PLASMA LIPIDS OF MALE WORKERS EXPOSED TO CEMENT DUST IN ABA

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ABSTRACT

Occupational exposure to cement dust causes many serious health hazards of which cardiovascular disorders are inclusive. This study is therefore aimed at evaluating the plasma lipids of cement dust exposed male workers in Aba. Hundred subjects comprising of fifty exposed workers and fifty non-exposed control group aged 21 – 61 years were randomly selected for this study and have been exposed for not less than eight years. The cement dust exposed workers were grouped based on their duration of exposure. They were measured for their weights (in kg), height (in meters), age, body mass index (BMI), plasma glucose and blood pressure. Lipid profile was measured using semi-automated analyzer and lipid ratios were calculated using lipid values .The data obtained were analyzed using Statistical Package for Social Sciences (SPSS version 25) and one way analysis of variance (ANOVA) with P-value equal to or less than 0.05 ($P \leq 0.05$). The mean total cholesterolTriglyceride LDL cholesterol were significantly high in cement dust exposed workers compared to control. Equally, observed in the study, was that there were no significant differences ($P>0.05$) in the diastolic blood pressure, lipid ratios and anthropometric indices measured. The study has shown that lipid profile, systolic blood pressure and fasting plasma glucose levels are significantly higher in cement workers as compared to the controls. Thus, this suggests that workers exposed to cement dust are at high risk of developing cardiovascular disorders. However, appropriate safety measures should be taken by cement workers to reduce the degree of exposure to cement dust, and also lipid profile analysis should be included as part of the routine medical laboratory test,so as to enable early detection and identification of workers at risk of developing cardiovascular disorders.

Key words: Cardiovascular disease, Hyperlipidemia, Triglyceride, Atherogenic index and Coronary artery

INTRODUCTION

Environmental and occupational pollution has always been a major cause of morbidity and mortality. The increased technological advancement, urbanization, industrial revolution, agricultural technologyhave, no doubt, improved our life styles, which couldnot be imagined decades ago, but these advancements have simultaneously polluted the natural environment. Environmental factors are being increasingly implicated in the pathogenesis of various biologicalderangements, including those disorders resulting to dyslipidemia. The incidence of occupational disease is constantly increasing throughout the world, especially in developing countries due to the lack of proper quality control documentation and the practical approach towards this mammoth problem (Fogarty *et. al.*, 2008).

Cement dust is a mixture of Calcium, Potassium, Silicon and Sodium which often include heavy metals like Aluminium, Cadium, Lead, Zinc, Iron and Chromium which in excess are hazardous to the environment with impact on human health. The cement dust chemicals enter the body of cement workers through different ways and cause internal derangements. Studies have shown that cement dust exposure can be associated with increased oxidative stress and impaired lipid metabolism (Khan *et. al.*, 2008). This further results to dyslipidemia and predispose the exposed workers to the risk of developing cardiovascular diseases. The cardiovascular risk factors include dyslipidemia, high blood pressure and diabetes. Other cardiovascular risk factors are non-modifiable and include age, gender and family history (Ademuyiwa, et al 2005)

Dyslipidemia is a single strong risk factor for the development of cardiovascular events and atherosclerosis is the most common. It has been described as a disease of the economically advanced societies, but recently, it has

found its way into the semi-urban societies and among its dwellers, who are at the increasing risk of developing cardiovascular accidents. Several factors, including cement dust exposure are implicated for increased risk. Cement production is a dusty operation leading to the exposure of factory workers to cement dust. The pollutants in the cement industry are emitted from the various production processes such as crushing, blending, storage and packing (Fryar *et. al., 2019).*

The aerodynamic diameter of cement particles makes it a potential health hazard as these are respirable in size and reaches internal organs particularly lungs leading to occupational lung diseases. This size distribution would make the trachea-bronchial respiratory zone, the primary target of cement deposition. The main route of entry of cement dust particles in the body is the respiratory tract and/or the gastrointestinal tract by inhalation or swallowing, respectively. Both routes, especially the respiratory tract are exposed to numerous potentially harmful substances in the cement exposed environment (Anne *and Fell, 2010* ,Fryar et. al., 2019).

