Biochemical Evidence of Aggravated Hepato-renal Damage in Alcohol Consuming Male Mechanics Occupationally Exposed to Petroleum Products

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Abstract

Background: Harmful lifestyle choices are sometimes found to co-exist with occupational hazard in some individuals yet the possible modulating effect of one on the other in some occupational types remains undetermined. The present study is designed to investigate synergetic effects of alcohol consumption and petroleum product exposure on hepato-renal function in auto-mechanics.

Material and methods: Of the 80 mechanics recruited for the study, 35 abstained from alcohol consumption; 45 others consumed alcohol whereas of the 75 participants not exposed to petroleum products, forty were alcohol consuming but unexposed to fuel, the remaining thirty-five were non-fuel, non-alcohol exposed subjects. Hepato-renal markers were determined using serum obtained from 5 mL of venous blood. Analysis of variance was used to test the statistical difference; p value of < 0.05 was considered significant.

Results: In alcohol and non-alcohol consuming auto-mechanics markers of hepato-renal function were significantly altered compared with control but these alterations were more pronounced in alcohol consuming mechanics.

Conclusion: Evidence obtained from the study indicates alcohol consumption is capable of aggravating hepato-renal abnormalities associated with petroleum product toxicity.

Keywords: alcohol, auto-mechanics, renal, hepatic

Introduction

Fuel (e.g. petrol) is a flammable liquid that is made up of a complex mixture of hydrocarbons such as benzene, toluene and xylene (1,2). These compounds are known for their general hazardous effects but the carcinogenic potency of benzene has also been identified (3). Therefore, exposure to these compounds may have a negative effect on the health of the exposed subjects (4). Occupational exposure of automobile mechanics to petrol and diesel is a lingering problem in many parts of the world (1,5,6), while it continues to be a serious problem, receiving attention from many experts in different fields, may not be unassociated with a lack of the basic knowledge on the part of these mechanics of the toxic nature of petroleum products.

A potentiating role of smoking in petrol-induced toxicity has been defined (7,8) but the modulating impact of alcohol consumption on petroleum product-induced toxicity in auto-mechanics has not been fully established. Petrol contains aromatic and aliphatic hydrocarbon compounds known for their toxic effects on a number of organs in the body. Being xenobiotics, their metabolism through the cytochrome P 450 (CYP 450) can result in excessive generation of free radical that can cause oxidative stress. When these toxic components (benzene, toluene and xylene) are co-administered or co-exposed with edible alcohol-ethanol, known to be an inducer of some of the enzymes of the CYP 450, the possibility of a
more pronounced degree of toxicity cannot be discounted. The aim of this study is to determine the impact of combined
effects of alcohol intake and prolonged exposure to fuels on hepatic and renal cells in automobile mechanics based within
Ibadan metropolis.

Materials and methods
A group of 80 adult automobile mechanics working in mechanic workshops were used for the pilot study, thirty-five of
whom abstained from alcohol consumption; the other forty-five consumed alcohol. They were male subjects recruited
from mechanic workshops situated at different locations within Ibadan metropolis. The unexposed group consisted of 75
healthy men that were recruited from general population, forty of them are alcohol consuming but unexposed to fuel, the
remaining thirty-five are fuel unexposed subjects who also have not consumed alcohol for more than 7 years. The two
alcohol consuming groups were matched, they were social (alcohol) drinkers who consumed alcohol at least once a week,
but not necessarily binge drinking. The minimum recruitment criteria were that the duration of fuel exposure was 5 years
or more, and the duration of alcohol consumption was also at least 5 years. Both the test and control subjects were age-
matched, as well as matched for alcohol consumption. The selection of the control and other groups was based on simple
random selection technique. None of the control subjects had any history of occupational or recreational exposure to
petrol vapours. All subjects gave informed consent prior to the commencement of the study. The study was conducted
according to revised Helsinki’s declaration.

