IOSR Journal of Pharmacy and Biological Sciences (IOSR-JPBS)
e-ISSN:2278-3008, p-ISSN:2319-7676. Volume 12, Issue 1 Ver. II (Jan. - Feb.2017), PP 06-09
www.iosrjournals.org

Evaluation of Haematological, Hepatic and Renal Function of Auto Drivers in Tirunelveli City,Tamilnadu, India.

Dr.A.Jeya jancy selvi ratnam[1], Dr.H.Kavingar kannan[2]
[1]*Associate professor, Department of Physiology, Tirunelveli Medical College, The Tamil Nadu Dr.MGR Medical University, India*
[2]*Postgraduate, Department of Physiology, Tirunelveli Medical College, The Tamil Nadu Dr.MGR Medical University, India*

Abstract
Background: *Episodes of severe air pollution in Asia have been reported in the scientific literature of recent times. The WHO 2005 report* Health effects of transport-related air pollution *provides the first comprehensive assessment of air pollution related to road transport and of the risks it presents to human health. Environmental pollution has many facets, and the resultant health risks include diseases in almost all organ systems. In this respect, auto rickshaw drivers are at risk, since they are continuously exposed to emissions from vehicles, due to the nature of their job. In view of this, this study is undertaken.*
Aim And Objectives: *The aim of the study is to evaluate the haematological, renal and liver functions of auto drivers in Tirunelveli city and compare it with age and socio demographically matched controls.*
Materials And Methods: *Following inclusion and exclusion criteria, twenty five auto drivers and twenty five controls were investigated for haematological, renal and liver functions.*
Results: *Red blood cell count, haemoglobin level, and Haematocrit level were significantly lower in auto drivers than the control group. Liver enzymes and renal functions showed statistically non significant difference between both groups except for alanine aminotransferase (ALT) which was significantly higher in auto drivers.*
Conclusion: *Work exposure to petroleum products inhalation has health implications as seen by the haematological, and liver function changes. Such group of workers need to be sensitized about the hazards of exposure, appropriate preventive strategies and periodic medical examination.*
Keywords: *Air pollution, particulate matter, carbon black, liver functions, drivers, renal functions.*

I. Background

Episodes of severe air pollution in Asia have been reported in the scientific literature of recent times. The WHO 2005 report Health effects of transport-related air pollution provides the first comprehensive assessment of air pollution related to road transport and of the risks it presents to human health[1]. Vehicle pollution has been a major factor causing the degradation of the environment around us, the air we breathe, and the soil we live on. In this respect, auto rickshaw drivers are at a risk, since they are continuously exposed to emissions from vehicles, due to the nature of their job [2]. Air pollution induces a wide range of toxic effects. As the World Health Organization (WHO) points out, outdoor air pollution contributes as much as 0.6 to 1.4 percent of the burden of disease in developing regions [3].

Although air pollutants are many, the most important are particle pollution (often referred to as particulate matter (PM)), ground-level ozone (O_3), carbon monoxide (CO), sulfur oxides (SO_x), nitrogen oxides (NO_x), and lead (Pb) which are found in the ambient air (also known as "criteria pollutants")[4,5]. These exposures are associated with a broad range of acute and chronic health effects varying from sub-clinical effects to premature mortality [6].

Hepatotoxicity and nephrotoxicity have been reported following human and animal exposure to unleaded petrol [7,8,9]. For instance, kidney adenoma[9], elevated serum activity of liver enzymes[10,11], urea, creatinine, and potassium, and decreased chlorine and sodium have been reported in laboratory animals[12]. Similarly, proteinuria, elevated serum activity of liver enzymes (aspartate aminotransferase, alkaline phosphatase, alanine aminotransferase), and total bilirubin and fatty liver changes have been reported in drivers and workers following exposure to unleaded petrol[13,14,15,16].

