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Influence of Occupational Exposure to Hydrocarbons and Automobile Exhaust on the Health of Automobile Workshop Workers

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Abstract: The effect of hydrocarbons and automobile exhaust exposure on the hematological, cardiovascular, and hepatic parameters of the workers of automobile workshops was studied. The blood samples collected from five groups of workers with different duration of exposure (6-10, 11-15, 16-20, 21-25, and >25 years) and a control group including university students were analyzed for hematological and clinical parameters. An exposure-dependent statistically significant linear increase was observed in hemoglobin level ($p=0.041$, $R2=0.8132$) and serum levels of alanine aminotransferase (ALT) ($p=0.000$, $R2=0.9604$) and creatine kinase (CK) ($p=0.000$, $R2=0.9759$) of the workers. However, the parameters of blood count and serum levels of aspartate aminotransferase, alkaline phosphate, lactate dehydrogenase, and total bilirubin levels remained unaffected. The long-term exposure to hydrocarbons resulted in a significant time-dependent increase in the hemoglobin, ALT, and CK levels of the workers. The findings indicate a risk of developing hematological, cardiovascular, and hepatic disorders in the automobile workshop workers.

Key Words: Hydrocarbon exposure; Automobile workshop workers; Hematological parameters; Hepatic parameters; Cardiac parameters.

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1. INTRODUCTION

In the modern age of technology, pollutants are perpetually damaging the ecosystem and also disturbing the biological functioning of living organisms including humans. The hydrocarbons and related compounds released from the burning of natural combustion fuels such as automobile fuels leave an alarming impact on living organisms (Baklanov et al., 2007). Automobile workshops are one of the major contributors to environmental pollution. Automobile workshop workers are continuously exposed to these hydrocarbons as they breathe in volatile hydrocarbons and automobile exhaust saturated environment (Wang et al., 2012).

Hydrocarbons present in the environment cause structural, functional, and compositional changes in the immune system (Bahadar, Abdollahi, Maqbool, Baeeri, & Niaz, 2014). It has been also reported that extreme exposure to petroleum products can cause coma and death (Levy, Pappano, & Berne, 2007). Chronic exposure to hydrocarbons causes hepatotoxicity and lung, kidney and immune dysfunction (Akintonwa and Oladele, 2003; Lippmann et al., 2011). The severity of abnormalities depends upon the route, duration, and amount of exposure (Dere, Suriye, & Hüseyin, 2003; Diggs et al., 2011).

Previously, studies have been performed to evaluate the effect of petroleum products gasoline, diesel oil, engine oil, and other hydrocarbons on different parameters of workers (El-Ghazaly, Ali, Dekinesh, Sedky, & Kabeil, 2016; Ezenwaji, Evelyn, Yenagoa, Bebe, & Chukwuemeka, 2013; Uboh, Akpanabiatu, Atangwho, Ebong, & Umoh, 2007; Zahid, Shad, Sheikh, & Nawaz, 2018). However, the hazardous effects of automobile exhaust on the hematological, hepatic, and cardiac profile of automobile workers have not been reported in the literature. Therefore, this study was carried out to access the deleterious effects of automobile exhaust on the hematological, cardiac, and hepatic health of the workers working in automobile workshops. The study would provide awareness for the human population particularly the workers of automobile workshops and the petroleum industry regarding the possible health effects of volatile hydrocarbons released in their working environment.

2. MATERIALS AND METHODS

2.1 Experimental design

In this study, the effect of exposure to hydrocarbons and automobile exhaust on the hematological, cardiovascular, and hepatic parameters of workers of automobile workshops was studied. Written consent and a questionnaire based on their medical history were filled by each participant. Overall, 95 individuals divided into control (university students, n=15) and workshop workers were included in the study. The

workshop workers were divided into five subgroups based on the duration of exposure to hydrocarbons (6-10, 11-15, 16-20, 21-25, and >25 years). The blood samples collected from the participants of the control and study groups of workers were analyzed for hematological parameters. The sera obtained from the blood samples were analyzed for some cardiovascular and hepatic parameters. One-way analysis of variance and regression analysis were used for statistical evaluation of experimental data

2.2 Collection of blood samples

The blood sample (5 mL) was obtained from every participant using a sterilized disposable syringe and immediately divided into two portions. One portion (3 mL) was transferred to a collection tube containing gel and clot activator and centrifuged at 4000 × g to obtain serum. The sera were preserved at 4°C throughout the study period and used for the analysis of hepatic and cardiovascular parameters. The remaining portion of blood (2 mL) was transferred to a plasma tube containing anticoagulant (EDTA solution) and preserved at 4°C for analysis of hematological parameters.

2.3 Hematological parameters

Complete blood count (CBC) including hemoglobin (Hb), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), platelet count (PLT), red blood cells (RBC), mean corpuscular volume (MCV), packed cell volume (PCV), hematocrit (HCT), and white blood cell count (WBC)/(TLC) and differential leukocyte count (DLC) were measured on an automated hematology analyzer (Sysmex XK-21). Each blood sample (2 mL) along with EDTA, and the control were injected to the analyzer to obtain the CBC results.

