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Mining communities, Metal toxicity, Occupational health, Public health, Biomonitoring, Community-based interventions.

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Mining Metals And Medicinal Interventions: Pharmacological Care For Heavy Metal Toxicity In Mining Communities

Abstract
Mining plays a major role in the global economy. However, it can also expose workers and local communities to harmful heavy metals, such as cadmium, arsenic, chromium, mercury, and lead. These metals can bioaccumulate in soil, water, and living organisms and have been linked to neurotoxicity, renal dysfunction, cardiovascular disease, and increased carcinogenic risk. Children, pregnant women, and informal miners are among the most vulnerable groups, as they may be exposed to mercury and other toxic metals through dust inhalation, contaminated water and food, or direct occupational contact. Pharmacological treatments, such as metal-specific chelation therapy and antioxidant supplementation, are useful for clinically significant toxicity, but they should not replace environmental and occupational control measures. In resource-limited mining regions, pharmacists play an important role in early identification, risk assessment, medication management, patient counseling, and community-level interventions. This review summarizes the existing literature on the sources, mechanisms, clinical manifestations, and management of heavy metal toxicity, with emphasis on the pharmacist's role in reducing health risks and supporting public and occupational health programs in mining communities.

Introduction

The mining industry has long been a cornerstone of human civilization and economic development, providing precious metals, rare earth elements, and fossil fuels. These resources support industrialization, infrastructure development, and technological innovation, and they contribute about 6% to global GDP [1]. However, these economic benefits are often accompanied by serious environmental and public health burdens. Mining environments, particularly gold and coal mining areas, may contain toxic heavy metals and metalloids such as lead (Pb), mercury (Hg), cadmium (Cd), arsenic (As), and chromium (Cr). These metals can accumulate in the bodies of workers and members of nearby communities [2, 3]. This exposure is often chronic and gradual, occurring through contaminated air, soil, and water, as well as through direct occupational contact. Long-term exposure to these metals has been associated with neurological, renal, and cardiovascular diseases [4]. The effects are not limited to miners alone; nearby communities may also be affected through environmental degradation, loss of productive land, and disruption of local ecosystems. However, systematic reviews have shown that only a limited number of qualitative studies have examined how local communities, community leaders, and health workers perceive the health effects of mining [5]. Therefore, reducing these risks is essential for protecting community health and promoting sustainable mining practices [6].

Heavy metals are naturally occurring elements that resist degradation in the environment and can bioaccumulate in soil, water and living tissues [7]. Lead, arsenic, mercury, cadmium, and chromium are of particular concern because of their harmful physiological and toxicological effects are well documented. The World Health Organization (WHO) recognizes arsenic, cadmium, lead, and mercury as chemicals of major public health concern. Furthermore, the International Agency for Research on Cancer (IARC) has recognized arsenic, cadmium compounds, nickel compounds, and hexavalent chromium (Cr(VI)) compounds as known human carcinogens (Group 1) [8, 9]. In 2021, it was estimated that 1.5 million people worldwide died from exposure to lead, primarily from cardiovascular effects, with children also showing significant neurodevelopmental impacts, particularly in low and middle income countries (LMICs) [10].

Heavy metal contamination remains a persistent problem in mining areas [11]. Artisanal and small-scale gold mining (ASGM) accounts for about 37% of anthropogenic mercury emissions. It is active in more than 70 countries, involving 10–15 million miners and producing 12–13% of global gold. Community exposure occurs through inhalation and consumption of contaminated water and food [12]. Biomonitoring studies indicate the presence of mercury, lead, cadmium, chromium, and arsenic in water, soil, crops, and human populations worldwide. Women in these communities are typically more exposed to arsenic and cadmium. Notably, hepatic, renal and neurological effects are reported at blood and tissue concentrations previously considered safe [13, 14]. Although the burden of heavy metal toxicity is considerable, health care interventions remain insufficient, especially concerning the pharmacist's role. Chronic exposures may present with non-specific symptoms including fatigue, mental confusion, anemia, neuropathy, and renal dysfunction, which may lead to delayed diagnosis and avoidable morbidity [15]. Nevertheless, pharmacist across community, hospital, and occupational health settings has a role to play in bridging these gaps. They can identify toxicity early, adjust pharmacologic therapy, such as chelation, track patient compliance, and prevent toxicity at the individual and community-levels [16]. They are especially important in remote or low resource mining areas where access to specialized occupational medicine services is limited [17].

