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Evaluation of Biomarkers Oxidative Stress Parameters among Market Women using Local Lamp in Ibadan

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Abstract

The increasing global awareness of ambient air pollution from carbon monoxide (CO) and its side effect on the wellbeing of an individual is enormous. Thus, the complications and the health risk associated with air pollution through carbon monoxide cannot be ignored in developing countries. The study aimed at evaluating biomarkers of oxidative stress among market women using local lamp. A total of 200 research participants were recruited for the study, with 100 as subjects and 100 controls. Blood sample was collected and assayed for oxidative stress (SOD, GSH and MDA). Analysis of results was done with statistical package for social sciences (SPSS) version 20. The study depicts there was a significant decrease in the oxidative stress level of SOD and GSH of the test group when compared to the control group ($p<0.05$), and there was a significant increase in lipid peroxidation level MDA of test group when compared to the control group ($p<0.05$). There was no relationship of (age, frequent use of local lamp, and duration of local lamp usage) by the market women which is the subject when compare to the non-market women which is the control.

Conclusion raised from this present study involve that CO poisoning through the use of local lamp is associated oxidative stress among market women.

Keywords: Biomarkers; Oxidative; Stress; Parameters; Market; Lamp; Ibadan

Introduction

Human activities pollute the air we breathe, the water we drink, and the soil in which plants grow. Industrial activities result in massive amounts of pollutants being discharged into the air, which are hazardous to human health [1]. Without a question, global environmental pollution is a multifaceted international public health issue. Clearly, in our time, global urbanization and industrialization are reaching unprecedented and unsettling proportions. Anthropogenic air pollution is one of the world's most serious public health threats, accounting for around 9 million fatalities per year [2]. Carbon monoxide (CO) is a chemical molecule made up of carbon and oxygen. It's a colorless, odourless gas that's about 3% lighter than air and deadly to all warm-blooded mammals and many other kinds of life. When carbon or carbon-containing things are burned with insufficient oxygen, carbon monoxide is produced. It interacts with hemoglobin in the blood, limiting oxygen absorption and causing asphyxiation. Even though the amount of air is theoretically sufficient, the reaction is not always complete, resulting in some free oxygen and carbon monoxide in the combustion gases. There is considerable evidence on carbon monoxide exposure in humans from the environment and at work, as well as the levels of the particular biomarker COHb in blood and dose–effect associations for the most important health impacts [1]. The brain, circulatory system, exercising skeletal muscle, and the growing fetus are the organs and tissues most commonly affected [3]. Oxidative stress is a condition in which the physiological redox equilibrium is disrupted, resulting in an excess of oxidative free radicals and their derivatives. When present in physiological quantities, reactive oxygen species (ROS) operate as signaling molecules. When ROS and derived oxidative species such reactive carbonyl (RCS) and nitrogen species (RNS) are in excess, they limit the bioavailability of the anti-atherogenic vascular signaling molecule nitric oxide (NO) and activate pro-atherogenic redox sensitive signaling cascades [4]. Oxidative stress has been associated with many diseases such as diabetes, hypertension, heart failure, Parkinson disease, renal disease, epilepsy, Alzheimer's and other neurodegenerative diseases by clinical and post mortem studies [5]. It's also implicated to acute medical and critical care, as seen by increased oxidant activity in the lungs of patients with acute respiratory distress syndrome, which leads to multi-organ failure and death [5]. Many neonatal illnesses, such as retinopathy of prematurity, bronchopulmonary dysplasia, necrotizing enterocolitis, and periventricular leucomalacia, are linked to oxidative stress [6]. The pathophysiological mechanisms that lead to cellular damage and toxicity are known to be triggered by oxidative stress. Oxidative stress plays a role in CO toxicity, and it is possible to argue that oxidative stress is the primary cause of CO-related neuronal injury. Even though several mechanisms have been offered, the essential mechanism of brain harm caused by CO poisoning is still unknown. CO poisoning causes late alterations that are akin to post-ischemic reperfusion damage. CO-induced tissue hypoxia may be followed by CNS reoxygenation injury or any other tissue or organ injury [7]. Over the years, it has been reported that pathophysiology of carbon monoxide (CO) toxicity may cause alteration of free radical-mediated or ROS mediated cellular (e.g. erythrocytes) injury, as shown in both experimental animal studies and clinical studies. CO is known to produces oxidative stress and causes lipid peroxidation which increases the production of ROS, and leads to cellular damage and neurotoxicity [8]. However, effect of CO generated from local lamp users in relation to oxidative stress is yet to be established in developing countries e.g Nigeria. This present study is aimed to evaluate biomarkers of oxidative stress among market woman using local lamp in Ibadan.

