

ARE ROADSIDE PETROL DISPENSERS AT RISK OF RENAL DYSFUNCTION? A STUDY FROM GOMBE, NORTH EAST NIGERIA.

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ABSTRACT

Background: Occupational exposure to toxic chemicals is a major public health concern worldwide. Gasoline, especially the leaded form is a leading cause of this occupational exposure in developing countries such as Nigeria. Exposure to gasoline has been shown to generate oxygen free radicals which are implicated in the pathogenesis and progression of many diseases including renal dysfunction. The objective of the study is to evaluate the possibility of increased risk of renal dysfunction among roadside petrol dispensers in Gombe, Nigeria. **Objectives:** To determine the risk of renal dysfunction among road side petrol dispensers in Gombe. **Materials And Methods:** A cross sectional analytic study, where serum levels of uric acid, urea and creatinine were compared between 90 road side gasoline dispensers and 90 matched controls. These were measured using standard colorimetric methods. **Results:** The mean age of the exposed and control groups are 29.03 ± 3.7 and 29.24 ± 3.5 years respectively. The plasma level of uric acid (5.35 ± 0.9 mg/dl) of the exposed group was significantly ($p < 0.05$) higher than the control (4.48 ± 0.9 mg/dl). There was no significant difference in the plasma levels of urea and creatinine in the two groups studied. **Conclusion:** This study has shown that road side gasoline dispensers may be at a higher risk of renal impairment. Creating awareness of this risk among roadside gasoline dispensers could help reduce the burden of renal disease associated with exposure to gasoline.

KEYWORDS : Occupational Exposure, Petrol, Chemical Exposure, Renal Function.

INTRODUCTION

Gasoline is one of the fractionated products of crude oil used for fuelling automobiles and some power generating machines. It is made from processed crude oil and is a pale brown

liquid with a strong odour. It can form explosive mixture in air. It is very complex, volatile and inflammable with many organic and inorganic saturated and unsaturated hydrocarbons containing 3 to 12 carbons¹⁻². The constituents depend on the location of origin, processing techniques, the season and the additives added (anti knock) to enhance performance. Its composition also varies with the source of crude petroleum, the manufacturer and time of the year. Commonly, gasoline contains about 62% alkanes, 7% alkenes and 31% aromatics, alcohols, ethers and additives².

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Studies have shown that chronic exposure to gasoline pollutant gases is more common in oil drillers, refinery workers, petrochemical industry workers, refuel station attendants, drivers and motor mechanics²⁻⁴. Increase plasma levels of some constituents of gasoline



like benzene, lead and cadmium is found to be associated with increase reactive oxygen production and the chronic diseases associated with it including renal impairment⁵. Other constituent of gasoline associated with renal diseases are alkylbenzenes (Toluene and xylene). They are single ring aromatic hydrocarbons with one or more saturated aliphatic side chains. They are mainly absorbed by inhalation and through the skin. Studies have found association of these compounds with various chronic diseases such as kidney diseases, hearing loss, CNS disorders, liver, and heart diseases⁵⁻⁶.

Other components of gasoline implicated in nephrotoxicity are the organic compounds of leads such as tetramethyl and tetraethyl lead which are alkyls of lead and were developed as octane enhancers. Tertiary butyl alcohol (TBA) has also been found to be associated with nephropathy and renal tubular acidosis. Some studies have also found them to be neurotoxic⁷. Other effects found by investigators are, haematologic alterations, hypertension, growth and development deficiencies, and impairment of immune system responses⁸.

Risk of occupational exposure to gasoline is found even in well-organized setting of gasoline stations. This risk could even be higher among roadside dispensers of gasoline in Nigeria. They often use their mouth to create vacuum pressure to dispense the products through pipes into receivers. Like other known xenobiotics, the chemical pollutants from gasoline vapours may be metabolically transformed into various metabolites in the body. Some of these metabolites may be very reactive, interacting in various ways with the metabolizing, transporting and excretive tissues to elicit toxic effects. The interaction of these metabolites with renal tissues may cause cellular injury and hence damage to the tissues⁵. Once the renal tissues are damaged, the overall functionality of the kidneys maybe compromised. Individuals with renal dysfunction may have a variety of different

clinical presentations⁹. Some of these presentations may be asymptomatic, only detected on routine laboratory investigations which may include abnormal serum catabolites (such as uric acid, urea and creatinine) and electrolytes like the sodium, potassium and chloride⁹.

