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Impact of Long-Term Exposure to Waterborne Toxic Elements (Cd, As, Hg) on Renal Function and Oxidative Stress Biomarkers

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ABSTRACT

Background: Persistent exposure to toxic heavy metals through contaminated drinking water is now a critical global environmental and public health issue. Cadmium (Cd), arsenic (As), and mercury (Hg) are elements with high nephrotoxic properties and are able to induce oxidative stress, renal dysfunction, and progressive cellular injury after long-term exposure.

Objective: To assess the effect of chronic exposure to waterborne Cd, As, and Hg on renal function parameters and oxidative stress biomarkers in the chronically exposed population.

Methods: A cross-sectional analysis was performed from January 2025 to August 2025 involving 160 participants. The exposed group included 100 individuals with long-term consumption of contaminated groundwater, while 60 healthy individuals consuming treated water served as controls. Blood and urine samples were collected to measure serum creatinine, blood urea nitrogen (BUN), serum uric acid, estimated glomerular filtration rate (eGFR), urinary albumin, malondialdehyde (MDA), superoxide dismutase (SOD), catalase, and glutathione peroxidase (GPx). The heavy metal levels were assessed using atomic absorption spectrophotometry.

Results: Cadmium, arsenic, and mercury levels in blood were found to be significantly higher in the exposed individuals as compared to the control ($p<0.001$). The renal function parameters were significantly elevated serum creatinine, BUN, serum uric acid, and urinary albumin, in addition to significantly decreased eGFR among the exposed participants. The antioxidant enzymes such as SOD, catalase, and GPx were found to be significantly reduced, while MDA levels were found to be significantly increased in both groups ($p<0.001$). Heavy metal concentrations, renal function markers, and oxidative stress markers showed good correlation.

Conclusion: Waterborne toxic elements can be a significant cause of renal dysfunction and nephrotoxicity in chronically exposed populations, likely through oxidative stress.

Keywords: Cadmium, Arsenic, Mercury, Renal Dysfunction, Oxidative Stress, Heavy Metal Toxicity

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INTRODUCTION

The pollution of the environment with toxic heavy metals has become a great global public health concern, especially in the rapidly industrializing parts of the world, where the pollution of groundwater is still on the rise because of mining, industrial pollution, farm effluents, and waste disposal [1]. Cadmium (Cd), arsenic (As), and mercury (Hg) are regarded as highly hazardous among the environmental toxicants, as they are persistent in the environment, non-biodegradable, and can be stored in human tissues following chronic exposure. The chronic exposure to these toxic elements via contaminated drinking water is one of the major pathways of exposure, and the exposure dose is a major risk factor for chronic systemic diseases in the world [2].

Industrialization and urbanization have raised environmental pollution and heavy metal contamination in groundwater resources in several areas of Asia [3]. Cadmium, arsenic, and mercury have been linked to industrial discharges from mining, smelting, battery manufacturing, metal working industries, and coal combustion. People who live in industrial and mining regions have a higher risk of chronic exposure because they depend on contaminated groundwater sources for a long time [4].

Heavy metal toxicity is one of the primary target organs affected by the kidneys due to their essential function in the filtration, concentration, and elimination of toxic substances [5]. Cadmium is taken up mainly by the cells lining the proximal tubules, leading to mitochondrial dysfunction, cell death, and degeneration. Arsenic exposure is associated with endothelial dysfunction, oxidative DNA damage, inflammatory activation, and impairment of kidney blood flow, and mercury exposure causes glomerular and tubular injury via oxidative and immune-mediated mechanisms. Exposure to these harmful metals can cause a progressive decline in renal filtration and can speed up the progression of chronic kidney disease [6].

Oxidative stress is currently thought to be one of the key mechanisms involved in nephrotoxicity of heavy metals [7]. Increased generation of Reactive Oxygen Species (ROS) by toxic elements leads to lipid peroxidation, damage to mitochondria, protein oxidation, and cellular membrane damage. In parallel, antioxidant defense systems are depleted due to inhibition of the endogenous antioxidant enzymes such as superoxide dismutase (SOD), catalase, and glutathione peroxidase (GPx). Malondialdehyde (MDA), an indicator of lipid peroxidation, is often found to be increased among subjects exposed to chronic heavy metal toxicity and relates to the extent of oxidative damage to the cells [8,9].

Several epidemiological and experimental studies have shown a strong association between heavy metal exposure and renal dysfunction, which includes elevated serum creatinine, increased blood urea nitrogen, albuminuria, and reduced estimated glomerular filtration rate (eGFR) [10]. The combination of various nephrotoxicants can have additive and multiplicative effects, worsening the oxidative stress and renal dysfunction. However, there are still few data on the effects of combined exposure to cadmium, arsenic, and mercury on biomarkers of oxidative stress and renal function in people living in environmentally polluted areas [11]. Therefore, the present study was conducted to investigate the impact of long-term exposure to waterborne cadmium, arsenic, and mercury on renal function parameters and oxidative stress biomarkers among chronically exposed individuals.

