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Impact of environmental arsenic exposure on human health - a narrative review

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Abstract

Background. Arsenic is a naturally occurring metalloid known for its toxicity. Due to industrial activity and environmental pollution, human exposure, mainly through contaminated water and food, has become a global health problem.

Aim. This review aims to present the main sources of arsenic exposure, its mechanisms of toxicity, and the clinical features and management of poisoning.

Material and methods. A narrative review of the literature was conducted, focusing on environmental exposure, pathophysiology, and clinical aspects of arsenic toxicity.

Results. Arsenic enters the body mainly through ingestion and inhalation, with groundwater and rice being key sources. The inorganic forms, especially trivalent arsenic, are the most toxic. After absorption, arsenic affects multiple cellular processes, leading to oxidative stress, enzyme inhibition, and impaired energy production. Acute poisoning typically presents with gastrointestinal symptoms, while chronic exposure leads to multisystem damage involving the skin, nervous, cardiovascular, and endocrine systems, and increases cancer risk. Diagnosis is based on

history and arsenic levels in urine or hair. Treatment includes chelation therapy and supportive care.

Conclusions. Arsenic exposure remains an important but often overlooked health risk. Early recognition and limiting exposure are crucial, and prevention should remain a priority in affected regions.

Keywords: arsenic, environmental exposure, groundwater contamination, rice contamination, arsenic toxicity, arsenicosis, oxidative stress, enzyme inhibition, acute poisoning, chronic exposure, skin lesions, carcinogenesis, chelation therapy, public health

1. Introduction

Arsenic is a chemical element classified as a metalloid, a substance which has properties of metals and nonmetals. In nature arsenic occurs in organic and inorganic forms. These forms occur naturally or due to human activities such as mining or smelting of metals, combustion of fossil fuels and emission of industrial waste containing arsenic. There are over 245 minerals, that contain arsenic, which occur all around the world [1]. This element exists mainly as trivalent arsenic (As^{3+}) and pentavalent arsenic (As^{5+}) which are the most common inorganic forms in the environment [2], [3]. Trivalent form has higher toxicity than pentavalent [3]. This metalloid can occur also in oxidation states of -3 and 0 [4]. The two major ways which arsenic enters the body are through the digestive and respiratory system. Approximately 95% of trivalent arsenic is absorbed through the gastrointestinal tract, significantly less is absorbed through the respiratory system. After absorption, 95% to 99% of the arsenic accumulates in erythrocytes and after that is transported to

other organs of the body. Most of the absorbed arsenic is converted by the liver to methylated form, which is less toxic than primary form and is eliminated in the urine [2]. Hundreds of millions people around the world are exposed to arsenic mainly through contaminated water and food, but also through occupational exposure. Due to arsenic-contaminated ground water and drinking water occurs in many countries mainly in southern Asia but also in Central and South American countries. Significant amounts of arsenic in groundwaters was also found in the southwest regions of the USA and some countries in Europe such as Serbia. This frequent exposure to arsenic leads to serious health problems due to its toxic effect mainly on skin, nervous, respiratory, cardiovascular and endocrine systems and even in some cases can induce cancer disease [5]. The aim of this review is to summarize the known pathogenesis of the arsenic toxicity, the possible exposure routes, clinical manifestations of the intoxication, diagnostic methods and treatment options.

2. Arsenic presence in environment

Arsenic (As) is a chemical element from group 15 of the periodic table, with its chemical properties being similar to the phosphorus [6]. Arsenic is a chemical element that occurs naturally in the Earth's crust, groundwater and also in seafood. Arsenic is naturally released to the environment due to processes such as volcanic activity, geothermal activity and rock-weathering. The most important sources of this toxic metalloid for people around the world are water, soil and food products, which are contaminated with arsenic due to increasing human activities connected with industry such as mining, smelting metals, fossil fuel combustion, and waste emission [3] [4]. Other include fish, which absorb the arsenic from water, with the levels of this metalloid in their meat reaching high levels [6]. Seafood mainly contains organic forms of arsenic, while inorganic forms are present only in small amounts. Organic arsenic compound, called arsenobetaine, occurs mainly in ray-finned fish, whereas in mussels dominate arseonsugars [7]. Inorganic arsenic valency can occur, depending on the redox state of the soil, as a trivalent arsenic (arsenite, As^{III}) and as a pentavalent arsenic (arsenate, As^{V}). Trivalent form dominates in anaerobic environment such as flooded fields and pentavalent dominates in aerobic soils [8]. Arsenic contamination, mainly in groundwater, has been noted on almost every continent in the world. More than 230 million people worldwide are at high risk of chronic exposure to arsenic, with about 180 million of them living in Asian countries [9]. Elevated arsenic concentrations in groundwater have been recorded in some countries such as Bangladesh, India (state of West Bengal), Vietnam, Argentina, Chile, China,

