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REVIEW ARTICLE

Acute versus Chronic Heavy Metal Poisoning in Industrial Workers: A Comprehensive Review

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ABSTRACT

The poisoning by heavy metal is considered to be a direct health hazard towards the occupational industrial workers present globally. The various forms of advancements which are made towards all the safety regulations followed by the management of exposure for the individuals employed in the industries like mining, smelting, production of battery, electroplating, welding, manufacturing of paint, tanneries and other recycling of the electronic waste is highly vulnerable towards the hazards that emits from the toxic metals which includes lead (Pb), mercury (Hg), cadium (Cd), arsenic (As), chromium (Cr), manganese (Mn) and nickel (Ni). This exposure certainly occurs from the inhalation of dust which is contaminated and also includes the fumes, dermal contract or any unintentional ingestion. This severity comes with the heavy form of metal toxicity that is the result of the acute poisoning made from the high level of exposure and other chronic poisoning made from the extended low level exposure that is completely also depended on the dosage, the duration of exposure and the entry made through routes. It can be understand in contrast with the chronic poisoning that also helps to develop over time and also may lead towards other form of irreversible damage made to the neurological, renal, hepatic, reproductive systems, and might be also associated with cancer. This review also certainly aims to provide for the contemporary insights and also the sources that is made with the exposure of the workplace exposure, the mechanism of toxicity, clinical manifestations, diagnostic approaches, treatment options, preventive measures, and future research directions. It also highlights towards the importance that helps to distinguish among the various acute and other chronic poisoning that also enhances the early detection followed by the workplace surveillance and other related preventive strategies.

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KEYWORDS

• Heavy Metal Toxicity • Occupational Exposure • Acute And Chronic Poisoning
• Clinical Manifestations • Industrial Hazards • Workplace Surveillance

INTRODUCTION

The introduction of industrialization also played a crucial role that helps for the advancement of the various economic developments, but this also increased the various occupational exposures made towards the hazardous substances, that are also made particularly towards the heavy metal. The naturally occurring metals are heavy metals with related elements that also have high atomic weights and densities that become so toxic and also when their level exceeds various physiological thresholds. The other metals that include lead, mercury, cadmium and other arsenic which do not hold any beneficial biological functions that leads to the formation of toxicity and other levels of concentrations. This expansion comes with the other industrial activities that stands out to be made with both environmental and other occupational formation of exposure towards this metals, and this also makes the heavy metal poisoning which is much concern for the public health.

The associated workforce comes with the most vulnerable form of occupational categories, that also faces exposure to the heavy metals and other activities which also includes mining, smelting, wielding, electroplating, battery manufacturing, pigment production, construction, chemical processing, and electronic waste recycling. The exposure which is made certainly occurs with the inhalation of the dust and other metal fumes, which is absorbed by the skin and ingestion that comes with the stemming made from the poor hygiene workplaces. Thus in contrast the various other organic chemicals which are to be metal breakdown, the heavy metals stands out to be persistent to the biological system, the accumulation is with the tissues including the bones, kidneys, liver and other form of central nervous systems that also increases the risk of chronic toxicity.

Various occupational health agencies and the World Health Organization further classify lead, mercury, cadmium, arsenic and hexavalent chromium which stands as the proxy toxicants due to their extensive form of industrial utilization and are also associated with various neurological disorders, chronic kidney disease, cardiovascular diseases, reproductive toxicity, respiratory illnesses and other several kinds of cancer. The various forms of regulatory standards also curtailed other severe poisoning cases which are also developed towards other countries, the occupational exposure also remains prevalent in other developing regions where engineering controls, biological monitoring, and the enforcement of workplace safety regulations may not be adequately implemented.

The heavy metal poisoning is also categorized towards various other acute and other chronic based that is on the severity and length of the exposure. The acute poisoning which is made with the unintentional contact and is also elevated in higher levels with the other toxic metals made during industrial mishaps, malfunctioning of equipment's, chemical leaks, or ventilation which is insufficient. The associated symptoms also helps to manifest with the hours made in a day and can also include various other abdominal discomforts, nausea and diarrhea encephalopathy, seizures, acute renal failure, respiratory difficulties, low blood pressure, and cardiovascular failure. One is required to make timely diagnosis and the commencement of the supportive treatment, this is also suitable with the chelation therapy and when it stands as necessary and are also crucial for minimizing morbidity and mortality.