MATERIALS AND METHODS

The study was a cross-sectional analysis, carried out at the Department of Public Health and Environmental Toxicology, University of South China, Hengyang, China, between January 2025 and August 2025. Ethical approval for the study was obtained from the Institutional Ethical Review Committee before commencement of data collection under reference number ERC/PH/2025/041.

A total of 160 participants aged between 25 and 65 years were enrolled through non-probability consecutive sampling. The participants were split into two groups: one with a history of exposure and another without a history of exposure. The exposed group consisted of 100 subjects living in areas where groundwater contamination had been documented, and who had drunk the local groundwater for at least five years. A control group of 60 healthy persons who drank treated municipal water not known to contain heavy metals.

To minimize the potential impact of confounding factors on renal and oxidative stress parameters, participants with a previous history of chronic kidney disease, diabetes mellitus, hypertension, chronic liver disease, autoimmune diseases, malignancy, occupational heavy metal exposure, smoking history, alcohol abuse, or current antioxidant supplementation were excluded.

A detailed questionnaire was used for obtaining demographic data, residential history, duration of groundwater exposure, occupational history, dietary habits, and clinical history. Water samples were taken from the residential areas of the participants in sterile polyethylene containers and brought to the laboratory for determination of cadmium (Cd), arsenic (As), and mercury (Hg) levels by atomic absorption spectrophotometry.

Venous blood and spot urine samples were collected under aseptic circumstances after an overnight fast. Automatic biochemical analyzers were used to measure the amounts of serum creatinine, blood urea nitrogen (BUN), serum uric acid, and urine albumin. The CKD-EPI formula was used to calculate the estimated glomerular filtration rate (eGFR).

Following the manufacturer's instructions, commercially available enzyme-linked immunosorbent assay (ELISA) kits were used to measure the levels of oxidative stress indicators. Lipid peroxidation was indicated by the levels of malondialdehyde (MDA), and the antioxidant defense state was evaluated by measuring the activities of antioxidant enzymes such as glutathione peroxidase (GPx), catalase, and superoxide dismutase (SOD).

Under typical laboratory circumstances, flame atomic absorption spectrophotometry was used to measure the amounts of heavy metals in blood and water samples. To ensure measurement accuracy and reproducibility, laboratory analysis was performed using quality assurance techniques.

SPSS version 26.0 was used to input and analyze the data. Mean ± SD was used to represent quantitative values, whereas frequency and percentage were used to portray categorical information. Groups were compared using the independent sample t-test, and correlations between heavy metal concentrations, renal function measures, and oxidative stress biomarkers were evaluated using Pearson correlation analysis. P-values ≤ 0.05 were considered statistically significant.

RESULTS

The current research included 160 participants: 100 with long-term exposure to polluted groundwater and 60 healthy controls who drank treated municipal water. Participants in the exposed group had a mean age of 47.3±9.8 years, compared to 44.7±8.9 years for the control group. The exposed and control groups were 58% and 55% male, respectively. Exposure to contaminated groundwater lasted an average of 8.7±3.1 years. There was no significant difference in age, gender distribution, or BMI between the two groups ($p > 0.05$). Individuals exposed to harmful materials had significantly longer exposure times to groundwater compared to controls ($p<0.001$), indicating long-term environmental exposure. Demographic and exposure characteristics of the study subjects are shown in Table 1.

The blood levels of heavy metals showed that the concentrations of cadmium (Cd), arsenic (As), and mercury (Hg) were significantly higher in those exposed to the contaminated groundwater. The mean Cd concentration for the exposed group was 6.94±1.52 µg/L, while for the controls, the concentration was 1.68±0.47 µg/L. In the same manner, arsenic and mercury concentrations were also clearly elevated in exposed individuals, indicating high systemic exposures to toxic metals from chronic environmental exposure. The differences were all statistically highly significant, $p<0.001$. Blood heavy metal levels are presented in detail in Table 2.

Renal function assessment showed that there was significant impairment in the heavy metal-exposed group for a long period. Mean serum creatinine levels were significantly elevated in the exposed group (1.72±0.41 mg/dL) compared to controls (0.89±0.17 mg/dL). Another significant change noted in the exposed group was an increase in the levels of blood urea nitrogen and serum uric acid. Furthermore, estimated glomerular filtration rate (eGFR) was significantly reduced in exposed participants, while urinary albumin excretion was substantially increased, indicating both glomerular and tubular renal injury. The results indicate progressive nephrotoxic effects also observed with chronic exposure to cadmium, arsenic, and mercury. The renal function results are shown in detail in Table 3.