Structured questionnaire was administered by a trained person. Information was obtained on the following: lifestyle
(smoking, coffee, alcohol); occupational (working hour per day, length of employment in petrol-exposed environment
(years); and type of protective measures employed). Enquiry was also made about the condition of the workplace. The
subjects considered ineligible for the study were those that met the exclusion criteria i) subjects that have been working
for less than 5 years in automobile mechanic workshop, ii) apprentices in mechanic workshop- in most cases they are
always not directly/ fully involved in tasks associated with this profession, iii) individuals with either liver or renal
disease.

A total of 5 mL of venous blood sample was drawn from each participant, dispensed into anticoagulant free bottle and
used for biochemical analyses. Each sample tube was capped, labeled and the blood left to clot for two hours after which
it was centrifuged to obtain serum that was stored at -20°C until the time of analysis. The determination of serum
activities of the markers of hepato-renal damage such as alkaline phosphatase, γ-glutamyltransferase, alanine
aminotransferase and aspartate aminotransferase (ALP & γ-GT, ALT, AST) were carried out. In addition serum levels of
other markers like bilirubin, total protein, globulin and albumin were determined. While serum activities of both AST and
ALT were estimated using the method of Bergmeyer (9) that of alkaline phosphatase (ALP) was by the method of Mc
Comb and Bowers (10). Levels of albumin and bilirubin were determined using standard bromocresol and modified
Jendrassik-Groff (11) methods respectively. Serum concentration of total protein was evaluated using biuret’s method
(12). Assessment of serum levels of creatinine and urea were by Jaffé reaction and diacetylmonoxime oxidase method
respectively. Hitachi® 902 automated machines (Roche Diagnostic, Germany) was used for these estimations.

The SPSS version 15 was used for statistical analyses. The Student’s t test and analysis of variance were used to test the
statistical significance difference of measured parameters among studied population. A p value of ≤ 0.05 was considered
statistically significant.

Results
The results of the study are presented in Table 1 below. In alcohol and non-alcohol consuming auto-mechanics urea, uric
acid, creatinine, ALT and AST were significantly higher (p<0.05) compared with control. Total protein was not
significantly different (p>0.05) in non-alcohol consuming mechanics compared with control yet albumin was significantly
decreased (p<0.05), but globulin was significantly higher (p<0.05) than control. On the other hand in alcohol consuming
participants both total protein and albumin were significantly lower (p<0.05) yet globulin was significantly increased (p<0.05) compared with control. In the same vein, while in non alcohol consuming mechanics alkaline phosphatase and γ-glutamyltransferase were not significantly (p>0.05) different compared with control, in alcohol consuming mechanics both parameters were significantly increased (p<0.05). In alcohol consuming non-mechanics while urea, creatinine and uric acid were not significantly different (p>0.05) compared with control all other parameters were significantly different; alkaline phosphatase, γ-glutamyltransferase, bilirubin, globulin, ALT and AST were significantly higher (p<0.05).

Data obtained from structured questionnaire administered by a trained person revealed that the recruited participants did not have any other lifestyle like cigarette smoking, coffee consumption etc. average working hour per day was >7 hours. Length of employment for individuals was > 5 years. Nearly all did not use any type of gear for protective measure. All the mechanics in both alcohol and non-alcohol consuming groups revealed that petroleum product contamination of the workshop environment was a common experience.

