Environmental Risks for PD: Manganese, Welding, Mining, and Parkinsonism

You can find out more about NPF's National Medical Director, Dr. Michael S. Okun, by also visiting the NPF Center of Excellence, University of Florida Center for Movement Disorders & Neurorestoration.

The recent publication, in the American Journal of Epidemiology, addressing metal emission and urban incidence of Parkinson’s disease underscores the public’s growing concern that manganese, copper, and other metals may play a role as an environmental cause of Parkinson’s disease. Willis et. al. investigated the relationship between copper, lead, or manganese emissions and Parkinson’s disease. The study covered nearly 30 million Medicare beneficiaries, and ultimately concluded that urban Parkinson’s disease incidence was greater “in counties with higher reported industrial releases with copper or manganese.” The authors cited several weaknesses of their own study including other unaccounted for risk factors, and no direct environmental metal measurements. Furthermore, there was no way to determine whether patients had actual Parkinson’s disease or just symptoms similar to Parkinson’s (i.e. parkinsonism).

The dramatic rescue of thirty-three Chilean miners was all over the news last month. Miners have been known to have an increased risk of exposure to manganese, and this has led to the question as to whether miners and welders carry a higher risk of developing Parkinson’s disease. Controversy has swirled around this issue in both medical and in legal circles, with several class action lawsuits, and one paper reporting the results of exposures in patients pursuing litigation (Racette, 2005). It is therefore important to inform the public about the differences between typical (also known as “idiopathic”) Parkinson’s disease and parkinsonism due to manganese exposure.

Parkinson’s disease is an entity that is usually diagnosed by an experienced neurologist following a detailed clinical history and examination that may reveal motor (tremor, stiffness, slowness, gait, balance difficulties, etc.) and also possibly non-motor dysfunction (loss of smell, depression, anxiety, cognitive decline, sleep disorder, bladder and bowel symptoms, sexual dysfunction etc.). Patients with idiopathic Parkinson’s disease usually respond well
to levodopa or dopamine agonists. Brain MRI scans in Parkinson’s disease are usually normal. Manganese exposure (in welders or in miners), in contrast, is known to have a largely different acute and chronic presentation.

Manganese toxicity and Parkinson’s disease has been reviewed in an article published by Dr. Joseph Jankovic who is at the NPF Center of Excellence at Baylor College of Medicine, Houston TX (Jankovic, 2005). Dr. Jankovic points out that in acute manganese intoxication “cough and shortness of breath, as well as headaches occur early. This early phase is also manifested by nonspecific symptoms such as asthenia, somnolence, insomnia, anorexia, and loss of sexual drive, as well as behavioral manifestations, characterized by irritability, impulsiveness, belligerence, obsessive-compulsive behavior, hallucinations, and other psychiatric symptoms (“manganese madness”). Other psychiatric abnormalities may include pseudobulbar affect with emotional incontinence which is manifested by inappropriate laughing and possibly crying. Later, hypomimia (decreased facial expression), action tremors, dystonia, myoclonus, speech, gait and balance problems, and other parkinsonian features may emerge. A variety of neuropsychological abnormalities have been reported, even in early stages of manganese poisoning, but no consistent pattern of behavioral or cognitive abnormalities has emerged.”

Patients with manganese toxicity usually have normal blood manganese levels unless acutely exposed, and they usually do not respond to dopamine replacement therapy (levodopa) or agonists. Additionally, manganese toxicity patients, unlike idiopathic Parkinson’s disease, usually have characteristic MRI abnormalities.

The most important thing for patients who have worked as miners or welders to be aware of is the differences in clinical presentations, imaging studies, and response to medications. These differences usually help in the differentiation of manganese parkinsonism from Parkinson’s disease. It is currently unknown and unexplored as to whether manganese exposure imparts a greater risk of the development of Parkinson’s disease (“second hit environmental hypothesis”), and we hope more research will focus on this area in the future.

For more information, read the 11/11/10 article in Medscape Today: Urban Exposure to Manganese, Copper May Raise Parkinson’s Risk

Selected References


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