Hungary, Mexico and also in the USA [2]. As a result, arsenic contamination of groundwater has been recognized as a serious public health problem worldwide due to its high toxicity to humans [9]. Because groundwater is the main source of drinking water, elevated levels of this metalloid above 10 μg/l in the ground induce chronic multi-systemic disease which is called arsenicosis [2]. Only in Bangladesh approximately 5,6 % of all deaths in this country are the result of chronic exposure to arsenic [9]. The main source of human exposure to arsenic is rice and rice-based products, because rice has the highest capacity to accumulate arsenic among the grains [10]. The problem of arsenic accumulation in rice plants is a serious global issue, as rice grains, rice milk, and rice bran are consumed by millions of people from infants to adults worldwide. The amount and forms of arsenic in grains depend on the irrigation system, geographical location and environment, rice genotypes, and the method of rice grain processing. Studies have shown that arsenic concentrations may vary, even within a single field, with the highest concentrations found near the irrigation water inlet [11]. The maximum acceptable values of inorganic arsenic for human consumption is 0.2 mg/kg for adults and 0.1 mg/kg for infants and children [12]. As a result of occupational activity, exposure to arsenic can occur in various types of human activity. The most common ones include production of non-ferrous metals (without aluminum), metal mining and smelting, agriculture, glass manufacturing and other activities related to industry [13]. The less common way of arsenic exposure is through herbicides, as well as through contaminated alcoholic beverages, such as beer, wine or whiskey [14]. The occupational hazard of the arsenic exposure includes professions such as miners, carpenters (wood preservatives), farmers (herbicides, pesticides), and factory workers (especially glass, semiconductors and lasers) [14], [15].

3. Arsenic exposure pathophysiology

Arsenate, due to their structural similarity to phosphates, enters cells via phosphate transporters, while Arsenite enters through aquaglyceroporins (AQP) and glucose transporters (GLUT) [2]. Ingested arsenic compounds travel from the digestive tract into the bloodstream, where they bind to erythrocytes [16]. Arsenic can spread all over the body, with an ability to pass the blood/brain barrier into the brain. The unmetabolized Arsenic is extremely toxic because it accumulates in tissues more easily [17]. When As reaches the liver, it undergoes a methylation process. Firstly, Arsenate (As^{5+}) is reduced by thioredoxin (Trx) / trx reductase (Th) system to arsenite (As^{3+}). During next pathway As^{III} is methylated twice by arsenic methyltransferase (AS3MT) and by

glutathione *S*-transferase Ω (GSTO) into dimethylarsenic acid (DMA), which is much less toxic and can be easily excreted by urine [18]. Trivalent arsenic is regarded as a more toxic compound, with some studies showing monomethyloarsonous acid (MMA) being more toxic to cells than arsenite [19]. Its forms, MMA and dimethyloarsonous acid (DMA), have a big affinity to sulfur-containing enzymatic parts, such as thol- and sulfhydryl groups. Moreover, it reacts easily with glutathione and cysteine. As a result, enzymes, such as pyruvate dehydrogenase, or glutathione reductase are inhibited, leading to the depletion of intracellular ATP, increase of the intracellular free radicals, eventually leading to increase of the concentration of the free radicals, which in turn damage the membranes, as well as DNA [20]. Pentavalent arsenate resembles chemically phosphorus atoms, therefore being able to replace it in the organic compounds. In cells, arsenate can react with glucose, creating glucose-6-arsenate, which in turn can inhibit hexokinase [21]. Moreover, it can disrupt the production of ATP in all tissues, especially in erythrocytes, by substituting phosphorus during the glycolysis, creating unstable intermediates, which in turn break down and halt the process, depleting the cells of their ATP [19]. Arsenic is toxic not only directly by accumulating in tissues, but also indirectly through the consequences of its metabolism. Those pathways lead to the depletion of compounds such as *S*-adenosylmethionine (SAM) or glutathione and accumulation of S-adenosylhomocysteine (SAH). The lack of SAM is one of the causes of hypomethylation of the genetic material, which with the reactive oxygen species leads to DNA damages, protooncogenes expression, which eventually leads to carcinogenesis [22].

