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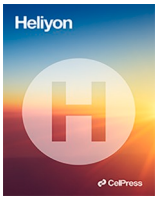

Case report

Acute onset parkinsonism after consumption of traditional Chinese medicine: A cause for vigilance

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ABSTRACT

Mercury poisoning is a rare yet critical toxicological emergency, typically associated with chronic exposure. This case report details the unusual presentation of acute parkinsonism in a 66-year-old woman who had been ingesting black pills, an unidentified kind of traditional Chinese medicine, obtained from a pirate radio source. The patient displayed symptoms such as acute onset frequent falls, unsteady gait, and slow movements, prompting a detailed medical examination. The patient's neurological assessment revealed classic parkinsonian features, including mask face, cogwheel rigidity, and bradykinesia. Subsequent laboratory investigations disclosed an elevated blood mercury level (47 μg/L), and imaging studies, including 99mTc-TRODAT-1 SPECT, confirmed bilateral putamina dysfunction consistent with secondary parkinsonism. Traditional medications of Parkinsonism provided minimal relief, leading to the introduction of chelation therapy with dimercaptosuccinic acid (DMSA), resulting in a significant improvement in symptoms following a 20-day course. The discussion emphasizes the distinctive clinical manifestations of organic and inorganic mercury poisoning, underscoring the delayed onset and central nervous system involvement in organic mercury toxicity. The unidentified black pills, known to exceed mercury standards, were identified as the likely source of mercury poisoning in this case. This report acknowledges the potential reversibility of certain causes of acute parkinsonism and highlights the importance of a thorough drug history and toxicology assessment in patients presenting with acute parkinsonism. This report also contributes to the existing understanding of mercury-induced parkinsonism and emphasizes the significance of timely intervention in managing similar cases.

1. Introduction

The onset of acute parkinsonism is an uncommon occurrence, often associated with various underlying factors. Potential causes

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include rapid onset dystonia-parkinsonism, post-encephalitic sequelae, vascular issues like stroke or subdural hematoma, acute metabolic disturbances, osmotic demyelination syndrome (including central and extra-pontine myelinolysis after rapid correction of hyponatremia), drug-related side effects (such as neuroleptic malignant syndrome secondary to antipsychotics), and exposure to toxins like cadmium, carbon monoxide, mercury, and organophosphates [1]. Notably, mercury poisoning, while infrequent, constitutes a critical toxicological emergency with diverse clinical manifestations contingent on its organic or inorganic form. Although neurological symptoms typically manifest in cases of chronic mercury intoxication, the acute and atypical nature of these presentations poses a diagnostic challenge, necessitating swift and accurate identification.

2. Case presentation

A 66-year-old woman presented to our hospital with complaints of frequent falls for 2 days. Associated symptoms included hand tremors, unsteady gait, slow movements, and depressed mood. She had a medical history of insomnia and anxiety disorder, and she had been regularly taking alprazolam and zolpidem. She mentioned that she had been taking a large amount of black pills (unidentified traditional Chinese medicine) from pirate radio with unknown ingredients for 2 weeks due to feeling fatigued. She reported no family medical history or consumption of alcohol. On neurological examination, mask face, cogwheel rigidity in four extremities, and bradykinesia of four limbs were observed. A comprehensive laboratory evaluation was conducted, including an assessment of heavy metals. The only abnormal finding was an elevated blood mercury level (Hg = 47 μg/L). Brain MRI did not identify any lesions consistent with her clinical symptoms. 99mTc-TRODAT-1 SPECT (TRODAT) revealed heterogeneously mild dysfunction of bilateral putamina with right inhomogeneity and left posterior segment reduction, which is compatible with secondary parkinsonism (Fig. 1). Under the impression of secondary parkinsonism induced by mercury intoxication, carbidopa (75mg/day) and levodopa (300mg/day) were prescribed for symptom relief. However, minimal symptomatic relief was noted from this regimen under regular follow-up for 3 weeks. Although the patient demonstrated high adherence to the prescribed medication, her response to carbidopa and levodopa was not ideal. While the medication slightly improved her tremors, other symptoms persisted and continued to affect her daily life. Chelation therapy with dimercaptosuccinic acid (DMSA) at 400mg BID (around 20 mg/kg/day) was added and extended over 20 days to reduce her blood mercury levels. The blood mercury levels decreased from 47 μg/L to 20.7 μg/L finally. Throughout continuous monitoring of the patient's conditions, no adverse effects were reported by the patient, and no unexpected reactions were observed. Following the treatment, her symptoms showed a near-complete and gradual recovery (Fig. 2).

