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The Silent Threat: Toxicological Effects of Heavy Metals on Human Health

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ABSTRACT: Metals are the oldest known toxins that harm humans and the environment. Some metals have bio-importance as trace elements, and the bio-toxic effects on humans are of greater concern. Heavy metals are known for their high atomic mass and toxicity in living organisms. Heavy metals become toxic by mixing in different environmental sources such as water, air, and soil. Heavy metals such as Arsenic, Lead, Zinc, Mercury, Cadmium, Copper, and Iron get exposed to humans through inhalation, ingestion and dermal contact, leading to accumulation in tissues and organs. The toxicological effect of heavy metals comprises a wide range of physiological systems, including the nervous, renal, hepatic, cardiovascular, and reproductive systems. They are primarily associated with inducing oxidative stress, disrupting cellular functions, and interfering with biochemical processes. Children, Pregnant women, and Occupational workers are particularly affected to the adverse effects of heavy metals. Due to the toxicity of heavy metals, there is a metal poisoning that has forensic importance to determine the cause of it, acute or chronic exposure, manner of poisoning, and hence awareness regarding the toxicity of heavy metals in humans. The objective of this study is to provide an overview of the toxicity of heavy metals and their toxicological effects in humans.

1. Introduction

Many heavy metals are listed in the periodic table because of their large atomic weight and density. Most of them reside in the biosphere, and their hazardous principle has been understood for ten years. They are released into the environment by anthropogenic resources, such as commercial and industrial[1]. Heavy metals are classified as essential or non-essential based on their roles in biological systems. The non-essential components are not necessary for the metabolic system and do not gain biological activities in living things. At low concentrations, these elements—which include arsenic, lead, mercury, and cadmium—have harmful consequences. Both plants and animals need the essential elements, which are needed in varying amounts and are fundamental to complete metabolism. Iron, copper, zinc, nickel, and chromium are among the 19 essential components in this[2,3]. Essential elements are sometimes referred to as trace elements because of their trace concentration (< 10 ppm)[4]. Although the body needs trace elements, people only need 1–100 mg per day, which is less than 0.01–0.02% of their total body weight[5,6]. When these substances are present in higher amounts, they endanger the health of living things[7].

According to the characteristics of the growth medium, food crops, which are the most important component of nutrition, can contain a range of both essential and non-essential components[8]. The majority of human exposure to heavy metals occurs through edible vegetables, which make up 90% of total intake. The remaining 10% is caused by skin contact and inhaling contaminated dust[9,10]. Metals are long-lived and nonbiodegradable; biological organisms cannot break them down, and they persist in bodily parts and the environment, endangering human health[11]. Numerous methods of treatment have emerged to stop heavy metal poisoning[12]. Heavy metals like arsenic, cadmium, mercury, and lead are chemical elements that pose the greatest threat to human health. Metals having a density of more than $5 g/cm^3$ is defined as heavy metals; since there is no universal definition[13].

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When heavy metals combine with one or more reactive groups, their harmful effects are strained on regular physiological functions. Through interaction with the necessary metals, the reaction occurs in the body with ligands that contain oxygen (OH, -COO, >C=O), sulphur (-SH, -S-S-), and nitrogen (-NH and >NH)[14,15,16]. Acute heavy metal toxicity is uncommon, but chronic low-grade toxicity is more harmful and leads to long-term disease. Certain impacts of heavy metals, such as neurotoxicity, nephrotoxicity, hepatotoxicity, and teratogenicity, directly affect behaviour by affecting the generation of neurotransmitters, changing different metabolic processes, and compromising mental and neurological function[17]. The blood and cardiovascular, endocrine, elimination pathways (colon, liver, kidney, skin), enzymatic, gastrointestinal, neurological, urinary, and reproductive systems are among those where heavy metal intoxication can cause damage and dysfunction. Target areas for toxic metals include structural proteins, enzymes, membranes, and DNA molecules[18].

2. Materials and Methods

The electronic search was conducted using Google Scholar concerning signs and symptoms, diagnosis, and mode of action by heavy metals such as arsenic, lead, mercury, copper, iron, barium, cadmium, as well as other chemical elements. The keywords ‘heavy metals’, ‘toxicity’, and ‘human health’ were crossed as well as their clinical and forensic signs. Furthermore, retrieved journal articles, books were reviewed to expand the source of information.

3. Objectives: Impact of heavy metals and their effects on humans

Heavy metal exposure in humans can occur through a variety of routes, including ingestion, inhalation, and skin absorption, and is caused by industrial operations. When ores are mined from the Earth's crust, they contain heavy metals that are removed as minerals. Heavy metals, which are found in most ores as sulphides and oxides, are present. Paints, cosmetics, and insecticides are examples of industrial goods that can serve as heavy metal sources[19].

3.1 Arsenic (As)

Arsenic, one of the important heavy metals, is semi-metallic and occurs naturally in both organic and inorganic forms as arsine (AsH3) and metalloid (As0)[20,21]. Arsenic compounds can be found in three different oxidation states: elemental, pentavalent (As5+), and trivalent (As3+). Arsenite has ten times the potential for toxicity compared to arsenate, but the element is harmless[22]. Methylated metabolites such as monomethyl arsenic acid (MMA), dimethyl arsenic acid (DMA), and trimethylarsine oxide are examples of organic arsenic compounds. Water contains the inorganic form of arsenic, which is more harmful than the organic form. Additionally, seafood naturally contains arsenic, which raises urine arsenic levels due to the presence of a harmless organic molecule called arsenobetaine[23,24]. Accidental ingestion of pesticides containing arsenic is the primary cause of acute arsenic poisoning, with homicide and suicide being less frequent causes[25].

Poisonous Compounds:

Arsenic acid, potassium arsenite (white), Sodium arsenite (white), Copper arsenite (Scheele's green), Copper aceto arsenite (Paris green), Arsenic trioxide (Sankhya-white), Arsenic trisulphide (Orpiment/Harital-yellow), Arsenic bisulphide (Realgar-red), and Arsenic triiodide (orange). Arsenic is present in rodenticides, weed killers, fly paper, fly powder, and fly water, and it can cause unintentional poisoning.

Mechanism of Action:

Trivalent substances work with sulfhydryl enzymes (SH-thiol group) to block oxidation and cell metabolism. They will prevent oxidative phosphorylation and Kreb's cycle, which will cause ATP to be depleted. The oxidative phosphorylation in the mitochondria is detached by pentavalent substances. Arsenic causes capillaries to dilate[26].

The dominant and more stable arsenite can bind to thiol or sulfhydryl groups of proteins and inactivate over 200 enzymes, which can affect various organ systems. However, it also prevents glucose from entering cells, acetyl coenzyme A from being produced, glutathione reductase, and thioredoxin reductase from synthesising. Arsenite replaces the phosphate that is necessary for biological processes[27,28]. Ingestion of tainted food and water, tobacco smoke inhalation, employment near a facility that produces or uses this metal, and skin contact can all result in high amounts of exposure to inorganic arsenic[29]. The diet and water are the main sources of exposure, with a consumption of roughly 12–50 µg/day; however, the recommended dietary requirement is between 12–25 and 12–50 µg/day[30]. Renal excretion removes both organic and inorganic substances from the body. While inorganic chemicals are eliminated over a few days, some of them remain retained in the body for months or longer. In contrast, organic molecules are eliminated more quickly.

ACUTE POISONING:

Signs & Symptoms: Faintness and depression, nausea, thirst, salivation, burning pain in the gastrointestinal tract, diarrhoea, pain and irritation of anus, oliguria, haematuria and dysuria, cramps of muscles, dehydration, coma and finally death.

With large doses: Sudden death due to shock or mainly narcotics manifestations- pain in muscle, delirium, coma, and death.

Subacute Poisoning: Neuritis, paralysis, cyclic vomiting syndrome disturbances along gastrointestinal tract manifestations.

Unusual manifestations: Locked jaw, increased temperature, loss of speech and memory, haemolysis, jaundice, anaemia.

Fatal dose: Trivalent is more hazardous at 180–200 mg.

Fatal period: Sudden death or within two to three hours in narcotic form; 12 to 48 hours in gastrointestinal form.