Just on the other hand, the chronic heavy metal poisoning also progresses gradually as due to the repeated exposure as it is relatively low over a span of months and years. As the heavy metals also help to accumulate the various tissues and also have extended towards the biological half lives. The chronic exposure which is correlated with the cognitive decline

also is peripheral neuropathy hypertension, chronic kidney disease, osteoporosis, endocrine dysfunction, infertility, immune dysregulation, and cancer. Thus the delayed symptoms also complicate the diagnostic process and also the result made under the recognition of the other occupational heavy metal toxicity.

The only mechanism which is responsible for the heavy metal toxicity stands out to be multifactorial. The common feature which is shared by the toxic metal are the indication of the oxidative stress that is made with the excessive production of the reactive oxygen species. The heavy metals also binds the sulfhydryl groups of proteins that also inhibit the antioxidant enzymes which is superoxide dismutase and glutathione peroxidase, also impair mitochondrial respiration, disrupt calcium homeostasis, interfere with DNA repair mechanisms, and activate inflammatory signalling pathways. The associated molecular events also contributes towards the apoptosis, fibrosis, carcinogenesis and other progressive dysfunction of various multiple system of organs. The heavy metals also helps to possess the unique toxicodynamic properties followed by the oxidative stress and other mitochondrial injury which actually represents the central mechanism that helps to underlay the acute and other chronic toxicity.

The recent evidences showcases the that chronic occupational exposure also helps to contributes other form of substantially greater form of disease which is a burden that is made with the acute poisoning. This improvement stands out to be industrial hygiene which stands out to be reduced in a catastrophic manner with the poisoning events, this however with the prolonged low doses of exposure that also continues to further affect millions of workers. The occupational environments also expose the workers to give the mixture of the heavy metals and not individual metals, this also helps to create the addictive synergistic toxic effect that also stands insufficient. The consequence made with the early biological monitoring with the exposure assessment, followed by the engineering controls, other personal protective equipment and also helps the works education to remain with the essential components and other related occupational health programs.

LITERATURE REVIEW

1. Historical Background of Heavy Metal Poisoning

The heavy metal poisoning is also recognized towards as the occupational disease made for centuries. The historical records also describes the poisoning of leads made among the roman miners and other pipe workers, while with the mercury poisoning it also became well documented with the miners and the associated hat makers made in the period of industrial revolution. The forms of rapid industrialization made during the nineteenth and twentieth centuries has significantly increased which is to the occupational exposure made to the metals through the form of mining, smelting, battery manufacturing, welding, pigment production, and electroplating. The various regulatory policies and the industrial hygiene also have a reduced form of poisoning effect formed in various countries. Hence the occupational exposure also remains with the major form of public health concerns made with the developing nations under the engineering controls and other biological monitoring stands out be inadequate. (Jaishankar *et al.,* 2014; Jomova *et al.,* 2025).

The variation which is made with the organic toxicants with the heavy metals in the industrial environments. As the organic chemicals that can also be metabolized and further eliminated and the heavy metals are not biodegradable and does not contain biological tissues over a period of time. As a consequence the low occupational exposure might also result in the clinical toxicity after any form of prolonged employment. (Balali-Mood *et al.,* 2021; Narayanan, 2026).

2. Occupational Sources and Routes of Exposure

The industrial workers also encounter the heavy metals that are made in the numerous occupational settings. The mining and processing of ore also further exposes the workers towards lead arsenic, cadmium, and mercury-containing dust. This also includes the battery manufacturing and any other form of recycling which are the major sources of lead and cadmium exposure, as wielding and stainless steel fabrication also exposes the workers towards chromium, manganese, and nickel fumes. The electroplating industries also use the chromium and nickel compounds

in extensive manner, on the other hand the manufacturing, production of pigment and the tanneries also continues to further utilize the chromium and compounds of lead present in several regions. The informal recycling made to the electronic waste also grows as the mixed exposure of metal as of dismantling, burning and the extraction of acid that also releases the multiple toxic metals simultaneously. (Jaishankar *et al.,* 2014; Narayanan, 2026).

