*Original Research Article*

Effects of Occupational Exposure to Heavy Metals on Oxidative DNA Damage in Some Artisans in Rivers State

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ABSTRACT

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| **Aim:** To assess the effects of occupational exposure to heavy metals on oxidative DNA damage in some artisans in Port Harcourt, Rivers State, Nigeria.**Study design:** Cross-sectional study**Place and Duration of Study:** Automobile workshops, welding workshops, and painting workshops, located in Mile 3 mechanic garage, Elekahia and Rumuagholu., between June 2023 and January 2024.**Methodology:** A total of 100 subjects were recruited for this study based on specific inclusion and exclusion criteria. The exposed group was made up of three different types of artisans (mechanics, welders, and painters) with a total of 75 exposed individuals, each group comprised of 25 artisans each, while the non-exposed (control) group were 25 individuals who were mainly students and office workers. All the participants for the study were asked to fast for 8 hours after which 10 ml of venous blood samples were collected into plain sample bottles. Separate serum samples were used to assay 8-OHDG, total superoxide dismutase, cardiac troponin I, total antioxidant capacity and total oxidant status. Statistical analysis was performed using GraphPad prism version 8.3. and p-values less than 0.05 were considered statistically significant.**Results:** The results of 8-OHDG, cardiac troponin, total superoxide dismutase, total oxidant status and oxidative stress index showed a significant increase (P<0.05) in the mechanics, welders, and painters, while there was a significant decrease (P<0.05) in total antioxidant capacity in the painters and welders.**Conclusion:** In conclusion, according to this study, the exposure to heavy metals is capable capable of causing DNA damage because of DNA oxidation by reactive oxygen species generated by heavy metals. |

*Keywords: Occupational Exposure, Heavy Metals, Oxidative DNA Damage, Artisans, Rivers State, Nigeria*

1. INTRODUCTION

Heavy metals have been discovered to be one of the most toxic and harmful environmental pollutants, especially in developing countries. Most research dwells mostly on the imbalance in antioxidant levels and elevated levels of free radicals [1]. There have been information’s from epidemiologists in the past years indicating the dangers of welding fumes, however, until now, welding fume-induced health problems remain an occupational hazard that is yet to be solved. The adverse effects of heavy metals from welding fume exposure are observed in both humans and animals [2]. There is limited information regarding the effect of multiple metal exposures. It has been reported that some workers are routinely exposed to toxic metal and are thus said to constitute about 0.9% of the total global health burden with majority of cases in developing countries, between 0.5 and 1.5 million being due to occupational exposure [3]. Lead and cadmium poisoning are major potential public health problems throughout the world particularly in developing countries. Long-term exposure to these pollutants can increase the risk of developmental and reproductive disorders, immune system disorders, endocrine disruption, impaired nervous system function, cardiac diseases, and development of certain cancers. There is no adequate sensitization and public/industrial health education towards ensuring the understanding of the potential hazard of the various chemicals and metal pollutants they are exposed to [4].

The free radicals are important components in many metabolic, biological and physiological processes, they serve as antibacterial and antifungal agents, and they promote the normal division of cells and the expression of genes. When there are changes in the body, the balance between the antioxidants and reactive species will be disrupted, favoring the increased production of the reactive species leading to disease conditions associated with oxidative stress [5]. When the concentration of free radicals is above that than the body can bear, they disrupt cellular structures since the ROS have affinity for cells and important macromolecules; the disrupted cells fail to function properly, hence the accumulation of toxic compounds such as Malondialdehyde in the cells due to a failed redox signal. This process may progress as one ages, and it causes the aging process to speed up accompanied by some diseases like eye problems, Parkinson’s disease and diabetes. It has been discovered that antioxidants with low molecular weight are incapable of initiating their normal antioxidant activity by combating the free radicals [6]. The aim of this study was to assess the effects of occupational exposure to heavy metals on oxidative DNA damage in some artisans in Port Harcourt, Rivers State, Nigeria.