4. Signs and symptoms of arsenic exposure

After the ingestion of the arsenic and liver metabolism, around half of the ingested dose is excreted in the urine, with this process taking approximately 3 to 5 days. A small portion of inorganic arsenic is also eliminated from the body in its unchanged form. Studies have shown that after poisoning, the highest concentrations of arsenic are found in organs such as the liver and kidneys. As a result of chronic arsenic exposure, this metalloid accumulates mainly in liver, kidneys, lungs and heart while to a lesser extent in muscles, spleen, nervous systems and digestive tract. After about two weeks of consumption, arsenic accumulates in tissues containing keratin- hair and nails. Arsenic levels ranging from 0.1 to 0.5 mg/kg measured in hair signify chronic poisoning, and levels from 1.0 to 3.0 mg/kg acute intoxication [23].

Arsenic induces oxidative stress, mitochondrial damage, enzyme disruption and impairs signaling pathway in neurons, leading to a variety of neurological disorders such as encephalopathy, neuropathy, impaired verbal comprehension and memory problems [2]. This metalloid is also associated with other neurological disorders, such as Alzheimer's and Parkinson's disease, through its ability to form plaques and penetrate the substantia nigra [17]. Arsenic impact on the liver is mostly caused by ROS (Reactive Oxygen Species). ROS damages lipids, proteins and DNA, which induce death of hepatic cells. Besides hepatotoxicity, arsenic can also damage kidneys, which is called arsenic-induced nephrotoxicity. It is the result of arsenic related podocyte damage and dysfunction of endothelial cells. By inducing oxidative stress, which also causes pancreatic cells disfunction, arsenic leads to insulin resistance and decreased insulin synthesis. All of these mechanisms contribute to Diabetes mellitus development. Arsenic exposure can also induce hypertension due to decreased production and bioavailability of nitric oxide which causes vasoconstriction and leads to high blood pressure. Studies have shown that chronic arsenic exposure can cause infertility. Arsenic causes changes in hormone levels and disruption of a variety of reproductive processes in the human body, such as spermatogenesis and oogenesis. Skin changes have been noticed at arsenic concentrations in contaminated water ranging from 50 to 100 µg/L [24]. The earliest cutaneous manifestations of chronic arsenic exposure are pigmentary changes. The most common skin change among patients with arsenicosis was raindrop pigmentation, which generally appears on the chest, back of trunk and extremities as spotty hyperpigmentation. Less common skin manifestations include: leukomelanosis and keratosis [2]. About one month after chronic arsenic exposure, skin lesions called Reynolds-Aldrich-Mees lines occur in about 5% of patients. They are white horizontal bands across the fingernails. Other dermatological symptoms include eczematoid lesions, warts and alopecia [4]. Anemia, leukopenia and thrombocytopenia occur usually in chronic arsenic intoxication, sometimes in acute poisoning [25] Studies show that arsenic exposure increases the risk of developing genitourinary, lung, liver and skin cancers. Conversely, the correlation between arsenic exposure and the induction of liver, prostate, and kidney cancer is limited [17]. Moreover, epidemiological studies revealed correlation between exposure to inorganic arsenic at high doses through drinking water and increased risk of bladder neoplasms. The probable cause of the correlation is the fact that arsenic is excreted mainly in urine. The most common arsenic-induced skin cancers are Bowen’s disease, Basal Cell Carcinoma (BCC), and Squamous Cell Carcinoma (SCC). Aforementioned skin changes, such as Bowen’s

disease, are early indicators that can develop into invasive cancers in other organs, emphasizing the need for early diagnosis and intervention [2].