Treatment: Emetics, gastric lavage with ferric oxide, demulscents, morphine for pain, intravenous fluids.

Antidote: Physical- Demulcents; Chemicals- Freshly prepared hydrated Ferric oxide. It forms Ferric arsenite- a harmless salt; Pharmacological- British Anti-Lewisite, or BAL, is administered deep intramuscularly in the gluteal region at a rate of 4 hours per day for two days, 6 hours on the third day, and 12 hours per day until the tenth day. It is administered at a dose of 3 mg/kg in a 10% solution in Arachis or pea nut oil with benzoyl benzoate.

Post Mortem Appearance: Dehydration, jaundice, stomach appearance is known as red-velvety appearance – crimson plus/flea bitten appearance, ulceration and sub-mucosa petechial haemorrhages, degeneration of liver, heart and kidney.

Chronic Poisoning (arsenophagia, arsenicosis): Results due to - After effect of acute poisoning, consumption of small doses repeatedly, consumption of food and drinks contaminated with arsenic, people working in industry, using arsenic.

Forensic Importance: In the West, notably in the United Kingdom during the Victorian era, arsenic was highly regarded as the ideal murderous poison. Napoleon Bonaparte's case is among the most amazing, and it remained purely hypothetical until recently, when scientific evidence finally proved the reality without a reasonable doubt. Arsenic trioxide can be given without warning a victim of their poisoning because it is nearly colourless and tasteless in solution. Relative insolubility is the main disadvantage. However, using hot fluids like tea, coffee, chocolate, and soups can greatly increase the solubility. The sole disadvantage is that a large portion of the dissolved arsenic may precipitate out as a gritty layer as it cools. However, a number of factors have contributed to the decline in arsenic's popularity over time, including legal prohibitions on its sale, the development of advanced techniques that can identify even trace amounts in a dead corpse, and, of course, the creation of more effective poisons for evil purposes. Nowadays, the majority of documented cases of arsenic poisoning are unintentional. Murders are occasionally recorded in the meantime. Accidental poisoning can result from consuming tainted food or drink, or it can be caused by exposure at work or in the workplace. In the past, arsenic was found in some allopathic medications, and iatrogenic poisoning was fairly uncommon. Today's threats come from ayurvedic items, such as preparations that might have significant levels of arsenic that, if used for an extended period of time, could result in chronic poisoning. In India, drinking well water is one of the main causes of long-term unintentional poisoning. It has been claimed that large areas of numerous Asian nations are exposed to water tainted with arsenic, especially from tube wells[31].

Figure 1. Hyperkeratosis of soles (**A**) and palms (**B**)[32], Mee's lines (**C**)[33], diffused hyperpigmentation on the trunk (**D**), Bowen's disease on head (**E**) and palm (**F**), hyperkeratosis on the trunk (**G**)[34], palmar pits (**H**)[35], and raindrop pigmentation (**I**,**J**)[36].

3.2 Cadmium (Cd)

Similar to arsenic, lead, mercury, and chromium, cadmium (Cd) is a heavy metal that has no physiological use and is therefore frequently regarded as harmful. Traces of cadmium are found in nature, especially in the sulphide ores of copper, lead, and zinc. Although it is not easily found in ores, cadmium can be found in most zinc ores because it isomorphically substitutes for zinc. But since it is so widely available, detectable levels of cadmium can be found in food, drink, and breath[37,38]. Combustion from burning fossil fuels produces a byproduct of human activity, as does leachate from landfills, farms, and mining, particularly lead and zinc[39]. Cadmium contamination is frequently caused by its usage as a corrosive reagent in industry, as well as by its stabilisation with PVC goods and colour pigments and by the use of Ni-Cd batteries[40]. Humans can be exposed by ingestion and inhalation, however the primary health impacts documented in the literature to date are from occupational exposure (lung damage), inhalation from tobacco smoking, and dietary exposure (damage to the kidneys and bones)[41]. Food is the primary way that non-users are exposed to tobacco. This kind of exposure is found in foods such as peanuts, shellfish, leafy greens, sunflower seeds, cocoa powder, grains, cereals, soybeans, mushrooms, and potatoes. According to studies that track chemicals in the body, cigarette smokers frequently had elevated blood and urine levels. They were lower in non-smokers and moderate in ex-smokers. This is due to the metal's propensity to build up in tobacco leaves in comparatively significant quantities. Both the duration of exposure and the amount of exposure determine how harmful they are. After eating, 5–10% of the cadmium is absorbed. When iron, calcium, or protein levels in the diet are low, the percentage absorbed is higher.

In the liver, cadmium promotes the production of metallothionein. With a half-life of up to 10 to 30 years, the glomerulus secretes the cadmium-metallothionein complex, or Cd-MT, which is reabsorbed by the proximal tubule. It is a defence mechanism to reduce the toxicity of free cadmium, but after prolonged exposure, it is no longer produced, which raises intracellular levels. After then, they build up in the mitochondria and work at the Complex III level to block the respiratory chain. Over the course of a lifetime, the renal tubule cells' Cd levels rise. The kidneys, bones, and lungs are all impacted by chronic cadmium exposure. The kidneys store around half of the entire dosage. Because of this, the kidney is the ideal location for chronic cadmium buildup[42-45].

Acute Poisoning: Flu like symptoms known as cadmium blues, bronchitis, pneumonitis, pulmonary oedema, proteinuria and painful bony lesion-Ouch-Ouch disease in severe poisoning, vomiting**,** abdominal pain**,** convulsions start in 15-30 mins

Chronic Poisoning: Anaemia, renal failure, anosmia, increased chances of cancer, emphysema, osteoporosis, chronic rhinitis. Infertility[46], Diabetes mellitus type-2[47].

Treatment: DMSA (2,3-dimercapto-succinic acid), DMPS (2,3-dimercapto-1-propane sulfonic acid), BAL (British Anti-Lewisite, dimercaprol), emesis, stomach lavage by ingestion, and chelation with EDTA (ethylenediamine tetra acetic acid)[48].

High doses of cadmium, especially during pregnancy, reduce bone density. Cadmium also contributes to Itai-Itai illness, which is typified by increased excretion of calcium and phosphate, renal problems, and painful bone degradation (osteomalacia and osteoporosis)[49].

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3.3 Lead (Pb)

In Latin, the word for lead, plumbum (liquid silver), has given rise to the terms plumbism, colica Pictonum, Devon colic, painter's colic and saturnism for poisoning. Many Roman leaders probably suffered lead poisoning, which caused neurotoxicity and sterility[50-52]. Since lead may create major environmental damage and health issues, it is the most toxic heavy metal. Industrial processes like burning fossil fuels, mining, smelting, and manufacturing through recycling are the main causes of environmental contamination. The industrialization increased the use of many leaded products, among which gasoline and lead-based paints are the most conspicuous[53]. Inorganic lead enters the body by smoking, ingestion, or gastrointestinal absorption, which accounts for 20% of water and 65% of food, and inhalation or pulmonary absorption of contaminated air, which accounts for 15%. Inorganic substances cannot be absorbed via the skin, while organic substances may.

Poisonous Compounds: Tetra-ethyl lead (previously used as an anti-knocking agent in petrol), Lead tetra oxide (vermillion), Lead monoxide-mudrasang, Lead acetate (salt of Saturn), Lead carbonate (safeda), Lead nitrate, Lead sulphate, Lead chromate (chrome yellow), Lead sulphide (white surma), Lead chloride, Lead bromide and Lead iodide

Mechanism of Action: ATPases, ferrochelatase, aminolaevulinic acid dehydrase, and sulphydryl group are among the enzymes that are inhibited. The production of heme is suppressed. Lead fixation in the brain and peripheral nervous system is the primary cause of the chronic symptoms. Bones store lead as carbonate and phosphate. A capillary spasm.

According to the WHO, adults who have lead poisoning should be concerned about a level of 25 µg/dl of blood, while children should have a level of less than 5 µg/dl of blood. The amount that is absorbed depends on the age and health of the individual exposed. Lead is a carcinogen that damages the chromosomal structure and sequence, cellular processes that control genes, and the DNA repair pathway by producing reactive oxygen species (ROS). Because it removes zinc from some regulatory proteins, it has an impact on transcription[54].