Methodology

Materials

Materials used in this study include; Cotton wool, needle and syringe, plain bottle, Lithium heparin, methylated spirit, Gloves, micropipette, tourniquet, automatic micropipette, pipette tips, spectrophotometer, spectrophotometric cuvette, water bathe, vortex mixer, disposable test tubes, and Eppendorf centrifuge, NADPH reagent, NADPH diluents, assay buffer, hydrogen peroxide reagent, and microplates.

Study Area

The samples was collected from market women in Ibadan market, Oyo state after obtaining an ethical approval from Lead City University Research Ethics Committee Ibadan, Oyo state and an informed consent from the study participants.

Study design and Population

This study is a cross sectional study. A total number of 100 market women using local lamp (test) and 100 market woman who do not use local lamp (control) was recruited from different market in Ibadan metropolis, Oyo state, Nigeria.

Sample size calculation

The sample size for this study was obtained using the formula;

$$n = \frac{2 \times (Z_\alpha + Z_\beta)^2 \times P \times (1-P)}{(P_o - P_1)^2}$$

Where $P = (P_0 - P_1)\ |2$

*P*0 is the proportion of participants in the unexposed group exhibiting the outcome of interest.

*P*1 is the proportion of participants in the exposed group exhibiting the outcome of interest.

*P*0 =1% *P*1= 7.3% (Eberhardt et al., 2006)

Hence P = 4.05%

$Z\alpha = 1.96$ $Z\beta = 1.28$

$$n = \frac{2 \times (1.96 + 1.28)^2 \times 0.0405 \times (1-0.0405)}{(0.01-0.071)^2}$$

n= 219 study subjects

However, sample size of convenience for this study was 200. 100 subjects was used as control and 100 subjects served as test subject.

Ethical consideration

Ethical clearance was obtained from Lead City University Research Ethics Committee Ibadan, Oyo State. Individual subject that participated in this research was duly informed about the project and consent approval was obtained from such individuals.

Study Criteria

Inclusion Criteria: Adult market women using local lamp between the age of 18 and 55 and non-market women local lamp non-user of matching age were recruited in this study.

Exclusion Criteria: Subject outside the age bracket, subjects with a previous history of cancer, cardiovascular disease or chronic obstructive pulmonary disease was excluded from this research study.

Collection of Data

Questionnaire containing comprehensive questions relating to the participants' demography, knowledge of, attitude towards and belief about effect of using local lamp on their health status was administered to each of the research participants. Due to the level educational background among the market women, questionnaire was be prepared in English, Yoruba and Hausa language so that participants were free to choose the language with which they wish to be interviewed.

Sample collection

Two-third of the lithium heparin was filled with blood. The sample was centrifuged at 2500 r.p.m. Then the plasma was separated and analysed for oxidative stress parameters namely SOD, GSH and MDA

Sample Analysis

Superoxide Dismutase Activity

The level of super oxide dismutase (SOD) activity was determined.

Principle: The ability of superoxide dismutase to inhibit the autoxidation of adrenaline (epinephrine) at pH 10.2 makes this reaction a basis for a simple assay for this dismutase. Superoxide (O2 -) radical generated by the xanthine oxidase reaction caused the oxidation of epinephrine to adrenochrome produced per O2- introduced increased with increasing pH and also increased with increasing concentration of epinephrine. These results led to the proposal that autoxidation of epinephrine proceeds by least two distinct pathways, only one of which is a free radical chain reaction involving superoxide O2- radical and hence inhibitable by SOD.