There is paucity of studies that looked at occupational exposure to gasoline especially in Northern Nigeria and to our knowledge, there was no study conducted among roadside dispensers of gasoline. This study, therefore evaluated the renal function among roadside gasoline dispensers in Gombe State, Nigeria.

MATERIALS AND METHODS

Study Area

This is a cross sectional analytic study conducted in Gombe, the capital city of Gombe State, Nigeria. Gombe metropolis has an estimated population of 319,875 and lies within latitude 10° 17' N and 10° 283' N and longitude 11° 10' E and 11° 167'E of the Greenwich Meridian. Is a tropical area with two seasons (rainy – May to October and dry –November to April). The climate is Sudan Savanna with temperatures ranging between 18° to 39° and annual rainfall of about 954mm¹⁰. The study was conducted in the dry season between January and April, 2012.

Study Sample

Ninety apparently healthy roadside dispensers of gasoline and 90 matched controls in Gombe were recruited using random sampling method in two randomly selected motor packs in Gombe (Dukku and Bauchi packs). The study was explained to the participants and their voluntary consent was obtained.

Ethical Consideration

The study was approved by the joint Ethical Review Committee of the University of Ibadan/University College Hospital, Ibadan and Ethical Review Committee of Gombe State Ministry of Health.



Inclusion Criteria

Apparently healthy, full time roadside dispensers of gasoline that are one year and above in the trade were included in the study.

Exclusion Criteria

People with occupational exposure to gasoline or occupational exposure that can lead to renal dysfunction are excluded from the study. These include mining, welding, battery, roadside mechanics, petrol station attendants and people around Ashaka cement company. People with acute or chronic illnesses that can affect renal function were also excluded. They include malaria, diabetes, chronic renal failure, hypertension and cigarette smokers.

Anthropometric Measurements

Height: - This was measured to the nearest centimetre against a flat, vertical surface with the subjects standing upright. A sliding headpiece was brought to the vertex of the subject's head and the reading at this level was taken.

Weight: - This was taken with Salter bathroom scale placed on a flat surface. The reading was recorded to the nearest 0.5kg. Body mass Index (B.M.I) was then calculated using the formula
$$\text{BMI (kg/m}^2\text{)} = \frac{\text{weight in (kg)}}{\text{Height in (m}^2\text{)}}$$

Blood pressure was measured using digital sphygmomanometer. Random plasma glucose was done using Acucheck glucometer.

Sample Collection and Laboratory Procedures

Four milliliters of fasting venous blood was collected from each of the 180 participants into a heparinised plastic tube. Plasma was separated by centrifugation and frozen. Samples were stored at -20°C till the time of analysis.

Uric Acid, Urea And Creatinine Assay

Plasma uric acid, urea and creatinine were measured using standard colorimetric methods.

Statistical Analysis

The data was analyzed using SPSS (version 20 Chicago IL). Qualitative data were reported using percentages. The mean, standard deviation, skewness and kurtosis were used to measure the normality of distribution of the quantitative variables. The mean and standard deviation (SD) was reported for quantitative data and comparison was made between the cases and controls.

The normally distributed variables were compared between the two groups using two tailed unpaired Student's T-test. The level of significance was fixed at the 5% probability level. Pearson correlation coefficient was used to establish correlation between uric acid and the duration of exposure among the exposed groups.

RESULTS

Fifty per cent (90) of the respondents were roadside dispensers of gasoline. Among the controls, 34 (37.8%) were traders, 17(18.9%) were students, 10(11.1%) were teachers, 9(10%) were peasant farmers, and 20(22.2%) were other occupations (Tables 1). Fulanis made up 103(57.2%) of the respondents while 46(25.6%) were Hausa. Bolewa, Tera and others made up the remainder 30(17.2%). None of the exposed population uses face mask.

Table 2 shows the average duration of exposure among the exposed group. Plasma uric acid was significantly higher (P = 0.001) in the exposed (5.35 ± 0.9) than the control group (4.48 ± 0.9). Urea and creatinine were not significantly different between the exposed and the controls as detailed in table 3. Table 4 shows the Pearson correlation between total duration of exposure in hours and uric acid. There was a significant positive correlation between the duration of exposure and uric acid level (r = 0.63, p <0.001).



Table I: Occupational distribution of the respondents

| OCCUPATION | Frequency | Percent |
|-----------------|------------|--------------|
| GASOLINE SELLER | 90 | 50.0 |
| TRADER | 34 | 18.9 |
| TEACHER | 10 | 5.6 |
| FARMER | 9 | 5.0 |
| STUDENT | 17 | 9.4 |
| OTHERS | 20 | 11.1 |
| Total | 180 | 100.0 |

OTHERS: Doctors, nurses, fisher men, photographers, drivers and tailors.