Evaluation of markers of oxidative stress showed that there was a high degree of oxidative imbalance in the group exposed to toxic metals. Mean malondialdehyde (MDA) levels were significantly elevated in the exposed group (5.96±1.21 nmol/mL) compared to controls (2.84±0.69 nmol/mL), indicating increased lipid peroxidation and oxidative cellular injury. However, antioxidant defense enzyme activities such as superoxide dismutase (SOD), catalase, and glutathione peroxidase (GPx) were significantly decreased in the exposed group. All these findings suggest that there is a decrease in the body's own antioxidant defense mechanism as a result of chronic exposure to toxic metals and continued oxidative stress. The detailed analysis of oxidative stress biomarkers is given in Table 4.

Pearson correlation analysis also revealed a strong positive correlation between blood cadmium, arsenic, and mercury concentration and serum creatinine, blood urea nitrogen, urinary albumin, and malondialdehyde concentration. Heavy metal concentration showed a significant inverse relationship with all the antioxidant parameters, such as estimated glomerular filtration rate, superoxide dismutase, catalase, and glutathione peroxidase activity. All the above results suggest that the rise in heavy metal load strongly correlates with the gradual deterioration of renal function and severity of oxidative stress. The detailed correlation results are summarized in Table 5.

Overall results of the present study revealed that the systemic accumulation of cadmium, arsenic, and mercury was significantly increased in people chronically exposed to contaminated groundwater in comparison to healthy controls. Chronic exposure to these toxic elements resulted in significant loss of renal function, including serum creatinine increase, blood urea nitrogen increase, hyperuricemia, estimated glomerular filtration rate decrease, and urinary albumin excretion increase. Moreover, the exposed subjects showed severe oxidative stress, as evidenced by a significant increase in malondialdehyde and a significant decrease in the activities of antioxidant defense enzymes such as superoxide dismutase, catalase, and glutathione peroxidase. The reported positive association of the heavy metal contents with renal dysfunction markers also suggests that a direct link exists between an increase in the toxic metal burden and a progressive nephrotoxicity and oxidative cellular damage in chronically exposed subjects.

Table 1: Demographic and Exposure Characteristics of Study Participants

Variable	Exposed Group (n=100)	Control Group (n=60)	p-value
Age (years)	47.3±9.8	44.7±8.9	0.118
Male Gender	58 (58%)	33 (55%)	0.711
Female Gender	42 (42%)	27 (45%)	0.711
Duration of Water Exposure (years)	8.7±3.1	2.1±0.9	<0.001
BMI (kg/m²)	25.4±3.6	24.8±3.1	0.291

Table 2: Blood Heavy Metal Concentrations Among Study Participants

Parameter	Exposed Group	Control Group	p-value
Cadmium (µg/L)	6.94±1.52	1.68±0.47	<0.001
Arsenic (µg/L)	35.8±9.1	8.5±2.6	<0.001
Mercury (µg/L)	5.37±1.18	1.31±0.42	<0.001

Table 3: Renal Function Parameters Among Study Participants

Parameter	Exposed Group	Control Group	p-value
Serum Creatinine (mg/dL)	1.72±0.41	0.89±0.17	<0.001
Blood Urea Nitrogen (mg/dL)	39.4±8.7	22.3±5.1	<0.001
Serum Uric Acid (mg/dL)	7.1±1.4	4.8±1.0	<0.001
eGFR (mL/min/1.73m²)	66.9±13.8	104.7±15.6	<0.001
Urinary Albumin (mg/day)	95.6±31.2	24.1±8.8	<0.001

Table 4: Oxidative Stress Biomarkers Among Study Participants

Biomarker	Exposed Group	Control Group	p-value
MDA (nmol/mL)	5.96±1.21	2.84±0.69	<0.001
SOD (U/mL)	1.87±0.44	3.92±0.76	<0.001
Catalase (U/mL)	21.9±5.4	42.6±8.9	<0.001
GPx (U/L)	36.8±8.7	71.3±12.1	<0.001

Table 5: Correlation Analysis Between Heavy Metal Concentrations and Biomarkers

Variable	Serum Creatinine	eGFR	MDA	SOD
Cadmium	r=0.71	r=-0.74	r=0.77	r=-0.69
Arsenic	r=0.66	r=-0.68	r=0.72	r=-0.64
Mercury	r=0.61	r=-0.65	r=0.69	r=-0.59
p-value	<0.001	<0.001	<0.001	<0.001

DISCUSSION

The present study demonstrated a significant association between long-term exposure to waterborne cadmium (Cd), arsenic (As), and mercury (Hg) and progressive renal dysfunction accompanied by severe oxidative stress [1]. Exposed persons had significantly higher blood levels of toxic metals and impaired renal function (serum creatinine, blood urea nitrogen, estimated glomerular filtration rate (eGFR), and urinary albumin excretion). Moreover, significant oxidative imbalance was detected among the exposed group, consisting of increased malondialdehyde (MDA) and decreased antioxidant defense enzymes (superoxide dismutase (SOD), catalase, and glutathione peroxidase (GPx)) [2].