Acute Poisoning: Acute lead poisoning is extremely rare. The manifestations are similar to other metallic poisons except that diarrhoea is replaced by constipation and stools are black and foul smelling. The blood lead levels are elevated.

Gastrointestinal tract (GIT) Manifestations: Colicky pain, a sweet, metallic taste, burning in the throat, white or bloody vomiting, constipation, and a sensitive, constricted belly.

Urinary Manifestations: Oliguria, albuminuria, presence of lead in urine, presence of copro-porphyrin 3 in urine (red coloured urine).

CNS Manifestations: Headache, muscle cramps, convulsions, drowsiness, sleeplessness, and infrequently paralysis.

Sub Acute Poisoning: Consumption of repeated doses of soluble salts of lead results in:- Blue-line on gums known as "Burton's lines", face is pale, GIT and Urinary manifestations (as in acute), CNS manifestations are more pronounced, loss of appetite, renal failure[55-56].

Fatal Dose: Indeterminate, 0.5–20 g.

Fatal Period: Three to four days until a few months.

Treatment: Vitamins C and D, calcium gluconate, emesis, stomach wash, and morphine and atropine for abdominal pain.

Antidote: Physical- Demulcents; Chemical- Sodium sulphate or Magnesium sulphate; Pharmacological- One gram of $CaNa_2$.EDTA (calcium disodium ethylene-diamine tetra acetic acid) twice day for five days or five millilitres of a 50% solution intravenously administered twice daily as a 3% solution in saline. DMSA is helpful as well.

POST MORTEM APPEARANCE: Gastric mucosa is congested and sometimes it gets eroded**,** stools are black**,** gastroenteritis**,** and lead lines on X-ray.

CHRONIC POISONING (Plumbism/ saturnism): Mild Toxicity (blood lead: 40 to 60 mcg/ 100ml): Myalgia, Paraesthesia, Irritability, Abdominal discomfort; Moderate Toxicity (blood lead: 60 to 100 mcg/ 100ml): Arthralgia**,** Fatigue**,** Headache**,** Tremor**,** Anorexia, metallic taste, vomiting; Severe Toxicity (blood lead more than 100 mcg/ ml): Lead palsy**,** Lead colic**,** Lead encephalopathy**,** Facial pallor.

Forensic Importance**:** In the 18th and 19th centuries, lead-containing wine and cosmetics caused significant disruptions among the English, including members of the royal family. Nowadays, chronic lead poisoning is thought to be the biggest environmental health risk, especially for young children. Because their gastrointestinal systems may absorb a far higher dose of lead than an adult's, children are more vulnerable to lead poisoning. Due to the issue over the lead content in 2015, there is now a great deal of concern that food goods sold by international corporations may also contain lead levels that are higher than allowed. The aforementioned makes it evident that lead poisoning frequently occurs by unintentionally. In the medical industry, there have been relatively few cases of killings or suicide linked to lead-based products.

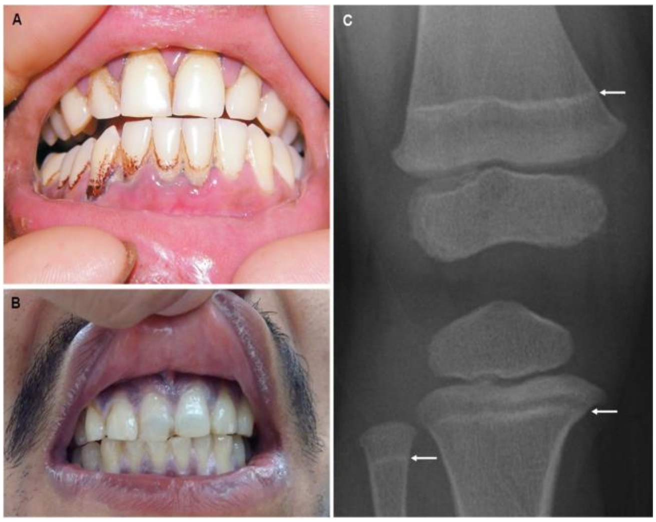

Figure 2. Features of Burton's line that are emphasised by deposits (**A**)[57], blue- purplish discoloration of gums (**B**)[58], and X-ray with arrows showing "lead lines" (**C**)[59].

3.4 Iron (Fe)

The most prevalent trace element in the human body, iron, is one of the most well-known elements required for homeostasis and is important for regular cellular metabolism. Numerous metabolic processes, including as cellular respiration, myelin creation, neuronal dendritic tree growth, and DNA, RNA, and protein synthesis, are catalyzed by it. Furthermore, iron functions as a cofactor for almost all enzymes[60-62]. The biological roles are based upon its chemical features, in particular its property to give rise to quite diverse coordination complexes with organic ligands dynamically and in an appropriately adaptable fashion, as well as its useful redox potential that may allow such transitions between the ferrous, Fe(II) and the ferric states, Fe(III)[63]. Ferric ammonium citrate, ferrous fumarate, ferrous gluconate, ferrous sulfate, and ferric chloride are examples of common iron salts. Many of these drugs cure iron-deficient anaemia, which can be caused by a number of different factors. Iron poisoning is closely associated with the majority of cases of excessive consumption of these salts.

Mechanism of Action: Gastrointestinal corrosion, suppression of mitochondrial activity, Massive post-arteriolar dilatation brought on by free iron leads to venous pooling and increased capillary permeability, which lowers plasma volume.

Table 1: The occurrence and presentation of signs and symptoms over diverse chronological stages.

Stage 1 (After 0.5-6 hrs):	Vomiting, pain in abdomen, bloody stools
Stage 2 (After 6-24 hrs):	No symptoms
Stage 3 (After 6-72 hrs):	Metabolic acidosis, jaundice, tachypnoea, hypotension, seizures
Stage 4 (After 12-96 hrs):	Hepatic and multi organ failure
Stage 5 (After 2-8 weeks):	Gastric stricture

Serum iron is the most important test: measured 2-6 hours after exposure because timely iron distribution into tissues and tissues may stabilize its serum level close to the normal limit. It fluctuates at peak levels and these vary within <350microg /dL, between 350-500microg /dl and >500microg / dl are present as mild and moderate systemic and toxic[64,65].

Fatal Dose: 20-30 gm.

Fatal Period: 1-2 days.

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Treatment: Gastric lavage with 5% sodium bicarbonate**,** Egg, milk and 1% magnesium hydroxide**,** Deferoxamine-6gm in 24hrs. 1gm intravenously or intramuscularly, followed by 500mg every 2 hrs- 2 doses, then 500mg 4-6 hourly**.** Whole bowel irrigation with polyethylene glycol 1.5-2 litres/ hr**,** Haemodialysis, and liver transplant in severe cases.

Post Mortem Appearance: Hemorrhagic gastric mucosal necrosis. When someone has ferrous sulfate poisoning, their stomach contents may seem bluish-green. Hepatic and renal necrosis, occasionally accompanied by bluish discolouration in cases of ferrous sulphate toxicity.

Forensic Importance: Acute iron poisoning has become quite dangerous in today's world, and toddlers are increasingly getting unintentionally poisoned. The majority of iron compounds have extremely vivid colours and delicious flavours, which will make them irresistible and ultimately deadly to such helpless victims. Western experience has shown that if sample childproof packaging is made publicly available, it will more effectively decrease children's accidental ingestion.

Figure 3. Skin hyperpigmentation—bronze coloration (**A**)[66], a black-greyish, rigid pancreas (**B**)[67].