The occupational exposure comes with the inhalation, dermal absorption and ingestion. The inhalation of airborne dust and the metal fumes is also considered to be the most significant route towards the industrial settings and of the associated fine particles of dust that reach alveoli and also enter the system of circulation. The dermal exposure which is made also occurs with the direct handling of the contaminated materials as it is with the ingestion that also results from the various workplace hygiene contaminated food or smoking without any hand washing. As once absorbed, many associated heavy metals also bind strongly towards the protiens and further accumulate the organs that also includes liver, kidney, bones and brain where it might also remain for years. (Balali-Mood *et al.,* 2021; Godt *et al.,* 2006).

2.3 Heavy Metals of Occupational Importance

Lead (Pb)

The lead also remains as one of the extensive occupational toxicants. The workers involved in the battery also helps to manufacture, followed by the smelting of lead construction, cable production, ammunition manufacture, radiator repair, and recycling industries experience the highest exposure. The lead interferes with the haem synthesis by the means of inhibiting inhibiting δ-aminolevulinic acid dehydratase (ALAD) and ferrochelatase, which also further results in anaemia. It also then effects the nervous system, kidneys and other cardiovascular system and reproductive organs. The chronic lead exposure is in association with the cognitive impairment, peripheral neuropathy, hypertension, nephropathy, and reproductive dysfunction (Jaishankar *et al.,* 2014; Balali-Mood *et al.,* 2021).

Cadmium (Cd)

Cadmium is mostly used under the nickel-cadmium batteries, pigments, electroplating, and refining of metal. This also possess and exceptionally form of long biological half-life, this is estimated to also exceed 15 years presently in humans, and this also allows for the progressive accumulation of the other renal tissues. The chronic cadmium exposure which also effects and damages the functioning of kidneys and bones that is also made in relation with osteoporosis, followed by other pulmonary disease and lung cancer. The classification of cadmium compounds is made by the International Agency for Research as Group 1 Human Carcinogens (Godt *et al.,* 2006; Jomova *et al.,* 2025).

Mercury (Hg)

Mercury exposure takes place in chlor-alkali industries, fluorescent lamp manufacturing, gold mining, laboratories, and some chemical industries. Elemental mercury vapour can easily transverse the blood-brain barrier, primarily impacting the central nervous system. Long-term workplace exposure may lead to behaviors such as erethism, and tremors, as well as, gingivitis, memory loss, and peripheral neuropathy. Short-term inhalation can cause acute pneumonitis and respiratory failure (Balali-Mood *et al.* 2021).

Arsenic (As)

Exposure to arsenic takes place during mining, smelting, pesticide production, semiconductor manufacturing, and wood preservation. Long-term exposure has been linked to distinct skin lesions, peripheral neuropathy, cardiovascular disease, diabetes mellitus, and cancers of the skin, lung, liver, and bladder. In cases of acute poisoning, individuals often exhibit gastrointestinal symptoms, hypotension, and multi-organ failure (Jaishankar *et al.,* 2014; Balali-Mood *et al.,* 2021).

Chromium (Cr)

Numerous sectors, including leather tanning, stainless steel welding, electroplating, and pigment manufacture, make extensive use of hexavalent chromium [Cr(VI)]. Reactive oxygen species, which can harm proteins and DNA, are produced by cells when Cr(VI) molecules easily pass through cell membranes. Asthma, nasal septum perforation, allergic dermatitis, and lung cancer have all been linked to prolonged occupational exposure (Balali-Mood *et al.,* 2021).

4. Mechanisms of Heavy Metal Toxicity

Toxicity is brought on by several common pathways, even though each heavy metal has distinct metaboliccharacteristics.

OxidativeStress

Oxidative stress is regarded to be the main cause of heavy metal poisoning. These metals cause an excess of reactive oxygen species (ROS), such as hydrogen peroxide, superoxide radicals, and hydroxyl radicals. An excess of ROS can overwhelm the body's natural antioxidant defensesystems, leading to lipid peroxidation, protein oxidation, mitochondrial damage, and DNA damage (Balali-Mood *et al.,* 2021; Jomova *et al.,* 2025).