Procedure: Sample (1mL) was diluted in 9 mL of distilled water to make a 1 in 10 dilution. An aliquot (0.2 mL) of the diluted sample was added to 2.5 mL of 0.05ml carbonate buffer (pH 10.2) to equilibrate in the spectrophotometer and the reaction started by the addition of 0.3 mL of freshly prepared 0.3mM adrenaline to the mixture which was quickly mixed by inversion. The reference cuvette contained 2.5 mL buffer, 0.3 mL of substance (adrenaline) and 0.2 mL of water. The increase in absorbance at 480 nm was monitored every 30 seconds for 150 seconds.

Methodology for glutathione peroxidase

Principle: Glutathione Peroxidase catalyzes the reduction of hydrogen peroxide (H2O2), oxidizing reduced glutathione (GSH) to form oxidized glutathione (GSSG). GSSG is then reduced by glutathione reductase (GR) and β-nicotinamide adenine dinucleotide phosphate (NADPH) forming NADP+ (resulting in decreased absorbance at 340 nm) and recycling the GSH. Because GPx is limiting, the decrease in absorbance at 340 nm is directly proportional to the GPx concentration.

Assay procedure

Standard Procedure for Microplate Assay: All reagents are brought to room temperature. After removing microplate from plastic bag, add 50 µL of diluted sample (or controls if present) to wells. 50 µL of working NADPH will be added each well. Then 50 µL of working H2O2 will be added to each well. Wait 1 minute, monitor A340 for 5 minutes with a recording interval of every 30 seconds. Calculate GPx activity from the net rate.

Determination of Thiobarbituric acid reactive substances (TBARS)

Lipid peroxidation levels were measured by the thiobarbituric acid (TBA) reaction. This method was used to measure spectrophotometrically the color produced by the reaction of TBA

with malondialdehyde (MDA) at 532 nm. For this purpose, TBARS levels were measured using a commercial Malondialdehyde Assay kit according to the manufacturer's instructions.

Principle of the assay

In the presence of acid, MDA reacts with TBA to produce a colored end product that absorbs light. The intensity of the color at 532 nm corresponds to the level of lipid peroxidation in the sample. Unknown samples are compared to the standard curve.

Procedure: Erythrocyte supernatant (50 µl) were added to test tubes containing 2 µl of butylated hydroxytoluene (BHT) in methanol. Fifty (50) µl of acid reagent (1 M phosphoric acid) was added and finally 50 µl of TBA solution was added. The tubes were mixed vigorously and incubated for 60 min at 60 °C. The mixture was centrifuged at 10,000 × g for 3 min. The supernatant was put into wells on a microplate in aliquots of 75 µl. Absorbance was measured with spectrophotometrically at 532 nm. TBARS levels were expressed as nmol/mg protein in various organs (brain, liver, pancreas and skeletal muscle), and as nmol/g hemoglobin in erythrocyte hemolysates.

Statistical Analysis

The data collected is analyzed statistically using the student t-test and the analysis of variance (ANOVA). Values will be deemed significant if P≤0.05. Correlation of parameters will be elucidated using the Pearson's correlation coefficient.

Results

(Figure 5) shows no significant correlation between duration of local lamp usage and SOD level among the study participants (p=0.496, $\chi 2$=3.384). (Figure 6) depicts no significant correlation between duration of local lamp usage and GSH among the study participants (p=0.311, $\chi 2$=4.781) (Figure 1-4).

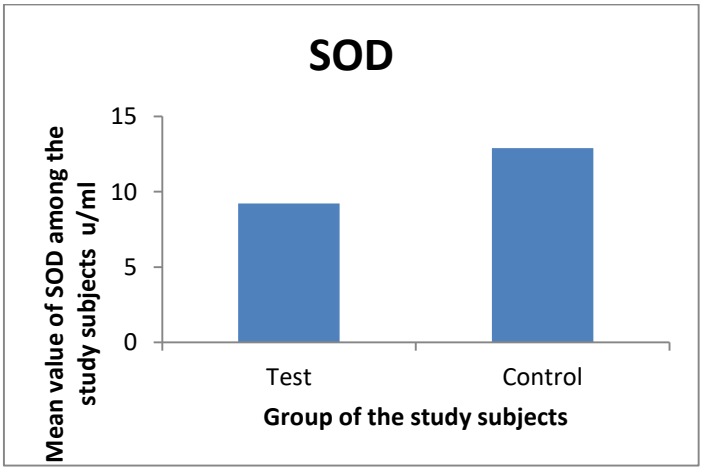

Figure 1: *Mean ± standard deviation of super oxide dismutase among the study subjects.*

