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EVALUATION OF RENAL FUNCTION PARAMETERS AND LIPID PEROXIDATION IN SOLID WASTE WORKERS IN YENAGOA, NIGERIA

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ABSTRACT

Environmental contamination due to solid waste disposal is a global issue that portends serious health problems to humans. This present study is designed to evaluate some renal function parameters and lipid peroxidation in solid waste workers in Yenagoa, Nigeria. Methods: A total of ninety (90) apparently healthy male recruits within the age range of twenty (20) and forty (40) years were used in this study. Sixty (60) recruits were solid waste workers, and thirty (30) served as control. Sera were collected, prepared and stored at -20oC prior to analysis which employed Ion Selective Electrode and Spectrophotometry. The result showed statistically significant ($P<0.05$) increase in the mean±SD values of creatinine (113.25±19.94), urea (4.03±0.89) and uric acid (178.35±4.74) in the tests when compared with controls (89.43±15.25, 2.51±1.42 and 158.5±2.95) respectively. There were slight decreases in the sera concentrations of sodium (135.07±9.35), potassium (3.85±0.42) and chloride (96.90±15.66) which nonetheless, were not statistically significant ($p>0.05$) in the tests when compared with controls (136.74±12.03, 4.08±0.47, 99.85±5.01 and 4.03±0.89) respectively. Lipid peroxidation product, Malondialdehyde (7.37±1.57) was significantly (0.001) elevated in the tests when compared with controls (5.88±0.42). In recruitts with duration of exposure ≥2years, sodium was significantly decreased compared with <2years of exposure. Creatinine, uric acid and malondiadehyde were significantly ($p<0.05$) increased among recruitts with ≥2years duration of exposure compared with <2years of exposure. Solid wastes have a negative effect on the renal function and lipid peroxidation which is dependent on the duration of exposure.

KEYWORDS: Solid Wastes; Renal function parameters; Lipid peroxidation.

INTRODUCTION

Poor sanitation and improper waste disposal pollutes the environment and therefore threaten the well-being of humans and animals alike. Solid waste management has been a chronic problem in developing countries, particularly in urban cities with high population density, high production of refuse and scarcity of land.[1] The activities of humans generate wastes that constitute a serious health problem to humans and the environment.[2] The collection and subsequent disposal of solid waste at municipal dumpsite is a common practice across the globe. Workers involved in the management process are exposed to high level of toxic chemicals, heavy metals and biological agents emanating from solid waste.[3]

During collection, disposal and processing of the solid waste at municipal dumpsites, workers are exposed to heavy metals from lead batteries, electrical equipment, and electronic waste, stainless steel or plastic products that pose a considerable health risk to these workers.[4] Several studies have confirmed the presence of more than thirty different metals in the incinerated ash and dust of untreated urban waste including arsenic, cadmium, chromium, lead, and mercury all of which are harmful to human health.[5] Exposure to high levels of heavy metals can result in acute and chronic toxicity, such as damage to central and peripheral nervous systems, blood composition, lungs, kidneys, liver, and may even cause death.[6,7] Cadmium and lead are priority environmental contaminants because they induces kidney damage and causes cancer.[8] Copper, zinc, nickel, and chromium have been studied to have adverse effects such as damage to the nervous membrane, kidney and liver failure, cancer, and fatigue.[9] These metals get into the body either by inhalation or ingestion or through skin contact.[10] Workers in the waste disposal field, in particular, can also be exposed to bioaerosols, gases, and vapours. Some gases such as ammonia, sulphur dioxide, nitric oxide, nitrogen dioxide and nitrous oxide are emitted from waste disposal sites during incineration where workers may become exposed through inhalation or contact with the skin.[11] Volatile organic compounds like benzene, toluene, dichloromethane, tetrachloroethylene, trichloroethylene, dichloroethane,

phthalates, butadiene and dimethylacetamide found in waste are carcinogenic, mutagenic and andreprotoxic chemicals[12] are also a threat to health. A wide range of organic pollutants such as PAHs (polycyclic aromatic hydrocarbons), PCBs (polycyclic aromatic hydrocarbons), dioxins, furans (polychlorinated biphenyls), phthalates, ketones, aldehydes, organic acids and alkenes are emitted from incinerators.[13]

