Evaluation of nephrotoxic effect of lead exposure among automobile repairers in Nnewi Metropolis

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ABSTRACT

Background: Lead toxicity is one of the most common occupational hazards that affect several organs of the body, kidney inclusive. Auto repairers are exposed to lead in petrol, radiator, leaded battery, lead soldering wire, and spray paints, thus this study was designed to evaluate lead-induced nephrotoxic effect among automobile repairers.

Methods: A total of 80 male subjects within the age range of 20 and 65 years were recruited for this study. 50 subjects were occupationally exposed automobile repairers, of which 15 were electricians, 21 mechanics and 14 panel beaters/spray painters, whereas 30 were non-exposed students and staff from Nnamdi Azikiwe University, Nnewi campus. Blood sample was collected from these individuals and their blood lead levels were determined alongside creatinine, urea, uric acid, sodium, potassium, chloride, and bicarbonate.

Results: The results showed that the mean levels of blood lead is significantly higher in automobile repairers than in control group (P <0.05). The comparison of blood lead level among different categories of automobile repairers (electricians, mechanics, and panel beaters/spray painters) showed no significant difference (P >0.05). The evaluation of the renal function markers show that, there were significant increases in the mean serum concentration of creatinine, urea, and uric acid in the study group compare to the control subjects (P <0.05). However, there was no significance difference in the mean levels of sodium, potassium, chloride, and bicarbonate in study group when compared to the control group (P >0.05).

Conclusion: Findings from this study show that blood lead level is high among automobile repairers above CDC recommended level for adults. This high blood lead level among automobile repairers may be responsible for raised levels of renal markers which may eventually lead to their renal damage.

Keywords: Nephrotoxicity, Occupational workers, Lead, Renal function markers
INTRODUCTION

Lead is a naturally occurring toxic metal found in the Earth’s crust. It is dense, ductile, malleable and corrosion-resistant and these properties have made it useful in building materials, pigments to glaze ceramics, water pipes, glass, paints and protective coatings, acid storage batteries and gasoline additives. Its widespread use has resulted in extensive environmental contamination, human exposure and significant public health problems in many parts of the world.

Human exposure to lead is estimated to account for 143000 deaths every year and 0.6% of the global burden of disease. It is one of the most widely distributed toxic metals in our environment in addition to mercury, cadmium and arsenic. Although its toxic effects have been known for centuries, occupational exposure to lead that results in poisoning, be it asymptomatic or clinically symptomatic, is still common in many countries of the world. It ranks as one of the most serious environmental poisons amongst the toxic heavy metals all over the world.

Auto repairers (commonly called mechanics) represent one risk occupation, having direct contact with lead in their daily work. Lead toxicity among automobile mechanics is a common event occurring in some developing countries of the world. It is more common in under developed countries as a result of reduced knowledge about the hazardous effect of lead and ineffective quality control measures to check lead toxicity. Automotive service and repair shops are the largest small quantity generators of hazardous waste.

Lead toxicity is one of the most common occupational hazards that affect several organs of the body, kidney inclusive. Kidney damage occurs with exposure to high levels of lead, and evidence suggests that lower levels can damage kidneys as well. The toxic effect of lead causes nephropathy and may cause Fanconi syndrome, in which the proximal tubular function of the kidney is impaired. Lead poisoning inhibits excretion of the waste product urate and causes a predisposition for gout, in which urate builds up. This condition is known as saturnine gout. There is nephrotoxicity as a result of exposure to lead; this is because the kidney is the main route by which lead is eliminated.

Auto repairers are exposed to lead in petrol, radiator, leaded battery, lead soldering wire, and spray paints. Nigeria being one of the developing countries is expected to have high blood lead level among automobile repairers as a result of reduced knowledge about the hazardous effect of lead and ineffective quality control measures to check lead toxicity, thus this study was designed to evaluate lead-induced nephrotoxic effect among automobile repairers.

