The Lethal Lure of Thallium: A Case Report

Abstract

Thallium toxicity is often missed due to its rarity. We present a case of thallium poisoning in a 29-year-old who presented with features consistent with the Guillain-Barré syndrome. He was treated successfully with Prussian blue, hemoperfusion, and hemodialysis.

Keywords: *Thallium, Hemodialysis, Hemoperfusion*

Introduction

Historically, thallium has been used as a depilatory agent and in the treatment of ringworm.[1] Its use is currently limited to industrial and medical purposes, but still widely used as rodenticide. It is often referred to as "poisoner's poison" as it is odorless, tasteless, and colorless.[2]

Case Report

A 29-year-old male with no comorbidities presented with acute paresthesia over the soles of his feet, which progressed over the course of a day. He was unable to walk the next day due to weakness and pain on both his feet. Upon first evaluation in a nearby hospital, diagnosis of acute peripheral neuritis was made. He was discharged on steroids, pregabalin, and alprazolam.

A week later, he was readmitted with complaints of fever, epigastric pain, constipation, oral ulcers, persistent pain over both legs, and tingling of fingers. A nerve conduction study showed demyelinating sensory and motor radiculoneuropathy involving both his lower limbs. Magnetic resonance imaging (MRI) of spine showed desiccation at multiple levels with no neural compromise and cerebrospinal fluid analysis revealed albuminocytological dissociation. He was diagnosed to have Guillain-Barré syndrome (GBS) and 2g/kg of intravenous immunoglobulin (IVIG) was given. After four doses, he showed some improvement and was discharged.

He was readmitted again after two weeks with complaints of hematemesis, constipation, pain, and worsening weakness of his lower limbs. Endoscopy showed features of reflux esophagitis. His deep tendon reflexes were now depressed in both his lower limbs with a power of 2/5, whereas his upper limb reflexes were normal. Repeat MRI and evaluation for sarcoidosis, porphyria, and lupus were negative. Suspecting atypical progressive GBS, he received three more doses of IVIG.

Due to persistent weakness and new onset of alopecia, screening for heavy metals was done. He had high thallium levels (>400 µg/L) in blood and urine. He was started on daily intermittent hemodialysis (HD) for one week along with oral Prussian blue (PB) (3g/day). Despite daily HD, his repeat blood and urine thallium levels were still high (>400 µg/L); hence, we opted for HD and hemoperfusion (HP) using HA230 cartridge in series (ten sessions).

His neurological status showed gradual improvement. After 22 sessions of dialysis and 10 sessions of HP, thallium levels progressively reduced to <16.87 µg/L. Upon discharge, he had some weakness of both the lower limbs requiring support. Follow-up after two months showed no neurologic deficit, except for mild neuropathic pain and foot drop, and his last serum thallium level was <2.5 µg/L.

The patient's mother tested negative for thallium, excluding household exposure. Despite thorough investigations, including interviews with the patient's colleagues (none of them exhibited similar symptoms) and law enforcement involvement, the exact source of the poisoning remains unidentified, underscoring the challenge of tracing environmental toxins.

Discussion

Thallium poisoning usually results from ingestion, leading to substantial accumulation in the kidneys and liver. This is due to thallium's large volume of distribution (3–10 L/kg) and its extensive enterohepatic circulation which contributes to its prolonged terminal half-life of two to four days.[3] Thallium's similarity to potassium allows it to bind to potassium sites with higher affinity, disrupting the Na^+/K^+-ATPase pump activity and leading to neurological manifestations such as painful sensorimotor dysfunction, numbness, tremors, and alopecia due to its effect on keratin cross-linking.[4,5]

Thallium poisoning is often misdiagnosed as GBS (50%), gastritis (41.7%), rheumatic diseases (8.3%), or skin diseases (8.3%),[3] which can delay appropriate treatment. Long-term complications, even in successfully treated patients, may include persistent pain, dysesthesia, tremors, depression, and anxiety.[4]

PB acts as an ion exchange resin, substituting potassium with thallium due to its larger ionic radius (0.147 nm vs. 0.133 nm), enhancing thallium excretion by interrupting enterohepatic recycling.[6] However, assessing PB's effectiveness in our case was challenging due to the unknown timing of exposure and initial thallium levels > 400 µg/L, which complicated accurate monitoring.

Even though thallium is nonprotein bound, management of poisoning is difficult due to its slow intercompartmental transfer and large volume of distribution, which often leads to rebound in serum levels despite treatment. High-flux dialyzers enhance thallium removal, and the EXTRIP (EXtracorporeal TReatments In Poisoning) workgroup supports extracorporeal therapy (ECTR) for severe thallium poisoning due to its superior effectiveness compared to endogenous elimination and for the lack of better alternatives.[3] Consequently, HD was used. Despite daily HD, thallium levels remained high, prompting the addition of HP with an HA230 cartridge.

HP improves thallium clearance through adsorption onto activated charcoal, addressing the slow elimination of thallium. Lin *et al*. highlighted the role of HP, particularly in cases with delayed admission, supporting its use in severe poisoning scenarios.[5] By combining HD for long-term toxin removal with HP for rapid clearance, we provided a comprehensive approach to managing thallium poisoning.

Conclusion

Thallium poisoning should be considered in differential diagnoses for patients with suspected GBS who do not respond to IVIG. Early and effective intervention is critical to prevent long-term neurological sequelae.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Conflicts of interest

There are no conflicts of interest.

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