnormalities in these cases. The study revealed decreased values of both blood serum and CSF levels of Mg in PEM children which denote some abnormalities in brain metabolism. Such abnormalities add further support for assumption of the link between dietary insufficiency and subnormal mental performance in PEM.

#### References

1. Moncherg, F., *Malnutrition Learning & Behaviour*, Scrimshaw, N.S. & Gordon, I. E. ed. Cambridge MIT Press 1968. - 2. Cravioto, I., B. Robles, Am. J. Ortho-psychiat. 34, 44 (1965). - 3. McLaren, D. S., U. S. Yarkin, A. A. Kanawati, S. Sabbagh, A. Kadi, J. Ment. Def. Res. 17, 273 (1973). - 4. Udari,

- P. M., *Ind. J. Child Health* **9**, 103 (1960). – 5. *Awwaad, S., E. Essawy, M. Awadalla, A. H. Hassan, M. Moustafa, E. Fadly*, *Gaz. Egypt. Ped. Assoc.* **16**, 211 (1968). – 6. *Davis, E. J. A., D. R. Harvey, J. S. Yu*, *Arch. Dis. Child.* **40**, 286 (1965). – 7. *Zuckermann, L., A. Marquardt*, *Proc. Soc. Exp. Biol. Med.* **112**, 609 (1963). – 8. *Jelliffe, N.*, *Clinical Nutrition*, 2nd ed. (New York 1962). – 9. *Neill, D. W., R. H. Neely*, *J. Clin. Path.* **9**, 162 (1956). – 10. *Andreason, E.*, *Scand. J. Clin. Lab. Invest.* **9**, 138 (1971). – 11. *Kwashima, S., W. H. P. Lewis*, *J. Lab. Technol.* **17**, 32 (1960). – 12. *Sandstead, A. H., M. K. Gabr, S. Azzam, A. S. Shukry, R. J. Weiler, O. Mohy-El-Din, N. Mokhtar, A. S. Prasad, A. El-Hifny, W. J. Darby*, *Am. J. Clin. Nutr.* **17**, 15 (1965). – 13. *Flink, E. B., R. McCollister, A. S. Prasad, J. C. Melby, R. B. Doe*, *Ann. Int. Med.* **47**, 956 (1957). – 14. *Metwalli, O. M., E. A. Eisa, A. S. Shukry, I. M. Fayad*, *Gaz. Egypt. Ped. Assoc.* **15**, 9 (1967). – 15. *Salem, S., A. S. Shukry, E. A. Eisa*, *J. Trop. Med. Hyg.* **75**, 240 (1972). – 16. *Brown, J. K.*, *Arch. Dis. Child.* **45**, 600 (1970). – 17. *Vitale, J. J., M. Nakamura, D. M. Hegsted*, *J. Biol. Chem.* **228**, 573 (1957). – 18. *Cravioto, J., E. R. De Licardie, H. G. Birch*, *Pediatrics* **38**, 319 (1966).

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