According to Landrigan et al.[14] the burden of diseases of waste-related exposures in underdeveloped countries is increasing and not sufficiently recognized. Several studies have reported the hazards or health effects associated with waste workers to include respiratory disorder, gastrointestinal dysfunction, and maladies of the skin, mucus membranes and skeletal system.[15] These dangerous substances found in solid waste are harmful to body tissues and organs, the kidney inclusive. Among the tissues and organs in the human body, the kidney and liver are the most sensitive to chemical toxicity. This could be attributed to the involvement of these organs in metabolism, detoxification, storage and excretion of xenobiotic and their metabolites, making them important targets of organs of xenobiotic-induced injuries.[16]

Yoshida `et al.[17] confirmed an increased serum creatinine and dioxins levels in male waste incinerator workers. A study by Staessen et al.[18] reported that biomarkers of renal dysfunction were positively correlated with lead (Pb^{2+}) levels in the blood of adolescents living near a lead (Pb^{2+}) smelter. Inhalation of dust containing heavy metals and contact with heavy metals present in solid wastes has been reported to cause oxidative stress[19] due to the excessive generation of Reactive Oxygen Species (ROS) and that leads to cellular damage. Oxidative stress results in lipid peroxidation. Malondialdehyde (MDA) is the main biomarkers of lipid peroxidation and its quantification is a direct indicator of oxidative stress.[20]

In the majority of cities in Nigeria (eg. Yenagoa), solid wastes are disposed indiscriminately in open dumps along the roads and streets, forming huge refuge hills.[21] This improper disposal of solid waste could present a great danger to the environment and human health.[22] To curb this menace, workers are employed to collect, dispose and process the solid waste which in turn predisposed them to hazardous health conditions[23] since these worker are inadequately equipped with the necessary protective tools to minimize the effects of exposure.

Studies on the short- and long-term effects of exposure to solid waste on public health have been focused all on identifying any associations between solid waste management workers and adverse effects on health.[24,25] Meanwhile, less attention has been paid to the risk posed in subjects who are directly involved in solid waste management, especially as regards the effects on the renal system. Epidemiological studies have linked exposure to dumpsites with increased health defects such as birth defects, reproductive disorders, liver disorder as well as increase in frequency of cancers.[26,27] However, there is paucity of data literature on the effect of solid waste exposure on the renal function and lipid peroxidation. Therefore, this present study is aimed to evaluate some renal function parameters and lipid peroxidation in solid waste workers in Yenagoa, Bayelsa State, Nigeria.

METHODS

Study Area

Yenagoa, the capital city of Bayelsa State, Nigeria lies between latitude 4° 50' to 5° 00' North and longitude 6° 11' to 6° 25' East. It is a small capital city, well-populated with very few industries, many commercial centres and schools (primary, secondary and tertiary), hospitals and residential buildings. The major occupation of residents of the town is civil service. Other occupations are farming, fishing and trading. There are several solid waste dump sites spread across the city with solid wastes forming refuge hills. This study was carried out on two private solid waste disposal companies and on the Bayelsa state Environmental Sanitation Authority solid waste disposal sector.

Subjects

The study was a cross-sectional comparative study carried out among 90 apparently healthy subjects of whom 60 were solid wastes management workers and 30 were non-workers. All the 60 subjects used for the study were male with age range of 25–45 years and residing in Yenagoa Bayelsa State, Nigeria. The duration of exposure was one (1) to seven (7) years. Subjects who consented to the study and without a medical history of any known metabolic disorders or infections were included for the study. Subjects who do not consent to the study. Also excluded are cigarette smoker and chronic alcohol drinkers. The ethical clearance was approved by the ethical committee Bayelsa State Environmental Sanitation Authority (BSEA) and the director of the solid waste disposal company (Brikari Limited Company).

Blood Sample Collection

Blood samples were collected from each of the ninety (90) subjects (after fulfilling the inclusion and exclusion criteria). A standard clean venipuncture technique was used to collect 10ml of venous blood from each participant into a plain tube. The samples were transported to the Research Laboratory of Medical Laboratory Science, Niger Delta University in a cool box containing ice bags. The samples were centrifuged for five (5) minutes at 2,500rpm and the supernatant serum was separated into separate tubes. The serum was stored at -20^0C and all analyses were carried out within 48hours of sample collection. Sodium, potassium, and chloride were analyzed using ion selective electrode machine. Randox Diagnostic kits were used for the assay of

creatinine, urea and uric acid. Malondialdehyde in serum was determined by the method of Shah and Walker's.[28]