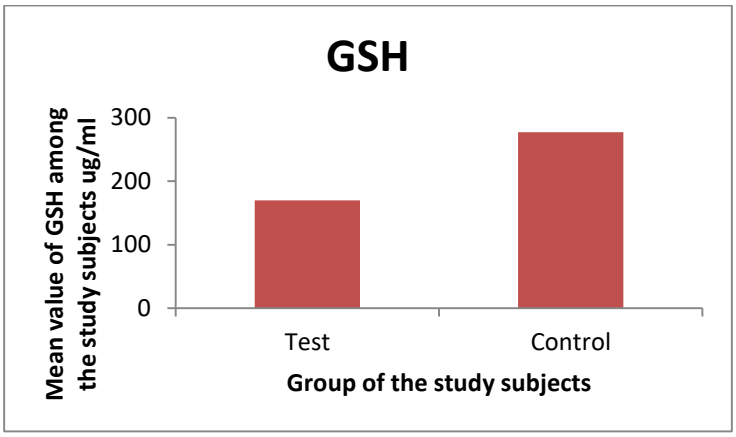

Figure 2: *Mean ± standard deviation of glutathione dismutase among the study subjects.*

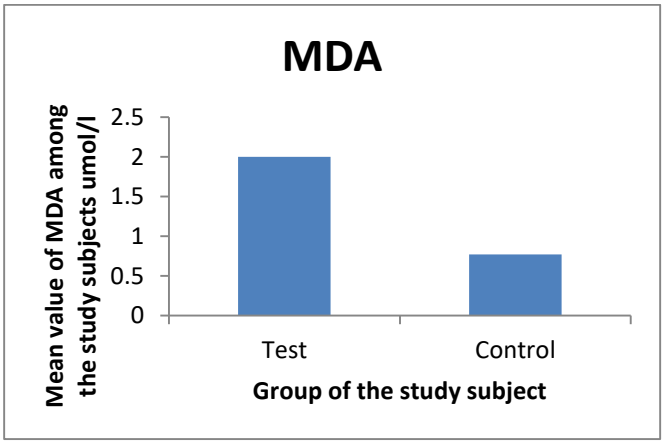

Figure 3*: Mean ± standard deviation of malonaldehyde among the study subjects.*

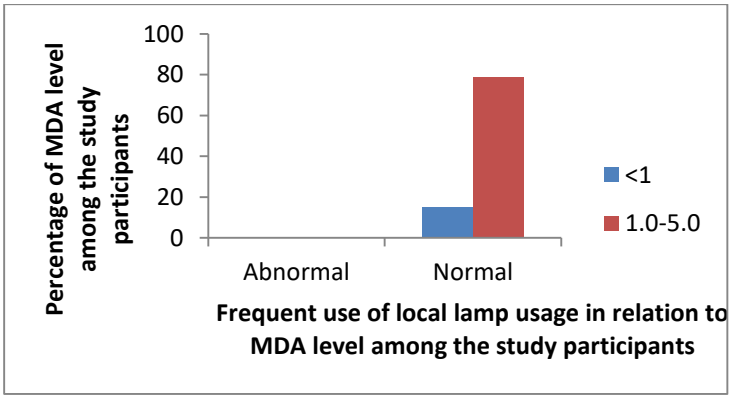

Figure 4*: Correlation on frequent of local lamp usage in relation to MDA among the study participants.*