3.5 Mercury (Hg)

A heavy metal that belongs to the periodic table's transition elements series is called mercury or hydrargyrum. There are three different kinds of mercury found in nature: elemental, metallic, or elementary mercury (Hg0), inorganic mercurous (Hg+1) and mercuric (Hg+2), and organic mercury compounds, such as methylmercury and ethyl mercury. The two last ones are made by microorganisms found in soil and water that methylate inorganic mercuric. Each type of chemical has unique chemical characteristics and levels of toxicity. Considering organic mercury compounds to be more dangerous than inorganic mercury, the order is as follows: metallic mercury (Hg0) < inorganic mercuric (Hg+2) < inorganic mercurous (Hg+1) < organic molecules. Elementary mercury is more hazardous since it is a liquid with a high vapour pressure at room temperature that is emitted into the environment as mercury vapour [68,69]. Both eating and inhalation are ways for these metals to enter the human body. These transmission methods include dental amalgams, industrial and agricultural processes, occupational activities, preventive medical procedures, and contaminated food, especially fish and seafood.

Poisonous Compounds: Mercuric oxide (sipichand), Mercuric cyanide, Mercuric nitrate, Mercuric sulphate, Mercuric methide, Mercuric sulphide (cinnabar, china sindur), Mercuric chloride (corrosive sublimate), Mercurous chloride (ras kapoor), Mercurochrome, and Mercury fulminate.

Mechanism of Action: Mercury is a powerful inhibitory factor for cellular enzymatic mechanisms because it reacts with the sulfhydryl group in the enzyme molecules. After being consumed, soluble forms of mercury are deposited in the kidney, liver, spleen, intestines, heart, muscles, and lungs. Whereas ingestion of insoluble forms will harm only the upper and lower gastrointestinal tracts. Mercury is a nephrotoxic poison.

Excretion phases of mercury and its compounds were dependent on their oxidation states. Elemental and inorganic mercury renal elimination (urinary) and some gastrointestinal (faecal), half-life 30–60 days. Although organic molecules were expelled in the faeces, their half-life was 70 days due to enterohepatic recirculation. The liver, brain tissue, and kidney hair hold the majority of the absorbed mercury[70,71].

Acute Poisoning: Features that distinguish from acute arsenic poisoning include strong metallic tastes, vomitus and diarrhoea are blood spotted; and symptoms develop within minutes.

Signs and Symptoms: The symptoms include metallic taste, burning, pain, and constriction in the mouth and upper gastrointestinal tract; a greyish-white, corroded oral cavity; nausea and vomiting, followed by a lot of bloody diarrhoea; anus pain; insensibility; convulsions;

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and collapse. Salivation, gum irritation, bad breath, and nephrotoxicity are symptoms if death is postponed. Salivation, stomatitis, vomiting, diarrhoea, coughing, dyspnoea, conjunctivitis, and ocular ulcers when inhaling vapours.

Fatal Dose: A corrosive sublimate (mercuric chloride) of around 1gm.

Fatal Period: Varies from a few hours to three to five days.

Treatment: Emetics, Gastric lavage, Intravenous fluid, Peritoneal or hemodialysis.

Antidote: Physical- Demulscents, Charcoal; Chemical- 5% sodium bicarbonate and sodium formaldehyde sulphoxylate solution; Pharmacological- BAL (British Anti-Lewisite) or Penicillamine.

Post Mortem Appearance: There are patches of blood leaking, the tongue and lips are greyish-white, the oesophagus and stomach are corroded and softened, and the colour is either black or greyish-white. Both the large and small intestines are clogged, ulcerated, and occasionally gangrenous. Kidney- Nephritis; Liver- Cloudy and swollen; Heart- Fatty degeneration and subendocardial haemorrhages.

Chronic Poisoning (Hydrargyrism): Late consequences of acute poisoning, careless medication administration, repeated tiny doses of mercury-containing salts, and mercury-using industry personnel

Signs and Symptoms: Gastrointestinal system issues, metallic taste, and excessive salivation (ptyalism/sialorrhea), Gums- blue streaks, ulceration, necrosis, and inflammation. Skin conditions include acrodynia, rosy cheeks, nose, hands, and feet, penetrating ulcers on fingers, nails, and knuckles. Kidney- Nephritis, Mercuria lentis, Uremia. Examining the anterior lens capsule under a slit lamp reveals a brownish hue. Danbury tremors, Hatter's shakes, glassblower's shakes, and other mercurial tremors that impact the fingers, tongue, face, arms, and legs. When no action is possible, concussion mercurialis is the most severe type. Erethism- Delusions, tremors, memory loss, sleeplessness, shyness, and irritability are symptoms of a disturbed personality. breakouts of the skin.

Forensic Importance: Mercury and its derivatives are among the most prevalent hazardous dangers in industry, agriculture, and the workplace. Iatrogenic poisoning was made more common in the past by the use of mercury in various forms to cure a number of illnesses, particularly syphilis. Calomel (mercurous chloride) is still occasionally advised as a laxative nowadays. Colitis, dementia, tremor, and renal failure can result with prolonged use. Thankfully, calomel teething powder-induced acrodynia is now quite uncommon. A separate type of harm is posed by domestic exposure, even if it is not as commonly reported nowadays. This could occur if someone processes gold ore at home or accidently spills mercury. The Hazardous Waste Management and Handling Rules of 1989 govern mercury and mercury waste in India; however, there is no particular policy that addresses mercury instruments used in the health sector exclusively. All hospitals in Asia will have all mercury-based medical devices (especially sphygmomanometers) gradually phased out over the next few years through the plan of the World Health Organization. Mercury sphygmomanometers have already been phased out of the European Union. Several private and Govt hospitals in India have already switched over to mercury-free health care.

Figure 4. Dermatological manifestations of acrodynia: maculopapular rash in the trunk (**A,B**)[72-73], exfoliation on fingers and pink discoloration of nails (**C**)[73] and toes (**D**)[73], hyperpigmentation with multiple cysts, chloracne-like lesions of face and neck (**E**)[74], swan-neck deformity of both fingers (**F**)[74], bluish grey-stained area on the skin (**G**)[75]

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3.6 Copper (Cu)

One naturally occurring metallic element is copper. Its typical concentration in soil is about 50 parts per million. Every animal and plant contain it, and people and animals can get trace amounts of this vital ingredient. Mining, smelting, and refining are the main processes that release copper into the environment. Copper emissions can also be attributed to the burning of fossil fuels and the businesses that create wire, pipes, and sheet metal. Numerous enzymes, including cytochrome C oxidase and ceruloplasmin, contain tiny amounts of copper. The action of the mineral affects tyrosinase, dopamine beta-hydrolase, zinc-copper superoxide dismutase, and other enzymes involved in its several functions as a transporter, detoxifier, antioxidant defence, immunological system, pigment, and melanin production. It tends to be harmful when the body has too much of it. The liver then stores the majority of copper, and the neurological system's organs—such as the ganglia, neurons, cerebellum, and hippocampus—are the target organs[76].

Poisonous Compounds: Copper carbonate (mountain green), copper sulphate (CuSO4, blue vitriol, blue stone), and copper subacetate (zangal or verdigris).

Mechanism of Action: It also affects enzymes' sulphydryl groups. It causes proteins to precipitate. It is advised that adults consume 0.9 mg (milligrams) of copper each day. According to studies, the median daily consumption from the average American diet is between 1 and 1.6 mg. Every copper source surpasses it by a safe maximum intake level of 10 mg/day (for extended exposure). A variety of foods, including shellfish, organ meats, nuts, legumes, and cocoa, contain copper.

Acute Poisoning:

Signs and Symptoms: Salivation, burning pain in the upper gastrointestinal tract, astringent metallic taste, nausea, vomiting (blue or green), diarrhoea, oliguria, haematuria, albuminuria, uraemia, jaundice, cramping, convulsions, and collapse were among the symptoms.

Fatal Dose: 15-30gms.

Fatal Period: 1-3 days.

Treatment: There is no use of emetics (copper salts are emetics), Gastric lavage, Diuretics, Intravenous fluids.

Antidote: Physical- Demulscents (forms copper albuminate with proteins); Chemical- Potassium ferrocyanide (forms insoluble Cupric ferrocyanide); Pharmacological- Penicillamine or BAL (British Anti-Lewisite) or EDTA (ethylenediamine tetra acetic acid).

Post Mortem Appearance: Jaundice, greenish-blue foam at the mouth and nose, and congestion in the stomach and oesophagus. The contents are greenish-blue, Intestinal haemorrhages and ulcerations are observed in the colon and large intestine. Liver and kidney deterioration in chronic poisoning.