Analysis of biochemical Parameters

Serum electrolytes were determined by the Ion Selective Electrode method as described by Bard & Faulker,[29] Urea was estimated by Urease-Berthelot Method according to Weatherburn,[30] Creatinine was determined spectrophotometrically according to the method described by Bartels and Bohmer,[31] Uric acid was estimated spectrophotometrically by Henry et al.[32] Malondialdehyde in serum was separated and determined as conjugate with TBA. Serum proteins were precipitated by TCA and then removed by centrifugation. The MDA – TBA complex was measured at 534 nm. Briefly, 1.0ml reagent 1 (17.5% TCA), reagent 2 (70% TCA) and regent 3 (Thiobarbituric acid 0.6%) was added to 1.0ml of serum and mixed. The reaction mixture was incubated in boiling bath for 15 minutes, allowed to cool, and then let to stand at room temperature for another 20 minutes. Then the tubes centrifuged at 2000 rpm for 15 minutes and the supernatant layer was read at 534 nm. Distilled water was used for the blank. The concentration of MDA (nmol/ml) was calculated by using the following formula: Concentration of the test= Abs (test) –Abs (blank) / 1.56 x 1000000 Shah and Walker's.[28]

DATA ANALYSIS

Statistical analysis was done by using descriptive and inferential statistics using chi-square test, one way ANOVA and Multiple Comparison-Tukey Test. The software used in the analysis was SPSS (Statistical Package for Social Sciences) version 17.0. All the results were tested at 5% level of significance.

RESULT

Table 1.0 shows effect or impact of solid waste on kidney function parameters in solid waste management workers in Yenagoa metropolis. The Result showed an increase in serum creatinine and urea and these were statistically significant ($p<0.05$) in the exposed subjects when compared with unexposed subjects. Sodium (135.07±9.35), potassium (3.85±0.42) and chloride (96.90±15.66) showed a non-significant ($p>0.05$) decrease in the exposed subjects when compared with the unexposed subjects (136.74±12.03, 4.08±0.47 and 99.85±5.01) respectively. Uric acid (178.35±4.74) showed statistically significant ($p<0.001$) increased in the test samples when compared with the unexposed subjects (158.5±2.95). Table 2.0 shows the effect of solid waste on lipid peroxidation in solid waste management workers in Yenagoa metropolis. The result revealed that lipid peroxidation product, Malondialdehyde (MDA) (7.37±1.57) increased statistically significance ($p<0.001$) in the test samples when compared with the unexposed subjects (5.88±0.42). Table 3.0 shows the effect of duration of exposure to solid waste on renal function biomarkers and lipid peroxidation in solid waste workers in Yenagoa metropolis. The Result revealed that sodium and creatinine were statistically significant ($p<0.05$). Lipid peroxidation product, Malondialdehyde (MDA) and uric acid showed a statistically significant ($p<0.05$) increase in solid waste management workers with occupational experience of two years and above as against those involved in less than two years. There was no significant difference ($p>0.05$) in potassium, chloride and urea levels in solid waste management workers with occupational experience of two years and above as against those involved in less than two years.

Table 1.0: Impact of solid waste on kidney function parameters of solid waste workers in Yenagoa metropolis.

VARIABLES	CONTROL (Mean ±SEM)	TEST (Mean ±SEM)	P-VALUE
Sodium (Na^+)	136.74±12.03	135.07±9.35	0.47
Potassium (K^+)	4.08±0.47	3.85±0.42	0.56
Chloride(Cl^-)	99.85±5.01	96.90±15.66	0.51
Creatinine	113.25±19.94	98.43±15.25	0.03
Urea	4.12±0.14	3.07±0.117	0.04
Uric acid	158.5±2.95	178.35±4.74	0.02

KEY: Results are expressed as Mean ± Standard Error of Mean (SEM).

Table 2: Effect of Solid Waste on lipid peroxidation in solid waste workers in Yenagoa metropolis.

Variables	CONTROL	TEST	P -Value
Malondialdehyde (MDA)	5.88±0.42	7.37±1.57	<0.001

KEY: Results are expressed as Mean ± Standard Error of Mean (SEM)

Table 3.0: Effect of Duration of Exposure to Solid waste on Renal Parameters and lipid Peroxidation product of Solid Waste Workers in Yenagoa Metropolis.