This review examines sources and routes of heavy metal exposure in mining communities, toxicological mechanisms, clinical manifestations, and pharmacological management strategies. This review highlights the role of pharmacists in disease prevention, health monitoring, and community-based interventions. Its findings may help inform public health policy on heavy metal toxicity, support improved occupational safety protocols for miners and nearby communities, and guide clinical practice in the screening, monitoring, and pharmacological management of heavy metal toxicity among mining populations.

2. Toxicological Profile of Heavy Metals in Mining Environments

2.1. Sources and Routes of Exposure

Heavy metal pollution is a result of the interconnected pathways associated with mining activities. Respirable dust is a primary route of exposure, formed during mechanical drilling, crushing, and blasting of ore-bearing rocks. Ore processing and smelting are significant emitters of metals into the air. Acid mine drainage (AMD) occurs when pyrite is oxidized, releasing cadmium, arsenic, and lead into surface and groundwater. The communities around mining areas are vulnerable to exposure via drinking water and food grown in the immediate vicinity [18]. In artisanal and small-scale mining (ASGM), additional exposures occur when mining personnel handle ore, use contaminated equipment, and bring dust home, affecting entire communities.

2.2. Metal-Specific Toxicological Profiles

Table 1 summarizes the major heavy metals, their primary sources, exposure pathways, affected organ systems, and associated toxic effects in mining communities.

i. Lead (Pb):

Lead exposure occurs most commonly in the mining and smelting of coal, gold, and silver ores. Inhalation of airborne lead particles damages pulmonary alveoli and can lead to chronic neurotoxic, hematologic, and renal effects. A community study in Peru found higher blood lead levels (BLL) among residents living near a mining site and identified associations with hemoglobin levels and DNA methylation changes, including changes in genes related to neurological development and function [19]. The World Health Organization (WHO) affirms that no safe level of BLL exists; even levels as low as 3.5 µg/dL have been correlated with measurable cognitive impairment in children [10, 20].

ii. Arsenic (As):

Arsenic is commonly released during the processing of gold and copper ores. Sources of exposure include drinking contaminated water and breathing particulate dust, as well as using arsenic-contaminated groundwater for irrigation and domestic purposes [21]. Chronic exposure may cause cardiovascular disease, impaired function of mitochondrial enzymes, oxidative stress, genotoxicity, and increased risk of various cancers [22, 23]. Children are highly vulnerable, with neurodevelopmental and cardiovascular effects reported at relatively low environmental levels.

iii. Mercury (Hg):

Elemental mercury vapor is the major route of exposure in ASGM. Mercury is highly neurotoxic, crosses the blood–brain and placental barriers, and impacts neurodevelopment in the basal ganglia, cerebellum, and developing fetus [24]. In aquatic ecosystems inorganic mercury may be converted into methylmercury by microorganisms, bioaccumulating in fish and resulting in cardiovascular and neurodevelopmental effects in local populations that eat contaminated fish. ASGM miners typically have significantly higher blood mercury levels, and clinical signs are tremor, ataxia and memory loss [25].

iv. Cadmium (Cd):

Cadmium is a common contaminant in zinc and lead ore processing and enters the body via both ingestion and inhalation. It binds to metallothioneins, accumulates preferentially in the kidneys and bones, with a renal cortex half-life of 10–30 years. Cadmium exposure causes proximal tubules apoptosis, proteinuria, osteomalacia and bone demineralization (a syndrome historically called Itai-itai disease). A biomonitoring study in the Kabwe area of Zambia documents high environmental cadmium concentrations and a high prevalence of kidney dysfunctions among the local residents [26, 27].

v. Chromium (Cr):

The main source of exposure to hexavalent chromium (Cr(VI)) is chromite mining and ferrochrome smelting [28]. Inhaled Cr(VI) is classified as a Group 1 pulmonary carcinogen by the International Agency for Research on Cancer (IARC) and is associated with respiratory tract irritation, skin ulceration, nasal septum perforation, and renal toxicity[29]. Occupational exposure to Cr(VI) has been linked with an increased risk of lung cancer at an OR of 1.32 in a SYNERGY pooled case-control study [30]. In Pakistan and Iran, environmental studies have shown that drinking water sources contain Cr(VI), Cd, Pb and Ni in concentrations above the WHO's guideline values, which are currently a health threat to people living in the vicinity of mining areas [31].

