Case Report

To Death by Thallium - A Case of Thallium Poisoning

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ABSTRACT

Thallium is a systemic, metallic toxicant, which has high affinity for sulphydryl groups of cells and potassium binding sites throughout the body. Alopecia, painful peripheral neuropathy, gastrointestinal symptoms and visual disturbances are characteristic manifestations following thallium exposure. However, details with regard to thallium toxicity or management are not widely known to physicians.

In this case report, we present a misdiagnosed case of thallium poisoning, wherein timely diagnosis and specific treatment could have saved a life.

Key Words: Homicidal poisoning; Thallium

INTRODUCTION

Thallium is a toxic metal that is used in the semiconductor industry and in the manufacture of optical systems and photoelectric cells. It is also used in jewellery pigments and to manufacture highly refractive optical glass, also used for cardiac perfusion imaging, and as a depilatory. In 1972, thallium and its compounds were banned for use in pesticides in the US. However, in India, thallium and its compounds are available for commercial use. Important derivatives include thallous sulphate, acetate, iodide, nitrate and carbonate. Most of these salts (particularly thallous sulphate) are odourless, tasteless and freely soluble in water. Humans may be exposed to thallium by ingestion, inhalation or dermal absorption. Thallium salts unfortunately possess several properties of a "perfect poison" – they are odourless, tasteless, freely

soluble in water, and produce toxic manifestations, which closely resemble natural disease.⁴

The Case: A 27-year-old recently-married female presented with disturbance of mood, loss of appetite, low grade fever and burning micturition since 2 weeks for which she was treated by a gynaecologist with norfloxacin. A week later, she grew progressively weak and required assistance to walk. She was shown to a psychiatrist, but there was no improvement. Over the next 2 days her condition worsened and she became drowsy and immobile.

She was hospitalized by her family members, and at admission she had an episode of focal upper limb seizures. Dropping blood pressure and other vitals indicated a need for ventilator support. CT scan and MRI of the brain revealed essentially normal findings. However, the CSF was reported to have high protein values. She was diagnosed with possible viral encephalitis and managed on those lines. She continued to detiorate both neurogically and haemodynamically over a period of time. A repeat MRI revealed areas of restricted diffusion bilaterally in cerebellar white matter, splenium of corpus callosum, right centrum semiovale and bilateral parafalcine frontal subcortical white matter.

Three weeks after the first symptoms, she began to lose hair. The treating dermatologist opined it was due to *Telogen effluvium*. However, despite all efforts there was no improvement. In view of hair loss and altered

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sensorium of uncertain aetiology, a sample of blood was sent for toxicology screening, suspecting a possible lead, arsenic or thallium poisoning. Two weeks later, the laboratory reported it to contain thallium with a value of 290 mcg/mL. The case was now registered as a medicolegal case.

In view of the fact that it appeared now to be a case of thallium poisoning, she was treated with Prussian blue, forced alkaline diuresis and even haemodialysis for a week. But the patient died, and a medicolegal autopsy was conducted. The main findings at autopsy were as follows:

- 1. External examination: The deceased measured 170 cm in length, was moderately built and nourished, and was fair in complexion. Rigor mortis was present throughout the body. Purplish-blue postmortem staining was present over the back of the body. There was total loss of scalp hair, marked facial pallor, maculopapular skin eruptions over the face, stomatitis and transverse white stripes on the finger nails.
- Internal examination: Brain was oedematous and cut section showed petechial haemorrhages in the white matter. Lungs were also found to be oedematous and cut section exuded frothy blood. Mucosa of stomach was congested and haemorrhagic at many places.
- Histopathological examination of the brain revealed widespread degeneration of nerve cells. Kidney showed evidence of tubular necrosis and liver showed fatty degeneration.
- 4. Cause of death was opined as "respiratory failure as a result of consumption of a compound containing thallium." The manner of poisoning remained unclear.

DISCUSSION

Thallium is a soft pliable metal, which is acquiring an increasingly notorious reputation as an "ideal homicidal poison." Fatal dose is around 1 g, depending on the type of thallium compound administered.

In mild cases, joint pains in the lower limbs, loss of appetite, stomatitis and drowsiness occur, which pass off in a few days. In severe cases, there is abdominal colic, vomiting, pain in the muscles, joints and nerves, lethargy, motor and sensory neuropathies, convulsions, psychosis, optic neuritis, tremors, delirium and coma. Hair loss occurs after 1–2 weeks. Large tufts tend to come away. Loss of outer third of eyebrows is said to be very significant.⁴ There may be cardiac manifestations. Maculopapular skin

eruption having butterfly distribution on face is characteristic. There may be Mees lines on the nails. Death usually occurs from respiratory failure due to paralysis of respiratory muscles.^{5,6}

The outstanding combination of alopecia and skin rash, painful peripheral neuropathy and mental confusion with lethargy (thallium triad) in any clinical case must immediately arouse the suspicion of chronic thallium poisoning. The progression of symptoms may sometimes mimic other conditions, particularly Guillain-Barre syndrome, acute porphyria, psychosis or thiamine deficiency. Diagnosis can be made by X-ray of the abdomen, which may reveal opacities in the GI tract or liver. Microscopy of the scalp hair may reveal a diagnostic pattern of black pigmentation of hair roots. A quick urine colour test combining a few drops of urine with a few drops of saturated bromine water, HCl, sulphosalicylic acid, rhodamine B and then centrifuged with the addition of benzene will produce a bright yellow or fluorescent red colour.

In the case being reported, the presenting signs and symptoms and their progression were very much in favour of chronic thallium poisoning, and early accurate diagnosis could have saved the patient's life. One of the major reasons for the clinical practitioners ignoring the grim reality of poisoning in India is that the diagnosis and treatment are woefully inadequate and information resources are very scanty.⁷

CONCLUSION

It is a known fact that a doctor is not liable for an error of judgment or of diagnosis, if he has acted with ordinary care and secured all necessary data on which to base a sound judgment. There is always room for a difference of opinion among doctors. However, the treating doctor must consider poisoning to be a possibility in the early stages when a clear cut diagnosis is not available keeping in mind that a handful of poisons mimic natural diseases. In India, toxicology is being taught mainly by the department of Forensic Medicine and Toxicology in all medical colleges. To overcome this problem of mis-diagnosis in poisoning cases, we suggest that it is time that medical institutes improved the training in toxicology to assist the treating doctor in every case of poisoning, as well as cases where poisoning is a possibility.

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