METHODS

Study population and design

This is a case-control study that was design to evaluate lead induce nephrotoxic effect and renal function markers between the test subjects and control subjects. A total number of 80 individuals randomly recruited into this study; 50 test subjects (exposed) and 30 control subjects (non-exposed). The study population comprised of individuals who are within the age bracket of 20 and 65 years. The control group was made up of individuals who are located in an environment with little or no lead exposure and also free from occupational exposure. The study group were made up of male automobile repairers in Nnewi metropolis from different mechanic villages (sites).

This research was conducted in the city of Nnewi, Anambra state of Nigeria where a good percentage of the population is involved in automobile repair sequel to its great involvement in automobile business. All consenting automobile mechanics were recruited for the study in their hall during their general monthly meeting and explanation about the study was given to them. Also close-ended questions were asked through questionnaire. An informed consent was obtained before recruitment.

The criteria for selecting test subjects for this research included: willingness to participate; must be a male; must be up to 20 years of age; must have worked for at least one year; must not be on any kind of chelation therapy or supplement that can chelate lead, must not have any history of kidney defect, and must not be a smoker, must be an occupational automobile repairer.

Sample collection, preparation and storage

5 ml of venous blood was collected from every participant and 1 ml of whole blood was dispensed into EDTA bottle for the analysis of blood lead while the remaining 4 ml was dispensed into a plain container. Blood was allowed to clot and separated by centrifugation and serum collected and stored frozen at -20°C until analysis of creatinine, urea, uric acid, sodium, potassium, chloride and bicarbonate using standard methods. All containers, needles and syringes used were lead-free. Questionnaire was issued to the participants to obtain other vital information about them.

Procedures

The uric acid was determined by the method of Henry et al., urea and creatinine were determined by the method of Coulombe and Faureas and Taursky respectively. Lead was determined by Atomic Absorption Spectrometry (AAS) as described by the method of Hassel while electrolytes (sodium, potassium, chloride and bicarbonate) were done using ion-selective electrode analyzer, 4000 (SFRI, France).
**Statistical analysis**

The data for both study and control groups were subjected to statistical analysis using the Statistical Package for Social Sciences (IBM SPSS version 20, USA). The mean, percentage, standard deviation, students’-t-test, ANOVA and Pearson correlation coefficient were used for the analysis and P <0.05 was considered significant.

The graphical presentations were done using SigmaPlot 12 (systat software, USA).

**RESULTS**

There is a significant increase in the mean serum level of lead, creatinine, urea and uric acid in automobile repairers than in the control subjects (P <0.05). Whereas, there were no significant differences in the mean serum level of electrolytes (Na⁺, K⁺, Cl⁻ and HCO₃⁻) between the two groups (P >0.05) (Table 1).

**Table 1: Comparison of lead level and other renal markers in automobile repairers and control subjects.**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Study group N=50</th>
<th>Control group N=30</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lead (µg/dl)</td>
<td>64.75 ± 24.78</td>
<td>15.10 ± 11.85</td>
<td>0.000</td>
</tr>
<tr>
<td>Creatinine (mmol/L)</td>
<td>98.96 ± 23.67</td>
<td>75.30 ± 24.46</td>
<td>0.003</td>
</tr>
<tr>
<td>Urea (mmol/L)</td>
<td>3.75 ± 1.53</td>
<td>2.80 ± 0.97</td>
<td>0.010</td>
</tr>
<tr>
<td>Uric acid (mmol/L)</td>
<td>200.60 ± 56.51</td>
<td>172.73 ± 36.89</td>
<td>0.010</td>
</tr>
<tr>
<td>Na⁺ (mmol/L)</td>
<td>135.86 ± 5.73</td>
<td>136.47 ± 2.97</td>
<td>0.591</td>
</tr>
<tr>
<td>K⁺ (mmol/L)</td>
<td>3.95 ± 0.35</td>
<td>3.66 ± 0.37</td>
<td>0.423</td>
</tr>
<tr>
<td>Cl⁻ (mmol/L)</td>
<td>95.33 ± 3.16</td>
<td>96.10 ± 2.28</td>
<td>0.247</td>
</tr>
<tr>
<td>HCO₃⁻ (mmol/L)</td>
<td>21.96 ± 2.10</td>
<td>22.67 ± 2.68</td>
<td>0.196</td>
</tr>
</tbody>
</table>