2. materialS and methods

**2.1 Experimental Design**

All qualified subjects for this study were classified into four groups including the control group and it was based on their different occupations (automobile mechanics, painters, welders), and the control group. Alcohol consumption by the test participants was classified as heavy (consumes large amounts of spirits, and beer regularly), moderate (consumes small amounts of spirits, and beer sometimes), and (no) for those that do not consume alcohol at all.

**2.2 Study Area**

The subjects for this study were recruited from automobile workshops, welding workshops, and painting workshops, located in Mile 3 mechanic garage, Elekahia and Rumuagholu, Port Harcourt, Rivers State Nigeria.

**3.3 Population of the Study (Sample Size)**

The sample size was determined using the Cochran Formula; no = z2pq / e2 [7].

Z2= Confidence level (1.96)2

P= Prevalence rate (0.11),

Q = 1-P (0.89),

e2 (margin of error) = (0.05)2

(3.84) (0.11) (0.89) / 0.0025 = 100

A total of 100 apparently healthy male subjects aged (18 –65 years) were selected for this study. They were divided into 4 groups including the control group, with each group comprising 25 subjects each. The test groups were (automobile mechanics, welders, and painters) who have been working for at least two years either as workers or as apprentices and they were selected based on specific inclusion and exclusion criteria designed for this study using the well-structured questionnaire, while the control group was made up of 25 subjects who were mainly students, business owners, and office workers.

**2.3.1 Test Group**

The test group comprises of 3 subgroups with 25 subjects in each group

Sub-Groups

Group 1: Automobile mechanics.

Group 2: Painters.

Group 3: Welders.

**2.3.2 Control Group**

Group 4: Non-occupationally exposed individuals (25 subjects).

**2.4 Sample Collection**

Ten milliliters (10 ml) of venous blood (fasting samples) were collected from each subject into a plain bottle. The serum obtained after centrifugation was stored at -20°C until analysis.

**2.5 Inclusion Criteria**

• Only males aged 18-65 years were included in the study.

• Individuals who have been exposed for at least 2 years during their job, including apprentices with not less than 2 years working experience.

• Individuals that smoke and consume alcohol were also included in this study.

**2.5 Exclusion Criteria**

• Subjects less than 18 years of age were not eligible to participate in this study.

• Subjects with history of already existing cardiovascular or lung diseases like atherosclerosis, hypertension, chronic obstructive airway disease (COPD), and reactive airway disease are ineligible for the study.

• Subjects with less than 2 years of working experience in the listed vocations.

**2.6 Determination of 8-Hydroxydeoxyguanosine (8-OHdG) by Enzyme Linked**

**Immunosorbent Assay (ELISA) Technique** [8]

**2.6.1 Principle**

This ELISA kit uses the Competitive-ELISA principle. The micro-ELISA plate provided in the kit has been pre-coated with 8-OHdG. During the reaction, 8-OHdG in the sample or standard competes with a fixed amount of 8-OHdG on the solid phase supporter for sites on the Biotinylated Detection Antibody specific to 8-OHdG. Excess conjugate and unbound sample or standard are washed from the plate, and Avidin conjugated to Horseradish Peroxidase (HRP) are added to each microplate well and incubated. Then a TMB substrate solution is added to each well. The enzyme-substrate reaction is terminated by the addition of stop solution and the color change is measured spectrophotometrically at a wavelength of 450 nm ± 2 nm. The concentration of 8-OHdG in the samples is then determined by comparing the optical density of the samples to the standard curve.

**2.7 Determination of Total Oxidant Status (TOS) Using Colorimetric Technique by** **Ozcan,** [9]

**2.7.1 Principle**

Under acid conditions, the oxidizing material in the sample can oxidize Fe2+ to Fe3+, which binds highly with xylenol orange to produce a blue-purple complex. When the pH of the solution is in the range of 2 to 3, its maximum absorption wavelength is around 590 nm, and the color depth is proportional to the content of oxidation substances in a certain concentration and a certain time, to indirectly calculate the total oxidation state of the sample.