Table 1: Heavy metals in the mining environment: sources, exposure routes, and target organs

Heavy metal	Common mining source	Major exposure route	Main target organs / systems	Key health effects
Lead (Pb)	Ore dust, smelting emissions, tailings, lead-rich waste rock	Inhalation and ingestion	Brain, bone marrow, kidneys, cardiovascular system	Neurotoxicity, anemia, hypertension, renal dysfunction, abdominal pain, and cognitive impairment in children [10, 32]
Mercury (Hg)	Artisanal and small-scale gold mining (ASGM), amalgam burning, cinnabar mining	Inhalation of vapor, ingestion of contaminated fish, dermal contact	Central nervous system, kidneys, and developing fetus	Tremor, memory loss, renal injury, fetal neurotoxicity, and acrodynia [24, 33]
Arsenic (As)	Gold and copper mining, acid mine drainage, contaminated groundwater	Ingestion of contaminated water/food and inhalation of dust	Skin, liver, lungs, bladder, nervous system, cardiovascular system	Skin lesions, neuropathy, gastrointestinal toxicity, cardiovascular effects, and lung/bladder cancer [22, 23]
Cadmium (Cd)	Zinc and lead smelting, phosphate mining, coal mining, mine tailings	Inhalation and ingestion of contaminated food/water	Kidneys, bones, and lungs	Proteinuria, proximal tubular dysfunction, osteomalacia, Itai-itai disease, and pulmonary toxicity [34, 35]
Hexavalent Chromium Cr(VI)	Chromite mining and ferrochrome smelting	Inhalation and dermal contact	Lungs, nasal septum, skin, and kidneys	Asthma, respiratory irritation, skin ulcers, nasal septum perforation, renal toxicity, and lung cancer [30]

2.3. Environmental and Community Implications

Mining communities suffer from a double burden – metal emissions into the atmosphere and long-term exposure to contaminated water and soil. Arsenic, lead, cadmium, and zinc are released into the environment from tailings, waste rock, smelting emissions, and ore processing and AMD [36]. Community-level exposures occur through inhalation of contaminated dust, incidental ingestion and dermal contact (especially in ASGM settings), ingestion of contaminated clothing, water, crops, and bioaccumulated metals via fish [18, 37, 38]. Heavy metal toxicity disproportionately affects children, pregnant women, malnourished individuals, and informal miners [39]. Despite the phasing-out of leaded fuel, BLLs remain elevated in LMICs and are largely attributable to lead emissions from mining

and smelting [40]. These effects underscore the need for occupational health protection and community biomonitoring, environmental cleanup and public health education.

2.4. Mechanisms of Toxicity

Heavy metal induced cell damage mechanisms are complex and overlapping. One of the primary mechanisms is the excessive generation of ROS and decreased antioxidant defense [3, 41]. Arsenic and cadmium are potent inhibitors of the enzyme pathways involved in heme biosynthesis, namely delta-aminolevulinic acid dehydratase (ALAD), and also inhibit glutathione-dependent antioxidant pathways and thiol-containing enzymes [42]. The combination of these effects causes lipid peroxidation, protein oxidation, mitochondrial dysfunction and DNA damage. Differences in clinical toxicity can be attributed to metal-specific mechanisms.

- Mercury is known to pass the blood-brain barrier and placenta, and to be neurotoxic, especially to the developing nervous system [43].
- Cadmium deposits are present in the renal proximal tubules and result in tubular apoptosis, proteinuria and chronic nephropathy [44].
- Hexavalent chromium induces oxidative DNA damage and DNA double-strand breaks, which can be a factor in the development of lung cancer [45].
- Arsenic affects the activity of mitochondrial enzymes, forms genotoxic metabolites, promotes oxidative stress, and contributes to cellular dysfunction and increased cancer risk [46].

Toxicity is therefore evident and overlapping across multiple organs. The principal organ systems affected by these toxicants include the nervous system, kidneys, lungs, liver, and cardiovascular system [47]. These overlapping toxicological profiles underscore the importance of patient- and metal-specific pharmacological management strategies. Figure 1 illustrates the potential sources of exposure, pathways, mechanisms, and organ-specific effects.