Chronic Poisoning (Hemochromatosis/ Bronzed diabetes/ Pigment cirrhosis):

Nausea, giddiness, headache, vomiting, diarrhoea, colicky discomfort, conjunctivitis, corneal ulceration, and a green or purple line on the gums (Clapton's line). Dermatitis, bronchitis, laryngitis, nephrotoxicity, anaemia, neuritis, muscle atrophy, and greenish bodily secretions.

Forensic Importance: India had a high rate of suicidal copper sulphate consumption till recently. It has, however, recently altered to much less. Chronic copper poisoning can occur under certain situations .

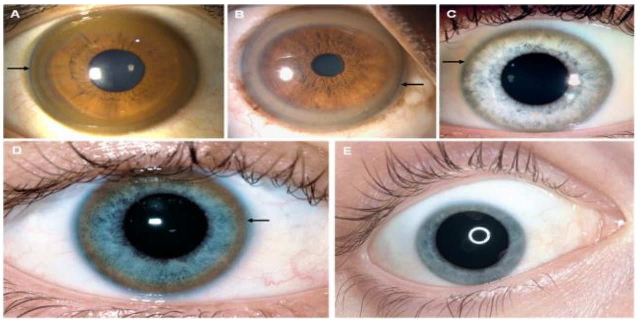

Figure 5. Kayser–Fleischer rings—(**A–E**)[77-80].

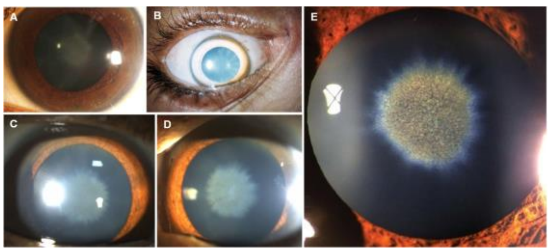

Figure 6. Sunflower cataracts seen with slit lamp—(**A–E**)[81-84].

3.7 Thallium (Tl)

When exposed to air, thallium, a soft, heavy metal with a tin-white, glossy appearance, tarnishes on the surface because black thallous oxide forms. It is highly poisonous and mostly utilised in the production of fake jewellery, dye glass, and optical lenses[85]. Historically, the main application for thallium was as a rodenticide. However, a number of nations have outlawed this use due to the rising number of unintentional intoxications. Additionally, the use of thallium in the treatment of dermatophytosis, gonorrhoea, TB, and syphilis was discontinued [86,87]. Since thallium salts are odourless and tasteless, they can be fully absorbed in liquids at a very quick rate, which is why they don't appear in routine toxicological reports. Because of this, it is a strong contender for criminal poisoning[88-90]. However, in the majority of Western countries, this only occurs infrequently[91].

The poisonings have mostly been caused by eating salt, although there have also been cases of overdosing from cocaine and heroin consumption, skin absorption, and inhalation from smelting dust or fume[92,93]. After coming into contact with thallium exposure, it swiftly spreads to every area of the body. Its activity is stopped by binding when it binds to the Na+/K+-ATPase channel, which happens ten times faster than its affinity for potassium[94].

Poisonous Compounds: Thallium (I) acetate ($TlC_2H_3O_2$), Thallium (I) carbonate (Tl_2CO_3), Thallium (I) chloride (TlCl), Thallium (I) nitrate ($TlNO_3$), Thallium (I) oxide (Tl_2O), Thallium (III) oxide (Tl_2O_3), Thallium (I) selenite (Tl_2SeO_3), Thallium (I) sulphate (Tl_2SO_4)[95-97].

It has been discovered that thallium is deposited in the brain, kidneys, testes, stomach, lungs, spleen, liver, bones, and scalp skin. Additionally, it is deposited in tears, hair, nails, and even breast milk. The levels of Tl in the various human organs are as follows: bone (600 ng/g), hair (150–650 ng/g), kidney (6.1 ng/g), liver (1.5 ng/g), brain (0.42–1.5 ng/g), and nail (1200 ng/g) [98]. Urine and faeces are the main ways in which this metal is eliminated. The most accurate technique for determining Tl levels in human bodies is thought to be the urine test. Typically, thallium levels in urine shouldn't be more than 1 mg/g creatinine. Additionally, it can remain in a person's system for up to two months after exposure after one hour[99].

Acute Poisoning: After injection, severe poisoning symptoms may appear 24 hours to 12 days later. Stomatitis, loss of appetite, stomach pain, vomiting, diarrhoea, and constipation are symptoms of mild instances. Although rare, bloody vomiting and diarrhoea can happen. These typically go away in a few days. Dry mouth, conjunctivitis, rhinitis, difficulty swallowing, colic-induced vomiting, diarrhoea followed by constipation, bilateral ptosis, neuropathic lower limb pain, oliguria, albuminuria, ataxia, delirium, convulsions, pulmonary oedema, and cyanosis are the symptoms in severe instances. Paralysis of the respiratory muscles causes respiratory failure and cardiac arrest, which in turn causes death from coma. Upon recovery, the patient may experience mental abnormalities, peripheral neuritis, optic atrophy, loss of sight and hearing, and glycosuria.

Fatal Dose: Twelve milligrams per kilogram of body weight is the minimum lethal dose for an adult.

Fatal Period: 24-30 hrs.

Treatment: The gastric aspirate should be carefully disposed of once the stomach has been cleaned with water. To improve fecal excretion, activated charcoal should be used often together with a salt purgative supplement or peglec powder. The most common application for ferric hexacyanoferrate ($Fe_4[Fe(CN)_6]_3$) is as an antidote for thallium poisoning. Prussian blue, iron blue, Chinese blue, Paris blue, Brunswick blue, and Turnbull's blue are other names for this combination[100]. It can be administered in two to four divided doses at a rate of 250 mg/kg/d. In patients treated with Prussian blue, 50ml of 15% mannitol must also be administered since thallotoxicosis frequently results in intestinal stasis of a very high degree, which causes constipation. Since potassium loading is known to enhance the risk of toxicity, potassium iodide or chloride should not be administered. It is important to keep the patient warm. Within 48 hours of consumption, intermittent hemoperfusion or haemodialysis might be beneficial. However, a single antidote is not effective in severe Tl poisoning cases and reports combinations of different administrations to show effectiveness in several cases[101].

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Post Mortem Appearance: Submucous petechial haemorrhages may cause inflammation of the stomach mucous membrane. There is congestion in the spleen. Additionally, the liver exhibits fatty degeneration and congestion. Congested kidney, enlarged glomeruli, hazy edema, and necrosis of some cells in the convoluted tubules are all present. There is congestion in the trachea and bronchi. Additionally, there are subpleural haemorrhages and lung congestion. There is fatty deterioration in the heart. There is congestion in the meningeal vessels. The cells of the thyroid, adrenal cortex, and hair follicles exhibit degenerative alterations and vascularization.

Chronic Poisoning: Workers who separate thallium from pyrite residue in chemical industries are susceptible to chronic poisoning. People who have often used depilatory creams containing thallium acetate for an extended period may also develop chronic poisoning. Restlessness, sleeplessness, apathy, exhaustion, anorexia, abdominal colic, limb aches, tachycardia, a high level of eosinophilia, lymphocytosis, peripheral neuritis, wasting muscles, proteinuria, tremors, and occasionally retrobulbar neuritis are among the symptoms. The nails exhibit alterations similar to those caused by arsenic, and the skin may be dry and scaly. Males have been shown to exhibit aspermia, whereas females have been observed to exhibit amenorrhea.

Forensic Importance: Thallium poisoning comes out as a serious forensic topic due of its high toxicity and also to its background of deliberate criminal use and challenges of diagnosing it. Thallium is the same as the poisoner's poison, which is a substance that cannot be smelled or tasted, and is also colourless. The symptoms usually come late in the disease period and are more specific, such as gastrointestinal troubles, hair loss, and neuropathy. In order to identify exposure to tell-tale signs of thallium in living tissues like hair and nails, forensic toxicologists employ cutting-edge technical techniques like inductively coupled plasma mass spectrometry (ICP-MS) or laser ablation ICP-MS. This helps them fight the time of intoxication[102-103].