**2.8 Determination of Total Antioxidant (TAC) Using Colorimetric Technique by Ozcan,** [9]

**2.8.1 Principle**

Fe3+-TPTZ (2, 4, 6-Tripyridyl -S- triazine) can be reduced by antioxidants and produce blue Fe2+-TPTZ under acidic condition. The antioxidant capacity of the sample can be calculated by the detection of the absorbance value at 593 nm.

**2.9 Determination of Cardiac Troponin I (Human TNNI3/cTn-I, Troponin I) by Enzyme Linked Immunosorbent Assay (ELISA) Technique** [10]

**2.9.1 Principle**

This ELISA kit uses the Sandwich-ELISA principle. The micro-ELISA plate provided in the kit has been pre-coated with an antibody specific to Human TNNI3/cTn-I. Standards or samples are added to the micro-ELISA plate wells and combined with the specific antibody. Then a biotinylated detection antibody specific for Human TNNI3/cTn-I and Avidin-Horseradish Peroxidase (HRP) conjugate was added successively to each micro plate well and incubated. Free components are washed away. The substrate solution is added to each well. Only those wells that contain Human TNNI3/cTn-I, biotinylated detection antibody and Avidin-HRP conjugate will appear blue in color. The enzyme-substrate reaction is terminated by the addition of stop solution and the color turns yellow. The optical density (OD) is measured with a spectrophotometer at a wavelength of 450 nm ± 2 nm. The OD value is proportional to the concentration of Human TNNI3/cTn-I. The concentration of Human TNNI3/cTn-I in the samples can be calculated by comparing the optical density of the samples to the standard curve.

**2.10 Determination of Total Superoxide Dismutase (T-SOD) by Enzyme Linked Immunosorbent Assay (ELISA) Technique** [10]

**2.10.1 Principle**

Xanthine and xanthine oxidase reaction systems produce superoxide anion free radical O2+. The superoxide anion free radical oxidizes hydroxylamine to produce nitrite, the color of the solution turns purple following the reaction of the developer. When the samples containing superoxide dismutase are measured, the superoxide dismutase specifically inhibits superoxide anion free radical. Nitrite formation is decreased following the inhibitory effect of superoxide dismutase. The values obtained from the sample tubes are lower than the control group. The total superoxide dismutase in the sample is calculated with the given formula.

**2.11 Statistical Analysis**

The software used for the statistical analysis was GraphPad prism (version 8.3), the analytical tools used were Mean, Standard deviation (SD), ANOVA, Tukey's Multiple Comparison Test. The Results were expressed as mean± standard deviation.

3. results and discussion

**Table 1: Comparison of 8-OHDG, Cardiac Troponin-I, and Oxidative Stress Markers of Controls, Mechanics, Welders, and Painters**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Subjects** | **8-OHDG (ng/l)** | **Cardiac Troponin-I (ng/l)** | **Total-SOD (U/ml)** | **TAC** **(U/ml)** | **TOS (µmol/H2O2 Eqiv./L)** | **OSI (%)** |
| Controls n=25 | 8.94 ± 0.80 | 0.82 ± 0.08 | 17.66 ± 3.48 | 86.23 ± 3.46 | 5.21 ± 0.81 | 5.91 ± 0.81 |
| Mechanics n=25 | 46.09 ± 8.063 | 1.33 ± 0.12 | 71.58 ± 5.58 | 77.20 ± 3.48 | 15.82 ± 2.30 | 21.58 ± 3.15 |
| Welders n=25 | 51.11 ± 12.43 | 1.66 ± 0.12 | 82.11 ± 4.96 | 68.16 ± 3.14 | 29.75 ± 3.95 | 45.58 ± 6.36 |
| Painters n=25 | 23.01 ± 10.45 | 1.76 ± 0.14 | 70 51 ± 5.79 | 61.88 ± 4.04 | 26.71 ± 3.27 | 52.22 ± 8.94 |
| p-values | 0.0046 | < 0.0001 | < 0.0001 | < 0.0001 | < 0.0001 | < 0.0001 |
| F-Values | 4.618 | 13.72 | 33.81 | 8.997 | 15.47 | 14.05 |
| Tukey'sMultiple Comparison Test | Summary | Summary | Summary | Summary | Summary | Summary |
| Control vs Mechanic | \* | \* | \*\*\* | Ns | \* | Ns |
| Control vs Welder | \*\* | \*\*\* | \*\*\* | \*\* | \*\*\* | \*\*\* |
| Control vs Painter | Ns | \*\*\* | \*\*\* | \*\*\* | \*\*\* | \*\*\* |
| Mechanic vs Welder | Ns | Ns | Ns | Ns | \*\* | \* |
| Mechanic vs Painter | Ns | \* | Ns | \* | \* | \*\* |
| Welder vs Painter | Ns | Ns | Ns | Ns | Ns | Ns |

