Magnesium Deficiency and Depression: Two Case Reports *

Arif VERİMLİ**, Hayrettin KARA**, Reşit KÜKÜRT**

ÖZET

Depresyonun tedaviye cevap vermemesinde ve kronik depresyonda önemli faktörlerden biri Medikal Ko-morbitedir. Bu durum ile ilgili medikal durumlardan biri metabolit metabolizmasıdır.

Biz resistan depresyon gösteren ve Hipomagnezemi tesbit ettiğimiz ve suplemantasyonu ile düzelen iki olguyu sunuyoruz.

SUMMARY

Medical co-morbidity is considered to be one of the reasons of chronicity or treatment resistance in depression. Solving the other problem may result in improvement of depression. Metabolic disorders constitute an example for this phenomenon. Disturbances of the electrolyte metabolism, especially magnesium deficiency is reported to have caused a wide range of psychiatric disorders, from dysphoria to psychosis.

We found out that two of our patients with a diagnosis of depression resistant to treatment had hypomagnesemia after routine laboratory tests. Following a course of magnesium replacement therapy, we observed that the depressive states of the patiens improved markedly. In this paper we present these two cases.

CASE 1:

55 years old, female

The patient was admitted to our hospital with complaints of a depressive nature that has been continuing for the last 1.5 years. In her past history she reffered to two other episodes 20 and 30 years ago respectively, that have resolved without any treatment In the psychiatric examination of the patient. who appeared thin, with a solemn/sunken facial expression, she was reluctant to communicate with the interviewer. Her attention and concentration were poor, her associations were slow and her mood was sad. She had nihilistic delusions, mainly that "her stomach has decayed and disapeared", which were consistent with her mood. The physical evaluation of the patient, who received a diagnosis of 'major depression with psychotic features' according to DSM-III-R criteria, revealed no abnormality. EEG was normal, biochemical, hematological and endocrine tests were all within normal limits. The only disturbance in the serum electrolyte metabolism was a magnesium deficiency (1,4 mg/dl) that persisted after repeat laboratory tests. CAT scan disclosed bilateral basal ganglia calsifications. Since the patient refused to eat anything and no improvement was obtained after a treatment of maprotillin 150 mg/day for 3 weeks, electrocovulsive therapy was started as an alternative. However, the clinical condition of the patient did not change after 10 days of ECT application. Hypomagnesemia was considered as a possible cause of depression. Magnesium replacement therapy was started. Improvement in her mood was observed from the third day of the treatment with iv magnesium sulphate. After two weeks of treatment a bood level of 2,1 mg6dl was achieved. By this time, her sunken expression has cleared up and her vegetative symptoms such as insomnia, anorexia etc. have vanished. She no longer had nihilistic delusions.

CASE 2:

27 years old male, collage student

The patient had complaints about his reluctance for work and school, a sense of pressure in his head, mental slowing, and failure at school for the last 3 years. Seven years ago, he had complaints about "a thingtness in his chest", which continued for 3 years. By that time, four years ago, he had persecutory delusions, and instructing auditory hallucinations. Full remission had been obtained by antipsychotic treatment, however his anhedonia, inferiority feelings,

^(*) XVIIIth C.I.N.P. Congress'de yayınlanmıştır.

^(**) Bakırköy Ruh ve Sinir Hastalıkları Hastanesi 5. Psikiyatri ve Mizaç bozuklukları Birimi.

and school failure turned out to be persistent for the last 3 years. His psychiatric evaluation revealed the following: decreased psychmotor activity, slow associatons, a sad mood impaired mental functioning, inferiority feelings, and delusions of being guilty. Despite vigorous search no abnormality in physical examination or laboratory tests was found, except a marked decrease in the level of serum manesium (0,45 mEq/dl). eeg was normal.

The patient has been using maprotillin 50 mg/day and pimozide 2 mg/day when he applied to our clinic. Pimozide was stopped and maprotillin dose was raised to 150 mg/day. Two weeks of adequate antidepressant treatment made no change in his symptoms. Maprotillin was also stopped and he was maintained on magnesium sulphate (iv) treatment. His inital score in Hamilton Depression Scale, before magnesiuum treatment was started was 20. After 8 days of replacement therapy, his serum magnesium level reached up to 2,6 mEq/dl and his HDS score decreased to 15. He admitted that he no longer felt anhedonia.

DISCUSSION AND CONCLUSION

Magnesium has vital importance in many cellular functions. Serum calcium, phosphate, and potassium levels are associated with the level of magnesium. The major reasons of magnesium deficiency are as follows: Insufficient oral intake, malabsorbtion, and excessive renal loss.

Hypomagnesemia and hypocalcemia have many common features. Their psychiatric manifestations are not spesific ranging from apathy to psychosis. Most commonly delirium and depression are observed (2,3). It is reported that a slight decrease in serum magnesium level may be observed in depression, and the treatment of depression will result in correction of this abnormality (2). It is also claimed that treatment resistant palpitations, neuromuscular and

neuropsychiatric disorders are due to the disturbance of blood-electrolyte imbalance caused by hypomagnesemia (4,5).

In a sample of 21 patients with hypoglisemia, depression was observed besides muscular weakness, refractory hypokalemia, an atrial fibrillation as a frequent manifestation (5-6). In a study of 27 patients with parsomnia due to magnesium deficiency (e.g. nocturnal automatisms, right terrors etc.) nocturnal EEG and polysomnograhic abnormalities were demonstrated. This may be considered as a clue to brain damage caused by hypomagnesemia (7). Nevertheless, the results of our study, specifically the remission of the psychiatric symptoms lead to the suggestions that hypomagnesemia does not cause irreversible damage in the brain.

Many psychiatric manifestations of hypomagnesemia have been reported in the literature, however, there is only one case report suggesting a direct correspondence between magnesium deficiency and depression, which was treated with magnesium replacement (2). The rarity of this clinical condition is perhaps the most striking point of our study.

REFERANCES

- 1- Galland L: Magnesium and inflammatory bowel disase Magnesium 7(2). 78-83, 1988.
- 2- Rausmussen HH, Mortensen PB, Jensen IW: Depression and Magnessium Deficiency. Int J Psychiatry Med 19(1) 57-63, 1988
- 3- Lishman WA: Organic Psychiatry: Psychological consequences of cerebral disorder. Blackwell Scien. Pub. 2nd ed London 1987 p 478
- 4- Berkelhammer C, Bear RA: A Clinical approach to comman electrolyte problems. Can Med Ass. J. 132 (4) 360-368, 1985
- 5- Flink EB: Magnesium Deficiency; Etiology and clinical spectrum/Acta Med Scand Supp 674: 125-137,1982
- 6- Sheehan, J., White A: Diuretic-associated hypomagnesemia. Br Med J. 285(6) 1157-9, 1982
- 7- Popoviciu L, Delost-Popoviciu D; etall: Parasomnias in patients with magnesium deficiency. Rom. J. Neurol Psychiatry 28(1)19-24, 1990