Figure 1. Characteristic scalp hair loss (**A,B**),[104-105] blackened hair root under light microscopy (**C**)[106], form pustular lesions, lip oedema, and angular stomatitis (**D**)[107], keratosis of soles (**E**)[108], and Mee's (**F**)[109].

4. Conclusion

Heavy metal toxicity is a growing health hazard for humans since these may accumulate in the environment and their increasingly used in industries. Exposure to heavy metals like lead, mercury, cadmium, and arsenic may show both acute and chronic harmful effects on health, damaging key organs of the body, neurotoxic effects, and carcinogenicity, coupled with neurophysiological impairment. These are also highly grave effects for the reason that they may occur with a very low level of exposure. Advances in toxicology have made it possible to bring mechanisms by which heavy metals are toxic, mainly oxidative stress, interference with essential metal ions, and epigenetic modification. It therefore emphasizes very stringent regulatory policies for the reduction of exposure, promoting environmental remediation actions, and encouraging the issue of much safer industrial practices. Future-oriented research agendas may develop the following priorities: exploration of new biomarkers for early detection of heavy metal toxicity, investigation on the long-term health risks from exposure to low doses, and scouring for therapeutic interventions such as chelation therapy and diet modification. As an overall multidisciplinary approach, this should be supplemented by public health

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experts as well as policymakers to mitigate the risk associated with heavy metal exposure on human health.

5. References

[1] Engwa, G.A., Ferdinand, P.U., Nwalo, F.N. and Unachukwu, M.N., Mechanism and health effects of heavy metal toxicity in humans. Poisoning in the modern world-new tricks for an old dog, 2019,10, 70-90.

[2] Ali, H., Khan, E. and Ilahi, I., Environmental chemistry and ecotoxicology of hazardous heavy metals: environmental persistence, toxicity, and bioaccumulation. Journal of chemistry, 2019, (1), 6730305.

[3] Koller, M. and Saleh, H.M., Introductory chapter: Introducing heavy metals. Heavy metals, 2018, 1, 3-11.

[4] Kaur R, Sharma S, Kaur N. Heavy metals toxicity and the environment. Journal of Pharmacognosy and Phytochemistry. 2019, 247-249

[5] Mehri, A., 2020. Trace elements in human nutrition (II)–an update. International journal of preventive medicine, 2020, 11(1), 2-9.

[6] Kulkarni, N., Kalele, K., Kulkarni, M. and Kathariya, R., Trace elements in oral health and disease: an updated review. Journal of Dental Research and Review, 2014, 1(2), 100-104.

[7] Jaishankar, M., Tseten, T., Anbalagan, N., Mathew, B.B. and Beeregowda, K.N., Toxicity, mechanism and health effects of some heavy metals. Interdisciplinary toxicology, 2014, 7(2), pp.60-72.

[8] Yang, Q.W., Xu, Y., Liu, S.J., He, J.F. and Long, F.Y., Concentration and potential health risk of heavy metals in market vegetables in Chongqing, China. Ecotoxicology and environmental safety, 2011, 74(6), 1664-1669.

[9] Khan, S., Reid, B.J., Li, G. and Zhu, Y.G., Application of biochar to soil reduces cancer risk via rice consumption: a case study in Miaoqian village, Longyan, China. Environment international, 2014, 68, 154-161.

[10] Kim, H., Song, B., Kim, H. and Park, J., 2009. Distribution of trace metals at two abandoned mine sites in Korea and arsenic-associated health risk for the residents. Toxicology and Environmental Health Sciences, 2009, 1, 83-90.

[11] Nabulo, G., Black, C.R. and Young, S.D., Trace metal uptake by tropical vegetables grown on soil amended with urban sewage sludge. Environmental Pollution, 2011, 159(2), 368-376.

[12] Singh, R., Gautam, N., Mishra, A. and Gupta, R., Heavy metals and living systems: An overview. Indian Journal of Pharmacology, 2011, 43(3), 246-253.

[13] Kuruvilla, A., Pillay, V.V., Adhikari, P., Venkatesh, T., Chakrapani, M., Jayaprakash Rao, H.T., Bastia, B.K., Rajeev, A., Saralaya, K.M., and Rai, M., Clinical manifestations of lead workers of Mangalore, India. Toxicology and industrial health, 2006, 22(9), 405-413.

[14] Nolan, K.R., 1983. Copper toxicity syndrome. Journal of Orthomolecular Psychiatry, 1983, 12(4), 270-282.

[15] Young, R.A., Toxicity profiles: toxicity summary for cadmium, risk assessment information system. RAIS, University of Tennessee, 2005.

[16] Duruibe, J.O., Ogwuegbu, M.O.C. and Egwurugwu, J.N., Heavy metal pollution and human biotoxic effects. International Journal of Physical Sciences, 2007, 2(5), pp.112-118.

[17] Flora, S.J.S., Mittal, M. and Mehta, A., Heavy metal induced oxidative stress & its possible reversal by chelation therapy. Indian Journal of Medical Research, 2008, 128(4), pp.501-523.

[18] Florea, A.M. and Büsselberg, D., Toxic effects of metals: modulation of intracellular calcium homeostasis. Materialwissenschaft und Werkstofftechnik: Entwicklung, Fertigung, Prüfung, Eigenschaften und Anwendungen technischer Werkstoffe,2005, 36(12), pp.757-760.

[19] Rehman, A.U., Nazir, S., Irshad, R., Tahir, K., ur Rehman, K., Islam, R.U. and Wahab, Z., Toxicity of heavy metals in plants and animals and their

uptake by magnetic iron oxide nanoparticles. Journal of Molecular Liquids,2021, 321, p.114455.

[20] Wu, X., Cobbina, S.J., Mao, G., Xu, H., Zhang, Z. and Yang, L., A review of toxicity and mechanisms of individual and mixtures of heavy metals in the environment, Environmental Science and Pollution Research, 2016, 23, pp.8244-8259.

[21] Tchounwou, P.B., Yedjou, C.G., Patlolla, A.K. and Sutton, D.J., Heavy metal toxicity and the environment, Molecular, clinical and environmental toxicology, 2012, 3, 133-164.

[22] Gorby, M.S., 1988. Arsenic poisoning. Western Journal of Medicine, 149(3), 308.

[23] Lai, V.W.M., Sun, Y., Ting, E., Cullen, W.R. and Reimer, K.J., Arsenic speciation in human urine: are we all the same?. Toxicology and applied pharmacology, 2004, 198(3), 297-306.

[24] Le, X.C., Lu, X., Ma, M., Cullen, W.R., Aposhian, H.V. and Zheng, B., Speciation of key arsenic metabolic intermediates in human urine. Analytical Chemistry, 2000, 72(21), 5172-5177.

[25] Sullivan, J.B. and Krieger, G.R. eds., Clinical environmental health and toxic exposures. Lippincott Williams & Wilkins, 2001.

[26] Singhal, S.K., Toxicology at a Glance. 10th ed. National Books, 2022, 94–111.

[27] Pratush, A., Kumar, A. and Hu, Z., Adverse effect of heavy metals (As, Pb, Hg, and Cr) on health and their bioremediation strategies: a review. International Microbiology, 21, 2018, 97-106.

[28] Yang, L., Li, X., Chu, Z., Ren, Y. and Zhang, J., Distribution and genetic diversity of the microorganisms in the biofilter for the simultaneous removal of arsenic, iron and manganese from simulated groundwater. Bioresource Technology, 2014, 156, 384-388.

[29] Balali-Mood, M., Naseri, K., Tahergorabi, Z., Khazdair, M.R. and Sadeghi, M., Toxic mechanisms of five heavy metals: mercury, lead, chromium, cadmium, and arsenic. Frontiers in pharmacology, 2021, 12, 643972.

[30] Mahmood, A. and Malik, R.N., Human health risk assessment of heavy metals via consumption of contaminated vegetables collected from different irrigation sources in Lahore, Pakistan. Arabian Journal of Chemistry, 2014, 7(1), 91-99.

[31] Pillay, V., Textbook of Forensic Medicine and Toxicology (19th ed.). Paras Medical Publisher, 2019, 554-573.

