CASE REPORT

Thallium poisoning: emphasis on early diagnosis and response to haemodialysis

U K Misra, J Kalita, R K Yadav, P Ranjan

Thallium poisoning is known for its diverse manifestations and these can delay the diagnosis if a clear history of poisoning is not forthcoming. A 42 year old man presented on the third day of illness with flaccid quadriaparesis and paresthesia, which were confused with Guillain-Barré syndrome. Because of associated loose motions, skin lesions, and liver and kidney dysfunction arsenic poisoning was considered. In the second week he developed ophthalmoplegia, nystagmus, and neck tremor and later developed alopecia, and thallium poisoning was suspected. His serum thallium level on the 18th day of illness was 40 980 µg/ml. He was subjected to haemodialysis, potassium supplementation, laxatives, and B complex supplementation. He showed significant improvement after haemodialysis and at three months he was able to walk with support. At six months of follow up he was independent for activities of daily living. Severe paresthesia, ophthalmoplegia, cerebellar and extrapyramidal signs, and alopecia are highly suggestive of thallium poisoning. Haemodialysis may be effective even in the third week of poisoning.

Thallium is a protoplasmic poison and may be effective in the treatment of arsenic, selenium, and lead poisoning. It is an important industrial poison and is used in various chemical industries. It is also used in optical lenses, for optical fireworks, in green coloured fireworks, in imitation jewellery. Many thallium salts are colourless, odourless, and tasteless hence it is a favoured homicidal poison. Soluble thallium salts such as sulphate, acetate, and carbonate have higher toxicity and their fatal dose is 10–15 mg/kg. Thallium is highly reactive heavy metal, which exists as monovalent and trivalent ionic forms. It is used in semiconductors, low temperature thermometers, and in imitation jewellery. Many thallium salts are colourless, odourless, and tasteless hence it is a favoured homicidal poison. Soluble thallium salts such as sulphate, acetate, and carbonate have higher toxicity and their fatal dose is 10–15 mg/kg. Thallium poisoning commonly occurs after oral ingestion but can also occur after inhalation of contaminated dust or after dermal absorption. Thallium is a protoplasmic poison and may disrupt the sulphhydryl group on the mitochondrial membrane and interferes with the functioning of sodium-potassium ATPase for which thallium has 10 times greater affinity than potassium. The clinical picture of thallium poisoning is non-specific and variable, depending on the dose and route of administration. In the early stage, thallium poisoning is managed by gastric lavage, laxatives, forced diuresis, haemodialysis, and Prussian blue. We report a patient with thallium poisoning who was referred to us with suspected Guillain-Barré syndrome; he responded well to haemodialysis in the third week. We highlight the distinguishing features of thallium poisoning, especially in the early stage of poisoning, and the utility of haemodialysis in management.

Thallium poisoning is managed by gastric lavage, laxatives, forced diuresis, haemodialysis, and Prussian blue.
Treatment and course
After confirming the diagnosis of thallium poisoning, the patient was subjected to haemodialysis. He underwent seven dialysis sessions, each lasting for 4–5 hours daily. He also received 20 mmol potassium chloride thrice daily, laxatives, and intravenous vitamin B complex. After the first dialysis, his consciousness improved and he responded to verbal commands. After the third dialysis, ptosis and nystagmus improved and after the seventh dialysis he was fully conscious and neck and trunk tremor subsided. After the seventh dialysis on the 28th day of illness he developed high fever with leucocytosis (17.0 × 10⁹/l) hence the haemodialysis was stopped. By this time his skin lesions on face had improved and his serum bilirubin, transaminase, and serum creatinine also normalised. He had prominent Mee’s lines. On the 36th day of illness the patient developed severe pain in his abdomen and constipation due to an obstructed inguinal hernia for which herniorrhaphy was done under general anaesthesia. After herniorrhaphy his abdominal symptoms improved. By day 45 he was almost completely bald (fig 3), withdrawn, and depressed but talked appropriately with a nasal twang. He had flaccid quadripareisis with muscle power III–IV, which was more marked distally and in the lower limbs. He had loss of pinprick sensations in glove and stocking distribution; joint position and vibration sense were also absent in the lower limbs. Ankle reflex was lost bilaterally. He had profound dysautonomia. The blood pressure from supine to sitting declined by 30/20 mm Hg; sinus arrhythmia was absent. Blood pressure did not rise on handgrip or cold pressor test. The patient was discharged on the 50th day with alopecia and peripheral neuropathy as prominent sequelae. On three months of follow up the patient was able to walk with support. Hair had appeared on his scalp. The limb power was grade IV, sensations were normal and ankle reflex absent, although knee reflexes were normal and plantar response flexor. Skin appeared normal. The other sequelae at this stage were nasal speech and visual impairment (6/60 bilaterally), though fundus was normal. P100 visual evoked potential was 140 and 138 ms on right and left side respectively. At six months of follow up the patient was independent for daily activities, although his visual impairment and bilateral absence of ankle reflex persisted.