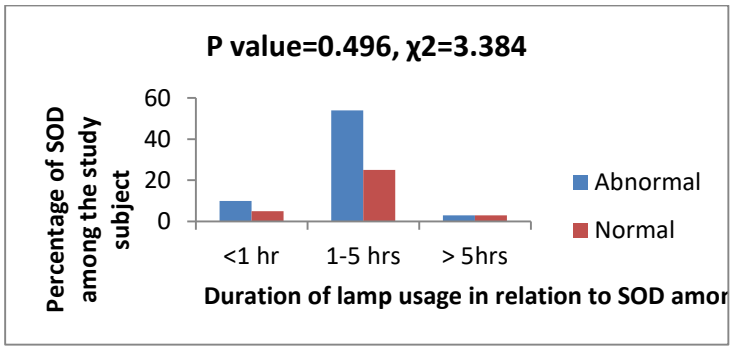

***Figure 5**: Correlation on duration of local lamp usage in relation to SOD among the study participants.*

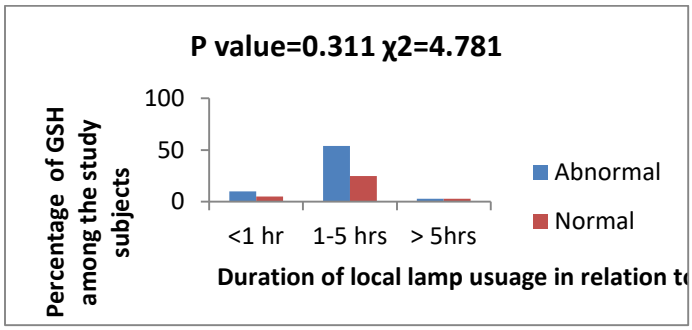

***Figure 6**: Correlation on duration of local lamp usage in relation to GSH level among the study participants.*

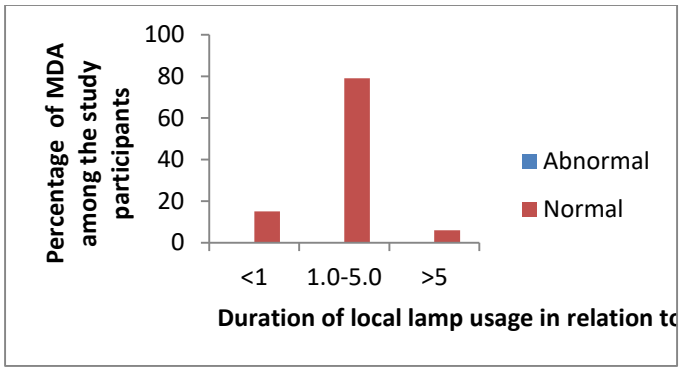

***Figure 7**: Correlation on duration of local lamp usage in relation to MDA level among the study participants.*

***Table 1**: Mean ± standard deviation of Oxidative Stress Parameters.*

Parameters	Test	Control	P-value
SOD	9.23±1.89	12.85±1.33	0.000*
GSH	169.82±14.57	277.39±35.50	0.000*
MDA	2.00±0.56	0.77±0.25	0.000*
Keys: SOD= Superoxide dismutase, GSH= Glutathione, MDA= Malonaldehyde Data presented as mean ± standard deviation * significant at p<0.05			