[32] Patel, K., Gin, A. and Scardamaglia, L., Palmoplantar keratosis caused by arsenic toxicity. The Medical Journal of Australia, 2021, 214(6), 258-258.

[33] Richir, M.C. and Langenhorst, B.L.A.M., 2011. Een vrouw met 'jaarringen'op haar nagels. Nederlands Tijdschrift voor Geneeskunde, 2011, 155(45), 34-37.

[34] Li, Y., Ye, F., Wang, A., Wang, D., Yang, B., Zheng, Q., Sun, G. and Gao, X., 2016. Chronic arsenic poisoning probably caused by arsenic-based pesticides: findings from an investigation study of a household. International journal of environmental research and public health, 2016, 13(1), 133.

[35] Das, S., Chowdhury, J. and Ghoshal, L. An introspection into the cutaneous manifestations of chronic arsenicosis as reported in a tertiary care centre in Kolkata. Journal of Pakistan Association of Dermatologists, 2014, 24(4), pp.286-291.

[36] Das, A., Toshniwal, A., and Majumdar, K., Raindrop pigmentation in chronic arsenic poisoning. Pigment, International Council for Harmonisation (ICH), 2014. Guideline for Elemental Impurities Q3D., 2020, 7, 1–75.

[37] Sinicropi, M.S., Amantea, D., Caruso, A. and Saturnino, C., 2010. Chemical and biological properties of toxic metals and use of chelating agents for the pharmacological treatment of metal poisoning. Archives of Toxicology, 2010, 84, 501-520.

[38] Who, C., Environmental health criteria. World Health Organization: Geneva, Switzerland, p.1992.

[39] Thompson, J. and Bannigan, J., Cadmium: toxic effects on the reproductive system and the embryo. Reproductive toxicology, 2008, 25(3), 304-315.

[40] Genchi, G., Carocci, A., Lauria, G., Sinicropi, M.S. and Catalano, A., Nickel: Human health and environmental toxicology. International journal of environmental research and public health, 2020, 17(3), 679.

[41] Mahurpawar, M., 2015. Effects of heavy metals on human health. Int J Res Granthaalayah, 2015, 530, 1-7.

[42] Zalups, R.K. and Ahmad, S., Molecular handling of cadmium in transporting epithelia. Toxicology and applied pharmacology, 2003, 186(3), 163-188.

[43] Wang, Y., Fang, J., Leonard, S.S. and Rao, K.M.K., Cadmium inhibits the electron transfer chain and induces reactive oxygen species. Free Radical Biology and Medicine, 2004, 36(11), 1434-1443.

[44] Svartengren, M., Elinder, C.G., Friberg, L. and Lind, B., Distribution and concentration of cadmium in human kidney. Environmental Research, 1986, 39(1), pp.1-7.

[45] Barbier, O., Jacquillet, G., Tauc, M., Cougnon, M. and Poujeol, P., 2005. Effect of heavy metals on, and handling by, the kidney. Nephron Physiology, 2005, 99(4),105-p110.

[46] Pařízek, J., Effect of cadmium salts on testicular tissue. Nature, 1956, 177(4518).

[47] Schwartz, G.G., Il'yasova, D. and Ivanova, A., Urinary cadmium, impaired fasting glucose, and diabetes in the NHANES III. Diabetes care, 2003, 26(2), 468-470.

[48] Bernhoft, R.A., Cadmium toxicity and treatment. The Scientific World Journal, 2013, 1, 394652.

[49] Nishijo, M., Nakagawa, H., Suwazono, Y., Nogawa, K. and Kido, T., Causes of death in patients with Itai-itai disease suffering from severe chronic cadmium poisoning: a nested case–control analysis of a follow-up study in Japan. BMJ open, 2017, 7(7), 015694.

[50] Guideline for Elemental Impurities Q3D; ICH, 2014; 1–75.

[51] Assi, M.A., Hezmee, M.N.M., Sabri, M.Y.M. and Rajion, M.A., The detrimental effects of lead on human and animal health. Veterinary world, 2016, 9(6), p.660.

[52] Bathla, S. and Jain, T., 2016. Heavy metals toxicity. International Journal of Health Sciences and Research, 2016, 6(5), 361-368.

[53] Hernberg, S., Lead poisoning in a historical perspective. American journal of industrial medicine, 2000, 38(3), pp.244-254.

[54] Silbergeld, E.K., Waalkes, M. and Rice, J.M., Lead as a carcinogen: experimental evidence and mechanisms of action. American journal of industrial medicine, 2000, 38(3), pp.316-323.

[55] Pearce, J.M.S., Burton's line in lead poisoning. European neurology, 2007, 57(2), pp.118-119.

[56] Herman, D.S.S., Geraldine, M. and Venkatesh, T., Evaluation, diagnosis, and treatment of lead poisoning in a patient with occupational lead exposure: a case presentation. Journal of Occupational medicine and Toxicology, 2007, 2, 1-4.

[57] Chawla, M.P.S. and Sundriyal, D., Burton's Line. New England Journal of Medicine, 2012, 367(10), 937-937.

[58] Verma, S., Santhosh, A.P., Shukla, S., Gupta, P., Mandavdhare, H.S., Dutta, U. and Sharma, V., Gastrointestinal: An oral clue to an unusual cause of pain abdomen: Burton's line. Journal of Gastroenterology & Hepatology, 2019, 34(4).

[59] Lau, K.K., Chow, T.Y.A., Chan, C.K., Chan, Y.C., Ng, C.H.V., Ng, S.H. and Tse, M.L., 2018. Hong Kong poison information centre: annual report. Hong Kong Journal of Emergency Medicine, 2017, 25(6), 313-323.

[60] Abhilash, K.P., Arul, J.J. and Bala, D. Fatal overdose of iron tablets in adults. Indian journal of critical care medicine: peer-reviewed, official publication of

Indian Society of Critical Care Medicine, 2013, 17(5), 311.

[61] Mackenzie, E.L.; Iwasaki, K.; Tsuji, Y. Comprehensive Invited Review. Antioxid. Redox Signal. 2008, 10.

[62] Gerlach, M., Ben‐Shachar, D., Riederer, P. and Youdim, M.B.H., Altered brain metabolism of iron as a cause of neurodegenerative diseases?. Journal of Neurochemistry, 1994, 63(3), 793-807.

[63] Papanikolaou, G. and Pantopoulos, K., Iron metabolism and toxicity. Toxicology and applied pharmacology, 2005, 202(2), 199-211.

[64] Baranwal, A.K. and Singhi, S.C., 2003. Acute iron poisoning: management guidelines. Indian pediatrics, 2003, 40(6), 534-540.

[65] Yuen, H.W. and Becker, W., Iron toxicity. In Stat Pearls [Internet]. Stat Pearls Publishing, 2023.

[66] Pittelkow, M.R. and Flores, S., Hemochromatosis, 2017.

[67] Chmieliauskas, S., Banionis, D., Laima, S., Andriuskeviciute, G., Mazeikiene, S., Stasiuniene, J., Jasulaitis, A. and Jarmalaite, S., Autopsy relevance determining hemochromatosis: case report. Medicine, 2017, 96(49), 8788.

[68] Carocci, A., Rovito, N., Sinicropi, M.S. and Genchi, G., Mercury toxicity and neurodegenerative effects. Reviews of environmental contamination and toxicology, 2014, 1-18.

[69] Morais, S., Costa, F.G. and Pereira, M.D.L., Heavy metals and human health. Environmental health–emerging issues and practice, 2012, 10(1), 227-245.

[70] Caito, S.W., Jackson, B.P., Punshon, T., Scrimale, T., Grier, A., Gill, S.R., Love, T.M., Watson, G.E., van Wijngaarden, E. and Rand, M.D., Editor's highlight: Variation in methylmercury metabolism and elimination status in humans following fish consumption. Toxicological Sciences, 2018, 161(2), 443-453.

[71] Bjørklund, G., Dadar, M., Mutter, J. and Aaseth, J., The toxicology of mercury: Current research and emerging trends. Environmental research, 2017, 159, 545-554.