The kidneys are very sensitive to heavy metal toxicity due to their important role in the filtration and elimination of toxic substances [3]. Chronic exposure to cadmium leads to the accumulation in the proximal tubular epithelial cells, which causes mitochondrial injury, inflammation, and apoptosis of the proximal tubular cells with subsequent tubular degeneration and loss of renal filtration [4]. Previous epidemiological and experimental studies have shown a strong association between Cd exposure and chronic kidney disease progression by similar nephrotoxic mechanisms. The high level of serum creatinine and albuminuria in the current study also indicates the nephrotoxic effect of chronic cadmium exposure [5].

Oxidative damage of tissues and renal failure have also been shown to be closely linked to the effects of arsenic exposure [6,7]. Exposure to arsenic for a prolonged period causes the production of reactive oxygen species (ROS), endothelial dysfunction, activation of inflammatory cytokines, and damage to DNA in the kidneys. Chronic oxidative stress can gradually cause a decrease in glomerular filtration and increase renal cellular damage [8]. In the present study, urinary albumin was higher, and eGFR was lower among exposed subjects as compared to other studies that have reported nephrotoxicity upon arsenic exposure [9].

Mercury toxicity also acts on renal injury via the mechanism of oxidative stress and direct injury of tubular cells [10]. Mercury is known to have a high affinity for sulfhydryl groups of cellular proteins and antioxidant enzymes, which leads to exhaustion of the endogenous antioxidant defense mechanisms and thus the cell becomes prone to lipid peroxidation [11]. The significantly high level of MDA observed in the exposed participants in this study showed serious oxidative damage to the membrane and increased lipid peroxidation in response to the chronic exposure to heavy metals [12].

The major and important result of the present study was that antioxidant enzyme activities (SOD, catalase, and GPx) showed a significant decrease in exposed persons [13]. The intracellular defence system against ROS is the first line of defence, that are antioxidant enzymes. Exposure to toxic metals continues over a long period of time and is overwhelming the antioxidant defense mechanisms, leading to continued oxidative stress and progressive cellular damage. Antioxidant enzyme activities have been decreased in populations exposed to environmental heavy metal contamination [14].

Heavy metal concentrations showed strong positive correlations with renal dysfunction markers, which further highlight the nephrotoxic effects of cadmium, arsenic, and mercury accumulations [15]. When toxic elements are encountered together, they may be synergistically toxic or

may act to increase oxidative stress and speed up the damage to the kidneys. These results suggest a major public health impact of chronic groundwater contamination in industrial and environmentally polluted areas [16].

The present study provides important evidence regarding the relationship between environmental toxic exposure, oxidative stress, and renal dysfunction [17]. To minimize kidney disease burden caused by heavy metals, exposed populations must be identified early, and environmental monitoring implemented. Long-term toxic effects might be prevented by public health interventions like the improvement of safe drinking water supply, the regulation of industrial waste, and regular health screening programs to vulnerable groups [18].

Some of the limitations of the study should also be noted. However, the cross-sectional design precludes drawing firm conclusions about the relationship between toxic metal exposure and renal dysfunction. Furthermore, full standardization of the assessment of dietary exposure and environmental confounding factors was not possible. Despite the limitations, the study offers valuable data on nephrotoxic and oxidative effects of chronic heavy metal exposure among populations who are exposed to the environment [20].

CONCLUSION

Chronic exposure to water-borne cadmium, arsenic, and mercury is strongly related to renal dysfunction and high levels of oxidative stress. Chronic exposure to toxic metals leads to hypercreatinuria, hyperuremia, proteinuria, impaired GFR, hyperlipidation, and deficiency of endogenous antioxidant enzymes. The results suggest that oxidative stress is a key factor in the nephrotoxicity caused by heavy metals. To reduce toxic metal-induced renal damage and prevent adverse health outcomes, continuous environmental monitoring, strict controls on industrial emissions, provision of safe drinking water, and early screening of the potentially exposed populations are essential.

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Authors' Contributions: D.L. contributed to conceptualization, study design, supervision, environmental toxicology assessment, interpretation of findings, and manuscript drafting. D.L. also contributed to data analysis, critical revision of the manuscript, and approved the final submitted version.

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Data Availability: The datasets generated and analyzed during this study are available from the corresponding author upon reasonable request.

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