Enzyme Inhibition

A variety of heavy metals have a pronounced tendency to bind with sulfhydryl (-SH) groups located in proteins and enzymes. This binding hampers the function of enzymes that play critical roles in cellular respiration , antioxidant defense, and energy metabolism. Specifically, lead inhibits the enzymes that are crucial for haem synthesis, whereas arsenic disrupts ATP(production by affecting oxidative phosphorylation (Jaishankar *et al.,* 2014).

Mitochondrial Dysfunction

Heavy metals hinder mitochondrial respiration by interfering with the activity of the electron transport chain, leading to reduced ATP production and the induction of apoptosis. This persistent dysfunction of mitochondria is a contributing factor to the progressive neurological decline, muscle weakness, and chronic organ dysfunction that are prevalent among workers with occupational exposure (Jomova *et al.,* 2025).

DNA Damage and Carcinogenesis

Cadmium, arsenic, chromium, and nickel disrupt DNA repair mechanisms, promote chromosomal instability, and modify gene expression via epigenetic mechanisms. continuous oxidative stress and ineffective DNA repair lead to increased mutation rates, contributing to occupational carcinogenesis. As a result, numerous heavy metals are designated as known or probable human carcinogens (Balali-Mood *et al.,* 2021; Jomova *et al.,* 2025).

Inflammation and Immune Dysregulation

Long-term exposure to heavy metals activates inflammatory pathways, which results in the persistent production of cytokines such as tumour necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-1β (IL-1β). This persistent inflammatory syndrome leads to tissue fibrosis, endothelial dysfunction, and growing damage to the kidneys, liver, lungs, and cardiovascular system. Furthermore, research indicates that heavy metals weaken both innate and adaptive immune responses, making people more vulnerable to viral and autoimmune diseases (Narayanan, 2026).

DISCUSSION

1. Acute Heavy Metal Poisoning

Acute heavy metal poisoning signifies the onset of detrimental effects resulting from one or more exposuresto elevated levels of heavy metals within a short timeframe, typically ranging from a few hours to several days. Acute poisoning at work often happens because of industrial mishaps, accidental chemical spills, equipment malfunctions, poor ventilation, explosions, or failing to use appropriate personal protective equipment (PPE). People employed in mining, smelting, electroplating, battery production, welding, pigment manufacturing, and electronic waste recycling encounter increased risks due to direct contact with toxic metals like lead (Pb), mercury (Hg), cadmium (Cd), arsenic (As), chromium (Cr), nickel (Ni), and manganese (Mn) (Balali-Mood et al., 2021; Agency for Toxic Substances and Disease Registry [ATSDR], 2020). Acute toxicity levels are affected by several factors, including the specific metal involved, the amount, duration of exposure, chemical makeup, route of exposure (inhalation, ingestion, or skin contact), and the individual's age, nutritional status, and pre-existing health conditions. In industrial environments, the primary avenue of exposure occurs via inhalation of metal dust and fumes, as ultrafine particles can rapidly access the alveoli and enter systemic circulation (Jaishankar *et al.,* 2014). The clinical manifestations vary depending on the particular toxic metal involved. Acute lead poisoning typically manifests as severe abdominal pain, nausea, vomiting, constipation, encephalopathy, seizures, altered mental state, anemia, and abrupt kidney injury.

Acute arsenic poisoning presents with intense gastrointestinalirritation leading to profuse watery diarrhea, vomiting, hypotension, dehydration, cardiac arrhythmias, peripheral neuropathy, and failure of multiple organs. The detrimental impacts mainly result from the suppression of pyruvate dehydrogenase and the disruption of oxidative phosphorylation, causing compromised energy generation in cells (Balali-Mood *et al.,* 2021). Exposureto mercury vapor can result in prompt respiratory problems such as coughing, chest pain, difficulty breathing, fever, and chemical pneumonitis. High-dose exposure can lead to pulmonary edema, respiratory failure, tremors, agitation, confusion, and kidney impairment. Similarly, inhaling cadmium oxide fumes during welding or metal refining can result in acute chemical pneumonitis, fluid accumulation in the lungs, and severe respiratory distress, often requiring intensive medical care (Clarkson & Magos, 2006; Godt *et al.,* 2006)