2.4 Hepatic parameters:

2.4.1 Total bilirubin

The level of total bilirubin (TB) was measured using a bilirubin kit (BR2361, Randox, County Antrim, Bt29 4QY, UK) on a spectrophotometer. In alkaline medium, bilirubin was treated with diazonium ion to form diazo-bilirubin complex. Caffeine was mixed with the complex for removal of unconjugated bilirubin bound with albumin. TB level was measure using following regression equation obtained from the standard curve of bilirubin (R2=0.9834).

TB(mg⁄dL)=(Abs.of sample)⁄6.019

2.4.2 Liver function enzymes

The serum levels of liver function enzymes including alkaline phosphatase (ALP), Aspartate aminotransferase (AST), and alanine aminotransferase (ALT) were determined by the spectrophotometric (Schumann and Klauke, 2003) method proposed by the International Federation of Clinical Chemistry using standard kits (ALP2, Cat No. 12173107122, Roche Diagnostic, Mannheim, Germany, AST, Cat No. 10851124 216, Roche Diagnostic, Mannheim, Germany, and ALT, Cat No. 10851132 216, Roche Diagnostics, Mannheim, Germany).

2.5 Cardiovascular parameter

2.5.1 Creatine kinase

Creatine kinase (CK) was determined by the spectrophotometric method by the NADPH dependent coupled reactions as described by the International Federation of Clinical Chemistry (IFCC) (Schumann and Klauke, 2003) using standard kit (CK/CK-MB kit-12008, Human Diagnostics, Magdeburg, Germany). The CK activity was calculated as:

CK (U⁄L)=((ΔA)⁄min[f0][() ×4125]]

2.5.2 Lactate dehydrogenase

Lactate dehydrogenase (LDH) activity was measured by using a spectrophotometric method based on NADH dependent reaction that converts pyruvate to lactate and NADH is oxidized to NAD+ (Decker and Lohmann-Matthes, 1988). The LDH activity was measured in terms of the amount of NAD+ formed using standard kit (LDH kit-12014, Human Diagnostics, Magdeburg, Germany). The LDH activity was calculated as:

LDH (U⁄L)=((ΔA)⁄min[f0][() ×8095]]

2.6 Statistical analysis

The results were presented as mean ± SD of the studied parameters. One-way analysis of variance (ANOVA) was used to differentiate the means on the basis of significant differences between various study groups by applying Turkey's multiple ranges tests at 95% confidence level using statistical software SPSS version 19.

3. RESULTS AND DISCUSSION

The Hb level of the workers at a selected level of the duration of exposure to hydrocarbons is presented in Table 1. Hb levels ranged from 14.39±1.04 to 15.73±0.97 g/dl. A statistically significant difference (p=0.041) was observed in the Hb levels of the control and the hydrocarbon-exposed workers. The Hb level of the workers increased linearly, with an increase in the duration of exposure to hydrocarbons up to 16-20 years with a relatively higher value of regression coefficient (R2 =0.8132) (Figure 1). The following generalized equation was developed to explain the relationship between the duration of hydrocarbon exposure and Hb level.

$$Hb\ (g/dL)=[Hb]\_sc\times DE+[Hb]\_0$$

Where Hbsc, DE, and HB0 are the hemoglobin sensitivity coefficient, duration of exposure, and level of hemoglobin at negligible exposure to hydrocarbons.

An exposure-dependent increase in the Hb level of the workers may be attributed to the possible induction of Hb to fulfill the requirement of oxygen in a hydrocarbon-saturated and oxygen-deficient environment. It may also be attributed to the hydrocarbon-induced oxidative stress causing inflammation and lysis of red blood cells which leads to an increase in Hb content in body fluid. The results are in agreement with previous reports that hemoglobin concentration is decreased with time-dependent exposure to different pollutants in automobile workshop workers (Khan et al., 2010). Recently, another study showed that lead exposure decreases the Hb level of battery workers (Kargar-Shouroki, Mehri, & Sepahi-Zoeram, 2022).

The levels of red blood cells (RBC), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), mean corpuscular volume (MCV), hematocrit (HCT) of the workers at selected levels of the duration of exposure to hydrocarbons are presented in Table 1. The RBC, MCH, HCT, MCHC, and MCV levels ranged from 5.02 × 106 to 4.81 × 106 cells/uL, 28.39 to 25.18 pg/cell, 44.59 to 46.92%, 32.46±1.34 to 33.63±1.09 g/dL, and 87.11 to 85.80 fL respectively. Statistically, no significant effect of hydrocarbon exposure was noticed on the erythrocyte count of the workers (p>0.05).

The level of white blood cells (WBC), eosinophils (EO), monocytes (MONO), neutrophils (NEUT), and lymphocytes (LYMPH) in the serum of the workers at selected levels of the duration of exposure to hydrocarbons are presented in Table 1. The WBC, EO, MONO, NEUT and LYMPH levels ranged from 7942 to 6275 cell/uL, 3.05 to 2.50%, 3.05 to 2.5%, 56.15 to 55.87%, and 43.0 to 37.54% respectively. Statistically, no significant effect of hydrocarbon exposure was noticed on the leukocyte count of the workers (p>0.05).

