

Short communication**Low serum zinc level in dpression***S.A. Mousavi*, H. Habibollahi**, F. Mahmoudian*****Abstract**

BACKGROUND: Major Depressive Disorder (MDD) is a common disorder, with a lifetime prevalence of about 15 percent, perhaps as high as 25 percent for women. The etiology of MDD is too complex to be explained totally by a single social, developmental, or biological theory. A variety of factors appear to work together to cause or precipitate depressive disorders. Various functions have been reported for trace elements such as zink in recovery or exacerbation of depression.

METHODS: In this experimental study, we studied 46 patients with MDD based on DSM IV criteria, among the patients referred to mental disorders clininc of Noor Hospital. Twenty Patients were men and 26 were women. Thirty two volunteers of general population were evaluated for depression with Beck depression test who did not show any depressive symptoms with this test. A blood sample of 5cc was obtained from each person and the serum zinc concentration was measured. Data gathered and analyzed with SPSS, logistic regression and chi-squar tests.

RESULTS: Serum zinc concentrations were 74 to 130 mg/dl in men and 60 to 128 mg/dl in women of control group. Serum zinc concentration was 30 to 60 mg/dl in depressive patients that it was lower in women than men. The difference between serum zinc concentrations of normal and depressive persons was meaningful ($P = 0.02$).

CONCLUSION: In our study, the serum concentration of zinc was about half of normal value. This study replicates previous findings that major depressed subjects show significantly lowered serum zinc concentration.

KEYWORDS: Depression, zinc.

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Major Depressive Disorder (MDD) is a common disorder, with a prevalence of about 15 percent, perhaps as high as 25 percent for women¹. The etiology of depression is too complex. A variety of factors appears to cause or precipitate depression. Patients with MDD have changes in their brain levels of norepinephrine, serotonin, and dopamine¹. Other factors such as GABA, vasopressin, and the endogenous opiate have been implicated in the pathophysiology of mood disorders¹. Adenylate cyclase, phosphatidylinositol, and calcium regulation may also be causally relevant¹. There is an evidence showing drugs antagonizing NMDA receptors, have antidepressant effects^{1, 2}. Various neuroendo-

crine dysregulations have been reported in patients with mood disorders¹. Genetic data strongly indicate that genetic factors may be involved in the development of mood disorders¹.

Various functions have been reported for trace elements such as zinc (Zn) in recovery or exacerbation of depression³. Zinc is an essential trace element, required for function of several hundred zinc metalloenzymes^{4, 5, 6, 7}. It has some functions with thyrozone and dopamine Beta hydroxilaze that they involve in depression producing mechanisms^{8, 9}. The amount of zinc is low in the CNS, most of that is on hippocampus, cerebral cortex, basal forebrain

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and limbic system. Any disturbance in pathways of these regions can induce anxiety and mood disorders¹⁰. Despite many reports about the hypozincemia in depression, there is not a definite explanation about it^{9, 10, 11}. Because we did not find any study about zinc deficiency in Iranian depressive patients, the aim of present study was to replicate the previous ones.

Methods

In this experimental study, we studied 46 patients with major depression, based on DSM IV criteria, who were selected by consecutive method among the patients referred to the Mental Disorders Clininc of Noor Hospital, between years of 1999 to 2000. Patients were evaluated for underling diseases such as neuroendocrine disorders, systemic infections, and inflammatory disorders. 20 Patients were men and 26 were women. 32 persons of patients families who did not have any history of acute infection and inflammatory diseases were volunteered to be the control group and were evaluated for depression with Beck depression test who didn't show any depressive symptoms with this test. The control group equalized with case group based on matching method for sex and age (± 3 years). Clinical symptoms and signs of hypozincemia were detected with a check list which was extracted from Harrison's Principles of Internal Medicine. A blood sample of 5cc was obtained from each person and the serum zinc concentration was measured. Data gathered and analyzed with logistic regression and chi-squar tests.