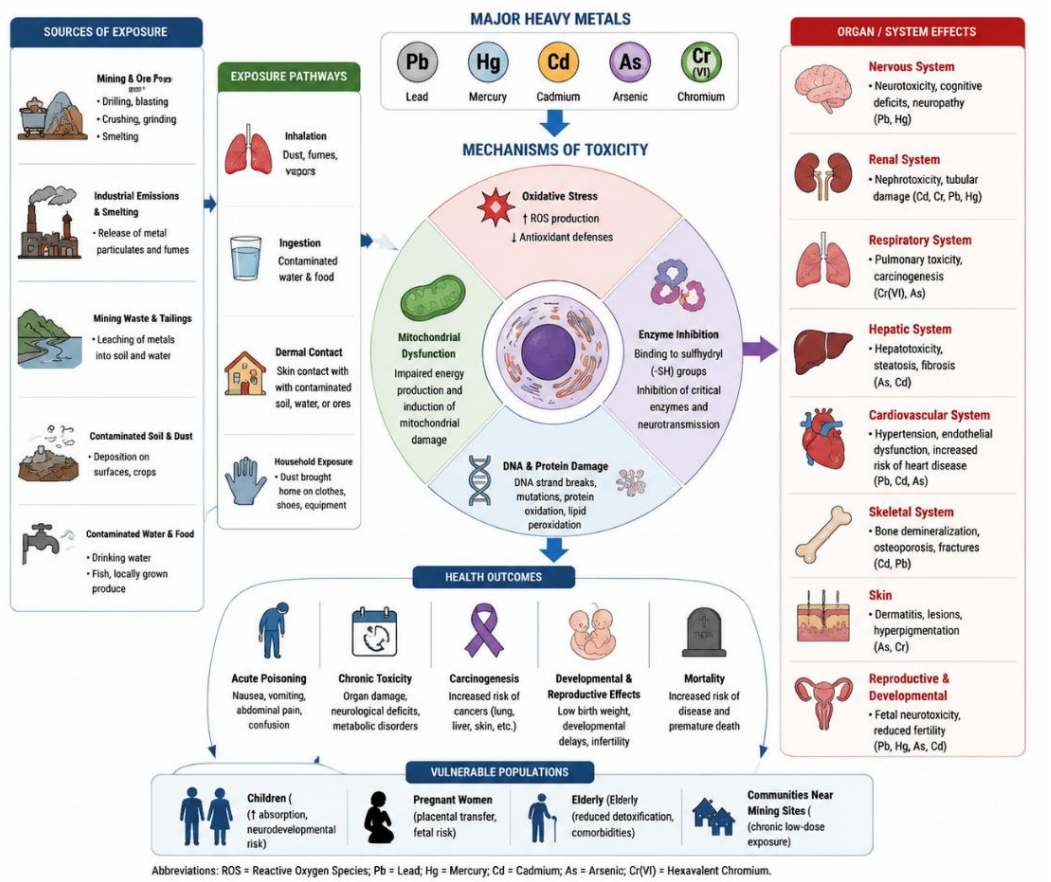

Figure 1: Sources, exposure pathways, mechanisms of toxicity, and organ/system effects of major heavy metals

3. Pharmacological Management of Heavy Metal Toxicity

The treatment of metal and metalloid poisoning includes removal from exposure, decontamination, supportive treatment, and selective chelation. Pharmacological treatment alone cannot offset ongoing occupational or environmental exposure. Treatment should be tailored to the specific metal, the chemical form, route and duration of exposure, clinical signs, validated biomarkers, comorbidities, and risk of further exposures. A consultation with clinical toxicologists or poison-control centers is recommended, especially when a chronic or polymetallic case is encountered [48, 49].

3.1. Chelation Therapy

Chelation is indicated for patients with clinically significant toxicity rather than all exposed individuals. Agent selection depends on the metal, chemical form, severity, timing, organ function, and patient tolerability.

Dimercaprol (British Anti-Lewisite, BAL): Administered parenterally for acute arsenic and inorganic mercury poisoning and, with $CaNa_2EDTA$, severe lead poisoning. Limited utility in chronic mercury toxicity; contraindicated in cadmium, iron, and selenium exposure, and in hepatic or renal insufficiency. Peanut oil is used in formulation, may be contraindicated in sensitive patients [50].

Calcium Disodium EDTA ($CaNa_2EDTA$): Used for severe lead poisoning, used alone or with BAL or succimer. Must be distinguished from Na_2EDTA, as inadvertent use may cause fatal hypocalcemia. Renal function, electrolytes, and trace-element levels should be monitored.

Dimercaptosuccinic Acid (DMSA, succimer): FDA-approved for pediatric lead poisoning (≥45 µg/dL). Off-label use for arsenic or mercury requires specialist guidance. Does not cross the blood–brain barrier; it is not the sole therapy for lead encephalopathy. Hydration, renal and hepatic function, hematology, and hypersensitivity should be assessed.