The results of platelet (PLT) count of the serum of the workers at selected levels of the duration of exposure to hydrocarbons are presented in Table 1. The PLT count ranged from 266739 to 202588 cell/µL. Statistically, no significant effect of hydrocarbon exposure was noticed on the platelet count of the workers (p>0.05).

The levels of hepatic parameters including TB, ALP, AST, and LDH activity in serum of the workers at selected levels of the duration of exposure to hydrocarbons are presented in Table 2. The TB, ALP, AST, and LDH levels ranged from 0.72±0.083 to 0.86±0.12 mg/dL, 185.65±13.57 to 224.66±43.82 U/L, 33.85±12.98 to 155.50±41.23 U/L and 261.60±85.69 to 351.66±179.72 U/L respectively. Statistically, no significant effect of hydrocarbon exposure was observed on above-mentioned parameters of the workers (p>0.05).

The level of alanine aminotransferase (ALT) in serum of the workers at selected levels of the duration of exposure to hydrocarbons is shown in Table 2. The ALT levels ranged from 37.10±13.38 to 183.00±50.83 U/L. Initially, the activity of ALT increased slowly with the duration of exposure, however during the duration of 11-15, 16-20, and 21-25 years of hydrocarbons exposure, there was a massive increase in activity of ALT that ranged from 97.04±19.86 to 135.47±6.97 and 180.23±30.29 U/L respectively. A statistically significant difference (p=0.00) was noticed in ALT levels of the control and the hydrocarbon exposed workers. The regression analysis showed a linear increase in serum ALT level in response to an increase in the duration of exposure to hydrocarbons with a relatively higher value of regression coefficient (R2=0.9604) (Figure 2). The following regression equation was obtained to study the relationship between the duration of hydrocarbon exposure and the ALT levels of the workers.

$$ALT\ (U/L) =[ALT]\_sc\times DE+[ALT]\_0$$

Whereas ALTTSC, DE, and ALT0 are alanine aminotransferase sensitivity coefficient, duration of exposure, and alanine aminotransferase level before exposure to hydrocarbons.

An exposure-dependent increase in the ALT level of the workers may be attributed to the hepatocellular injury that increases the extracellular enzyme concentration in the worker's serum. The exposure to hydrocarbons has been found to increase the serum levels of liver function enzyme of automobile workers (N and Db, 2010).

The level of creatine kinase (CK) in the serum of the workers at selected levels of the duration of exposure to hydrocarbons is presented in Table 2. The CK level ranged from 101.85±18.37 to 335.83±34.50 U/L. The CK level was greatly increased during the duration of 0 to 6-10 years of hydrocarbons exposure and ranged from 101.85±18.37 to 181.47±41.94 U/L respectively and then increased linearly. A statistically significant difference (p=0.00), was observed in CK levels of the control and the hydrocarbon exposed workers. The regression analysis showed a linear increase in serum CK level in response to an increase in the duration of exposure to hydrocarbons with a relatively higher value of regression coefficient (R2=0.9759) (Figure 3). The

following regression equation was obtained to study the relationship between the duration of hydrocarbon exposure and the CK level of the workers.

$$CK\ (U/L) = [CK]\_SC \times DE + [CK]\_0$$

Whereas CKSC, DE, and CK0 are creatinine kinase sensitivity coefficient, duration of exposure, and creatinine kinase level before exposure to hydrocarbons.

An exposure-dependent increase in the CK level of the workers may be attributed to the possible generation of free radicals due to the stress of the hydrocarbon-saturated environment. Previously, it has been reported that ultrafine particles present in automobile exhaust have adverse effects on the respiratory, cardiovascular and neurological systems of the workers (Ley, 2005; Møller et al., 2014; Valko et al., 2007).

The increase in Hb, ALT, and CK levels indicates an exposure-dependent damage to erythrocytes, hepatocytes, and cardiac cells of the workers working in the automobile workshops. The study suggests that long-term exposure to hydrocarbons and automobile exhaust may lead to hemoglobin deficiency and hepatic and cardiovascular damage. However, the non-significant difference in CBC TB, AST, ALP, and LDH suggests that these parameters have not been affected by the exposure to hydrocarbons and automobile exhaust.

6. CONCLUSION

In conclusion, the occupational exposure to hydrocarbons including gasoline, petrol, and engine oil resulted in a significant time-dependent increase in the hemoglobin, ALT, and CK levels of the workers working in the automobile workshops. The observed variations in these parameters may be attributed to the release of these proteins due to the hydrocarbon-induced oxidative stress causing inflammation and cell death. However, no significant effect of hydrocarbon exposure was observed on the CBC TB, AST, ALP, and LDH levels of the workers. These findings suggest that the workers working in automobile workshops are at a relatively higher risk of developing hematological, cardiovascular, and liver disorders due to their exposure to hydrocarbons and automobile exhaust. The study suggests formulating the safety and preventive measures for these workers.

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