***Keys:*** *8-OHDG= 8-Hydroxyl-2’deoxyguanosine, Total SOD=Total Superoxide Dismutase, TAC= Total Antioxidant Capacity, TOS= Total Oxidant Status, OSI= Oxidative stress index, \*=Significant (P<0.05), NS=Not Significant (p>0.05). Reference Range: 8OhDG: 10-300 ng/L, Cardiac Troponin-I: <14ng/L, Total-SOD: 1.56-100 U/ml, TAC: 0.62-145.2 U/ml, TOS: 2.5-100 µmol/H2O2 Eqiv./L, OSI: Normal <40, Borderline: 41-65, High: 66-120, Very High: >121.*

**Table 2: Comparison of 8-OHDG, Cardiac Troponin-I, and Oxidative Stress Markers to Heavy Intake of Alcohol by Controls, Mechanics, Welders, and Painters**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Subjects** | **8-OHDG (ng/l)** | **Cardiac Troponin-I (ng/l)** | **Total-SOD (U/ml)** | **TAC** **(U/ml)** | **TOS (µmol /H2O2 Eqiv./L)** | **OSI** **(%)** |
| Controls | 31.52 ± 5.33  | 1.2 ± 0.31 | 63.12 ± 11.87 | 74.12 ± 5.51 | 15.98 ± 4.41 | 23 ± 7.16 |
| Mechanics n=5 | 61.77 ± 19.47 | 1.5 ± 0.20 | 87.48 ± 5.59 | 66.11 ± 4.86 | 49.01 ± 15.19 | 49.01 ± 15.19 |
| Welders n=9 | 13.03 ± 2.93 | 2.08 ± 0.19 | 64.03 ± 7.09 | 52.67 ± 9.5 | 27.17 ± 5.64 | 72.05 ± 25.12 |
| Painters n=5 | 9.09 ± 1.09 | 0.66 ± 0.12 | 11.59 ± 1.66 | 90.54 ± 18.36 | 4.78 ± 0.68 | 5.19 ± 0.59 |
| p-values | 0.0089 | 0.0002 | <0.0001 | 0.0021 | 0.0067 | 0.0099 |
| F-Values | 4.661 | 9.755 | 36.94 | 6.447 | 4. 672 | 4.642 |
| Tukey's Test | Summary | Summary | Summary | Summary | Summary | Summary |
| Control vs Mechanic | Ns | Ns | Ns | Ns | Ns | Ns |
| Control vs Welder | Ns | Ns | Ns | Ns | Ns | Ns |
| Control vs Painter | Ns | Ns | \*\*\* | Ns | Ns | Ns |
| Mechanic vs Welder | \* | Ns | \* | Ns | Ns | Ns |
| Mechanic vs Painter | \*\* | \*\* | \*\*\* | \* | \*\* | Ns |
| Welder vs Painter | Ns | \*\*\* | \*\*\* | \*\* | Ns | \*\* |

