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Vitamin D Deficiency

In Patients with Early Onset Parkinson's Disease

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Vitamin D insufficiency is reported to be more common in individuals with Parkinson's disease than in healthy controls. Evat, et.al, measured the vitamin D status of patients who had developed early, non-disabling, Parkinson's disease and who were diagnosed within five years of entering the study. Vitamin D levels in the affected group were found to be low, which is similar to the findings of other reported studies. However, vitamin D levels did not decline further over time during the progression of the condition. (Evat, ML, et al. *High Prevalence of Hypovitaminosis D Status in Patients With Early Parkinson Disease. Arch. Neurol* 68,3,2011.)

Through hair tissue mineral analysis (HTMA) studies here at Trace Elements, we have categorized Parkinson's disease (PD) under a sympathetic dominant neuro-endocrine category. That is, conditions manifesting in patients with sympathetic dominance typically have an increased requirement for vitamin D when compared to individuals with a parasympathetic dominance. Early Parkinson's disease typically manifests with a sympathetic mineral pattern showing a low tissue calcium level as well as low calcium to phosphorus and calcium to potassium ratios along with an elevated sodium to magnesium ratio, indicating excessive adrenal and thyroid activity. Other health conditions manifesting in sympathetic categories are listed below, which can also be said to have higher vitamin D requirements.

Sympathetic Dominant Disease Manifestations and Increased Vitamin D Needs:

Anxiety	Arthritis (Rheumatoid)
Allergies (Histamine)	A.L.S.
Cushing's Disease	Cardiovascular Disease
Malignancies (Metastatic)	Hyperactivity (ADD-ADHD)
Diabetes (Type I)	Hypertension
Hyperthyroidism	Heavy Metal Accumulation
Hyperglycemia	Hyperadrenia
Hypoparathyroidism	Hypovitaminosis
Hypervitaminosis	Co-Factor Deficiency
Infections (Bacterial)	Multiple Sclerosis (Type 1)
Osteoporosis (Type I)	Parkinson's Disease
Ulcers (Peptic)	Seizures
Cystic Fibrosis	Cellular Immune Suppression
Tourette's Syndrome	Humoral Immune Dominance
Metabolic Syndrome X	Manic Depression

HTMA studies of individuals diagnosed with Parkinson's disease show that men are affected slightly more than females at about fifty-eight percent. Metabolically, it appears that PD is distributed almost equally in Sympathetic and Parasympathetic categories. However, these studies do not distinguish early onset PD from chronic PD. Even though we classify PD as a sympathetic dominant condition, long-term progression of the condition and medications can cause a shift toward parasympathetic dominance.

Mechanism of Parkinson's Disease

The cause of PD is unknown although it is associated with a reduction in dopamine-producing cells in the brain. The

condition is known to run in some families. Secondary Parkinsonism can be caused by other diseases such as stroke, heavy metals and environmental toxins. Symptoms of PD can also be caused by medications, such as antipsychotic drugs, metoclopramide a drug used to treat gastric reflux. Neurological diseases, especially PD can be difficult to diagnose and can often be misdiagnosed. A PD diagnosis is often arrived at by excluding other diseases rather than by confirmation. Autopsy findings of Lewy Bodies in the midbrain confirm the diagnosis of PD. Otherwise, the diagnosis is arrived at by signs and symptoms or response to medications. However, signs and symptoms can be caused by many factors and may be similar to other neurological conditions.

Low HTMA Copper and Parkinson's Disease

Very low HTMA copper levels are found in patients diagnosed with PD, a factor we also deem a contributor to the development of Parkinson's. Copper's role is associated with neurotransmitter production, protection of tissues from free radical damage through activation of superoxide dismutase, and with the production of ceruloplasmin, which has been found low in patients with Parkinson's Syndrome. (New Insights Into Parkinson's. JAMA 273,20,1995.) HTMA studies at our laboratory show a mean copper level of 0.8 milligrams percent (mg%) in one-hundred twenty-five patients diagnosed with PD compared to an ideal mean of 2.5 mg%. Further, test results show a marked elevation of the tissue zinc to copper ratio averaging 15.45:1 compared to an established ideal of 8:1. Neurological disturbances have been reported in individuals with induced copper deficiency due to the use of denture creams containing high amounts of zinc. Discontinuation of the cream resulted in improvement of symptoms ranging from ataxia, sensory loss, paresthesia, myeloneuropathy and anemia. Correction of the copper deficiency resulted in neurological improvements. (Spain, RI, et al. When Metals Compete: A Case of Copper-Deficiency Myeloneuropathy and Anemia. Nat.Clin.Pract.Neurol. 5,2, 2009.) (Nations, SP, et al. Denture Cream: An Unusual Source of Excess Zinc, Leading to Hypocupremia and Neurological Disease. Neurol. 71,9 2008.)