Air pollution is increasingly documented as a threat to public health in most developing countries. Evaluation of current air quality levels, regulatory standards and scientific literature on outdoor and indoor air pollution, and health effects are important to identify the burden, develop and implement interventions and to fill knowledge gaps. Hence this study is undertaken to study the effects of vehicle pollution on haematology, renal and liver functions of auto drivers.

II. Aim And Objectives

The aim of the study is to evaluate the haematological, renal and liver functions of auto drivers in Tirunelveli city and compare it with age and socio demographically matched controls, who are not occupationally exposed to vehicular exhaust.

III. Materials And Methods

Before the commencement of the study, ethical approval was obtained from the Ethical Review Committee of the Institution. All the participants gave informed consent to participate in the study. Questionnaires were distributed and accurately filled; candidates who met the criteria for participation in this study were enrolled in the study. Following inclusion and exclusion criteria, twenty five auto drivers and twenty five controls were investigated for haematological, renal and liver functions.

Inclusion criteria: male auto drivers with no less than 5 years experience as drivers in the age group of 25 to 55 years. Exclusion criteria: alcohol and tobacco use, liver and kidney diseases, any major medical illness, malignancy, hepatotoxic drug usage, industry near the residence. Controls: same age group males not occupationally exposed to vehicle exhaust. Exclusion criteria same. Each participant was subjected to the following: Interview: a questionnaire was used to collect the following information: socio demographic and occupational profile of workers; usage of personal protective equipment and reasons for non usage; general health status; and respiratory complaints. Laboratory investigation: 5 mL blood sample was taken from each participant through venipuncture which was then divided into the following

Two mL blood collected in a plastic tube containing ethylenediaminetetraacetic acid (EDTA) for complete blood picture.

- Three mL blood collected in a dry plastic tube for kidney function tests (urea and creatinine) and liver function tests (including ALT, aspartate aminotransferase (AST),and serum albumin). The sample was allowed to clot naturally to separate the serum for analysis and was stored upright at room temperature until it was transported to the laboratory for analysis. In the laboratory, each sample was centrifuged and stored in the freezer at −70°C until being processed.

IV. Results

Socio Demographic Data Of Autodrivers

AGE (years)	Number	Percentage
25 ----35	6	24
36----45	12	48
46----55	7	28
Total	25	100
Experience as driver(Years)		
5 -----10	8	32
11 -----15	10	40
16 -----20	5	20
20 and above	2	8
Educational qualification		
Below 10^{th} std	21	84
Above 10^{th} std	4	16
Safety Knowledge		
Yes	6	24
No	14	56
Don't care	5	20

Comparison of haematological parameters

Groups	WBC($\times 10^9$/L) Mean SD	RBC(mil/mcL) Mean SD	Platelet($\times 10^9$/L) Mean SD	Hb% Mean SD	PCV% Mean SD
Autodrivers	8.4±1.17	4.8±0.60	332.3± 62.38	14.08±1.32	41.08±2.87
Controls	8.3±1.22	5.3±0.51	318.6± 55.73	16.72±1.52	44.56±3.41

PARAMETER (case Vs control)	p value	t value
WBC Count	0.161	+1
RBC Count	0.00191	-3.04
Platelet Count	0.2081	+0.82
Hb%	<.0001	-6.5
PCV	0.0001	-3.9

Comparison of renal function parameters

Groups	Urea mg/dl Mean SD	Creatinine mg/dl Mean SD
Autodrivers	25.84±5.20	0.87±0.27
Controls	23.38±3.25	0.80±0.17

PARAMETER (case Vs control)	p value	t value
Urea	0.1784	+0.93
Creatinine	0.202	0.84

Comparison of hepatic function parameters

Groups	AST U/L Mean SD	ALT U/L Mean SD	Total protein Gm/dl Mean SD
Autodrivers	30.48±5.62	42.6 ±30.68	5.26 ±0.54
Controls	29.56± 7.87	30.48 ± 15.05	5.40 ± 0.46

PARAMETER (case Vs control)	p value	t value
AST	0.1340	1.12
ALT	0.0166	2.19
Alb	0.188	-0.89