VARIABLES	< 2 years (Mean ± SEM)	≥2 years (Mean ± SEM)	P-value
Sodium (Na^+)	135.77±7.27	134.58±6.89	0.03
Potassium (K^+)	3.93±0.35	3.79±0.46	0.25
Chloride(Cl^-)	96.11±11.94	97.46±18.17	0.53
Creatinine	107.24±14.85	113.25±19.94	0.02
Urea	3.27±0.89	3.51±1.42	0.77
Uric acid	159.7±8.22	162.8±2.23	0.03
Malondialdehyde (MDA)	6.83±1.17	8.13±0.96	0.00

KEY: Results are expressed as Mean ± Standard Error of Mean (SEM).

DISCUSSION

The kidney is a vital organ involved in excretion, homeostatic and endocrine regulations.[33] It is the primary organ of target in heavy metal toxicity because of its ability to reabsorb and concentrate divalent ions and metals. Studies of both acute and chronic exposure have confirmed that heavy metal such as lead, cadmium, mercury and arsenic cause nephropathies, with various levels of severity ranging from tubular dysfunctions to severe renal failure which could occasionally lead to death.[34] The kidneys maintain electrolytes concentrations by filtering electrolytes and water from blood. It reabsorbs 99% of plasma filtrate including the electrolytes (Na^+, K^+, HCO3- and Cl) and excretes any excess into the urine. Alteration in the architecture of the renal tubules could occur as a result of the accumulation of heavy metals such as lead, cadmium, mercury and arsenic which could lead to reduced tubular reabsorption of micronutrients including electrolytes.[35] In the present study, the mean serum electrolytes (Na^+, K^+, and Cl^-) level showed no statistically significant ($p>0.05$) decrease in the exposed recruits compared to the unexposed. This could be associated with the poor sensitivity of electrolytes as renal function biomarkers and the reserved ability of the kidney to maintain normal physiology even after loss of about 50% of nephron.[36] Furthermore, the slight decrease in the serum electrolytes (Na^+, K^+, and Cl^-) observed in this study could be linked to the accumulation of heavy metals (lead, cadmium, arsenic and mercury) in the renal tubules and induced a condition known as Fanconi syndrome characterized by a decreased Glomerular Filtration Rate (GFR), excessive loss of major ions like Na^+, K^+, and Cl^- and water, proteinuria and glycosuria, and thus reduced tubular reabsorption of Na^+, K^+, and Cl^-.[34] This slight reduction could also be attributed to the ability of lead, cadmium, arsenic, and mercury to cause an increased plasma renin activity leading to excessive loss of water and electrolytes. This finding is in agreement with the work done by Amah et al.[36] who reported a non-significant difference in the mean level of sodium (Na+), potassium (K+) and chloride (Cl-) of lead exposure among automobile repairers in Nnewi. However, it contradicts the observations of both of whom reported a significant increase in the levels of sodium (Na+), potassium (K+), and chloride (Cl-) in heavy metal-induced injury in mice and rats.[37,38]

Creatinine is a biomarker of renal function which tends to be elevated in damaged kidneys. It is excreted in urine by the kidney in healthy individuals. Any renal impairment may lead to retention of creatinine in the circulation. Studies have shown that occupational exposure to heavy metals such as lead, arsenic, mercury, cadmium and other toxic chemicals have been demonstrated to cause proximal tubular damage and nephropathies.[39,40] Heavy metals generated from solid waste exist in two forms in plasma: protein bound and ionized forms. The luminal fluid in the early proximal tubule of the nephron can contain the two forms. In prolonged exposure to solid waste containing heavy metals, the protein bound form is conjugated with metallothionein and glutathione, which are then released into the circulation by the liver and the kidney. These heavy metals (lead, cadmium, arsenic, and mercury) are subsequently reabsorbed through +an endocytotic process in segment S1 of the proximal tubule of the nephron and can lead to chronic inflammation, fibrosis and renal failure.[41,42] A report by Salazar-Flores et al.[39] revealed that Hg, Pb and As contribute significantly to oxidative stress by stimulating the generation of free radicals, oxidation of biomolecules, deregulation of pro-oxidant proteins, and activation of pro-inflammatory molecules, which ultimately lead to renal damage accompanied by creatinine retention in circulation. In this present study, the serum creatinine level of the solid waste workers was significantly elevated compare to the unexposed subjects. This elevation could be due to the bioaccumulation of heavy metals in the renal tubules, which could have resulted in chronic inflammation, production of free radicals and renal damage. This is in agreement with the work done by Yoshida et al.[17] who

confirmed an increased serum creatinine and dioxins levels in male solid waste workers. However, this is contrary to the observation of Dioka et al.[43] carried out on artisans occupationally exposed to lead in which there was a decrease in mean serum creatinine level of exposed subjects compare to non-exposed.

