Thallium Poisoning Presenting as Paresthesias, Paresis, Psychosis and Pain in Abdomen

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Abstract

Due to involvement of multiple systems, thallium poisoning is notorious for complexity and seriousness as symptoms of toxicity are non-specific and diverse. Alopecia and painful neuropathy are its cardinal features, others being gastrointestinal disturbances, encephalopathy, tachycardia, ataxia, hepatorenal and cardiac damage etc.

We report a case of thallium poisoning who presented initially with gastrointestinal symptoms and later developed neurological features (peripheral neuropathy and delirium). Various diagnoses were entertained in this case and thallium poisoning was suspected only after he developed alopecia and neuropsychosis. He made a significant recovery by conservative management in spite of delay in diagnosis.

We conclude that a high level of suspicion should be kept for thallium poisoning, especially in patients with painful, peripheral neuropathy and gastrointestinal symptoms which appear earlier than alopecia, since prognosis is more rewarding with early diagnosis, leading to complete recovery. ©

INTRODUCTION

Thallium poisoning is rare, usually found in context of suicide, homicide or after accidental exposure on entering food chain via rodenticides or insecticides. Most of thallium salts are colorless, tasteless and odorless, which makes them potentially an ideal poison. It is one of the most potent neurotoxin and involves multiple systems, including cardiac, renal, neurological and gastrointestinal; thus early diagnosis is difficult and is often delayed till alopecia appears. We present a case in which thallium toxicity was not suspected until he developed severe neuropsychiatric symptoms and alopecia.

CASE REPORT

A 33 years sales manager complained of an irritating abnormal sensation on the tip of tongue immediately after consumption of sweets at a dinner party. Within 24 hrs, he developed severe tingling and burning sensation, followed by weakness in all four limbs, which was insidious in onset, but gradually progressed over 2-3 days limbs. By day 7, he was unable to move his limbs and got confined to bed. Simultaneously he developed difficulty in speaking in form of decreased volume and nasal twang. He was hospitalized where a diagnosis of Guillain-Barre syndrome (LGBS) was suspected, after nerve conduction studies suggested axonal neuropathy. On day 12 of illness he observed pustular rash over the face along with severe headache. The same day he also developed pain in abdomen, vomiting and diarrhea. These gastrointestinal symptoms persisted for 2-3 days and they stopped after treatment with intravenous fluids. He also had retention of urine. The patient was also investigated for porphyria.

After 20 days from the onset of illness, he developed giddiness and blurring of vision. He was given intravenous methyl prednisolone. Within next 2-3 days he developed rapid loss of scalp hair (alopecia), especially in occipital area. Methyl prednisolone was stopped as relatives insisted that hair loss was due to it. By day 32 of illness, most of his scalp hairs were lost and he virtually became bald. Meanwhile he started having tremors (titubation) of neck and head, which were present initially only in sitting position but later also while he was lying down. Due to further deterioration in form of unconsciousness, he was referred to tertiary care center with presumptive diagnosis of acute disseminated encephalomyelitis (ADEM). Over this period there was considerable loss of appetite and weight (8 kg). He remained afebrile throughout this illness.

Patient presented at our hospital after 41 days from the onset, in altered sensorium. There was irrelevant talking, agitation, gross restlessness and aggressiveness. Examination revealed pustular rash over face, alopecia (Fig. 1) and congestion in both eyes. There was sinus
tachycardia (140/min) but blood pressure was normal (130/80 mm of Hg). He had mild, non-tender hepatomegaly. Neurological examination revealed delirium, irritability, and agitation. He had hypophonia and dysarthria in the form of a slow scanning speech. The gag reflex was diminished. Other cranial nerves (including fundus) were normal. There was generalized wasting and hypotonia in all limbs. Power was II/V in all 4 limbs. Weakness was predominantly distal. All deep tendon reflexes were decreased and planter response was not elicitable. Titubation was present; He had alldynia in soles and impaired posterior column (decreased joint position and vibration sense) in all four limbs. He had a broad based ataxic gait (which was possible only with assistance).

On investigations, hemogram and blood counts were normal, ESR was increased (48 mm for 1st hour), and liver transaminases were elevated. Urine for porphobilinogen was negative. ECG showed sinus tachycardia. EEG revealed generalized slow wave activity. Cranial CT and MRI were normal. Nerve conduction studies were suggestive of axonal neuropathy in both common peroneal and median nerves. Sensory nerves were normal. EMG showed neurogenic pattern of denervation. Acuity of vision - ortho dextra (OD) – finger counting (FC) 2 feet, ortho sinistra (OS) - FC 2.5 m. Field of vision (Goldman perimetry) was constricted, more so in the right eye superiorly. Color vision was defective. On fundus examination, there was bilateral diffuse pallor of disc. Indirect laryngoscopy showed congestion in vocal cords and vestibular folds.