Results

Serum zinc concentrations were 74 to 130 \pm 0.9 mg/dl in men and 60 to 128 \pm 0.9 mg/d in women of control group. Serum zinc concentration was 30 to 60 \pm 0.9 mg/dl in depressive patients that it was lower in women than men. The difference between serum zinc concentrations of normal and depressive person was meaningful ($P = 0.02$). Nobody had diarrhea 6 depressive women (23 percent) and 4 depressive men (15.4 percent) had eczema, while nobody in control group showed eczema and this

difference was meaningful ($P < 0.05$). 12 percent of women in normal group ($n = 2$) and 6.6 percent of normal men ($n = 1$) had taste problems while 54 percent of depressive women ($n = 16$) and 70 percent of depressive men ($n = 14$) showed taste problems; the difference was significant too ($P < 0.0005$). 12 percent of normal women ($n = 2$) and 20 percent of normal men ($n = 3$) had olfactory disorders while 46 percent of depressive women ($n = 12$) and 60 percent of depressive men had olfactory problems that this difference was also meaningful ($P < 0.0005$).

Discussion

In our study, serum concentration of zinc was about half of normal value. This study replicates previous findings that major depressed subjects show significantly lowered serum zinc concentration^{10, 11}.

In our study, the evaluation of clinical manifestations of hypozincemia shows that there are many symptoms and signs of lowered serum zinc concentration in depressive patients which indicates hypozincemia. Possible causes for decreased serum zinc concentrations in depression are:

- 1- Anorexia and subsequent malnutrition^{9, 11}. In our patients this hypothesis is less likely, since we did not find significant anorexia or weight loss in our patients and there were not any symptoms and signs of nutritional deficiencies.
- 2- Hypothalamic-pituitary-adrenal (HPA) hyperfunction, which frequently occurs in depression⁹. In our study we excluded patients with any endocrine disorders, so this hypothesis is less likely too.
- 3- Immune responses as the most plausible hypothesis. In our study we evaluated all patients clinically and paraclinically for systemic infections and inflammatory disorders. So, at least our patients did not have any acute inflammatory reactions and this hypothesis is less likely in our patients too¹¹.

Because, in our study, we did not find any explanation for hypozincemia in depressive patients, similar to above studies, it may imply

a relationship between physiopathology of depression and lowered zinc serum concentra-

tion which needs more evidences from future studies.

References

1. Barrett JE, Barrett JA, Oxman TE, Gerber PD. **The prevalence of psychiatric disorders in a primary care practice.** *Arch Gen Psychiatry* 1988; 45(12):1100-1106.
2. Ormel J, VonKorff M, Ustun TB, Pini S, Korten A, Oldehinkel T. **Common mental disorders and disability across cultures. Results from the WHO Collaborative Study on Psychological Problems in General Health Care.** *JAMA* 1994; 272(22):1741-1748.
3. Boosalis M, Stuart M, McClain CJ. Zinc in the elderly. In: Jorley JH, Glick Z, Rubenstein LZ, editors. *Geriatric Nutrition*. New York: Raven Press, 1995: 115-121.
4. Powell SR. **The antioxidant properties of zinc.** *J Nutr* 2000; 130(5S Suppl):1447S-1454S.
5. Hennig B, Toborek M, McClain CJ, Diana JN. **Nutritional implications in vascular endothelial cell metabolism.** *J Am Coll Nutr* 1996; 15(4):345-358.
6. Okita M. **Chronic hepatic disease and dietary instruction.** *Hepatol Res* 2004; 30S:92-95.
7. Harrison's Principles of Internal Medicine. 15 ed. New York: McGraw-Hill Company, 2000.
8. McClain CJ, Kasarskis EJ, Jr., Allen JJ. **Functional consequences of zinc deficiency.** *Prog Food Nutr Sci* 1985; 9(1-2):185-226.
9. Maes M. The immune pathophysiology of major depression. In: Honig A, van Praag HM, editors. *Depression: Neurobiological, Psychopathological and Therapeutic Advances*. Chichester: John Wiley, 1997: 197-215.
10. McLoughlin IJ, Hodge JS. **Zinc in depressive disorder.** *Acta Psychiatr Scand* 1990; 82(6):451-453.
11. Maes M, Bosmans E, Meltzer HY, Scharpe S, Suy E. **Interleukin-1 beta: a putative mediator of HPA axis hyperactivity in major depression?** *Am J Psychiatry* 1993; 150(8):1189-1193.