**Table 2: Association between the duration of exposure and other parameters in automobile repairers.**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>R</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration of exposure vs. lead</td>
<td>0.403</td>
<td>0.027</td>
</tr>
<tr>
<td>Duration of exposure vs. creatinine</td>
<td>0.068</td>
<td>0.712</td>
</tr>
<tr>
<td>Duration of exposure vs. urea</td>
<td>0.028</td>
<td>0.879</td>
</tr>
<tr>
<td>Duration of exposure vs. uric acid</td>
<td>-0.105</td>
<td>0.567</td>
</tr>
<tr>
<td>Duration of exposure vs. sodium</td>
<td>0.094</td>
<td>0.610</td>
</tr>
<tr>
<td>Duration of exposure vs. potassium</td>
<td>-0.053</td>
<td>0.773</td>
</tr>
<tr>
<td>Duration of exposure vs. chloride</td>
<td>0.082</td>
<td>0.654</td>
</tr>
<tr>
<td>Duration of exposure vs. bicarbonate</td>
<td>0.208</td>
<td>0.252</td>
</tr>
</tbody>
</table>

Result for Pearson correlation between the duration of exposure and blood lead level, creatinine, urea, uric acid and electrolyte (Na⁺, K⁺, Cl⁻ and HCO₃⁻) in the test (exposed) subjects. The result for this correlation analysis shows that, there was a significant correlation between duration of exposure and blood lead level (P <0.05). However, there were no significant correlations between the duration of exposure and serum concentration of creatinine, urea, uric acid and electrolytes (Na⁺, K⁺, Cl⁻ and HCO₃⁻) in the exposed subjects (P >0.05) (Table 2).

Graphical presentation of result for the blood lead level for different specialization among the automobile repairers shows that there was no significant difference (P >0.05) in the mean blood level of lead among the different specializations in the test subjects (Figure 1).

**Figure 1: Comparison of lead levels among different specialties of automobile repairers.**

Graphical presentation of percentage distribution of individuals among the test subjects who mouth pipette fuel (87%), wash hand with fuel (100%), wear nose mask (3%) and drink alcohol (63%) (Figure 2).

**Figure 2: Presentation of percentage distribution of individuals among the test subjects who mouth pipette fuel, wash hand with fuel, wear nose mask and drink alcohol.**

Graphical presentation of percentage distribution of individuals among the test subjects who experienced abdominal pain (15%), diarrhoea (19%), vomiting (3%), muscle pain (73%) loss of appetite (3%) and headache (80%) (Figure 3).
In a work carried out by Olayiwola, 3,14,15 statistically significant difference in their blood lead levels, though the mean blood lead level is higher for automobile repairers, the one group and control subjects. This could be due to poor sensitivity of electrolytes as renal function markers and the reserve capability of the kidney to maintain normal function even after lost of about 50% of nephron. Conversely, work done by Odigie et al.16 in rats exposed to lead shows that there were statistically significant difference (P <0.05) in serum mean levels of Na⁺, K⁺, and Cl⁻ in the test group compared to control.

CONCLUSION

Findings from this study show that blood lead level is high among automobile repairers above CDC recommended level. This high blood lead level among

Urea is another marker of the kidney function which concentration in the plasma tends to increase in impaired kidney state. Result from this work shows that the mean serum urea concentration in test subjects was significant higher than that of control subjects. Meanwhile, both means are found within reference range. The findings from this work is not consistent with work done by Dioka et al.,17 though, their results were within the reference range, but there was no statistical significance difference in the mean serum level of urea between the exposed and control subjects. Increase serum urea could be due to slow impairment of glomerular basement membrane caused by the deposition and accumulation of lead in the kidney.