Table II: Average duration of exposure among the exposed population

| | Mean | Std. Deviation |
|------------------------------|-------|----------------|
| DURATION OF EXPOSURE (YEARS) | 6.4 | 2.4 |
| AVERAGE WORK HOURS PER DAY | 7.9 | 0.71 |
| TOTAL EXPOSURE (HOURS) | 18332 | 6931.3 |

Table III: Student's t- test for equality of means between exposed and controls

| VARIABLE | EXPOSED | CONTROLS | P-VALUE |
|-------------------|------------|------------|---------|
| | MEAN (SD) | MEAN (SD) | |
| Uric acid(mg/dl) | 5.35(0.9) | 4.48(0.9) | 0.001 |
| Urea(mg/dl) | 36.19(5.1) | 35.10(5.6) | 0.167 |
| Creatinine(mg/dl) | 0.76(0.13) | 0.77(0.15) | 0.526 |

TABLE IV: Pearson correlation (r) of duration of exposure and uric acid

| | R | p |
|------------------------------|-------------|--------------|
| Total exposure (hours) | 0.63 | 0.000 |
| Duration of exposure (years) | 0.61 | 0.000 |

R (Pearson correlation), p (p value)



DISCUSSION

This study found significantly higher levels of uric acid among the exposed group compared to the control group with significantly positive correlation with the duration of exposure. The mechanism of increased serum uric acid concentration in conditions associated with exposure to hydrocarbons has been largely described as unknown¹¹⁻¹⁶. Some suggested it could be due to increased breakdown of nucleic acids in DNA and RNA caused by oxidative injury. This is in addition to the renal tubular damage that may cause increase in reabsorption or decrease secretion of uric acid by the tubules^{3,17-18}. Other suggested mechanisms are increased metabolism of adenosine generated by ischaemic tissues¹⁹, loss of the inhibition of xanthine oxidase caused by nitric oxide²⁰, and impaired oxidative metabolism²¹. The uric acid elevation may be a protective response, to reduce the harmful effects of reactive oxygen species (ROS) generated by the petrochemicals. Therefore, although both clinical and experimental evidences suggest that uric acid has antioxidant properties, it is conceivable that its antioxidant activity could be overcome by the pro-oxidant and pro-inflammatory effects of ROS accumulation under certain conditions²².

The higher levels of uric acid among the roadside petrol dispensers found in this study is a risk for renal dysfunction which may be mediated through inducing hypertension by uric acid²³. Some studies have found uric acid to be an independent predictor of microalbuminuria which is by its self a predictor of renal dysfunction²⁴, and is also found to have correlated with electronic GFR (eGFR)²⁵. Renal dysfunction may also be mediated through urate crystals deposits in the kidney²⁶.

The finding in this study of higher levels of uric acid among the petrol exposed group is similar to the finding of some researchers in Nigeria. In Nnewi, Dioka et-al found elevated uric acid

among occupationally exposed individuals. However, urea and creatinine were not elevated. This is in keeping with our findings. The considerable reserve capacity of the kidneys and uric acid been an early marker for renal dysfunction may explain the lack of significant differences in urea and creatinine between the two groups¹⁵. In Port Harcourt, Nigeria, Alasia et-al made a finding of elevated uric acid, urea and creatinine among occupationally exposed individuals. Similar findings were observed in Gaza when petrol station workers were studied^{14,18,27}. Impaired markers of renal function were also observed in Owerri¹¹, South-East Nigeria among petrol station attendants which also agrees with the finding in our study. Although the effects of inhaled gasoline on renal tissue might not be dependent on age and sex, it is probable that the effects could depend on the exposure time, as suggested by the finding of positive correlation between uric acid and the duration of exposure in this study. Exposure time dependent renal dysfunction was also observed among petrol station attendants in Owerri, Nigeria¹¹. This is possibly as a result of the phenomenon of bioaccumulation which is associated with the direct transfer of compounds through body surface into the circulatory fluids in a process known as bioconcentration²⁸.

LIMITATIONS OF THE STUDY

Measurement of urinary benzene or its metabolites, lead or cadmium would have given a more objective level of exposure than the duration used in the study. A study that will monitor the progression of the renal function indices or their reversal by interventions is needed to produce a more causal relationship.

CONCLUSION

In conclusion, this study has demonstrated the probability of some risk for renal impairment among roadside gasoline dispensers. Further study to establish a link between gasoline exposure and serum urea and creatinine is recommended. ■



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