Dimercaptopropanesulfonic Acid (DMPS): Not FDA-approved but used in some countries for arsenic and mercury exposures. Increases urinary excretion, but effectiveness varies with metal species, exposure pattern, and established organ damage. Limited Central Nervous System (CNS) penetration; does not reverse established neurotoxicity [48].

Polymetallic exposures require caution, as chelators may alter the distribution or excretion of co-occurring metals. Treatment should be guided by a toxicologist with repeated clinical and laboratory assessment to minimize nephrotoxicity and essential-element depletion.

3.2. Adjunctive Antioxidant and Supportive Therapies

One of the most important mechanisms of cellular damage in toxic-metal exposure is oxidative stress, which can lead to organ damage and neurotoxicity. Adjunctive antioxidants have been explored as a way to reduce these effects, but they are not a substitute for avoiding exposure, supportive care, or clinically indicated chelation [51].

N-Acetylcysteine (NAC): Replenishes intracellular glutathione, scavenges reactive oxygen species (ROS), and modulates redox signaling. Some clinical and preclinical studies indicate protection from oxidative injury induced by arsenic, lead, and mercury. Oral and well-tolerated, NAC is already established in other clinical indications and has been used in clinical practice. It should be used with caution as an adjunct to existing therapies, although efficacy in systemic metal removal has not yet been proven [52].

Vitamins C and E: Reduce markers of oxidative stress in small human and preclinical trials of exposure to lead or cadmium. Vitamin C is able to reduce hexavalent chromium (Cr VI) to Cr III in vitro and also decrease the generation of ROS, although its efficacy in systemic toxicity by Cr VI has not yet been proven. These vitamins are not therapeutic antidotes, but rather nutritional or investigational adjuvants [53].

Alpha-Lipoic Acid (ALA): Antioxidant, anti-apoptotic, and anti-metal binding activities seen in preclinical studies in cadmium models; clinical data in human use are unavailable; it is regarded as investigational [51].

Further supportive therapy is needed and is dependent on the organs involved and clinical complications. This includes fluid and electrolyte replacement, seizure control, stabilization of the respiratory and cardiovascular system, renal and hepatic support, and correction of anemia and nutritional deficiencies. Essential elements lost to chelation therapy should be replaced during chelation therapy with mineral supplementation. Table 2 provides a summary of medical treatments, such as chelators and complementary antioxidant drugs, their indications, routes of administration, considerations and references.

Table 2: Pharmacological treatments for heavy-metal toxicity: chelators and adjunctive therapies.

Therapy	Indications	Route/ Formulation	Key Notes / Considerations
Dimercaprol (BAL)	Acute arsenic & inorganic mercury poisoning; severe lead (with $CaNa_2EDTA$)	Parenteral	Avoid in cadmium, iron, selenium exposure; caution in patients allergic to peanut oil
Calcium Disodium EDTA ($CaNa_2EDTA$)	Severe lead poisoning, alone or with BAL/DMSA	IV/IM	Monitor renal function, electrolytes, trace elements; distinguish from Na_2EDTA to avoid hypocalcemia

Dimercaptosuccinic Acid (DMSA / Succimer)	Pediatric lead ≥45 µg/dL; off-label arsenic or mercury	Oral	Does not cross blood–brain barrier; monitor renal/hepatic function; not sole therapy for lead encephalopathy
Dimercaptopropanesulfonic Acid (DMPS)	Arsenic and mercury exposures (not FDA-approved in some countries)	Oral/IV	Limited CNS penetration; effectiveness varies with metal species and established organ damage
N-Acetylcysteine (NAC)	Adjunctive therapy to reduce oxidative stress	Oral/IV	Replenishes glutathione, scavenges ROS; adjunct only, not a chelator
Vitamins C & E	Adjunctive therapy to reduce oxidative stress	Oral	Nutritional support; vitamin C reduces Cr(VI) to Cr(III) in vitro; not therapeutic antidotes
Alpha-Lipoic Acid (ALA)	Antioxidant and potential metal-binding activity (preclinical)	Oral	Investigational; no human trials yet

3.3. Emerging Pharmacological Approaches

Standard chelators are not effective in reaching cells and the central nervous system and may also have the potential to deplete essential elements from the body. Researchers are investigating targeted delivery systems to address these challenges. Preliminary experiments indicate that nanoparticle-based strategies could enhance the effectiveness of chelators. Polymeric nanoparticles, lipid-based carriers, or protein nanoparticles can generate higher bioavailability, greater stability, targeting of chelators to specific tissues or lower general toxicity [54].

