Thallium (Tl)
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Introduction

Thallium was discovered by Sir William Crookes in 1861 while he was trying to extract selenium from the by-products of sulfuric acid production. Crookes named the new element "thallium" from the Greek *thallos*, meaning "green shoot" after the bright green spectral emission lines that identify the element. In 1862, Claude-Auguste Lamy independently isolated thallium [1].

Thallium has been used as a therapeutic agent to treat syphilis, gonorrhea, tuberculosis and ringworm. It was also used as a depilatory for excess hair. In the early 20th century, a product (Koremlu) was marketed in the United States for the combined treatment of ringworm and as a depilatory agent. By 1934, 692 cases of thallium poisoning had been reported, with at least 31 deaths [2,3]. Thallium was also used as a rodenticide in both the US and the UK, but this was banned in 1965 after multiple unintentional poisonings [4]. Unfortunately, such poisonings are still reported in other countries where thallium is used as a rodenticide and as an ant killer.

Currently, thallium is used in the manufacture of electronic components, optical lenses, semiconductor materials, alloys, gamma radiation detection equipment, imitation jewelry, artist's paints, low temperature thermometers, and green fireworks. Thallium is also used as a radiological contrast agent in myocardial imaging and in the visualisation of tumors. Thallium exposure may occur at smelters, in the maintenance and cleaning of ducts and flues and through contamination of cocaine, heroin and herbal products. The main source of exposure to thallium in the general population is as a result of atmospheric pollution from, for example, coal-burning power plants [6]. Environmental concerns are growing, as thallium is both a waste product of coal combustion and also of the manufacture of cement.

Thallium is a soft and pliable heavy metal which is colourless, odourless and tasteless, and which has no metabolic role. Thallium has a similar ionic radius to that of potassium (Tl 0.147 nm vs K 0.133 nm), which is one of the reasons for its toxic properties, since it can enter cells through the normal potassium channels, especially when there is an intracellular deficiency of potassium [1].

Thallium metabolism.

Thallium salts are rapidly and nearly completely absorbed by all routes, with gastrointestinal exposure being the most common route to produce toxicity. It appears in the urine of rats within 1 hour of oral dosing. In man, the highest tissue concentrations are found in the kidney. Excretion is via the urine, stool and hair. There are few studies in man, but the biological half life is reported as 30 days. There are extensive studies on human thallium levels from the NHANES data and it appears that both blood and urine are useful biomarkers of its exposure [6].

Thallium, in spite of having no biological function, enters cells by a process governed by its similarity in charge and ionic radius to potassium. Although the exact mechanism of its toxicity has not been established, thallium interferes with energy production at essential steps in glycolysis, the Krebs cycle and oxidative phosphorylation. Additional effects include inhibition of the sodium-potassium-ATP pump and binding to sulfhydryl groups. The major symptoms of toxicity consist of a rapidly progressive, ascending, extremely painful sensory neuropathy and of hair loss (alopecia). Unlike most metal salts, gastrointestinal symptoms are minor, with constipation is more characteristic than diarrhoea. Many other findings such as an autonomic neuropathy, cranial nerve abnormalities, altered mental status, motor weakness, cardiac,

hepatic, and renal effects have been described, all of which may be secondary to inhibition of the intracellular Krebs cycle. Thallium also crosses the placenta freely and produces abnormalities as well as fetal death and congenital abnormalities [5].

Treatment of thallium toxicity

There are no controlled trials of treatments in thallium-poisoned patients. Thus, the literature is predominated by very small animal studies and case reports with very limited data. Traditional metal chelators such as dimercaprol (British Anti-Lewisite), penicillamine and DMSA (dimercaptosuccinic acid) may cause redistribution of thallium into the central nervous system. The use of activated charcoal may be a useful adjunct to therapy, but animal studies suggest that Prussian blue is the most effective agent [5].

Specimen requirements

Blood for thallium analysis should be collected into a trace element-free (dark blue top) BDH venoject tube. For urine determinations a sample from a 24-hour or 6-hour urine collection should be submitted.

Methodology

Thallium determinations are carried out by inductively coupled plasma-mass spectrometry (ICPMS).

Interpretation of results

The reference interval for whole blood Tl is ≤ 0.30 nmol/L.

The reference interval for urine Tl is ≤ 0.42 umol/mol creatinine.

There is no established statutory maximum permitted level for Tl in drinking water.

References

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