V. Discussion

The transport sector is an important source of emissions of a wide range of gaseous air pollutants and of suspended particulate matter (PM) of different sizes and compositions. Tailpipe emissions of primary particles from road transport account for up to 30% of fine PM (less than 2.5 µm in aerodynamic diameter: PM2.5) in urban areas. Other emissions from road transport (such as those from re suspended road dust and the wear of tyres and brake linings) are important contributors to the coarse fraction of PM (2.5-10 µm in aerodynamic diameter: PM10-2.5). Road transport is also the most important source of emissions of nitrogen dioxide and benzene in cities. Exposure to transport-related air pollution varies, as some groups may be more exposed, depending on how long they stay in polluted areas and what they do while there.

The effects of air pollution on red blood cells have been investigated mostly in children [17,18]. Air pollutants present in the environment largely damage cell immunity and change the intensity and course of iron metabolism in the body, which results in iron-deficient anemias with very low values of haematocrit and haemoglobin. Also, lead can damage the erythrocytes' membranes, resulting in anemia. Various organs including skin, eyes, and digestive tract can be exposed to ambient PM or CB. Air pollution induces a wide range of toxic effects. In addition to the well known harmful effects it can induce liver toxicity in persons who are continuously exposed to hydrocarbons. Whereas it is conceptually easy to visualize how air pollutants might trans locate from the lung into systemic circulation, detailed mechanisms are less easily explained since the constituents of PM are quite diverse and the toxico dynamics of individual components could vary widely. Water soluble fractions of PM could trans locate into extra-pulmonary circulation is one possibility[19,20,21]. The second possibility is that inhaled insoluble nano particles directly cross the alveolar-capillary barrier, circulate in the blood stream, and deposit on blood cells or on the surface of vascular endothelial cells in nonspecific organs [22]. Such interactions might result in pro thrombotic effects[23] on the hepatic microcirculation[24]. The third possibility is that inhaled PM particles are first in contact with immune cells such as alveolar or bronchiolar macrophages and thereby stimulate innate immune responses, releasing pro-inflammatory cytokines into the blood stream. Such an inflammatory milieu could contribute to the disease progression in organs such as liver[22]. However, the exact translocation pathways of the various constituents of PM or CB are not fully understood.

Exposure to PAHs is associated with a high risk of renal dysfunction and cancer. Many researchers have confirmed nephro toxicity with chronic exposure of PAHs [25]. Experimental data have demonstrated both glomerular and tubular toxicity[26-27]. Systemic exposure by inhalation has been demonstrated by detection of the pyrene metabolite, 1-hydroxylpyrene in urine of exposed subjects [28]. In a cross sectional study by Taberghar occupational medicine associates[29], wood fetching workers exposed to creosote had an increased incidence of haemeturia, although renal function of this cohort was not altered. Hydrocarbons are also known to be hepatotoxic [30]. The hepatotoxicity results after hydrocarbon undergoes phase 1 metabolism, thereby inducing free radicals formation. These free radicals subsequently bond with hepatic macromolecules and ultimately cause lipid peroxidation. This metabolite creates a covalent bond with the hepatic macromolecules, thereby initiating lipid peroxidation [31]. Liver function tests can be abnormal within 24 hours after ingestion and clinically apparent jaundice can occur within 48-96 hours [31].

VI. Conclusion

Air pollutants have been associated with increased morbidity although direct evidence of causation has been lacking until recently. Several animal models provide strong evidence that PM or CB, indicators of air pollution, can induce various diseases and act to exacerbate existing lesions in organs that are accessible to the constituents of air pollution. Among those organs, the liver is one of the vulnerable target organs since its microvasculature allows ready access to hepatocytes and inhaled PM pollutants can be translocated from the alveolar space into the bloodstream. Direct effects of PM or CB on hepatocytes include the induction of

oxidative stress and DNA strand breaks. In addition, airborne PMs contribute to the pathogenesis of steato hepatitis by alteration of lipid metabolism and induction of a pro-inflammatory milieu, resulting in exacerbation of non-alcoholic steato hepatitis (NASH) . Although direct evidence for these associations has been reported, more extensive and detailed studies are needed in liver models since the constituents of PM or CB are diverse and the roles of individual constituents on liver pathophysiology are currently unknown. Furthermore, therapeutic or prophylactic strategies for liver protection against exposure to air pollutants should be considered if we are not able to reduce air pollution in our environment.