***Keys:*** *8-OHDG= 8-Hydroxyl-2’deoxyguanosine, Total SOD=Total Superoxide Dismutase, TAC= Total Antioxidant Capacity, TOS= Total Oxidant Status, OSI= Oxidative stress index, \*=Significant (P<0.05), NS=Not Significant (p>0.05). Reference Range: 8OhDG: 10-300 ng/L, Cardiac Troponin-I: <14ng/L, Total-SOD: 1.56-100 U/ml, TAC: 0.62-145.2 U/ml, TOS: 2.5-100 µmol/H2O2 Eqiv. /L, OSI: Normal <40, Borderline: 41-65, High: 66-120, Very High: >121.*

**Table 3: Comparison of 8-OHDG, Cardiac Troponin-I, and Oxidative Stress Markers to Moderate Intake of Alcohol by Controls, Mechanics, Welders, and Painters**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Subjects** | **8-OHDG (ng/l)** | **Cardiac Troponin-I (ng/l)** | **Total-SOD (U/ml)** | **TAC****(U/ml)** | **TOS(µmol /H2O2 Eqiv./L)** | **OSI (%)** |
| Mechanics n=15 | 53.8 ± 14.56 | 1.34 ± 0.16 | 71.91 ± 7.02 | 73.39 ± 4.56 | 14.54 ± 3.01 | 20.82 ± 4.29 |
| Welders n=8 | 26.81 ± 2.79 | 1.9 ± 0.17 | 84.34 ± 7.33 | 68.25 ± 6.46 | 31.5 ± 6.38 | 47.49 ± 8.87 |
| Painters n=14 | 29.92 ± 18.71 | 1.6 ± 0.18 | 73.57 ± 9.14 | 61.85 ± 5.15 | 25.56 ± 4.28 | 45.12 ± 9.22 |
| Controls | 9.09 ± 1.09 | 0.66 ± 0.12 | 11.59 ± 1.66 | 90.54 ± 5.81 | 4.78 ± 0.68 | 5.19 ± 0.59 |
| p-value | 0.1885 | 0.0002 | <0.0001 | 0.0059 | 0.0003 | 0.0003 |
| F-value | 1.666 | 8.122 | 16.31 | 4.99 | 7.899 | 7.862 |
| Tukey's Test | Summary | Summary | Summary | Summary | Summary | Summary |
| Mechanics vs Welder | Ns | Ns | Ns | Ns | \* | Ns |
| Mechanics vs Painter | Ns | Ns | Ns | Ns | Ns | \* |
| Mechanics vs Control | Ns | \* | \*\*\* | Ns | Ns | Ns |
| Welder vs Painter | Ns | Ns | Ns | Ns | Ns | Ns |
| Welder vs Control | Ns | \*\*\* | \*\*\* | Ns | \*\*\* | \*\* |
| Painter vs Control | Ns | \*\* | \*\*\* | \*\* | \*\* | \*\* |

***Keys:*** *8-OHDG= 8-Hydroxyl-2’deoxyguanosine, Total SOD=Total Superoxide Dismutase, TAC= Total Antioxidant Capacity, TOS= Total Oxidant Status, OSI= Oxidative stress index, \*=Significant (P<0.05), NS=Not Significant (p>0.05). Reference Range: 8OhDG: 10-300 ng/L, Cardiac Troponin-I: <14ng/L, Total-SOD: 1.56-100 U/ml, TAC: 0.62-145.2 U/ml, TOS: 2.5-100 µmol/H2O2 Eqiv./L, OSI: Normal <40, Borderline: 41-65, High: 66-120, Very High: >121.*