Iron /Copper Imbalance and Neurological Disease

Clinically, low HTMA copper levels and/or an elevated tissue iron to copper ratio is seen in patients with neurological symptoms. Studies have revealed a distinct difference in the hair tissue copper status in patients with multiple sclerosis compared to non-affected control groups. (Ryan, DE et al: Trace Elements in Scalp-Hair of Persons with Multiple Sclerosis and of Normal Individuals. Clin. Chem. 24, 11, 1978.) An imbalance between copper and zinc as well as copper and iron is seen in HTMA patterns of individuals diagnosed with A.L.S. and

contributes to a reduction in anti-oxidant activity, particularly SOD, contributing to neurological disturbances. (Mutations in the Copper- and Zinc- Containing Superoxide Dismutase Gene are Associated with "Lou Gehrig's Disease." Nutr. Rev. 51,8, 1993.) Autopsied brain tissue from individuals who died with Parkinson's Disease and a control group who had no history of neurological disease before death revealed a 35% increase of iron in the substantia nigra of the parkinsonian brain as compared to the controls. The copper content was reduced by 34%. (Dexter DT, et al: Increased nigral iron content in postmortem parkinsonian brain. Lancet ii, 1987.) Researchers found that lipid peroxidation was increased in the parkinsonian nigral tissue, adversely affecting dopamine-containing cells. (Dexter D, et al: Lipid peroxidation as cause of Nigral cell death in Parkinsons Disease. Lancet II, 1986.) (F, Agid et al: Lipid peroxidation as cause of Nigral cell death in Parkinsons Disease. Lancet II, 1986). Since excess iron promotes lipid peroxidation and copper inhibits it, we feel an imbalance of Fe/Cu may play a role in the development of PD in some individuals.

Neurological disturbances that produce symptoms similar to PD have also been associated with excess tissue manganese. (Banta, RG et al: Elevated Manganese Levels Associated with Dementia and Extrapryramidal Signs. Neurol. 27,3, 1977.) Additionally, excess aluminum is known to produce degenerative processes in the central nervous system. Aluminum retention in the brain has been related to neurological conditions such as Alzheimer's disease and Amyotrophic Lateral Sclerosis (ALS). (Mitani, K: Relationship Between Neurological Diseases Due to Aluminum Load, Especially Amyotrophic Lateral Sclerosis, and Magnesium Status. Magnesium Res. 5,3, 1992.) Lastly, many heavy metals are CNS toxins and which have been associated with contributing to neurological symptoms.

Dietary Factors and PD

It should be noted that vitamin D is just one component of PD and should not be expected to be a corrective or curative treatment even if vitamin D levels are returned to normal. As with any disease the cause or contributors are multiple and cannot be simplified to a single entity or treatment. When PD is viewed from a metabolic standpoint from HTMA patterns we can more specifically address the nutritional needs of patents that may be suffering from this condition.

Vitamin D needs can be readily assessed from HTMA studies by a low tissue calcium level and low tissue calcium to phosphorus ratio. Also knowing factors that can antagonize calcium absorption and retention can provide a further guide for nutrient evaluation. For example, foods high in phytates can adversely affect calcium absorption and therefore, individuals with a sympathetic mineral dominant pattern should markedly

reduce their intake of grains and cereals. Also, high vitamin A intake should be avoided since high intake can adversely affect calcium retention.

Low tissue copper levels can be exacerbated by factors that antagonize copper, such as fructose, zinc, vitamin C, niacin, and iron.

Elevated tissue iron or high iron to copper ratios can be caused by bacterial infections, as well as high intake of the previously mentioned copper antagonists. Alcohol use increases the retention of iron. High iron sources of foods and water should be reduced in patients with PD who show an elevated iron to copper ratio. High iron herbs such as black cohosh, goldenseal, comfrey and others should be avoided as well as iron cooking utensils.

Practitioners should not only assess individual nutrient requirements of PD patients, but their medical history as well. Medications can be a major cause and contributor to the mineral imbalances found in patients suffering from this condition.

Conclusion

Again, the diagnosis of neurological disease can be difficult as it is known to be caused by any number of factors. Therefore, it is suggested that anyone who has been diagnosed with Parkinson's disease be periodically reviewed, since over time the condition may often be found not to actually be Parkinson's. This is good advice for anyone exhibiting neurological symptoms or who has been diagnosed with any neurological disease, as medications prescribed for the condition in the long run would be less than helpful and most likely harmful, while not addressing the underlying contributing factors or causes.