3. Discussion

Organic mercury and inorganic mercury poisoning are compared in various aspects, including the mechanism of absorption, the speed of onset, the organs primarily affected, and the distinct clinical manifestations they present. Inorganic mercury poisoning primarily occurs through absorption via the respiratory tract and occasionally through the skin or gastrointestinal (GI) tract in smaller percentages [2]. It remains in the body for about two months before being excreted through urine and feces. Conversely, methylmercury, a significant organic mercury form, is highly absorbed through the GI and respiratory tract. Once absorbed, it swiftly distributes throughout tissues within 30 hours and is mainly excreted through feces via bile. Its half-life ranges from 45 to 70 days. Metallic and organic mercury can readily cross the blood-brain barrier, while inorganic mercury cannot, leading to distinct clinical presentations. In cases of inorganic mercury poisoning, symptoms may manifest as profuse vomiting and diarrhea, potentially progressing to hypovolemic shock, oliguric renal failure, and, in severe instances, fatality. Conversely, organic mercury toxicity can exhibit delayed symptoms, emerging weeks after exposure, and typically affects the central nervous system. Manifestations include paraesthesia, headaches, ataxia, dysarthria, visual field constriction, blindness, and hearing impairment [3]. Diseases like Minamata

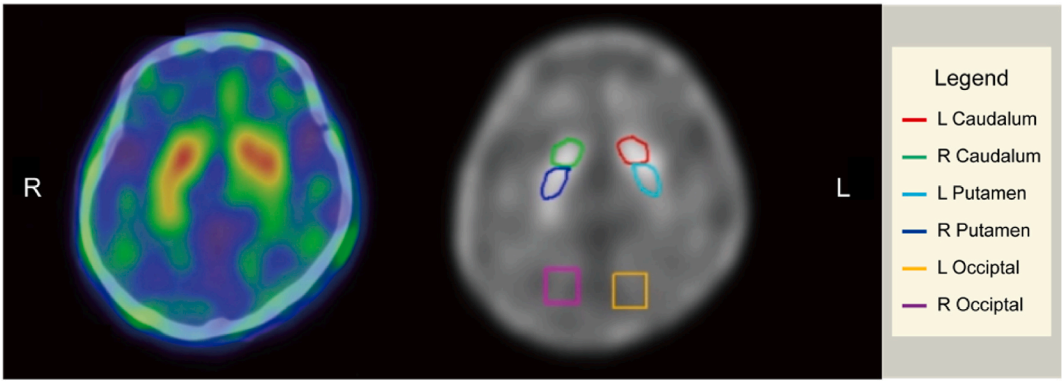

Fig. 1. Technetium-99m-labeled tropane derivative single-photon emission computed tomography (SPECT) disclosed mild to moderate reduction of dopamine transporter-1 avidity in the bilateral putamina, especially the left posterior segment. Heterogeneous dopamine transporter-1 avidity in the bilateral caudate head. Heterogeneous dopamine transporter-1 avidity in the bilateral caudate body, with a relatively more pronounced reduction on the left side.