**Table 4: Comparison of 8-OHDG, Cardiac Troponin-I, and Oxidative Stress Markers to No Intake of Alcohol by Controls, Mechanics, Welders, and Painters**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
|  **Subjects** | **8-OHDG (ng/l)** | **Cardiac Troponin-I (ng/l)** | **Total-SOD (U/ml)** | **TAC (U/ml)** | **TOS (µmol /H2O2 Eqiv./L)** | **OSI (%)** |
| Mechanics n=5 | 37.54 ± 7.35 | 1.44 ± 0.26 | 79.04 ± 15.58 | 91.74 ± 7.04 | 19.52 ± 6.23 | 22.44 ± 7.15 |
| Welders n=8 | 63.41 ± 32.19 | 1.56 ± 0.21 | 73.84 ± 12.45 | 70.38 ± 5.67 | 26.09 ± 3.92 | 39.8 ± 7.11 |
| Painters n=6 | 13.82 ± 2.24 | 1.85 ± 0.33 | 69.98 ± 10.05 | 69.83 ± 6.96 | 29.77 ± 8.38 | 50.07 ± 18.75 |
| Controls n=25 | 8.84 ± 1.15 | 0.93 ± 0.11 | 21.39 ± 5.92 | 82.59 ± 4.51 | 5.04 ± 1.28 | 5.94 ± 1.30 |
| p-value | 0.0523 | 0.0101 | 0.0002 | 0.0758 | 0.0003 | 0.0011 |
| F-value | 2.88 | 3.733 | 9.487 | 2.541 | 8.775 | 6.995 |
| Tukey's Test | Summary | Summary | Summary | Summary | Summary | Summary |
| Mechanics vs Welder | Ns | Ns | Ns | Ns | Ns | Ns |
| Mechanics vs Painter | Ns | Ns | Ns | Ns | Ns | Ns |
| Mechanics vs Control | Ns | Ns | \*\* | Ns | Ns | Ns |
| Welder vs Painter | Ns | Ns | Ns | Ns | Ns | Ns |
| Welder vs Control | Ns | Ns | \*\* | Ns | \*\* | \* |
| Painter vs Control | Ns | \* | \*\* | Ns | \*\*\* | \*\* |

***Keys:*** *8-OHDG= 8-Hydroxyl-2’deoxyguanosine, Total SOD=Total Superoxide Dismutase, TAC= Total Antioxidant Capacity, TOS= Total Oxidant Status, OSI= Oxidative stress index, \*=Significant (P<0.05), NS=Not Significant (p>0.05). Reference Range: 8OhDG: 10-300 ng/L, Cardiac Troponin-I: <14ng/L, Total-SOD: 1.56-100 U/ml, TAC: 0.62-145.2 U/ml, TOS: 2.5-100 µmol/H2O2 Eqiv./L, OSI: Normal <40, Borderline: 41-65, High: 66-120, Very High: >121.*

This study reported an increase in 8 hydroxy-2’-deoxyguanosine (8-OHDG) and cardiac troponin in mechanics when compared with the controls, this finding is consistent with a similar research of Sirikamol et al. [11], they reported significant higher levels of 8-OHDG in some artisans that work in an automobile industry were batteries are repaired and recycled, the result also agrees with the findings of Szymanska et al. [12], who also reported significant increase in 8-OHDG concentration in some artisans exposed to arsenic heavy metal. 8-OHDG is a marker of oxidative DNA damage, and it is usually released into the urine or blood for excretion, when DNA is damaged, the nucleoside base 8-OHDG is synthesized to facilitate the repair of damaged DNA molecules, the increase in 8-OHDG in the serum of mechanics may be as a result of the generation of reactive oxygen species due to the exposure of the mechanics to arsenic heavy metal, when the levels of reactive oxygen species accumulated in the body passes the threshold of the antioxidant defence system, it is capable of inducing cellular responses that initiates the onset of disease processes, cancers, and improper functioning of some cells and organs, [13]. When reactive oxygen species attack DNA molecules, the body defence system immediately repairs the damaged DNA and during the process, 8-OHDG is formed, if it remains in the body for a long time especially during the period of DNA replication it may cause an abnormal binding of G:C to A:T leading to genetic mutations.