Table 1: Serum levels or activities of markers of hepatic and renal damage of different studied groups.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control Non-alcohol consuming mechanics</th>
<th>Alcohol consuming automobile mechanics</th>
<th>Non-alcohol consuming automobile mechanic</th>
<th>Alcohol consuming non-mechanics</th>
<th>F-value</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gamma-glutamyltransferase (IU/L)</td>
<td>36.43±5.73</td>
<td>60.49±17.3</td>
<td>37.46±5.09</td>
<td>45.33±6.66</td>
<td>6.980</td>
<td>0.026</td>
</tr>
<tr>
<td>Alkaline phosphatase (IU/L)</td>
<td>56.31±16.23</td>
<td>72.14±9.68</td>
<td>59.04±3.66</td>
<td>63.63±17.24</td>
<td>16.532</td>
<td>0.017</td>
</tr>
<tr>
<td>Total protein (g/L)</td>
<td>75.38±28.04</td>
<td>70.59±6.99</td>
<td>73.13±29.27</td>
<td>73.87±23.94</td>
<td>3.109</td>
<td>0.042</td>
</tr>
<tr>
<td>Albumin (g/L)</td>
<td>37.34±5.49</td>
<td>25.27±8.04</td>
<td>36.00±11.16</td>
<td>31.80±4.89</td>
<td>15.641</td>
<td>0.038</td>
</tr>
<tr>
<td>Globulin (g/L)</td>
<td>38.04±4.78</td>
<td>45.32±14.27</td>
<td>43.13±3.84</td>
<td>42.07±8.10</td>
<td>34.002</td>
<td>0.029</td>
</tr>
<tr>
<td>Bilirubin (µmol/L)</td>
<td>15.33±2.10</td>
<td>27.55±8.10</td>
<td>19.10±2.28</td>
<td>18.17±3.62</td>
<td>1.573</td>
<td>0.039</td>
</tr>
<tr>
<td>Alanine aminotransferase (IU/L)</td>
<td>37.55±7.09</td>
<td>52.69±27.34</td>
<td>44.43±5.48</td>
<td>41.79±5.00</td>
<td>14.030</td>
<td>0.005</td>
</tr>
<tr>
<td>Aspartate aminotransferase (IU/L)</td>
<td>32.84±6.30</td>
<td>60.19±15.30</td>
<td>48.28±8.10</td>
<td>39.19±4.09</td>
<td>1.206</td>
<td>0.023</td>
</tr>
<tr>
<td>Uric acid (mmol/L)</td>
<td>172.91±15.21</td>
<td>198.28±56.00</td>
<td>206.23±49.52</td>
<td>180.73±85.40</td>
<td>16.330</td>
<td>0.053</td>
</tr>
<tr>
<td>Creatinine (µmol/L)</td>
<td>20.27±3.82</td>
<td>27.47±4.33</td>
<td>25.36±4.06</td>
<td>22.01±7.06</td>
<td>19.064</td>
<td>0.047</td>
</tr>
<tr>
<td>Urea acid (mg/dL)</td>
<td>24.65±4.56</td>
<td>30.00±6.33</td>
<td>28.02±6.91</td>
<td>22.81±4.71</td>
<td>6.608</td>
<td>0.036</td>
</tr>
</tbody>
</table>

Results are expressed as mean ± standard deviation. *P < 0.05 is significant.
Discussion

Acute renal failure (ARF) is a more common manifestation of the renal cells when exposure to nephrotoxic agents takes place. An important feature of renal failure is a rapid decline in glomerular filtration rate and an elevation in the blood levels of nitrogenous compounds such as urea, creatinine, and uric acid. Nephrotoxicants are known to induce renal vasoconstriction thereby decreasing the quantity of blood that gets to the glomerulus, which in turn may result in a reduction in the amount of blood filtered (13). Some other nephrotoxicants cause glomerular injury; leading to an alteration in filtration rate, and consequently presenting as a reduction in the amount of filtrate that enters the tubules. But when it is the tubular cells that are involved and their functional integrity altered, their permeability may increase. This simple defect induces the filtrate to backleak into the interstitium and eventually into the circulation, producing a noticeable reduction of the glomerular filtration rate (GFR) without direct glomerular involvement.

Many other nephrotoxicants can reduce the adhesion of tubular cells to each other, making them to prevent filtrate reabsorption and thereby elevating the pressure within the tubule leading to a resistance of movement of filtrate into the tubule. While the liver is known for extensive regenerating ability and large functional capacity, the kidney is also able to overcome substantial loss of function. Increase of GFR by 40–60 percent by a kidney is common if the other suffers loss of function. This increase can be to the extent that the reabsorption of water and solutes by the nephrons is elevated to the extent that the osmotic balance is comparable to normal subjects. While in few cases these coping mechanisms protect the whole organism in the short term, they may result in chronic renal failure in the long run especially when exposure to nephrotoxicants persists. A persistent elevation in glomerular pressure can result in sclerosis of the glomerulus (14) and the degeneration of the capillary loops. Some of these processes might have played a role in the significant increase in the levels of markers of renal damage observed in auto-mechanics as over a hundred different toxic compounds constitute gasoline and other petroleum products.

