CASE REPORT

Acute psychotic presentation of Manganese Toxicity

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Abstract:

Manganese poisoning is a toxic condition due to excessive exposure to manganese which can present with neurological or psychiatric symptoms. Here we report a 51-year-old male with occupational exposure to manganese, brought to the Emergency Room with two days history of acute onset of behavioral disturbances. There was a history of taking Siddha medications for the last 4 months for polyarthralgia with minimal improvement. A diagnosis of organic psychotic disorder due to Manganese intoxication was made. MRI Brain plain and contrast were normal. The heavy metal screen was suggestive of high Manganese of 42 mg/ml and Cobalt of 4.4 ng/ml EEG was suggestive of generalized epileptiform abnormality. The patient was asked to refrain from further exposure to heavy metals and treated with Sodium valproate and neuro vitamins with gradual improvement in symptoms. This case report emphasizes the need for detailed evaluation and ruling out other medical conditions in a case presenting with first episode of psychosis.

Keywords: *depression, anxiety, prevalence, predictors, school, students, Chennai, south India*

INTRODUCTION

Manganese is an essential dietary trace element and useful for normal body functioning in humans. It regulates many functions of several enzyme systems by acting as catalyst. Beyond a certain level ingestion or inhalation of manganese leads to adverse health consequences.[1] The primary cause of excess manganese exposure in general population is inhalation of particulate matter containing Manganese. Hence people working in industries using manganese or living near to mining activities are most vulnerable to Manganese toxicity.[2]The acceptable range of manganese levels in blood is 4-15 mcg/L in blood, urine is 1-8 mcg/L, and in serum 0.4-0.85 mcg/L.[3] Any excess manganese in the

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body accumulates in brain, specifically in basal ganglia. A T1-weighted MRI of globus pallidus region of the basal ganglia in the brain typically shows a distinctive symmetrical high-signal lesion in the individuals with Manganese toxicity.[4]

The symptoms of manganese toxicity are usually insidious in onset and neurological symptoms and signs appear over months and years. The symptoms range from symptoms of irritability, aggressiveness, hallucinations, tremors, difficulty in walking, and facial spasms to a permanent neurological disorder. Manganese inhalation can even cause adverse cognitive effects like concentration and memory problems. When children in the formative years get exposed, they can have neurological, respiratory, and reproductive symptoms with neurodevelopmental problems.[5]

The neurological effects of manganese toxicity typically begin with feelings of weakness, lethargy and as the disease progresses more neurological signs appear. However, the presentation of Manganese toxicity is varied, not all individuals develop similar signs. The most common presentation is neurological extrapyramidal symptoms of slow and clumsy gait, speech disturbances, a masklike face, and tremors. In some individuals, the symptoms spontaneously disappear on cessation of exposure, in few the symptoms persist for many years post-exposure to Manganese and as the disease progresses, they develop severe muscle tension and rigidity and may be completely and permanently disabled.[6] In rare circumstances the presentation is a syndrome of psychological disturbances like hallucinations, and psychosis. Though Manganese neurotoxicity has some clinical similarities to Parkinson's disease, they have hypokinesia and tremor which are different from Parkinson's patients. They have tendency to fall back when pushed, less frequent resting tremor, more frequent dystonia, and don't typically respond to antiparkinsonian drugs.

Many studies have reported the subclinical neurological effects like decreased performance in neurobehavioral tests, impaired eye-hand coordination, hand steadiness, decreased reaction time, postural instability, and lower levels of cognitive flexibility in individuals having lower Manganese exposure levels. Most of the case reports regarding Manganese toxicity reported neurological symptoms and there are very few case reports of psychotic phenomena as a presentation of chronic Manganese ingestion.

CASE REPORT

A 51-year-old gentleman, a diploma holder in Information technology, employed in the heavy metal industry as a crane operator for more than two decades belonging to middle socioeconomic status Tamil speaking urban background, was brought to our emergency room by his family members with two days history of acute onset behavioral disturbances like muttering and smiling to self, making inappropriate gestures, spitting behavior with thoughts of reference and sleep disturbance. Further exploration of history revealed episodes of confused behavior with disorientation to time and place with probable visual hallucinations along with decreased sleep, decreased appetite in the last 2 months. There was no history suggestive of prior medical illness or substance abuse. He was on Siddha medications for the last 4 months for polyarthralgia with minimal improvement reported. As there was no personal or family history of psychiatric illness and acute presentation of behavioral disturbances in the background of fluctuating orientation, a central nervous system disorder was suspected.