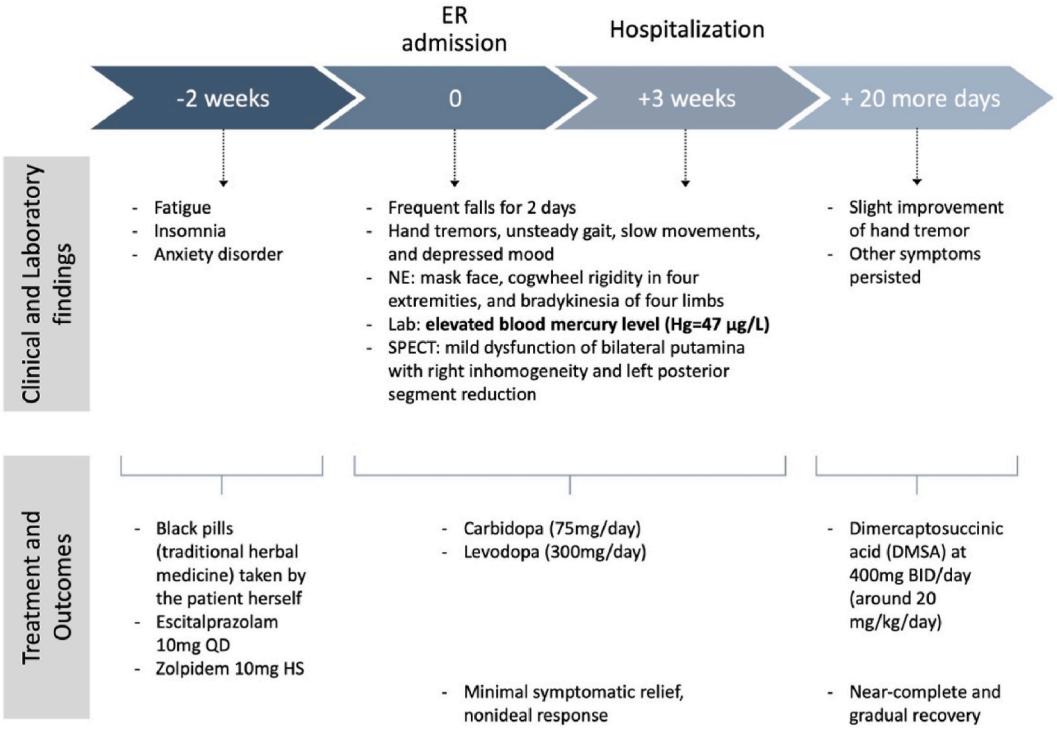

Fig. 2. Timeline of the patient's clinical presentations, laboratory findings, treatment, and outcomes.

Disease, induced by methylmercury, specifically damage the central nervous system, distinguishing it from inorganic mercury poisoning, which primarily targets the kidneys. Despite these distinctions, mercury exposure has been associated with classic parkinsonian symptoms, including ataxia, tremors, and myoclonus [4]. Research indicates that mercury, in various chemical forms, exerts neurotoxic effects, contributing to neurochemical and neuropathological changes akin to those observed in Parkinson's disease [5]. This includes the loss of dopamine neurons, tubulin and axon degeneration, mitochondrial dysfunction, and α-synuclein aggregation [6].

Acute onset parkinsonism, characterized by rapid development of tremor, rigidity, and bradykinesia, can be a rare but severe consequence of mercury intoxication [5]. While the exact mechanisms remain under investigation, several studies have demonstrated the potential for mercury to accumulate in the basal ganglia, leading to neuronal damage and disruption of dopamine signaling pathways characteristic of Parkinson's disease. Cases of mercury-induced parkinsonism have been reported in individuals with occupational exposure, accidental ingestion, and even through traditional medicine practices involving mercury-containing remedies. The black pills (Chinese herbal medicines) from the pirate radio incident are known to contain mercury levels exceeding national standards (with organic mercury being more prevalent), likely contributing to the observed mercury poisoning [7,8]. When confronted with a patient presenting acute onset parkinsonism, infectious and immune mechanisms (viral, autoimmune, paraneoplastic), drug-induced, toxic-induced, and psychiatric causes are common etiological considerations. The evaluation and diagnosis of acute parkinsonism necessitate a comprehensive approach involving cerebrospinal fluid analysis, toxicology screenings, standard blood tests, and imaging studies. Given the potential for reversible forms of these conditions, acute parkinsonism should be treated as a medical emergency. Prompt identification and chelation therapy are crucial for minimizing neurological damage and potentially improving symptoms. Secondary parkinsonism usually has a suboptimal response to dopaminergic treatment. Treating the underlying cause of secondary parkinsonism is usually more crucial. However, the long-term prognosis can vary depending on the severity of intoxication and individual response to treatment.

4. Conclusion

The case underscores the critical importance of obtaining a detailed drug history and the need for vigilance in conducting toxicology assessments when managing patients with acute parkinsonism. Further research is needed to elucidate the precise molecular mechanisms underlying mercury-induced parkinsonism and develop more effective treatment strategies for this potentially debilitating condition.

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Ethics statement

- All participants/patients (or their proxies/legal guardians) provided informed consent to participate in the study.
- The patient provided informed consent for the publication of their anonymized case details and images.

Data availability statement

Data included in article/supp. material/referenced in article.

CRediT authorship contribution statement

Ko-Ying Kuo: Writing – original draft. **Ying-Sheng Li:** Writing – original draft. **Poyin Huang:** Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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