The result of this study reported elevated levels of 8-OHDG in welders, this corroborates with the reports of Ting-Tao et al. [14] they recorded elevated levels of 8-OHDG in ship yard welders, the increased levels of 8-OHDG in welders may be attributed to the fact that welding fumes are made up of a mixture of harmful components and heavy metals which are released into the air following the heating of metals by welders, these ultra-fine particles of welding fumes once inhaled gains entrance into the lungs and lodge deep into the alveolar of the lungs from there they gain entrance into the blood circulation. The accumulation of these fine particles of welding fumes leads to the formation of free radicals and ROS, the reactive oxygen species oxidizes different cellular components, including nucleic acid and other genetic materials of the DNA molecule causing oxidative DNA damage, the Haber-weiss and fenton reaction pathways are the major pathways chromium uses to generate free radicals. The lesion caused by oxidative DNA damage caused by ROS causes the formation of 8-OHDG as the antioxidant defence system savages the damaged DNA molecules. 8-OHDG can be excreted into the urine or plasma, hence the presence of 8-OHDG in urine or serum indicates oxidative DNA damage.

The high levels of cardiac troponin observed in the mechanics, painters, and welders when compared with the controls is in agreement with the findings of Ai-Min et al. [15], and Chowdhury et al [16] they both evaluated the effects of environmental heavy metals on the onset of cardiovascular diseases and they both reported that the chronic exposure to heavy metals was capable of causing different types of cardiovascular diseases, however the results from the study of Monrad et al. [17], contradicts the results of this study, they reported that the exposure to heavy metals is not capable of putting the exposed individuals at risk of developing cardiovascular diseases. The increase in cardiac troponin I in the artisans (mechanics, welders, and painters) may be due to the presence of accumulated high levels of heavy metals which may induce the production of reactive oxygen species, and free radicals, these ROS are capable of causing lipid peroxidation and the modification of low density lipoprotein cholesterol (LDL-C) to an oxidized form (ox LDL-C), the ox-LDL-C are a threat to the cardiovascular system as the immune cells can react with them to form another intermediate called foam cells which begins the formation of plaques in the arteries of the heart leading to blocked arteries and atherosclerosis [18]. Cadmium is a very toxic heavy metal capable of inducing many oxidative reactions. It has also been discovered to cause disruption and damage in the vascular arteries, veins, and capillaries altering the natural mechanisms of the heart’s endothelium leading to vasoconstriction, inflammation of the endothelium and thrombosis (endothelial dysfunction), all these may support the onset of atherosclerosis, heart attack, and other heart disorders. Lead is also capable of initiating the onset of oxidative stress due to its ability to rapidly stimulating the generation of reactive oxygen species, this process can lead to the breakdown of useful proteins and nucleic acid [19].

The increased levels of Total Superoxide dismutase (TSOD) in all the three groups of artisans did not correlate with the findings of Bot et al. [20] they reported lower levels of SOD, and GSH, in some artisans (mechanics, painters, petrol attendants, and battery repairers) in Jos and higher levels of MDA when compared with the controls. The results from the study of Olufunsho et al. [21] also disagree with the results of this study. They also recorded decreased levels Of SOD and elevated levels of MDA, in painters exposed to lead, however, Moro et al. [21] reported elevated levels of SOD, MDA, and CAT in painters exposed to lead and other chemicals. The increased levels of SOD observed in the artisans when compared with the controls may be as a result of the body trying to mop off the excess reactive oxygen species and free radicals generated by the heavy metals, superoxide dismutase is a cellular enzymatic antioxidant and it functions by modifying superoxide anions to hydrogen peroxide which is later excreted from the body by the action of the enzyme catalase.

Total oxidant status (TOS) was elevated in all the three groups of artisans, this may be due to frequent exposure to heavy metals and the constant build-up of free radicals and reactive oxygen species, overwhelming the antioxidant defence system of the body to eliminate them [23].