On examination, the patient was having fluctuating sensorium, perplexed affect, though he was able to follow simple commands. He was having tremulousness of the whole body, rigidity in all four limbs, dysarthria.

MRI Brain plain and contrast were normal. The heavy metal screen was suggestive of high Manganese of 42 mg/ml and Cobalt of 4.4 ng/ml Blood investigations revealed low serum sodium levels of 129 mmol/liter, high serum uric acid levels of 50 mg/dL, low serum vitamin B12 levels of 142pg/ml. ECG and ECHO were normal. Blood viral markers were negative. Serum Procalcitonin, anti-TPO, Ammonia was normal. USG and CT abdomen were suggestive of fatty liver with hemangioma and cystitis of the urinary bladder. The autoimmune panel was negative. Other routine hematological and biochemical tests including complete hemogram, renal function test, and liver function test were normal except for slightly low total protein of 6.2 gm/dL EEG was suggestive of generalized epileptiform abnormality. The patient was treated with intravenous Vitamin B12, folate, thiamine, sodium valproate, and antibiotics. After 2 months of discharge, the patient was re-evaluated. There were no psychotic or mood symptoms. There were no signs or symptoms indicative of parkinsonian syndrome.

DISCUSSION

In our 51-year-old patient, an elevated serum manganese level of 42 mg/ml was found. It appeared to be caused by overexposure to manganese as he was working in the heavy metal industry for more than 20 years. There is no history suggestive of environmental exposure to Manganese other than occupational

exposure. Therefore, a diagnosis of organic psychotic disorder due to Manganese intoxication was made. An observational study on neuropsychiatric manifestations of chronic manganese poisoning reported psychotic disturbances are seen in both acute and insidious onset manganese poisoning but comparatively more in acute presentations.[7] The psychotic disturbances ranged from mild psychotic disturbances to florid psychosis. Most of them had spontaneous laughter or crying with disturbed sleep. In our case the patient presented with irritable mood, smiling to self and thoughts of reference along with insomnia. The neurological symptoms were fluctuating orientation with motor symptoms of tremors, rigidity, and dysarthria suggesting extrapyramidal involvement. Though there was no clinical history of seizures, given episodic fluctuations in orientation in the last two months and generalized epileptiform activity in EEG, Valproate treatment was initiated, and the patient responded well without any residual extrapyramidal symptoms. The neurological sequelae reported in the literature included predominantly extrapyramidal symptoms with permanent deficits and there were no reports of possible seizure activity secondary to manganese intoxication and complete recovery of extrapyramidal symptoms. Excess oral intake or inhalation of manganese can lead to accumulation of manganese which can have adverse effects on the respiratory, reproductive system, and neurological effects. However, the duration and mode of exposure will affect the presentation of symptoms.[8] In this case, he was asymptomatic till 4 months before the emergency room visit and presented with acute onset psychotic symptoms in the background of polyarthralgia, sleep disturbance, and irritability, and later fluctuating orientation and confused behavior. He was evaluated for the presence of neurological signs in the ER despite the psychotic presentation. He was treated with Siddha medications without significant improvement but continued to work which could have worsened the toxicity. As we did not test the medication for the content of manganese, we are unable to say if that was contributory. The sudden onset psychotic symptoms and extrapyramidal symptoms made the family seek medical consultation. Atypical presentation in terms of age, symptom profile, occupational exposure, presence of neurological symptoms, and absence of past or family history of psychiatric illness prompted us to do a detailed neurological and heavy metal screening.

CONCLUSION

Psychiatric symptoms can be produced by several medical or neurological conditions other than psychiatric disorders. Psychiatrists are usually asked to evaluate the patients presenting with first episode psychosis, presenting with disturbances of behavior, affect, or cognition. But often, because of the severity of symptoms or the imminent threat to the patient or their caregivers, detailed neuro-psychiatric evaluation gets delayed. Further treatment and management depend on the working diagnosis made. Hence failure to detect underlying medical/neurological conditions can lead to significant and unnecessary morbidity and mortality. This case highlights the importance of detailed history on occupational hazards and screening for heavy metal toxicities when patients present with acute psychiatric symptoms.

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CONFLICT OF INTEREST

Nil.

Competing interests

Authors have declared that no competing interests exist.

Authors' contributions

This work was carried out in collaboration among all authors. All authors have read and approved the final manuscript.

Consent

As per international standard or university standard, patients' written consent has been collected and preserved by the author(s).

Ethical approval

As per international standard written ethical approval has been collected and preserved by the author(s)

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