The molecular mechanisms associated with acute heavy metal toxicity involve the rapid emergence of oxidative stress triggered by excess reactive oxygen species (ROS), inhibition of antioxidant enzymes, mitochondrial dysfunction, disturbance of calcium equilibrium, protein denaturation, and DNA damage. This oxidative harm results in cell necrosis, inflammation, vascular endothelium dysfunction, and apoptosis, ultimately resulting in organ failure if the exposure is not promptly addressed (Balali-Mood *et al.,* 2021; Jomova & Valko, 2011). Prompt identification and quick action are essential for lowering disease and mortality rates. The management protocol starts with the immediate removal from the source of exposure, securing stabilization of airway, breathing, and circulation (ABC), gastrointestinal decontamination if required, supportive intensive care, correction of electrolyte disturbances, and administration of metal-specific chelators. Chelation therapy employing calcium disodium EDTA, dimercaprol (BAL), succimer (DMSA), or dimercaptopropane sulfonate (DMPS) is recommended only when laboratory findings and severity indicate significant systemic toxicity (Flora *et al.,* 2008).

2. Chronic Heavy Metal Poisoning

Chronic heavy metal poisoning can be brought on by months or years of exposure to relatively small concentrations of dangerous metals. Unlike acute poisoning, chronic toxicity often occurs without evident symptoms because many heavy metals have long biological half-lives and gradually accumulate in target organs. Because symptoms typically don't show up until significant tissue damage has happened, chronic poisoning is the most prevalent occupational issue among industrial workers (Järup, 2003). There are distinct patterns in the distribution and build-up of heavy metals in tissues. Lead is mostly deposited in cortical bone, which retains 90–95% of the entire body burden in adults. Cadmium mostly builds up in the renal cortex because of its strong affinity for metallothionein proteins. It has a longer biological half-life.

Mercury tends to accumulate in the central nervous system, whereas arsenic is broadly distributed throughout soft tissues before methylation and urine clearance. While chromium (VI) molecules enter cells through sulphate transporters, manganese accumulates in the brain's basal ganglia and impairs neurological function (Nordberg *et al.,* 2022; Jaishankar *et al.,* 2014).

Long-term occupational exposure has been linked to multisystem diseases. Neurological symptoms include Parkinsonian symptoms, tremors, anxiety, depression, peripheral neuropathy, cognitive deficits, memory loss, focus issues, and poor psychomotor performance. Long-term lead exposure has been linked to reduced IQ, hypertension, and faster cognitive decline, whereas chronic manganese exposure causes manganism, a neurological illness that resembles Parkinson's disease (Flora e*t al.,* 2012; Jomova *et al.,* 2025).

Renal damage is most severe after prolonged exposure to lead and cadmium. Cadmium's damage to the proximal renal tubules results in proteinuria, a drop in glomerular filtration rate (GFR), renal tubular dysfunction, and chronic kidney disease. Lead nephropathy is characterised by progressive interstitial fibrosis and irreversible nephron loss (Godt *et al.,* 2006; Järup, 2003).

Long-term exposure to heavy metals also has a major impact on cardiovascular disease. Epidemiological studies have connected prolonged exposure to lead, cadmium, and arsenic to increased risks of peripheral

vascular disease, stroke, hypertension, and ischaemic heart disease. The processes include endothelial dysfunction, oxidative stress, chronic inflammation, decreased nitric oxide production, and vascular calcification (Alissa & Ferns, 2011).

Reproductive toxicity is a significant occupational danger. Exposure to heavy metals alters ovarian physiology, interferes with endocrine function, affects spermatogenesis, decreases sperm motility, raises the likelihood of spontaneous abortion, and adversely impacts foetal development. The International Agency for Research on Cancer (IARC) has designated cadmium, arsenic, nickel compounds, and hexavalent chromium as Group 1 human carcinogens due to their capacity to damage DNA, including epigenetic modifications, induce genomic instability, and obstruct DNA repair mechanisms (IARC, 2012; IARC, 2018).