In view of painful peripheral neuropathy, cranial nerve palsy, alopecia and behavioral abnormalities, presumptive diagnosis of thallium poisoning was made. Blood sample for thallium estimation was sent. Treatment for thallium poisoning was initiated with intravenous potassium chloride, furosemide, laxatives, charcoal tablets and vitamin supplements while waiting for reports. Serum calcium levels which were regularly monitored, since hypocalcaemia often develops, were normal. Blood thallium level by atomic absorption spectrophotometry (AAS) was 109 µg/ dl (sample taken on 42nd day of illness).

The patient showed symptomatic improvement within a week of therapy. He became conscious, the ataxia and head tremor started decreasing within 4 days. There was decrease in irritability and aggressiveness within one week and his appetite improved. At the time of being relieved after 2 weeks of hospital stay, paresthesias had decreased, the power in all limbs had improved to IV/V, but the patient required support for walking. The color vision remained defective and there was no improvement in visual acuity at that time. The speech improved but the nasal twang persisted. Insomnia and depression disappeared. The hair started growing after about 2 months. In subsequent follow up after 6 months, he had recovered significantly and the alopecia disappeared (Fig. 2). He had become independent as there was no residual neurological deficit, including vision (both color and acuity of vision).

**DISCUSSION**

Thallium salts are used in some rodent poisons, fireworks, imitation jewellery, switching devices and chemical catalyst.1,2 Painful peripheral neuropathy and alopecia are the two of its cardinal symptoms.13 The other associated symptoms are gastrointestinal and cardiac problems which appear early. Ataxia, tremors,
arthetosis, cranial nerve palsy, and rarely coma, convulsions and death are other important complicating features of thallium poisoning.4

Most cases of thallium toxicity occur after oral ingestion or by inhalation of fumes. The exact mechanism of toxicity is unclear but postulations include ligand formation with protein thiol groups, inhibition of cellular respiration, disruption of calcium homeostasis, interaction with riboflavin and riboflavin based cofactors.1 Once the poison has been introduced into the system the cells, tissues and organs which absorb it, get injured and damaged. The pathogenic factors determining the nature and severity of the illness are the quantity of poison absorbed, strength and quality of the immunodefensive reaction of cell, tissues and organ and the varying individual tolerance to the poison.3,4 The elimination half time of thallium is between 1.7 and 30 days.1 Due to long terminal half-life, it may act as a cumulative poison. The normal total blood thallium concentration is under 2 µg/dL and concentration greater than 100 µg/dL are toxic.1 High level of thallium concentration is seen in blood, urine, liver and kidney. The recommended method of diagnosing is estimation of thallium level in a 24 hour urine sample, which unfortunately could not be done in our case, so a random blood level estimation was done.

In humans, the most characteristic sign of this toxicity is alopecia but it usually appears, as it was evident in our case after 2 to 3 weeks.3,5 Painful paraesthesia of hands and feet appears early; similarly gastrointestinal symptoms are also reported to occur within a few days.1,6 These two symptoms are nonspecific but can be a clue to early diagnosis if high index of suspicion is maintained. Other signs like ophthalmoplegia, cerebellar and extrapyramidal signs, present in our case are also highly suggestive of thallium poisoning.4,5 Immediate cardiovascular complications include hypertension, sinus tachycardia, ECG abnormality and ventricular fibrillation.1,5 Heart is the main target of thallium in early stage of acute poisoning and in our case it manifested as severe tachycardia (heart rate of 150/min).

Neuropsychological symptoms in the form of irritability with aggressive outbursts have been reported previously.3,6

Recommended treatment of thallium poisoning is administration of Prussian blue (potassium ferric ferrocyanide).1,8 Potassium therapy in the form of potassium chloride was attempted in this case, since Prussian blue could not be procured. It increases the rate of loss of thallium from body compartment and reported to be probably an alternative in treatment as evident in our case.2,6

We conclude a high level of suspicion should be kept for thallium poisoning, especially in patients with painful peripheral neuropathy and gastrointestinal symptoms which appear much earlier than alopecia, since the prognosis is more rewarding with an early diagnosis, leading to complete recovery.

REFERENCES


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