Encapsulation of the drug MiADMSA in solid lipid nanoparticles (Nano-MiADMSA) is one such possible approach. In animals, this formulation was more effective in arsenic removal from blood and target organs and more effective in improving the activity of antioxidant enzymes than MiADMSA alone. These enhancements were also reflected in improved neurobehavioral function in arsenic-exposed rats, which implies that nano-formulations might also overcome some of the most vital constraints of classic chelation therapy [55].

Plant-derived compounds like curcumin, silymarin, and quercetin have been reported to have a protective effect in experimental models of heavy-metal toxicity, including when used with nanoparticles. All these compounds have antioxidant effects, anti-inflammatory effects, and liver- and kidney-protective effects by modulating pathways such as Nrf2, upregulation of metallothionein, and inhibition of apoptosis [53].

These are promising results that still need to be applied to clinical practice in humans. Their effects are difficult to predict in people because they vary in formulation, bioavailability, dose, and study design. They should be further evaluated in controlled human trials before being recommended for general use. Therefore, when designing novel nanocarriers or plant-based auxiliary agents for targeted therapy to the CNS, attention must be paid to minimizing systemic toxicity and considering their synergistic effect with conventional chelation therapy, particularly in cases of chronic or multi-metal exposure.

4. The Pharmacist's Role

Pharmacists can play a role in prevention, early recognition, referral, and clinical management of heavy metal poisoning in remote mining communities. Access to pharmacies is variable in rural and geographically isolated areas, but community pharmacies could act as a key point of contact for miners and their families [56]. In addition to medication distribution, pharmacists can perform initial risk assessment for exposure, review medication use, offer counseling, and help with referral, tracking, and communication with other healthcare professionals and patients [57, 58].

Heavy-metal exposure programs through pharmacists have not, however, been well evaluated in mining populations. These activities should be viewed as an evidence-based extension of an existing scope-of-practice model rather than a fully validated service model. Their implementation requires toxicology training, sufficient resources, clear scope-of-practice guidelines, standardized referral pathways and will entail working alongside physicians, toxicologists, laboratory staff, occupational-health specialists and public health officials [59]. Figure 2 provides a conceptual overview of the pharmacist-centered workflow for managing heavy-metal exposure in

mining communities.

4.1. Early Detection and Risk Identification

Heavy-metal poisoning can develop insidiously, with initial nonspecific symptoms such as fatigue, headache, abdominal discomfort, anemia, tremor, cognitive changes, peripheral neuropathy or impaired renal function [60]. Relevant information covers miner's exposure type, duration, metals used, frequency of exposure, use of personal protective equipment, workplace hygiene, and potential contamination of food, drinking water, soil, clothing, or the home environment [6, 61]. Elemental mercury is a specific concern because it is used in artisanal and small-scale gold mining and the mercury vapor released by heating amalgamated gold may pose a threat to miners, family members, and people who live close to the processing area [33, 62].

Medication reconciliation is also important because adverse drug reactions, nutritional deficiencies, and underlying illnesses may resemble metal toxicity. Pharmacists should review prescription medicines, over-the-counter products, herbal preparations, and other substances used by the patient. They should also ask about non-occupational exposure sources, including contaminated food or water, traditional remedies, cosmetics, and mercury-containing skin-lightening products [63].

Patients with a credible exposure history, compatible symptoms, or membership in a high-risk group should be referred for clinical assessment and appropriately selected biomonitoring. Biomarker selection depends on the suspected metal, its chemical form, exposure route, and time since exposure [14]. Blood lead concentration is used to assess lead exposure, whereas urinary arsenic is generally more informative than serum arsenic because arsenic is rapidly cleared from the bloodstream. Urinary mercury is commonly used to assess exposure to elemental or inorganic mercury, while hair mercury may provide information about longer-term methylmercury exposure [48]. Results must be interpreted together with the exposure history, clinical findings, dietary sources, and limitations of the selected biomarker.

Children and pregnant women require particular attention because exposure during development may cause long-term harm. The blood lead reference value of 3.5 µg/dL identifies children whose concentrations are higher than those of most children in the United States; it is intended to guide follow-up and exposure prevention rather than define toxicity or determine the need for chelation [64]. Mercury can cross the placenta and reach the developing fetus. Pregnant women and children with suspected exposure should therefore be referred promptly for clinical assessment, source identification, and appropriate testing [48].