Discussion

Oxidative stress is defined as a disturbance in the balance between the production of ROS (free radicals) and antioxidant defenses in the body. The alteration in the balance between oxidants and antioxidants in favor of oxidants is termed as "oxidative stress" [9]. Regulation of reducing and oxidizing state is critical for cell viability, activation, proliferation, and organ function. In recent years, there has been more evidence that pathophysiology of CO poisoning may be the result of increased free radical-mediated or ROS mediated neuronal or cellular injury, as shown in both experimental animal and clinical studies [1, 10, 11, 12]. In this present study, we evaluated some of biomarkers of oxidative stress such as catalase, (CAT), superoxide dismutase (SOD) and malonaldehyde (MDA) among market woman using local lamp in Ibadan metropolis. Glutathione (GSH) and superoxide dismutase (SOD) are vital cellular redox buffer and they plays a pivotal role in the detoxification of MDA but also NO and other products of ROS-induced lipid peroxidation, such as 4-hydroxynonenal (4-HNE) [13]. In our study, we compared the level of antioxidant (SOD and GSH) in the test group to the control group. We observed significant decrease in the level of antioxidant of the test group exposed to CO poisoning through local lamp usage when compared to the control group. This outcome is consistent with the result of Teksam et al., [14] and Amiegheme et al., [15] who reported that the level of antioxidant enzymes including GSH-Px, GR, GSH, SOD and anti-ROS significantly decrease with time. Therefore, it was concluded that increased lipid peroxidation and decreased antioxidant enzymes can be responsible for CO-mediated delayed neuron damage [14]. The decrease in serum concentration of SOD observed in this study showed that the production of ROS is higher than the compensatory roles of SOD. The result of the study concurred with Ismail et al., [16] and Elhelaly et al., [17] which reported a decrease in concentration of SOD in comparison to the control with a significant negative with COHb concentration [16, 17]. Glutathione is utilized in vision and other photo-action in eye and also crucial in oxidative stress prevention [16]. The decrease as seen in the serum could be due to the insufficency of glutathione in the blood to counteract ROS produced by the CO [18]. In our study, plasma MDA level as a lipid peroxidation marker was found to significantly increase in the test subjects when compared to the control group. The outcome of significant increase in lipid peroxidation could be attributed to carbon monoxide poisoning from study subjects using local lamp. The outcome of our study corroborate with Yavuz et al., [19] who showed that MDA level was found increased at six (6) hours in rat after carbon monoxide (CO) poisoning. In another study by Teksam et al., [14], it was reported that plasma MDA level as a lipid peroxidation marker was found

increased during the initial period of poisoning. But, there was no significant difference at sixth hours after the poisoning. Findings from this present study depicts that CO absolutely increases lipid peroxidation among the market women using local lamp. However, it is still questionable whether the lipid peroxidation increased immediately or time-dependent after the CO poisoning [12, 15]. The socio-demographic pattern of age in this study shows that the highest percentage of age was between 41-50 years (32%). Our outcome in this study was to the report of Zengin et al., [20] who reported an average age of 35 years as the mean age among the study subjects on assessment of antioxidant status in patients with carbon monoxide poisoning. Also, another findings by Ntaji et al., [21] reported an average age of 24 years on knowledge of CO exposure. Previous study carried by Abbey et al., [22], stated that majority of his research participated 357 (72.86%) were in the age category of 25-34 years; that was followed by 103 (21.02%) at 35-39 years of age on maternal exposure to CO in the first trimester of pregnancy in the core Niger Delta.

***Table 2**: Distribution of socio-demographic status of the study participants.*

Parameter	**Test**	**Control**
Age	**Frequency (%)**	**Frequency (%)**
21-30	18 (18.0)	22 (22.0)
31-40	30 (30.0)	36 (36.0)
41-50	32 (32.0)	28 (28.0)
51-60	14 (14.0)	1 (1.0)
>60	6 (6.0)	13 (13.0)
Total	100 (100.0)	100 (100.0)
Marital status		
Single	14 (14.0)	4 (4.0)
Married	69 (69.0)	70 (70.0)
Divorced	6 (6.0)	18 (18.0)
Widowed	11 (11.0)	8 (8.0)
Total	100 (100.0)	100 (100.0)
Ethnicity		
Igbo	1 (1.0)	5 (5.0)
Yoruba	99 (99.0)	95 (95.0)
Total	100 (100)	100 (100.0)
Religion		
Islam	65 (65.0)	52 (52.0)
Traditional	2 (2.0)	0 (0.0)
Christian	33 (33.0)	48 (48.0)
Total	100 (100.0)	100 (100.0)

***Table 3**: Correlation of age in relation to SOD and GSH among the study participants.*

Parameter	**SOD**				
Age	**Abnormal**	**Normal**	**Total**	**χ2**	**P-value**
21-30	14	4	18	4.540	0.338
31-40	16	4	30		
41-50	27	5	32		
51-60	14	0	14		
>60	6	0	6		
Total	87	13	100		
	GSH				
Age	**Abnormal**	**Normal**	**Total**	**χ2**	**P-value**
21-30	12	6	18	5.521	0.238
31-40	16	14	30		
41-50	26	6	32		
51-60	9	5	14		
>60	4	2	6		
Total	67	33	100		

