

Copper and Iron Levels in Wilson's Disease Cases

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Abstract: *Wilson's disease is a genetic disorder that prevents elimination of extra copper and causes build-up signs in various organs such as liver, brain, eyes etc. and may cause life-threatening organ damage. The predominant symptoms of this disease manifested as mood change, depression, dark urine or light-colored stool, jaundice, brownish ring around the edge of cornea, tremors, stiff muscles, incoordination etc. The vital role of copper in formation of red blood cells and making immune system healthy. It also act as antioxidant and helps to reduce free radical injuries to cells. Deficiency of copper may cause neurological signs, polyneuropathy such as sensory ataxia i.e. irregular coordination due to proprioceptive loss. Iron role in growth and development, to make hemoglobin, carries oxygen etc. neurodegenerative disorders may occur in iron accumulation in brain and may manifest as dystonia, dyarthria etc. the present study was undertaken to assess the levels of copper and iron.*

Keywords: Copper, Iron, Wilson's disease, neurological disorder,

1. Introduction

Iron and copper are important cofactors for various enzymes in the brain, involved in neurotransmitter synthesis and myelin formation^[1]. Copper is an essential nutrient involved in energy production, connective tissue formation and neurotransmission, it also plays important role in erythropoiesis thus iron and copper deficiency may cause anemia^[2]. Abnormal metabolism and low levels of iron and copper may result in impairment of motor function^[3]. Therefore, the present study was planned to assess the levels of copper and iron in neurological disorders like Wilson's disease.

2. Materials and methods

In all 50 control cases and 10 Wilson's disease cases blood samples were assessed for their copper and iron status. The serum samples were subjected for copper and iron assessment. Estimation of copper^[4] ^[5] and iron^[6] was done. The comparison of control and epilepsy status of calcium, phosphorous and magnesium was done by using t test^[7].

3. Results and Discussion

Serum copper and iron levels were significantly decreased in Wilson's disease cases than control groups (Table 1). The impaired transport might have interfere incorporation of copper into the copper protein ceruloplasmin which causes decreasing serum ceruloplasmin^[8]. Low serum ceruloplasmin intensifies suspicion of Wilson disease^[9]. Serum iron concentration was very low and ferritin concentration was very high^[10]. Therefore, assessment of copper and iron is important in Wilson's disease cases.

Table 1: Comparison of (Mean \pm S.E.) of control and Wilson's disease cases

Parameters	Control (n=50)	Wilson's disease (n=10)
Copper ($\mu\text{g/dl}$)	116.648 \pm 4.903	77.500 \pm 4.550**
Iron ($\mu\text{g/dl}$)	150.672 \pm 2.516	76.901 \pm 4.400**

**= $p < 0.01$

References

- [1] Skjørringe T, Møller LB, Moos T. Impairment of interrelated iron- and copper homeostatic mechanisms in brain contributes to the pathogenesis of neurodegenerative disorders. *Front Pharmacol.* 2012 Sep 25; 3:169. doi: 10.3389/fphar.2012.00169. PMID: 23055972; PMCID: PMC3456798.
- [2] Gulec S, Collins JF. Molecular mediators governing iron-copper interactions. *Annu Rev Nutr.* 2014; 34:95-116. doi: 10.1146/annurev-nutr-071812-161215. Epub 2014 Jun 2. PMID: 24995690; PMCID: PMC4316823.
- [3] Kwik-Urbe C. L., Gietzen D., German J. B., Golub M. S., Keen C. L. (2000). Chronic marginal iron intakes during early development in mice result in persistent changes in dopamine metabolism and myelin composition. *J. Nutr.* 130, 2821–2830 [PubMed] [Google Scholar]
- [4] Ventura, S. & King, E. J. Copper, determination in blood serum *The Biochemical Journal* lxi MAY 1951, VOLUME 48, No. 5, Cambridge University Press London: Bentley house, n.w. 1 New York: 51 Madison avenue
- [5] S. Ventura and J. C. White The determination of iron and copper in single serum samples, *Analyst*, 1954, **79**, 39-42
- [6] Ramsay WN. The determination of iron in blood plasma or serum. *Biochem J.* 1953 Jan;53(2):227-31. doi: 10.1042/bj0530227. PMID: 13032059; PMCID: PMC1198133.
- [7] Snedecor G. W., and Cochran, W. G. *Statistical Methods.* USA: The Iowa State University Press, 1980. pp. 232-237.
- [8] Patil M, Sheth KA, Krishnamurthy AC, Devarbhavi H A review and current perspective on Wilson disease. *J Clin Exp Hepatol.* 2013 Dec;3(4):321-36. doi: 10.1016/j.jceh.2013.06.002. Epub 2013 Jul 6. PMID: 25755520; PMCID: PMC3940372.
- [9] Brewer GJ. *Wilson's Disease: A Clinician's Guide to Recognition, Diagnosis, and Management.* Boston, MA: Kluwer Academic Publishers; 2001.
- [10] Ogimoto M, Anzai K, Takenoshita H, Kogawa K, Akehi Y, Yoshida R, Nakano M, Yoshida K, Ono J. Criteria for early

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identification of aceruloplasminemia. Intern Med.
2011;50(13):1415-8. doi: 10.2169/internalmedicine.50.5108.
Epub 2011 Jul 1. PMID: 21720062.