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Letter to the Editor

Breakthrough: Zinc Therapy for Copper Toxicosis in Wilson's disease and probably also for Copper Toxicosis in Alzheimer's disease

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DEAR EDITOR

This letter is to improve the state of knowledge in the field of 2 neurodegenerative copper diseases.

1. Wilson's disease with neuro degeneration of the lenticular nuclei in the basal ganglia of the brain.
2. Alzheimer's disease with neurodegeneration of the cerebral cortex.
3. Improving knowledge on zinc therapy for free (non-ceruloplasmin bound) copper toxicosis in both neurodegenerative disorders.

WILSON'S DISEASE

Wilson's disease is a hereditary copper toxicosis. It is a recessively inherited inborn error of metabolism. The copper metabolism is disturbed resulting in increased levels of free copper in blood and in urine. Ceruloplasmin, a copper binding metalloprotein, plays an important role in copper metabolism and in WD treatment. It binds free highly toxic unbound copper in the liver and the serum. A low level ceruloplasmin in serum is an essential diagnostic sign and plays an important role in the diagnosis of Wilson's disease. The diagnosis of Wilson's disease may be easy: increased level of copper in the urine is easy to detect. Furthermore most patients with neurodegenerative WD have easy to detect typical brown coloured Kayser-Fleischer rings in the corneas. These rings are a sure sign of elevated copper levels in the blood. The clinical signs of neurologic Wilson's disease are caused by degeneration of the basal ganglia, that lead to movement disorders such as dystonia, parkinsonism, rigidity and tremor. However, diagnosis of WD is often far from easy because it is a typical orphan disease: it is rare and often forgotten to include in the differential diagnostic considerations.

In 1961 the Dutch neurologist Gerrit Schouwink discovered that zinc is the treatment of choice for WD. Zinc supplements are most effective in lowering the toxic levels of unbound free copper in blood and urine, by stimulating the excretion of copper via the stools. Schouwink developed the hypothesis that zinc supplements stimulate the forming of metal binding metallothioneïns in the mucosal cells in the gut that would bind copper out of passing food and passing blood from the arteries.

Before Schouwink's eminent discovery it was believed – and it still is - that WD is a copper accumulation disease and that chelating agents like BAL (British Anti Lewisite) and penicillamine were take out the accumulated copper. However, chelating agents increase the level of toxic free copper in the blood resulting in increase of free copper in the urine. This was abusively regarded to be favourable for the course of the disease. But instead of improving the condition of the patient it became worse than before treatment. Zinc stimulates the binding of free copper in metallothioneïns in the mucosal cells in the gut. It this manner it lowers the toxic free copper level in the blood.

ZINC THERAPY FOR FREE COPPER TOXICOSIS IN ALZHEIMER'S DISEASE

The discoveries that zinc therapy is an extremely effective and very safe method to provide causal treatment of copper intoxication in Wilson's disease opened a new way to think about a causal treatment of Alzheimer's disease. Alzheimer's disease is a chronic neurodegenerative free copper toxicosis. During the conference in the Department of Neurology in the Fatebenefratelli Hospital in Rome in June 19th in 2006 I presented the paper entitled "Zinc therapy for free copper toxicosis in Alzheimer's disease". I defended the idea that non-ceruloplasmin bound free copper is the essential causal factor in the pathogenesis of Alzheimer's neurodegeneration of the cerebral cortex.

Although there has not been a scientific evidence for the use of zinc therapy for Alzheimer's disease I am convinced that there are sufficiently enough indications that it is wise to start to advise the use of zinc supplementations for Alzheimer's disease, as a prevention and as a cure.

Regards

Tjaard Hoogenraad