4.2. Pharmacist Support in Chelation Therapy and Treatment Monitoring

Removal of the exposure source and provision of supportive care are the foundations of heavy-metal poisoning management. Chelation therapy should be reserved for confirmed or strongly suspected clinically significant poisoning and initiated by, or in consultation with, a clinician experienced in medical toxicology [65]. As outlined in Section 3, chelator selection depends on the metal involved, severity and timing of exposure, patient age, and organ function. Chelation does not replace exposure control and may cause harm when used for nonspecific symptoms or invalidated laboratory results. Within a multidisciplinary team, pharmacists play a critical role in verifying chelator selection, dose, route, treatment schedule, weight-based calculations, contraindications, interactions, and monitoring requirements [66]. Hospital pharmacists may also prepare parenteral chelators, assess formulation compatibility and stability, and support safe administration according to institutional protocols and specialist toxicology recommendations.

Monitoring should be tailored to the toxic metal, chelating agent, length of treatment and clinical condition. Laboratory tests that may be indicated include a complete blood count, renal and hepatic function tests, electrolytes, urinalysis, neurological and gastrointestinal symptoms, and serial blood and urinary metal levels [67]. Because of its potential for nephrotoxicity, calcium disodium EDTA should be monitored closely in the kidney. Succimer can produce gastrointestinal effects, increased liver enzymes, skin reactions and occasionally hematological changes. Chelation can also cause greater loss of important elements such as zinc, copper, iron, and calcium. These elements should be evaluated as clinically indicated and supplementation should not be routinely given. Any abnormal lab values, neurologic or renal symptoms that get worse, treatment intolerance, or poor results should be reported to the treating clinician promptly [68].

4.3. Patient Counseling and Community Pharmacy Practice

Pharmacist counseling should focus on the importance of preventing further exposure rather than simply relying on the drugs. Pharmacists can support the occupational-health recommendations for the use of personal protective equipment, ventilation, hand hygiene, safe handling of chemicals, and preventing take-home contamination from work clothing, shoes, tools, dust, or equipment [6, 61].

Adherence, adverse effects, warning symptoms, and laboratory and clinical follow-up should be discussed with patients receiving treatment. Pharmacists should also provide information that adequate iron, calcium and zinc intake may be supportive if there is a deficiency but not a substitute for exposure control, biomonitoring or medically recommended treatment. Structured exposure questionnaires are available for use by community pharmacists to identify high risk miners, pregnant women, children and households in close proximity to processing and waste disposal areas. They can assist with referrals, follow-up recommendations, and provide targeted education on household contamination and safer work practices. In hospital and clinical settings, the pharmacist can assist in access to chelators, double checking doses, tracking any toxicity associated with therapy, and communication with the physician, lab, and public health services. Pharmacists can also be involved in adverse event reporting, occupational health campaigns, exposure registries, and local referrals. However, there is limited direct evidence of pharmacist-initiated heavy-metal programs in mining communities. Future research should assess their

feasibility, clinical effectiveness, cost-effectiveness, acceptability and impact on treatment outcomes and referrals completed [69].

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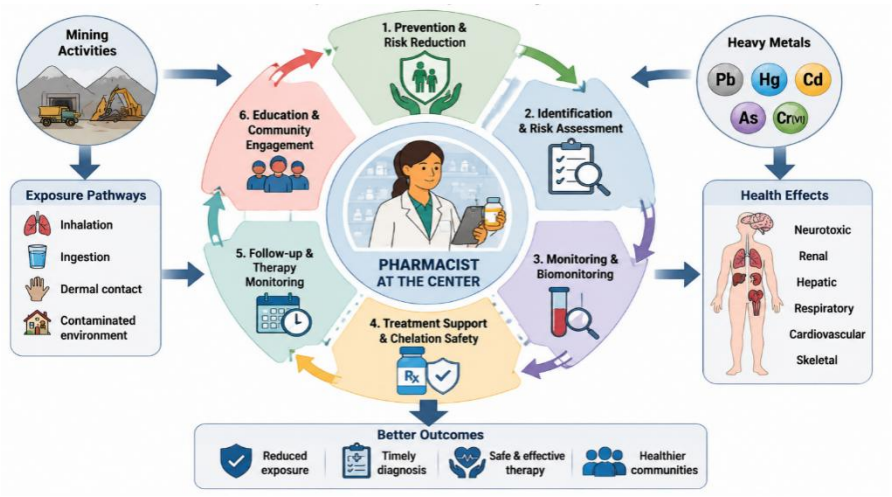

Figure 2: Pharmacist's role in the prevention, identification, management, and follow-up of heavy-metal toxicity in mining communities.