The parenchymal cells which constitute majority of the cells that make up the liver are the main but not the only cell type affected by toxic agent, and they are known to suffer different types of cell injury. The type of lesion or effect featured by experimental animals exposed to a particular toxic agent depends on the chemical involved, the dose, and the duration of exposure. Hepatocellular degeneration and death is common with various hepatotoxicants, involving organelles and structures within the liver cell. From the results of this study it seems as if the harmful effects of contact to chemicals at the workplace may be enhanced by lifestyle as in this case-alcohol consumption. Compared with non-alcohol consuming mechanics the synthetic ability of the liver of alcohol consuming mechanics was profoundly affected as reflected by a significantly low levels of total protein and albumin. Moreover, excretory machinery were more overwhelmed as a result of alcohol exposure in auto-mechanics with respect to bilirubin level, and there seems to be a higher inflammatory response as globulin fraction was significantly increased in alcohol consuming mechanics compared with their non-alcohol consuming counterparts.

Alanine aminotransferase (ALT) and aspartate aminotransferase (AST), alkaline phosphatase activity, and gamma glutamyltransferase (GGTP) are usually employed in standard clinical setting to determine possible hepatotoxicity. The advantage of employing a battery of tests to assess chemical-induced hepatotoxicity is that each of them responds differently in each category of liver injury. While hepatic injury affecting parenchymal cell membrane can result in dramatic elevations in serum AST and ALT activities (15), significant increases in alkaline phosphatase is characteristic of cholestatic injury (16,17), where increases in ALT and AST may be limited or nonexistent. Alcohol induced-liver injury is characterized by increases in AST activity that is usually greater than ALT activity, whereas for other types of hepatocellular injury ALT activities are higher. The two groups of mechanics featured significantly higher activities of AST and ALT than control but alcohol consumption seemed to have caused a more pronounced elevation in membrane damage. Chaphalkar et al. (18) also noted that alcohol consumption may cause significant increase in activities of AST and ALT occurring from membrane damage of hepatocellular cells.

Serum γ- glutamyltransferase activity, an extremely sensitive marker of hepatobiliary involvement, may be increased simply by drinking alcoholic beverages as revealed by Lieberman et al. (19). Their observation seems to support the result...
of this study in which both alcohol consuming groups (alcohol consuming mechanics and alcohol consuming non-mechanics) had significantly higher $\gamma$-glutamyltransferase compared with either control or non-alcohol consuming mechanics. Since $\gamma$-glutamyltransferase is not particularly specific because it is elevated in both hepatocellular and cholestatic injury. Therefore, using this enzyme in combination with other tests has proved useful in probing the mechanisms involved in different types of chemical-induced liver injury. The renal markers of alcohol-consuming mechanics were comparable with those of non-alcohol consuming automobile mechanics (i.e. not significantly different). Meanwhile, compared with control, urea, creatinine and uric acid were significantly higher in both alcohol-consuming and non-alcohol consuming mechanics, an indication that components of petroleum products may be capable of inducing renal damage. In agreement with this observation are data obtained from several studies carried out on experimental animals in which renal damage were reported consequent to exposure to different petroleum products (20).

Conclusion

The results of this study imply that it is essential to look for ways to reduce exposure to gasoline in automobile mechanics, especially by encouraging them to use protective gears. Moreover, adoption of alternative fuels that could be cleaner, safer to human health and are environment friendly may certainly benefit automobile mechanics as well. The more pronounced effects associated with alcohol group urges the necessity to educate automobile mechanics of the danger of gasoline exposure and to motivate them to change their lifestyle so as to reduce the detrimental effect of co-exposure to alcohol and petroleum and its attendant further deterioration of their state of health.

References


