D67 A Case of Manganese-Induced Parkinsonism: The Need for Safety in the Workplace

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Occupational exposure to manganese and the need for medical management of manganese-associated toxicity are well-established. After attending this presentation, attendees will understanding how both remain problematic in industrial settings.

This presentation will impact the forensic science community by illustrating the need to improve security rules in workplaces and to regularly inspect workplace healthiness in order to avoid possible exposure to toxic substances.

The case involved a welder who developed Manganese-Induced Parkinsonism after being exposed for twenty-six years. The welder worked in the steel industry, from the age of fifteen to forty-one years. His duties included arc and gas welding, using manganese-containing electrodes. This activity emitted welding fumes containing high levels of manganese.

At the age of thirty-six, the welder began to show extra-pyramidal rigidity, balance dysfunction, tremor at rest, bradykinesia, symmetric impairment, amimia, and “coq au pied.”

Subsequently, psychiatric disorders, including major depression and manic depression-psychosis also appeared.

The welder underwent several clinical and instrumental examinations, including MRI brain scans, that indicated T1-weighted hyperintensive signal in the globus pallidus, bilateral.

These results, atypical of idiopathic Parkinson’s Disease (PD), in association with the young age of the patient and his working activity, alerted the suspicion of Manganese-Induced Parkinsonism. Therefore, the welder had blood and urine tests which revealed high levels of manganese, confirming the work-related exposure.[1-6]

Instrumental research and laboratory tests, supported by clinical findings and circumstantial data, were essential to clarify the cause of extra-pyramidal symptoms.

Manganese is a heavy metal widely distributed in the environment by wind erosion and the manufacturing process. More than 90% is utilized in the manufacture of steel: it is alloyed with steel to increase strength, hardness, wear resistance, and with other metals to form highly ferromagnetic materials.
Manganese compounds are used as coloring, catalysts, oxidizers, and antiseptics. In biology, manganese is a trace element, a co-factor for a large variety of enzymes with many functions.[1,3,6]

Occupational exposure generally occurs by inhalation, then it is carried by the bloodstream to high-metabolism rate organs. Manganese half-life is approximately between 40 and 400 days and it is eliminated through fecal and bilious channels, a tiny amount by the urinary tract.

Toxic effects of manganese are related to its neurotropism; however, the basis for the selective neurotoxicity of manganese remains incompletely understood. It causes neuronal injury, especially in the globus pallidus, and also in other basal ganglia structures such as caudate and putamen, less frequently in the substantia nigra.

It primarily interferes with dopamine biology (the primary cellular energy metabolism), decreasing its receptors levels.[1,3,5,6]

An acute manganese-related disease is the metal-fume fever, resulting from inhalation of volatile metal oxides produced during welding or cutting of metal materials. The symptoms are generally nonspecific flu-like complaints including fever, fatigue, and muscle ache. Symptoms are self-limiting and typically resolved within 24 hours.[1,7,8]

Chronic manganese exposure produces an excessive accumulation of the metal in the brain that results in a neurological syndrome with cognitive, psychiatric, and movement abnormalities. The initial symptoms, manifesting within one to two years after the first exposure, are aspecific. Welders may also present neuropsychiatric disorders, popularly called “manganese madness.” Over the long term, manganese-exposed workers exhibit an extrapyramidal syndrome that resembles PD: symptoms include gait disturbance (“coq au pied”), balance dysfunction, symmetric impairment, masked face, relative absence of tremor, grimace, bradykinesia, and muscular rigidity.[1,3,5,6]

MRI brain scans in manganese-intoxicated patients demonstrate a characteristic signal abnormality on T1-weighted studies that are not seen in normal individuals or in patients with PD or other forms of parkinsonism. Instrumental examinations show a hyperintensive signal, particularly in the globus pallidus. Similar changes are noted in the substantia nigra, caudate nucleus, and putamen.[5,6] Diagnosis is based on case history and neurological evaluation: in particular, hyperkinetic movements and the “coq au pied” step give evidence of a Manganese-Induced Parkinsonism rather than PD or Parkinsonism induced by other causes.

Prognosis is generally severe because neurological injuries are progressive and worsening, even after exposure cessation.[1,3,5,6]

In conclusion, this case study demonstrates the need for proactive interventions in order to improve workplace safety, minimizing manganese exposure and, therefore, preventing the onset of Manganese-Induced Parkinsonism.

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The disease is progressive and disabling, causing social disadvantages, especially tragic in view of the young age of onset.

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