Dermatology	**Oncology**	**Endocrinology**	**Cardiology**	**Nephrology**	**Gastroenterology**
Raindrop-like hyperpigmentation	Basal cell carcinoma	Hypothyroidism	Hypertension	Chronic kidney disease	Liver cirrhosis
Mees' lines	Squamous-cell carcinoma	Diabetes	Atherosclerosis		
Palmaro- plantar hyperkeratosis	Lung cancer	Infertility	Ischemic heart disease		
Eczema	Bladder cancer		Cardiac arrythmias		
Alopecia	Liver cancer		Cardiomyopathy		

Tab. 1 List of symptoms of chronic arsenic exposure

In addition to the long-term effects of arsenic exposure, it is worth mentioning the classic manifestations of acute arsenic poisoning. Typical acute arsenic toxicity manifests itself as gastroenteritis and hypotension, as a result of dehydration. The most common gastrointestinal symptoms of poisoning include abdominal pain, nausea, vomiting and diarrhea resembling rice water (cholera-like diarrhea). These symptoms appear within a few minutes to a few hours after arsenic ingestion, and usually resolve within approximately 12 hours, but might persist for up to several days after arsenic exposure. In addition to the gastroenteritis symptoms, signs from other organs may be seen. After arsenic inhalation, the symptoms include coughing, shortness of breath and chest pain. Usually hours to days after arsenic exposure proteinuria, hematuria and symptoms

of acute renal failure can be observed. Neurologic signs may occur typically 1-3 weeks after arsenic exposure, and include headaches, confusion, memory loss, seizures, and delirium. Loss of deep tendon reflexes, temperature and vibratory sensation have been reported in severe arsenic toxicity [4].

Gastrointestinal	**Cardiovascular**	**Pulmonary**	**Renal**	**Neurologic**	**Hematological**
Nausea	Hypotension	Pulmonary oedema	Proteinuria	Sensorineural polyneuropathy	Leukopenia
Vomiting	QTc prolongation		Haematuria	Loss of deep tendon reflexes	Thrombocytopenia
Cholera-like diarrhea	QRS widening		Acute renal failure	Headache	Anemia
Abdominal pain				Confusion	
				Memory loss	
				Seizures	

Tab. 2 Acute and subacute symptoms of arsenic poisoning

The diagnosis of the arsenic exposure relies on both medical history, as well as laboratory findings. To differentiate between chronic and acute intoxication, arsenic levels in urine and hair should be tested. Exposure can be confirmed at levels exceeding 50 µg/L in urine, with levels exceeding 1000 µg/L often occurring in acute intoxication. A 24-hour urine collection should be done to measure the arsenic levels throughout the treatment [26]. Treatment includes chelating agents, such as dimercaprol, 2,3-dimercapto-1-propanesulfonic acid, or 2,3-dimercaptosuccinic acid, until levels of arsenic in urine drop below 50 µg/L [20], [27].

To reduce exposure to toxic arsenic, established occupational exposure limits should be complied. In areas where there is a risk of excessive amounts of naturally occurring arsenic in groundwater, wells must be tested. Chromated- Copper- Arsenate (CCA) treated wood should not be burned in wood stoves and fireplaces [28]. It is also very important to avoid eating untested food, which may contain arsenic, such as rice, or chicken [29].

5. Conclusion

Arsenic is a naturally occurring metalloid, which contributes greatly to the man-made environmental pollution. Exposure to this element can occur through various ways, including occupational hazards, drinking water exposure, or eating food containing arsenic. With its diverse toxic properties, its impact on the human organism can be either acute, or chronic, including most of the tissues and organs. The knowledge about the symptoms and treatment of the intoxication should be a part of the physicians' practice, as arsenic exposure still haunts hundreds of millions of people worldwide.

Disclosure

Author Contributions:

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Methodology: Kacper Szada-Borzyszkowski, Konstancja Owczarenko

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AI: AI was utilized for two specific purposes in this research. Text analysis of clinical reasoning narratives to identify linguistic patterns associated with specific logical fallacies. Assistance in refining the academic English language of the manuscript, ensuring clarity, consistency, and adherence to scientific writing standards. AI were used for additional linguistic refinement of the

research manuscript, ensuring proper English grammar, style, and clarity in the presentation of results. It is important to emphasize that all AI tools were used strictly as assistive instruments under human supervision. The final interpretation of results, classification of errors, and conclusions were determined by human experts in clinical medicine and formal logic. The AI tools served primarily to enhance efficiency in data processing, pattern recognition, and linguistic refinement, rather than replacing human judgment in the analytical process.

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