5. Prevention and Community-Level Interventions

5.1. Environmental and Occupational Controls

To prevent heavy metal toxicity, it is important to manage the contamination sources. Environmental interventions encompassed the secure containment of tailings, prevention and treatment of acid mine drainage, stabilization or rehabilitation of contaminated land, dust control, and periodic monitoring of soil, air and downstream water quality. These measures are necessary to prevent re-exposure when contamination is present in the workplace or environment where the person is working. The hierarchy should be used to determine the order of occupational controls to be used – that is, to eliminate the hazard, substitute it with a different material or method, and/or engineer the control. Examples of relevant interventions are wet drilling and other dust-suppression techniques, local exhaust ventilation, separation of processing from residence areas, facilities for hygiene in the workplace, appropriate respiratory protection and periodic biological monitoring of exposed workers. Reduction of mercury usage is especially critical for artisanal and small-scale gold mining operations. Another mercury-free processing method involves gravity concentration and subsequent borax smelting which could limit mercury exposure in appropriate environments. However, their effectiveness depends on the characteristics of the ores, technical skills, availability of equipment, economic viability, and acceptance by miners and local communities [70, 71].

5.2. Household Protection and Health Education

Mining contaminants can be introduced into homes in the following ways: work clothing, footwear, tools, dust, soil, water, and food. Children may be at greater risk from take-home exposure due to frequent hand-to-mouth contact and exposure to contaminated floors or soil. Workers should change contaminated clothes and boots before entering living areas, wash exposed skin after work hours, keep tools away from home, and not wash heavily contaminated work clothing with household laundry. Other protective measures are washing hands before eating, wet cleaning not dry sweeping contaminated surfaces, covering or replacing contaminated soil if pos sible, using safe water sources and keeping children away from mine waste, tailings, ore processing, and contaminated soil [24]. Community education should be designed to fit local mining practices, languages, literacy and resources. Practical steps to reduce exposure in the workplace and home can be reinforced by community health workers, schools, mining cooperatives, local authorities, and healthcare workers. Education programs should also clarify the indications for medical evaluation and when that is necessary or when a medical evaluation has been performed, and where appropriate testing and referral is available.

6. Challenges, Limitations, and Future Directions

There is a lack of evidence to support the management of heavy-metal toxicity in mining populations because most chelation studies are conducted in acute poisoning, childhood lead exposure, or non-mining occupational settings. This is due to their uncertain use for chronic, recurrent and polymetallic exposure in mining communities. Concurrent exposure to lead, mercury, arsenic, cadmium and other metals may also make the interpretation of biomarkers, toxicity assessment, selection of chelators and response to treatment more complicated. Limited laboratory capacity, scarcity of trained workers, restricted access to toxicologists, variable availability of diagnostic materials and chelators, treatment expenses, and lost to follow-up in remote mining areas further restrict management. However, pharmacist participation can be limited due to poor toxicology education, lack of access to patient information, resource shortages, and regulatory uncertainty. Future research should focus on studies

specifically targeting miners, pragmatic trials, validated pharmacist-assisted screening and referral processes, and better biomarkers for mixed-metal exposures. Other practical models, including the use of mobile clinics, teletoxicology, community-based specimen collection and integrated systems of pharmacy–laboratory referral, should also be assessed for safety, feasibility, cost-effectiveness and acceptability. Expansion of pharmacists' roles should involve formal education, clinical protocols, regulatory approval, and inter-professional collaboration.

6. Conclusion

Heavy metal exposure in the context of mining communities is a complex occupational and public health issue that demands coordinated interventions at the environmental, occupational, household, and clinical levels. Successful management begin with defining and controlling the source of contamination, then with appropriate biomonitoring and supportive care and medically supervised chelation if clinically warranted. Pharmacists can contribute to early assessment of exposure risk, medication review, patient counseling, referral, chelation-safety review, treatment monitoring, and through patient and health care professional communication. Their accessibility may be particularly valuable in remote mining areas where specialist toxicology and occupational-health services are limited. However, pharmacist-led services should be assessed directly in mining communities and should be contained within well-defined regulatory and inter-professional boundaries. Secure waste management, mercury and dust control, safer processing, household protection, easy access to testing, efficient referral system, and long-term health monitoring will likely be the greatest contributors to disease burden reduction. Occupational and environmental controls cannot be replaced by pharmacological measures, but pharmacological interventions remain important for clinically significant poisoning.

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