Reference

[1]. WHO. WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide, Global update 2005, Summary of risk assessment. Geneva: World Health Organization; 2005.
[2]. Respiratory Effects of Air Pollutants among Nonsmoking Auto Rickshaw Drivers of Patiala City (Punjab State, India) Aditya Jain, Ramta Bansal, Avnish Kumar, K. D. Singh4Journal of Dental and Medical Sciences (JDMS) ISSN: 2279-0853, ISBN: 2279-0861. Volume 1, Issue 5 (Sep-Oct. 2012), PP 01-04.
[3]. World Health Report 2002. Geneva: WHO.
[4]. Bruce N, Perez-Padilla R, Albalak R. The health effects of indoor air pollution exposure in developing countries. Geneva: World Health Organization; 2002. p. 11.
[5]. Senevirathne SRDA. Air pollution: a case study of environmental pollution. Journal of College of Community Physicians of Sri Lanka. 2003;8(1):1–9.
[6]. American Thoracic Society. What Constitutes an Adverse Health Effect of Air Pollution? American Journal of Respiratory Critical Care Medicine. 2000;161:665–673.
[7]. Perigo, J.F. and Prado, C. Evolution of occupational exposure to environmental levels of aromatic hydrocarbons in service stations. Ann Occup Hyg. 2005; 49: 233–240.
[8]. Adami, G., Larese, F., Venier, M., Barbieri, P., LoCoco, F., and Reisenhofer, E. Penetration of benzene, toluene and xylenes contained in gasoline's through human abdominal skin in vitro. Toxicol In Vitro.2006; 20: 1321–1330.
[9]. Benson, J.M., Gigliotti, A.P., March, T.H., Barr, E.B., Tibbetts, B.M., Skipper, B.J., Clark, C.R., and Twerdok, L. Chronic carcinogenicity of gasoline vapour condensate (GVC) and GVC containing methyl tertiary-butyl ether in f344 rats. J Toxicol Environ Health A. 2011; 74: 638–657.
[10]. Ayalogu, O.E., Igboh, N.M., and Dede, E.B. Biochemical changes in the serum and liver of albino rats exposed to petroleum samples (gasoline, kerosene, and crude petroleum). J Appl Sci Environ Mgt.2001; 5: 97–100.
[11]. Patrick-Iwuanyanwa, K.C., Onyemaenu, C.C., Wegwa, M.O., and Ayalogu, E.O. Hepatotoxicity and nephrotoxic effects of kerosene and petrol contaminated diets in Wistar albino rats. Res J Environ Toxicol. 2011; 5: 49–57.
[12]. Uboh, F.E., Akpanabiatu, M.I., Ndem, J.I., Alozie, Y., and Ebong, P.E. Comparative nephrotoxic effect associated with exposure to diesel and gasoline vapours in rats. J Toxicol Environ Health Sci. 2009; 1:68–73.
[13]. Dere, E. and Ari, F. Effect of benzene on liver functions in rats (Rattus norvegicus). Environ Monit Assess. 2009; 154: 23–27.
[14]. Akintonwa, A. and Oladele, A.A. Health effect of exposure to hydrocarbon on petrol filling station attendants in Lagos. Nig Q J Hosp Med. 2003; 13: 88–92.
[15]. Lippmann, S.J., Richardson, D.B., and Chen, J.C.M. Elevated serum liver enzymes and fatty liver changes associated with long driving among taxi drivers. Am J Ind Med. 2011; 54: 618–627.
[16]. Nwanjo, H.U. and Ojiako, O.A. Investigation of the potential health hazards of petrol station attendants in Owerri Nigeria. J Appl Sci Environ Manage. 2007; 11: 197–200.