Urea is another biomarker of the renal function which concentration in the plasma tends to increase in impaired kidney condition. The result from this current study shows that the serum urea level in the exposed subjects was significantly higher than the unexposed subjects. However, the results falls within the reference range. This increase in urea levels observed in this study could be linked to the slow impairment of glomerular basement membrane caused by the accumulation of heavy metals like lead, mercury, cadmium and arsenic found in solid waste in the kidney. The finding of elevated serum urea in the current study is consistent with the observation of Amah et al.[36] who confirmed a significant elevation of serum level of urea in heavy metal exposed automobile repairers in Nnewi Metropolis. However, this result disagree with the work done by Dioka et al. [43] who reported a statistical non-significant difference in the serum level of urea of heavy metals exposed subjects compared with the control subjects.

Uric acid is a by-product of purine metabolism and the purine (adenosine and guanosine) are released from DNA in their triphosphate forms.[44] Uric acid has been known as one of the antioxidants found in plasma.[45] The kidney is responsible for the secretion and excretion of uric acid. About 98% of filtered urate is reabsorbed and 6-10% is secreted from the S2 segment of the proximal tubules. Cadmium (Cd^{2+}) and other heavy metals are known nephrotoxicant and have been demonstrated to cause renal injury.[46] The deposition of lead in the proximal tubules of the nephron causes the alteration in morphology and physiology of the cell, thus decreasing renal tubular secretion of uric acid into the lumen.[36] In the present study, there was a statistically significant elevation of the serum levels of uric acid in the exposed subjects compared with the unexposed subjects. This elevation of uric acid in the exposed workers in this study may be attributed to heavy metal-induced renal impairment or may be linked to the effect of reactive oxygen species on DNA. Oxidative attack on DNA results in deoxyribose oxidation, strand breakage, removal of nucleotides, variety of modification in the organic bases of the nucleotides and DNA-protein crosslink.[47] The increased release of adenosine and guanosine phosphates from damaged DNA may be responsible for the elevated uric acid level in the exposed subjects in this study. This finding is in tandem with the observation of Godwin et al.[48] who reported an increased serum uric acid level in e-waste exposed workers.

Lipid peroxidation is the systematic oxidation by free radicals fatty acids in the cell membranes. This is known to limit cells function by decreasing the fluidity of the membrane. An end-product of lipid peroxidation, Malondialdehyde (MDA) is used as an indicator to access the extent of lipid peroxidation. Several authors have reported that heavy metal (lead, cadmium, mercury and arsenic) toxicity aggravates oxidative stress which causes different pathological processes including lipid peroxidation. Yiin et al.[49] confirmed that that lipid peroxidation increases in lead toxicity. Lead and mercury interact with renal cells and inhibits delta-aminolevulinic acid synthase and increases the levels of Reactive Oxygen Species (ROS) such as superoxide anion, hydroxyl radical and hydrogen peroxide that cause kidney damage.[39] In the current study, lipid peroxidation product, malondialdehyde (MDA) was significantly elevated in the solid waste workers compared to the unexposed subjects. This elevation may be linked to the presence of these toxic metals generated in solid wastes which have high affinity for cell membranes and mitochondria phosphorylation.[50] This process lead to the production of reactive oxygen species (ROS) and thus causes oxidative stress. The accumulative oxidative stress produced by exposure to solid waste causes damage of cellular components including DNA damage, DNA mutation and transformation to malignant cell. Our finding is in agreement with Gargy et al.[51] who reported an elevated level of lipid peroxidation in children working in garbage dumpsite. It also in agreement with the findings of Godwin et al.[48] who revealed a significantly elevated lipid peroxidation in e-waste workers.

CONCLUSION

This present study has shown that solid waste contain heavy metals and other hazardous chemicals that may have a negative impact on the kidneys and thus enhance lipid peroxidation in solid waste workers: which appear to be dependent on the duration of exposure.

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Conflict of Interest

The authors declare that there is no conflict of interests regarding the publication of this manuscript.

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