The main cause of chronic toxicity is oxidative stress. The ongoing production of reactive oxygen species (ROS) causes lipid peroxidation, mitochondrial failure, chronic inflammation, the activation of apoptotic pathways, and fibrosis in the affected tissues. These pathological processe account for the progressive and often irreversible nature of chronic heavy metal poisoning (Balali-Mood *et al.,* 2021).

3. Acute v/s Chronic Heavy Metal Poisoning

There are significant differences between acute and chronic heavy metal poisoning in terms of exposure parameters, clinical symptoms, diagnosis, prognosis, and treatment, despite the fact that both conditions are brought on by exposure to the same harmful substances (Jaishankar *et al.,* 2014).

Table 1: Comparative Overview of Acute and Chronic Heavy Metal Toxicity and Associated Parameters

Parameter	Acute Heavy Metal Poisoning	Chronic Heavy Metal Poisoning
Exposure pattern	Single or short-term high-dose exposure	Long-term low-dose repeated exposure
Duration	Hours to days	Months to years
Common cause	Industrial accidents, spills, inhalation of fumes	Continuous occupational exposure
Symptom onset	Rapid	Gradual and insidious
Primary mechanism	Acute oxidative injury and cellular necrosis	Cumulative tissue accumulation, chronic oxidative stress
Clinical features	Abdominal pain, vomiting, seizures, pulmonary edema, acute renal injury	Neuropathy, nephropathy, hypertension, osteoporosis, infertility, cancers
Diagnosis	Clinical emergency with laboratory confirmation	Occupational history plus biomonitoring
Treatment	Emergency stabilization and chelation therapy	Exposure reduction, long-term monitoring, selective chelation
Outcome	Often reversible if treated promptly	Frequently irreversible due to cumulative organ damage

The body of research repeatedly shows that the burden of occupational disease caused by chronic heavy metal poisoning is far higher than that caused by acute poisoning. Even though severe poisoning incidents have decreased due to improvements in industrial hygiene, prolonged exposure to low levels of heavy metals is still common, particularly in low-and middle-income countries where occupational safety regulations may not be adequately enforced (World Health Organization [WHO], 2021; Järup, 2003).

Mixed-metal exposure, in which workers are exposed to many heavy metals at once, is a significant new problem. Through overlapping pathways such oxidative stress, mitochondrial dysfunction, inflammatory signalling, and poor DNA repair, this combined exposure may result in cumulative or synergistic harmful consequences. The health concerns connected to mixed exposures are the main focus of current occupational exposure limits (Balali-Mood *et al.,* 2021).

In conclusion, research from epidemiological studies, occupational surveillance programs, and investigations shows that preventing chronic exposure through engineering controls, routine biomonitoring, periodic medical evaluations, and worker education offers the best chance of reducing the global burden of heavy metal-related occupational diseases (Nordberg *et al.,* 2022).

4. Diagnosis of Heavy Metal Poisoning

To detect heavy metal toxicity in industrial workers, a thorough approach comprising occupational history, clinical examination, laboratory testing, biomonitoring, and environmental exposure evaluation is needed. Since many clinical signs of heavy metal poisoning lack specificity, getting a complete occupational history is often the most crucial step in reaching a diagnosis. It is crucial to accurately record the kind of industry, duration of employment, specific job responsibilities, use of personal protective equipment (PPE), workplace ventilation, previous occupational exposures, smoking habits, and environmental exposure (Jaishankar *et al.*, 2014).

Clinical symptoms can vary according on the metal involved, the duration of exposure, and the affected organ systems. Acute poisoning often presents as gastrointestinal symptoms, respiratory distress, altered mental status, seizures, or acute kidney injury, whereas chronic poisoning typically manifests as fatigue, peripheral neuropathy, hypertension, cognitive decline, chronic kidney disease, anaemia, infertility, and musculoskeletal disorders (Balali-Mood *et al.*, 2021).

Laboratory Investigations

In a laboratory environment, the evaluation of metal levels in biological material is primarily responsible for the diagnosis.