[17]. Ponka A. Lead in the ambient air and blood of children in Helsinki.Sci Total Environ 1998; 138: 1-5.
[18]. Linn WS,Gong H,Shamoo DA et al. Chamber exposures of children to mixed ozone, sulphur dioxide and sulfuric acid. Arch Environ Health 1997; 52:179-87.
[19]. Wallenborn J.G., Kovalcik K.D., McGee J.K., Landis M.S., Kodavanti U.P. Systemic translocation of70zinc: kinetics following intratracheal instillation in rats. Toxicol. Appl. Pharmacol. (2009);234:25–32. doi: 10.1016/j.taap.2008.09.024.
[20]. Sharma R.P., Flora S.J., Drown D.B., Oberg S.G. Persistence of vanadium compounds in lungs after intratracheal instillation in rats. Toxicol. Ind. Health. (1987);3:321–329. doi: 10.1177/074823378700300304
[21]. Mani U., Prasad A.K., Suresh Kumar V., Lal K., Kanojia R.K., Chaudhari B.P., Murthy R.C. Effect of fly ash inhalation on biochemical and histomorphological changes in rat liver. Ecotoxicol. Environ. Saf.(2007);68:126–133. doi: 10.1016/j.ecoenv.2006.10.013.
[22]. Mills N.L., Donaldson K., Hadoke P.W., Boon N.A., Mac-Nee W., Cassee F.R., Sandström T., Blomberg A., Newby D.E. Adverse cardiovascular effects of air pollution. Nat. Clin. Pract. Cardiovasc. Med. (2009);6:36–44. doi: 10.1038/ncpcardio1399.
[23]. Khandoga A., Stoeger T., Khandoga A.G., Bihari P., Karg E., Ettehadieh D., Lakatos S., Fent J., Schulz H., Krombach F. Platelet adhesion and fibrinogen deposition in murine microvessels upon inhalation of nanosized carbon particles. J. Thromb. Haemostasis. (2010);8:1632–1640. doi: 10.1111/j.1538-7836.2010.
[24]. Khandoga A., Stampfl A., Takenaka S., Schulz H., Radykewicz R., Kreyling W., Krombach F. Ultrafine particles exert prothrombotic but not inflammatory effects on the hepatic microcirculation in healthy mice in vivo. Circulation. (2004);109:1320–1325. doi: 10.1161/01.CIR.0000118524.62298.E8 .
[25]. Ravnskor U. Experimental glomerulonephritis induced by hydrocarbons exposure. A systemic review. BMC nephrology; 2005; 6:15
[26]. Nauz A. Alejandro N.F. Falahatpisheh M.H. Kerzee J.K. Roths J.B. Ramos K. S . Distruption of glomerular cell-cell and cell-matrix interactions in hydrocarbon nephropathy. American journal of physiology; 2005; 289(6); F1291-303
[27]. Tang Y. Donnelly K.C. Tiffany-castiglioni E. Mumtaz M.M. Neurotoxicity of polycyclic aromatic hydrocarbons and simple chemical mixtures.Journal of toxicology and Environmental health; 2003; 66(10); 919-940
[28]. Elovaara E. Heikkila P. Pyy L. Mutanen P. Riihimaki V. Significance of dermal and respiratory uptake in creosote workers; exposure to polycyclic aromatic hydrocarbons and urinary excrection of 1-hydroxypyrene. Occupational and Environmental Medicine;1995; 52 (3):196-203.
[29]. TOMA . Tars are complex combinations of polycyclic aromatic hydrocarbons exposure levels from any source;1982
[30]. Sofer S. Milder L. and Gorodischer R. Acute pancreatitis in children following exposure to polycyclic aromatic hydrocarbons; 2003
[31]. Halliday A.N, Pettke T and Rea D.K. Remediation of polycyclic aromatic hydrocarbon.contaminated from incomplete combustion of hydrocarbon fuels; 2004.