DISCUSSION
Thallium poisoning has a distinctive clinical picture comprising skin manifestations, alopecia, neuropathy, and other systemic manifestations. The typical clinical picture unfolds by 2–3 weeks of acute poisoning. By then, precious time for therapeutic intervention is lost. In the early stage, thallium poisoning simulates Guillain-Barré syndrome, porphyria, myocardial infarction, diabetic neuropathy, arsenic poisoning, lead poisoning, systemic lupus erythematosus, carbon monoxide poisoning, and organophosphate poisoning.¹ Our patient was also suspected of having Guillain-Barré syndrome but severe dysaesthesia, associated nausea, vomiting, severe constipation, and behavioural abnormalities raised doubt about this diagnosis.

Peripheral neuropathy is quite characteristic and an early feature of thallium poisoning. It is consistent with distal symmetrical axonopathy with secondary loss of myelin. In our patient the nerve conduction studies were normal except peroneal motor conduction velocity, which was unrecordable. In these patients, small fibre involvement cannot be excluded and this could account for preservation of sensory conduction velocity at the same time resulting in the severe dysaesthesia. Histopathological findings in thallium neuropathy have revealed axonal degeneration with secondary demyelination.² Nerve biopsy in our patient also revealed axonal degenerations. In thallium poisoning cranial neuropathy resulting in ptosis, external ophthalmoplegia, dysautonomia due to vagal nerve involvement, facial weakness due to seventh cranial nerve palsy, and optic neuropathy have been reported. Our patient had severe visual impairment and prolonged P100 latency of visual evoked potential due to retrobulbar neuritis. In one study up to 25% of patients with severe thallium poisoning have been reported to develop optic neuropathy.³
Severe dermatitis, stomatitis, and neuropathy in our patient was consistent with riboflavin deficiency. Skin lesions similar to thallium poisoning have been reported in riboflavin deficiency. Thallium interferes with riboflavin homeostasis, forming an insoluble complex and intravascular sequestration of riboflavin. In our patient hyperkeratotic lesions on palms and soles, ichthyotic lesions on his legs, and acniform lesions on his face were apparent by the end of the second week and alopecia appeared on the 18th day. Interaction between the sulphphydryl group and thallium result in abnormality in form and function of structural proteins; this accounts for disturbances of hair growth, alopecia, growth of nails, and Mee’s lines.

The presence of gastrointestinal, skin, liver, kidney, and peripheral nerve dysfunction in our patient at one stage simulated arsenic poisoning. Alopecia is quite characteristic of peripheral nerve dysfunction in our patient at one stage. The involvement of basal ganglia in thallium poisoning can result in tremor, chorea, and extrapyramidal motor disturbances and rigidity. Such central nervous system abnormalities are not reported in arsenic poisoning.

In our patient, the diagnosis of thallium poisoning was possible in the third week. By this time the initial therapeutic measures such as gastric lavage by activated charcoal, forced diuresis, and Prussian blue are not helpful in eliminating thallium. In thallium poisoning three phases have been described: first is the phase of intravascular distribution which lasts for four hours. The second phase of central nervous system distribution lasts for 4–48 hours and the third phase is the elimination phase, which begins 24 hours after poisoning. Thallium elimination mainly occurs through large and small bowel, although there is some enteric absorption as well. Renal excretion also mirrors total body thallium and can be enhanced by forced diuresis. Prussian blue is considered as specific antidote and chelates the intestinal thallium. Potassium supplementation are invaluable in the management of thallium poisoning. The characteristic alopecia manifests later. Haemodialysis, forced diuresis, laxatives, B complex, and potassium supplementation are invaluable in the management of thallium poisoning.

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Authors’ affiliations
U K Misra, J Kalita, R K Yadav, P Ranjan, Department of Neurology, Sanjay Gandhi Post Graduate Institute of Medical Sciences, Lucknow, India,
Correspondence to: Dr J Kalita, Department of Neurology, Sanjay Gandhi PGIMS, Rae Bareli Road, Lucknow 226014, India; jkalita@sgpgi.ac.in.
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REFERENCES

Learning points
• In a patient with neuropathy severe dysesthesia, constipation and alopecia point towards thallium poisoning.
• Haemodialysis is useful even in the third week of illness.

Our patient also showed remarkable improvement after haemodialysis.

The wide clinical spectrum of thallium poisoning makes it difficult to suspect in its early stage especially in the absence of a reliable history. Dysesthesiae, neuropathies, and cranial nerve palsy (ptosis, ophthalmoplegia, nasal speech, dysautonomia) and extrapyramidal features are highly suggestive of thallium poisoning. The characteristic alopecia manifests later. Haemodialysis, forced diuresis, laxatives, B complex, and potassium supplementation are invaluable in the management of thallium poisoning.
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