Typical tests consist of:

- Blood lead level (BLL)
- Blood mercury concentration
- Urinary mercury concentration
- Blood cadmium concentration
- Urinary cadmium concentration
- Complete blood count (CBC)
- Renal function tests (serum creatinine, blood urea nitrogen)
- Liver function tests
- Electrolyte profile
- Urinalysis for proteinuria and tubular dysfunction

Blood metal concentrations often indicate recent exposure, while urine concentrations are important for assessing ongoing exposure and the body load of various metals. However, when interpreting data, individual variability, exposure timing, and biological half-life should always be considered (ATSDR, 2020; Nordberg *et al.*, 2022).

Ancillary Diagnostic Tests

Additional testing may be necessary to assess organ-specific toxicity, including:

- Using nerve conduction tests to investigate peripheral neuropathy
- Pulmonary function tests to evaluate inhalation exposure
- Use electrocardiography to detect cardiac arrhythmias.
- Using neuropsychology to assess cognitive impairment
- Ultrasound of the kidneys
- Magnetic resonance imaging (MRI) in specific neurological situations
- X-rays of the bones in cases of chronic lead exposure

Early detection is critical because many toxic consequences become permanent after significant tissue damage has occurred.

5. Biomonitoring

Biomonitoring is an essential component of occupational health surveillance because it enables the early detection of excessive heavy metal exposure before clinical illness emerges. Biological monitoring improves environmental monitoring by identifying the actual internal dose that workers absorb (Apostoli, 2002).

Conventional Biomarkers

The most commonly used biomarkers are:

Table 2: Heavy Metal Biomarkers and their Clinical Significance

Heavy Metal	Biomarker	Clinical Significance
Lead	Blood lead level	Recent exposure and body burden
Cadmium	Urinary cadmium	Cumulative renal burden
Mercury	Blood and urine mercury	Recent exposure
Arsenic	Speciated urinary arsenic	Recent exposure
Chromium	Urinary chromium	Occupational exposure
Nickel	Urinary nickel	Industrial exposure

Blood lead concentration remains the international standard for monitoring occupational lead exposure, whereas urinary cadmium reflects cumulative kidney accumulation because of its prolonged biological half-life (ATSDR, 2022).

Emerging Biomarkers

- Recent developments in molecular toxicology have revealed a number

of intriguing biomarkers that can identify subclinical toxicity before irreversible organ damage occurs. These include oxidative stress indicators like glutathione, malondialdehyde, and superoxide dismutase.

- Markers of DNA damage (8-hydroxy-2′-deoxyguanosine)
- Inflammatory cytokines (TNF-α, IL-6, IL-1β)
- Metallothionein expression
- MicroRNAs (miRNAs)
- Epigenetic alterations

Metabolomic and proteomic profiles

Despite these biomarkers' potential, their frequent usage in occupational surveillance is still limited due to their high cost, lack of standardisation, and insufficient validation (Balali-Mood *et al.,* 2021).

Periodic biomonitoring is particularly important for workers in industries like battery manufacturing, mining, electroplating, welding, pigment production, and electronic waste recycling where continual exposure is expected.

6. Treatment

Treatment for heavy metal poisoning depends on the specific metal involved, the level of exposure, the clinical symptoms, and the duration of intoxication. The primary objective of treatment is to minimise tissue damage, prevent further exposure, and promote the elimination of the harmful metal (Flora *et al.,* 2008).

Removal from Exposure

Withdrawing quickly from the contaminated surroundings is the first and most important treatment step. Workers should be relocated to a safe area while workplace hazards are examined and addressed.

Supportive Care

- Supportive treatment includes:
- Airway stabilization
- Oxygen therapy
- Intravenous fluid replacement
- Electrolyte correction
- Seizure management
- Mechanical ventilation when indicated
- Renal replacement therapy for severe renal failure
- Nutritional support

Chelation Therapy

Chelation therapy enhances the removal of heavy metals from the urine by forming water-soluble complexes.

Chelating agents that are commonly used include :

Table 3: Chelating Agents and their Major Therapeutic Indications

Chelating Agent	Major Indications
Calcium disodium EDTA	Lead poisoning
Succimer (DMSA)	Mild-to-moderate lead poisoning
Dimercaprol (BAL)	Arsenic, mercury, severe lead poisoning
DMPS	Mercury and arsenic poisoning
Penicillamine	Copper poisoning and selected heavy metal intoxications

Chelation therapy should be administered only when indicated because inappropriate use may mobilize metals from tissue stores, increasing toxicity. Clinical presentation and test confirmation of high body load should be the basis for treatment decisions (Flora *et al.,* 2008).

