Indian Journal of Forensic and Community Medicine 2022;9(4):178–180

Content available at: https://www.ipinnovative.com/open-access-journals

Indian Journal of Forensic and Community Medicine

Journal homepage: https://www.ijfcm.org/

Case Report

Thallium in rodenticide- banned or not..!!!- Two case reports

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ARTICLE INFO

Article history:
Received 11-11-2022
Accepted 26-11-2022
Available online 09-01-2023

Keywords:
Rodenticide
Peripheral neuropathy
Alopecia
Potassium chloride
Hemodialysis

ABSTRACT

Thallium poisoning is considered rare among all heavy metals. They are banned in view of their criminal use as an homicidal agent and a potential threat to environment. Their illegal use are still under practice and reported from time to time. We report two cases presented with similar manifestations i.e., gastrointestinal symptoms, peripheral neuropathy and alopecia which are characteristic of thallium toxicity which was later confirmed biochemically in blood and urine specimen. They were both treated successfully with activated charcoal, potassium chloride and hemodialysis.

1. Introduction

Heavy metal poisoning is a major hazard for the environment and public health. On account of their toxic, non-degradable and bio-accumulative properties, developing countries like India are facing the huge health burden on the population and the entire ecosystem. Thallium is a toxic heavy metal, which was accidentally discovered by Sir William Crookes in 1861 by burning the dust from a sulfuric acid industrial plant.[1] Since then, thallium has been used extensively, and human exposure to thallium compounds has occurred naturally. In the initial time period, it was used as medicinal agents for ringworms and rodenticides but soon it was banned because of the increasing cases of its homicide, herbal products contamination and drug abuse. Presently it is used in industrial set-ups, as radioisotope (Tl-201) in cardiac perfusion scan, optical lenses, extreme cold thermometers and electric lighting.[2,3] It is also useful in the manufacture of imitation jewellery, pigments, and fireworks (green color). In India also, cases of thallium poisoning have been observed even in the recent past despite its banned status. The biological half-life of thallium has a wide range of 1 to 30 days and depends on the entry route into the body, initial constipation, and enterohepatic circulation.[4] Shortly after ingestion typical presentation starts with acute gastroenteritis such as abdominal pain and vomiting which was followed by the intermediate stage, the distribution of thallium into the central nervous system within several days of exposure, causes paraesthesia, hyperalgesia that gradually develops, especially in the legs, followed by muscle weakness ascending from the lower extremities, indicative of heavy metal toxicity. Thallium also interferes with cross-linking of keratin, responsible for changes occurring in hair, skin, and nails.[5] Other manifestations of Tl exposure may include autonomic instability, involvement of cranial nerves, and AKI acute kidney injury. Severe cases develop altered mental status, coma with loss of airway-protective reflexes, respiratory muscle paralysis, and cardiac arrest in the later stages. The severity of symptoms depends

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https://doi.org/10.18231/j.ijfcm.2022.037

on the route of exposure and the victim's dose and age.

Here we report two similar cases of mother and daughter living alone, consumed wheat flour for 15 days which was brought by one of her in-laws relative, that was later found to be intoxicated with thallium containing rodenticide.

2. Case Report

Our first patient was a 45yr old female, resident of Aligarh known case of hypothyroidism, presented with abdominal pain along with nausea and vomiting since 10 days and pins and needle prick sensation associated with burning in both feet for 7 days. The second patient was her mother, 68year old, known case of hypertension, presented with similar complaints of abdominal pain with nausea, vomiting, burning feet and pins and needle prick sensation bilaterally. They both also complained of loss of hair from the scalp for the last 3-4 days (Figure 1). No history of colicky type of abdominal pain, fever, weakness in any body part, rashes, palpitations, headache and fever. No intake of any drug other than medications for hypertension by first patient and hypertension medications by her mother. No history of radiation exposure.

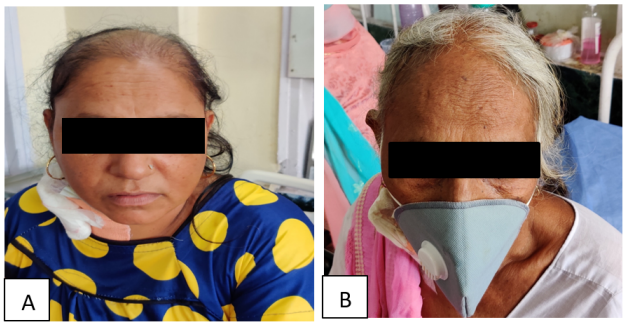

Fig. 1: Alopecia in frontal and parietal region. Patient-1 daughter(A) and patient-2, mother (B)