Keys: SOD= Superoxide dismutase, GSH= Glutathione,
Data presented as mean ± standard deviation
* significant at p<0.05

***Table 4**: Correlation of age in relation to MDA among the study participants.*

Parameter	**MDA**				
Age	**Abnormal**	**Normal**	**Total**	**χ2**	**P-value**
21-30	4	15	19	2.919	0.712
31-40	9	27	36		
41-50	4	24	28		
51-60	0	1	1		
>60	4	12	16		
Total	21	79	100		

Keys: MDA= Malonaldehyde
Data presented as mean ± standard deviation
***significant at p<0.05**

***Table 5**: Correlation of frequent use of local lamp in relation SOD and GSH among the study participants.*

	SOD				
Frequent use of local lamp	**Abnormal**	**Normal**	**Total**	**χ2**	**P-value**
Always	10	1	11	7.289	0.200
Sometimes	77	12	89		
Total	87	13	100		
	GSH				

Frequent use of local lamp	Abnormal	Normal	Total	$\chi 2$	P-value
Always	6	5	11	2.742	0.740
Sometimes	61	28	89		
Total	67	33	100		

Keys: SOD= Superoxide dismutase, GSH= Glutathione,
Data presented as mean ± standard deviation
* significant at $p<0.05$

This study depicts the highest percentage of age of those who uses local lamps are elderly women and most are married considering their marital status with respect to their age followed by single, widow and divorced. Ethnicity diversity among the study subjects depicts that majority of the subjects are Yoruba (99%) followed by Igbo (1%). This outcome was expected because the study was carried out among Yoruba tribe in the south west of Nigeria. The religious status of the study subjects shows that most of the study subjects practices Islam religion (65%), followed by Christianity (33%) and traditional religion (2%). The highest percentage of study subject that practices Islam in this study could be attributed to the fact that where the study was carried out is mostly dominated by Muslims. Regarding correlation between age and oxidative stress (SOD and GSH), there was no significant correlation between age and SOD ($p=0.388$, $\chi 2=4.540$), also there was no correlation between age and GSH ($p=0.238$, $\chi 2=5.521$) among the study participants. In is consistent with a similar study by Ramadhani et al., [23] who reported no significant effects on age, gender, smoking habit, and body mass index on SOD and GPX activities for the study subjects. Although in our present study was on SOD, GSH, and MDA, and not GPX. In vitro and in vitro studies by Ruan et al. [24] have suggested that MDA is a stable end product of lipid peroxidation that is associated with aging. However, in our present study, There was no significant correlation between age and MDA among the study participants ($p=0.712$, $\chi 2=2.919$). Regarding the correlation between the frequent use of the local lamp with oxidative stress parameters, this study shows no significant correlation between frequent use of local lamp and SOD ($p=0.200$, $\chi 2=7.289$) and GSH ($p=0.740$, $\chi 2=2.742$) respectively. Furthermore, regarding the correlation between duration of local lamp usage with oxidative stress parameters, this study depicts no significant correlation between duration of local lamp and SOD ($p=0.496$, $\chi 2=3.384$) and GSH ($p=0.311$, $\chi 2=4.781$) respectively.

Conclusion

This study showed that CO poisoning through the use of local lamp is associated with increased lipid peroxidation of MDA level and decreased antioxidant enzymes such as SOD and GSH among the market women. Therefore, it is established that oxidative stress parameters may be useful in evaluating intoxications for long-term outcomes as an early biochemical marker in carbon monoxide poisoning. Further studies must be done to establish the accurate therapeutic measures to eliminate carbon monoxide poisoning due to local lamp exposure, and reduce oxidative stress among the market women.

Conflict of Interest

The authors declare no conflicts of interest. The authors alone are responsible for the content and the writing of the paper.

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Authors' Contributions

The entire study procedure was conducted with the involvement of all authors.

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