Long-Term Monitoring

Periodic follow-up is necessary for industrial workers recuperating from heavy metal poisoning, and this includes:

- Neurological assessment
- Renal function monitoring
- Cardiovascular evaluation
- Repeat biomonitoring
- Occupational rehabilitation

7. Prevention

The best strategy for lowering occupational heavy metal toxicity is prevention. A hierarchy of controls that prioritises reducing or eliminating exposure over wearing personal protective equipment is supported by occupational health organisations (WHO, 2021).

Engineering Controls

The best preventative methods are engineering initiatives, which include:

- Local exhaust ventilation systems
- Process enclosure

- Dust suppression
- Wet drilling techniques
- Automation of hazardous processes
- Closed chemical handling systems

Administrative Controls:

- Administrative measures include:
- Worker rotation
- Exposure time limitation
- Routine equipment maintenance
- Workplace hygiene policies
- Standard operating procedures
- Hazard communication programs

Personal Protective Equipment

When exposure cannot be totally eliminated by engineering measures, the following PPE should be used:

- Respirators
- Chemical-resistant gloves
- Protective clothing
- Safety goggles
- Protective footwear

Medical Surveillance

Programs for comprehensive occupational surveillance must consist of: Pre-employment medical examinations

- Periodic clinical evaluations
- Routine biomonitoring
- Environmental monitoring
- Health education

Worker education significantly reduces occupational exposure by promoting hand hygiene, smoking cessation during work, proper PPE use, and awareness of early symptoms of toxicity (Jaishankar *et al.,* 2014)

8. Research Gaps and Future Directions

Even while occupational toxicology has made great strides, there are still a number of crucial knowledge gaps.

First off, even though industrial workers are frequently exposed to complex combinations of harmful metals, the majority of studies on occupational exposure tend to concentrate on individual metals. The existing occupational exposure limits do not adequately account for the additive or synergistic toxicity that may arise from the interactions between these linked metals (Balali-Mood *et al.,* 2021).

Moreover, current biomarkers primarily identify exposure after a significant accumulation has taken place. More sensitive biomarkers that can identify early molecular damage before clinical diseases appear are desperately needed. (WHO, 2021).

Future research should focus on:

- Development of highly sensitive molecular biomarkers.
- Toxicogenomics and precision occupational medicine.
- Artificial intelligence–assisted exposure prediction.
- Wearable sensors for real-time monitoring.
- Safer industrial technologies.
- Green chemistry alternatives to toxic metals.
- Longitudinal studies on mixed-metal exposure.
- Evaluation of gene–environment interactions influencing susceptibility.

These advances may substantially improve early diagnosis, individualised risk assessment, and prevention strategies for industrial workers.

CONCLUSION

Despite advancements in industrial hygiene and regulatory frameworks, heavy metal poisoning remains a serious occupational health concern for industrial workers. Accidental exposure to excessive amounts can result in acute poisoning, which usually presents as a medical emergency with problems in the neurological, respiratory, gastrointestinal, circulatory, and renal systems. On the other hand, long-term low-level exposure causes chronic poisoning, which develops gradually and frequently results in irreversible neurological damage, chronic renal disease, reproductive dysfunction, skeletal deformities, cardiovascular disease, and occupational malignancies.

Heavy metal toxicity is primarily caused by oxidative stress, mitochondrial dysfunction, inflammation, enzyme inhibition, and DNA damage, according to the research. Because

chronic poisoning often shows no symptoms until serious organ damage has occurred, prompt identification through occupational history, laboratory evaluations, and biomonitoring is essential.

Preventive approaches, including engineering controls, biological monitoring, PPE, worker education, and strict enforcement of occupational safety regulations, are the most effective ways to lower exposure to heavy metals. Intriguing avenues for better prevention and early diagnosis are presented by developments in toxico-genomics, precision occupational medicine, and molecular biomarkers.

Ongoing collaboration between toxicologists, industrial hygienists, occupational physicians, employers, and lowering the global burden of illnesses linked to heavy metals.

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