Clinical examination in both the patients revealed alopecia in frontal and parietal area of scalp. Loss of lateral one third of eyebrows was also seen. Nails and skin examination were normal. Neurological examination showed impaired vibration and position sense below knee joint and diminished ankle reflexes bilaterally. Rest CNS and other systemic examination were unremarkable in both the patients. Ophthalmic examination was normal. All routine blood investigations done in both patients including CBC, Liver and kidney function tests were normal. Urine routine and microscopy examination was normal. Anti HCV and HBsAg were non-reactive, HIV-Negative by rapid card test. Vitamin B12 and Folic acid levels were normal. Thyroid function test was normal and ANA was Negative. Chest X-ray, ECG and USG- abdomen were within normal limits. The typical clinical features of abdominal symptoms, painful sensory peripheral neuropathy and alopecia with the history of recent use of rodenticide brought by their relative were suspicious of heavy metal toxicity and thus lead us to send thallium levels in serum and urine which showed markedly raised levels. (Table 1)

Treatment comprised of multi dose activated charcoal. Intravenous fluids with forced diuresis and Syrup Potassium chloride were given along with rest of the supportive treatment. A total of six cycles of Hemodialysis were given to both of these patients.

Their symptoms of pain and paresthesia improved gradually and serum thallium levels came down to <0.1 microgram/litre in patient-1 and <0.5 microgram/litre in patient-2. Both the patients were followed up after 3 months, neurological motor and sensory examination were normal. In later period, they both lost to follow-up.

3. Discussion

Thallium is odourless, colourless, tasteless heavy metal. Exposure occurs by oral, dermal, and inhalation routes. Water-soluble salts are more toxic than the less water-soluble salts (e.g., thallium sulphate is more toxic than sulphide). Dermal exposure may even occur through rubber gloves.[6] The lethal thallium dose in humans is reported to be 10-15 mg/ kg; deaths can occur in adults with doses as low as 8 mg/kg.[7] It is rapidly distributed into the intracellular space, exhibiting an initial apparent half-life of 5 minutes.[8,9] Distribution into other peripheral compartments, including the central nervous system (CNS), occurs over 24 hours.[8–10] Tl has a large apparent volume of distribution (3–10 L/kg).[8,11,12] Once distribution is complete, Tl is detectable in almost all organs, with highest concentrations in kidney and liver, followed by bone, stomach, intestine, spleen, muscle, lung, testes, and brain.[9,11] Major routes of elimination are primarily unchanged via the bile and feces (51%) and urine (26%), but it is also excreted in sweat, saliva, tears, and breast milk and appears in hair and nails.[10] Although Tl appears in urine within an hour of exposure, its large apparent volume of distribution and extensive enterohepatic recirculation result in a long terminal elimination half-life, commonly reported to be between 2 and 4 days.[4,9,11,12] More prolonged half-lives of 10–15 days have been reported.[8,9]

The mechanisms of thallium intoxication include the substitution of potassium in various enzymatic and electrochemical processes such as sodium-potassium-adenosine triphosphatase, high affinity for the sulfhydryl or thiol group of mitochondrial membranes, resulting in inhibition of ATP generation at the Kreb's cycle and oxidative phosphorylation.[13] In addition, thallium disrupts calcium homeostasis.

Treatment with Prussian blue (or activated charcoal) interrupts the enterohepatic cycling of thallium, thus enhancing faecal elimination of the metal.[14] Urinary thallium excretion is enhanced by forced diuresis with

Table 1: Thallium levels in serum and urine of both the patients

	Patient-1	Patient-2	Normal range
Serum Thallium level:	335 microgram/litre	136 microgram/litre	(<10 microgram/litre)
Urinary thallium level:	2422 microgram/litre	2320 microgram/litre	(<2 microgram/litre)

potassium repletion, which should be undertaken cautiously due to precipitation of neurological and cardiac toxicity.[15] Because of its small size and its lack of protein binding, thallium is considered dialyzable.[14] Therefore, haemodialysis should be considered early in massive toxicities. In our hospital as Prussian blue was not readily available, we considered hemodialysis as the main therapy which was able to remove thallium effectively from the body and all symptoms of intoxication were resolved gradually.

Published data on the prognosis for severe thallium poisoning is limited, especially regarding the reversibility of neurological and vision impairment.[16] However, overall, the long-term prognosis of neurological impairment seems to be relatively good for patients who survive the acute phase of thallium poisoning.

4. Conclusion

Apart from the industries, criminal use of thallium is still practised as a rodenticide as seen in our two cases. The presence of classical features of acute abdominal discomfort, progressive painful sensory polyneuropathy and alopecia make the clinical suspicion of Thallium poisoning which can be further confirmed by demonstrating thallium levels in blood and urine samples. Role of hemodialysis was found effective in thallium elimination and improving outcome.

5. Source of Funding

None.

6. Conflicts of Interest

None.

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Cite this article: Verma P, Verma A, Verma A, Surbhi. Thallium in rodenticide- banned or not..!!!- Two case reports. *Indian J Forensic Community Med* 2022;9(4):178-180.