The result of this study indicated no significant difference in the mean values of TAC in the mechanics when compared with the controls, however, the welders and painters had significant lower levels of TAC when compared with the controls, also, higher levels of TOS, and OSI was also reported in the artisans in this study, this report correlates with the findings of Catherine et al. [24], and Ishiaq [25], the researchers recorded decreased levels of total antioxidant capacity (TAC) and higher levels of oxidative stress index (OSI) in some artisans. Total antioxidant capacity (TAC) measures both the endogenous (natural enzymatic) and exogenous (vitamins) antioxidants in the body, it was expedient to analyse this parameter as it encompasses all the antioxidant contents (both known and unknown) in the human body, hence it served as a very good marker instead of analysing different specific antioxidants, the significant decreased levels of TAC is attributed to the struggles of the body’s antioxidant defence system to regulate the levels of reactive oxygen species, but an increase in the generation and accumulation of free radicals due to the constant exposure to heavy metals may lead to higher levels of ROS as observed in the significant increase in TOS (total oxidant status) and lower levels of TAC because of the overwhelming effects in the antioxidant defence system. The elevated levels of Oxidative stress index in the artisans are attributed to the high levels of TOS, and lower levels of TAC, this report contradicts that of Arinola & Akinbiinu, [26], they reported high levels of TAC which may be as a result of increased levels of oxidants.

The result of this study also indicated that 8-OHDG (marker of DNA oxidation), cardiac troponin, and oxidative stress markers was elevated in the artisans that consume heavy amounts of alcohol when compared with the controls, alcohol may also play a role in the generation of reactive oxygen species during the process of its metabolism, alcohol is metabolized in the liver by the enzymatic action of alcohol dehydrogenase which converts the ingested alcohol to a toxic compound called acetaldehyde, this compound undergoes further enzymatic action to form acetate, which forms a molecule of nicotinamide adenine dinucleotide hydrogen (NADH), this leads to the generation of ROS, and more demand for oxygen. The acetaldehyde formed may also have a negative effect on macromolecules in the body like lipid and protein molecules and this reaction may also initiate the formation of ROS [27], this may be the reason for the elevated oxidative stress markers and cardiac troponin seen in the artisans that consume heavy amounts of alcohol. The result of this study showed that the painters that consume heavy amounts of alcohol are at more risk of developing diseases associated with oxidative stress.

The mean values of 8-OHDG, cardiac troponin, and oxidative stress markers of the artisans that consumed moderate quantities of alcohol showed that the welders had higher levels of TOS, when compared to the mechanics, and the painters had higher levels of OSI when compared to the mechanics, when the welders and painters were individually compared with the controls, cardiac troponin and all the oxidative stress markers were elevated in the artisans, this may be due to the different type of chemical components they are exposed to and the alcohol intake. TSOD, TOS, and OSI, was also significantly higher in the welders and painters when compared with the mechanics and controls that do not consume alcohol, this may suggest that the oxidative stress observed may not be completely related to alcohol intake but due to the exposure to the toxic chemicals and heavy metals they work with. The mechanics that do not consume had significant higher values of TSOD, while the welders had higher levels of TOS, and OSI, the painters had higher values of cardiac troponin, TOS, and OSI, this indicated that the oxidative stress observed in the artisans is more related to the exposure to heavy metals than alcohol intake.

4. Conclusion

The study showed that the exposure to heavy metals caused an increase in DNA oxidation, oxidative stress markers, total oxidant status, and a decrease in total antioxidant capacity and total superoxide dismutase. Oxidative stress can lead to the development of various pathological conditions.

Competing interests

Authors have declared that no competing interests exist.

Consent

All authors declare that written informed consent was obtained from the patient (or other approved parties) for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editorial office/Chief Editor/Editorial Board members of this journal.

Ethical approval

The ethical approval for this study was obtained from the Health Research Ethics Committee, Rivers State Hospitals Management Board (RSHMB). The participants of the study were required to sign an approved informed consent form before their participation in the study, and identification numbers were assigned to each participant instead of their names to protect their privacy.

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