The mean serum concentration of uric acid is significantly higher in automobile repairers than in control subjects. This finding is in consonance with other research works.17,18 The deposition of lead in the proximal tubules of the nephron causes the alteration in structure and function of the cell, thereby decreasing renal tubular secretion of uric acid into the lumen. These effects of lead can lead to increase plasma accumulation of uric acid which can subsequently lead to gout.9 Uric acid has been suggested as one of the antioxidants in plasma.19 Therefore elevations in uric acid levels in the exposed subjects in this study may also be as a result of antioxidant response to lead toxicity.

The mean serum electrolytes (Na⁺, K⁺, Cl⁻ and HCO₃⁻) level was not significantly different in both automobile group and control subjects. This could be due to poor sensitivity of electrolytes as renal function markers and the reserve capability of the kidney to maintain normal function even after lost of about 50% of nephron. Conversely, work done by Odigie et al.16 in rats exposed to lead shows that there were statistically significant difference (P <0.05) in serum mean levels of Na⁺, K⁺, and Cl⁻ in the test group compared to control.

Figure 3: Presentation of percentage distribution of individuals among the test subjects who experienced abdominal pain, diarrhoea, vomiting, muscle pain, loss of appetite and headache.

DISCUSSION

Apart from the usual sources of lead exposure which include food, pipe borne water (from leaded pipes), fume from vehicles, cosmetics, and old paints, one of the major sources of lead toxicity is occupational exposure. Automobile repairers (commonly called mechanics) are regularly exposed to different kinds of heavy metals including lead. In a work carried out by Olayiwola, wherein sand from different mechanic villages were sampled, he found out that mean concentration of lead as high as 91 mg/kg can be found in the soil where automobile activities are being carried out.6

In this study, the mean blood level of automobile repairers was significantly higher than the control group. More so, the mean blood lead level of the automobile repairers was found to be above the CDC’s reference range of <10 mcg/dL) for adults.13 The results obtained in this study is consistent with the results of other studies carried out on the determination of the blood lead levels of occupationally exposed individuals.3,14,15 This increase in blood lead level might be due to reduced knowledge about the hazardous effect of lead and ineffective quality control measures to check lead toxicity since these works were done in developing countries.

Among the different specialization of these automobile repairers, the one-way analysis of variance shows that there is no significant difference in their blood lead levels, though the mean blood lead level is higher for mechanics followed by panel beaters/painters with the least being electricians. This is contrary to the findings of Suwansaksri et al.15 and Yalemsew et al.,3 where spray painters have a higher mean blood value compared to mechanics. Statistical analysis of their questionnaire shows that only 3% of the automobile repairers wear nose mask, 87% mouth pipette fuel while 100% wash hand with fuel. This is an indication that the major sources of exposure are inhalation, ingestion and dermal absorption.

This study shows that the mean serum creatinine level of the automobile repairers was significantly high compared to that of control subjects. This is in line with a study done to determine the effect of lead on the kidneys.16 This however, is contrary to the findings of Dioka et al.17 carried out on artisans occupationally exposed to lead in which there was a decrease in mean serum creatinine level of exposed individuals compared to non-exposed. Although the mean serum level of creatinine is significantly high compared to that of control in this study, both are however, within the normal range.

The results obtained in this study is consistent with the results of other studies 3,14,15 means are found within reference range. The findings from this work is not consistent with work done by Dioka et al.,17 though, their results were within the reference range, but there was no statistical significance difference in the mean serum level of urea between the exposed and control subjects. Increase serum urea could be due to slow impairment of glomerular basement membrane caused by the deposition and accumulation of lead in the kidney.
automobile repairers may be responsible for raised levels of renal markers which may eventually lead to their renal damage. We advocate government involvement in increase awareness on the toxicity of lead among automobile repairers so that proper workplace regulation for lead will be adhered to by these workers.

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Conflict of interest: None declared
Ethical approval: The study was approved by the ethical board of the faculty of health sciences and technology, Nnamdi Azikiwe University, Nnewi campus

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