[72] Carman, K.B., Tutkun, E., Yilmaz, H., Dilber, C., Dalkiran, T., Cakir, B., Arslantas, D., Cesaretli, Y. and Aykanat, S.A., Acute mercury poisoning among children in two provinces of Turkey. European journal of pediatrics, 2013, 172, 821-827.

[73] Lai, O., Parsi, K.K., Wu, D., Konia, T.H., Younts, A., Sinha, N., McNelis, A. and Sharon, V.R., Mercury toxicity presenting as acrodynia and a papulovesicular eruption in a 5-year-old girl. Dermatology Online Journal, 2016, 22(3).

[74] Do, S.Y., Lee, C.G., Kim, J.Y., Moon, Y.H., Kim, M.S., Bae, I.H., and Song, H.S., Cases of acute mercury poisoning by mercury vapor exposure during the demolition of a fluorescent lamp factory. Annals of occupational and environmental medicine, 2017, 29, 1-8.

[75] Stone, C., Angermann, J. and Sugarman, J., 2021. Erethism Mercurialis and reactions to elemental mercury. Cutis, 2021, 107(4), 190-198.

[76] Strachan, S., Trace elements. Current Anaesthesia & Critical Care, 2010, 21(1), pp.44-48.

[77] Arora, N., Bhat, P., Goel, R., Pannu, A.K., Malhotra, P. and Suri, V., Kayser–Fleischer ring. QJM: An International Journal of Medicine, 2020, 113(5), 361.

[78] Chandra, A. and Bhattacharjee, M.S., Kayser–Fleischer Rings in Wilson's Disease. New England Journal of Medicine, 2021, 385(14), 46.

[79] Bigdon, E., Feuerstacke, J., Steinhorst, N.A. and Spitzer, M., Diagnosis of Kayser Fleischer Ring: Can Early Diagnosis Improve the Outcome of Wilson's Disease?. Klinische Monatsblatter fur Augenheilkunde, 2019, 237(10), pp.1237-1239.

[80] Schrag, A. and Schott, J.M., Kayser–Fleischer Rings in Wilson's Disease. New England Journal of Medicine, 2012, 366(12), 18.

[81] Koay, C.L., Zahari, M. and Lee, W.S., Kayser–Fleisher Ring and Sunflower Cataract in a Child

with Wilson's Disease. Pediatrics & Neonatology, 2017, 58(1), 97-98.

[82] Deguti, M.M., Tietge, U.J., Barbosa, E.R. and Cancado, E.L., The eye in Wilson‘s disease: sunflower cataract associated with Kayser-Fleischer ring. Journal of hepatology, 2002, 37(5), p.700.

[83] Goel, S., Sahay, P., Maharana, P.K. and Titiyal, J.S., Ocular manifestations of Wilson’s disease. BMJ Case Reports CP, 2019, 12(3), p.e229662.

[84] Ram, J. and Gupta, A., Kayser-Fleischer ring and sunflower cataract in Wilson disease. JAMA ophthalmology, 2014, 132(7), pp.873-873.

[85] Modi, J.P. and Kannan, K. Modi: A Textbook of Medical Jurisprudence and Toxicology. 27th edn. LexisNexis. 2022

[86] Link, V.B. and Mohr, C.O., Rodenticides in bubonic-plague control. Bulletin of the World Health Organization, 1953, 9(5), 585.

[87] Nelson, L.S., Howland, M.A., Lewin, N.A., Smith, S.W., Goldfrank, L.R. & Hoffman, R.S, Pesticides. In: Goldfrank’s Toxicologic Emergencies, 11th ed. New York, NY: McGraw-Hill Education. 2019.

[88] Desenclos, J.C., Wilder, M.H., Coppenger, G.W., Sherin, K., Tiller, R. and VanHook, R.M., 1992. Thallium poisoning: an outbreak in Florida, 1988. Southern medical journal, 1992, 85(12), 1203-1206.

[89] Peter, A.J. and Viraraghavan, T., Thallium: a review of public health and environmental concerns. Environment international, 2005, 31(4), pp.493-501.

[90] Sharquie, K.E., Ibrahim, G.A., Noaimi, A.A. and Hamudy, H.K., Outbreak of thallium poisoning among Iraqi patients. Journal of the Saudi Society of Dermatology & Dermatologic Surgery, 2011, 15(1), 29-32.

[91] Rusyniak, D.E., Furbee, R.B. and Kirk, M.A., Thallium and arsenic poisoning in a small midwestern town. Annals of emergency medicine, 2002, 39(3), 307-311.

[92] Baldwin, D.R. and Marshall, W.J., Heavy metal poisoning and its laboratory investigation. Annals of clinical biochemistry, 1999, 36(3), 267-300.

[93] Yumoto, T., Tsukahara, K., Naito, H., Iida, A. and Nakao, A., A successfully treated case of criminal thallium poisoning. Journal of clinical and diagnostic research: JCDR, 2017, 11(4), p.OD01.

[94] Yang, G., Li, C., Long, Y. and Sheng, L., Hair loss: evidence to thallium poisoning. Case reports in emergency medicine, 2018, (1), 1313096.

[95] Downs, A.J. ed., Chemistry of aluminium, gallium, indium and thallium. Springer Science & Business Media, 1993.

[96] IPCS (International Programme on Chemical Safety), Thallium: Environmental health criteria, vol. 182. Geneva: World Health Organization, 1996.

[97] ATSDR (Agency for Toxic Substance and Disease Registry), Toxicological profile for thallium. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA, 1992.

[98] Tyagi, R., Rana, P., Khan, A.R., Bhatnagar, D., Devi, M.M., Chaturvedi, S., Tripathi, R.P. and Khushu, S., Study of acute biochemical effects of thallium toxicity in mouse urine by NMR spectroscopy. Journal of Applied Toxicology, 2011, 31(7), pp.663-670.

[99] Karbowska, B., Presence of thallium in the environment: sources of contamination, distribution and monitoring methods. Environmental monitoring and assessment, 2016, 188, pp.1-19.

[100] Cvjetko, P., Cvjetko, I. and Pavlica, M., Thallium toxicity in humans. Arhiv za higijenu rada i toksikologiju, 2010, 61(1), 111-118.

[101] Riyaz, R., Pandalai, S.L., Schwartz, M. and Kazzi, Z.N., A fatal case of thallium toxicity: challenges in management. Journal of medical toxicology, 2013, 9, pp.75-78.

[102] Mulkey, J.P. and Oehme, F.W., A review of thallium toxicity. Veterinary and human toxicology, 1993, 35(5), pp.445-453.

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JCHR (2026) 16(1), 484-500 | ISSN:2251-6727

[103] Zhang, A. and Liu, X., Case study of chronic thallium poisoning using hair analysis. Journal of Forensic Medicine, 2015, 31(3), pp.188-195.

[104] Pelclová, D., Urban, P., Ridzoň, P., Šenholdová, Z., Lukáš, E., Diblík, P. and Lacina, L., Two-year follow-up of two patients after severe thallium intoxication. Human & experimental toxicology, 2009, 28(5), 263-272.

[105] Bandino, J.P. and Elston, D.M., Acute-onset alopecia. Cutis, 2019, 103(4), E24-E26.

[106] Ibrahim, D., Froberg, B., Wolf, A. and Rusyniak, D.E., Heavy metal poisoning: clinical presentations and pathophysiology. Clinics in laboratory medicine, 2006, 26(1), pp.67-97.

[107] Lu, C.I., Huang, C.C., Chang, Y.C., Tsai, Y.T., Kuo, H.C., Chuang, Y.H. and Shih, T.S., Short-term thallium intoxication: dermatological findings correlated with thallium concentration. Archives of dermatology, 2007, 143(1), 93-98.

[108] Misra, U.K., Kalita, J., Yadav, R.K. and Ranjan, P., Thallium poisoning: emphasis on early diagnosis and response to haemodialysis. Postgraduate medical journal, 2003, 79(928), pp.103-105.

[109] Almassri, I. and Sekkarie, M., Cases of thallium intoxication in Syria: A diagnostic and a therapeutic challenge. Avicenna Journal of Medicine, 2018, 8(03), 78-81.