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Hence, early identification and diagnosis of dyslipidemia at its earliest stage among individuals exposed to the risk factors is a worthwhile cardiovascular preventive measure. It was based on these, thatthe study was carried out to monitor the lipid profile of workers expose to cement dust.

MATERIALS AND METHOD

This study was carried out in Aba metropolis, Abia State. It is mainly an Urban settlement and surrounded by small villages and town. The indigenous people are the Ngwa (Falola *et al.*, 2008).

The studywas designed to access the plasma lipids of male cement dust exposed workers in Aba metropolis. Hundred male subjects participated in the study. Fifty people occupationally exposed to cement dust were recruited as the test subjects. Fifty age-matched apparently healthy male subjects who are not occupationally exposed to cement dust were randomly selected from the staffs and students of Abia State University Teaching Hospital as controls. The cement workers were grouped based on their duration of exposure to cement dust of not less than without years.

All respondents for lipid and glucose estimations were asked to do an overnight fasting of 12 hours. About 7 millilitres (7 ml) of fasting blood samples were collected by venipuncture techniques from the antecubital vein into sterile containers under aseptic conditions. About 2 ml of blood was stored in fluoride oxalate containing bottles for glucose estimation and 5 ml of blood was stored in lithium heparin bottle for lipid assay.

Plasma glucose, High density lipoprotein (HDL), Total Cholesterol (TC), Triglyceride (TG), Low density lipoprotein (LDL) were determined using the methods of Tietz, (1995) and Atherogenic index (AIP) was calculated.

Data was analyzed using Statistical Package for Social Sciences (SPSS) (Indrayan and Kumar, 2017). The differences between the groups were compared using one-way analysis of variance (ANOVA) and Student t-test with a P-value less than or equal to 0.05 ($P \leq 0.05$) which was considered as being statistically significant. Results were expressed as mean ± SD (Standard Deviation).

RESULTS AND DISCUSSION

COMPARISON OF MEAN ± STANDARD DEVIATION OF PLASMA GLUCOSE, LIPID PROFILE, ANTHROPOMETRIC INDICES, BLOOD PRESSURE AND LIPID RATIOS IN CEMENT DUST EXPOSED WORKERS AND CONTROLS.

Parameters	Cement workers(n=50)	Controls (n=50)	Calc.t	Crit.t	P(≤0.05)	Sig.
Glu (mmol/L)	5.89 ± 0.57	5.43 ± 0.83	3.246	1.98	0.002	Sig
TC (mmol/L)	4.80 ± 1.26	3.92 ± 0.78	4.185	1.98	0.0001	Sig

TG (mmol/L)	1.18 ± 0.50	0.86 ± 0.34	3.695	1.98	0.0004	Sig
HDL (mmol/L)	1.22 ± 0.50	1.01 ± 0.31	2.475	1.98	0.015	Sig
LDL (mmol/L)	3.06 ± 1.14	2.53 ± 0.84	2.630	1.98	0.01	Sig
CRI-I	4.42 ± 1.74	4.31 ± 1.62	0.335	1.98	0.735	NS
CRI-II	2.93 ± 1.63	2.73 ± 1.14	0.679	1.98	0.499	NS
AC	3.42 ± 1.74	3.26 ± 1.56	0.467	1.98	0.641	NS
AIP	-0.019 ± 0.24	-0.081 ± 0.24	1.280	1.98	0.204	NS
Weight (kg)	65.08 ± 5.99	65.6 ± 7.56	0.377	1.98	0.707	NS
BMI (kg/m^2)	24.04 ± 1.83	24.19 ± 2.47	0.346	1.98	0.730	NS
Systolic (mmHg)	127.48 ± 8.94	121.94 ± 8.22	3.194	1.98	0.002	Sig
Diastolic (mmHg)	81.42 ± 6.57	79.82 ± 5.94	1.265	1.98	0.209	NS

KEYWORDS: The following abbreviations stand for:

Crit. t-Critical t value

Calc. t-Calculated t value

Sig- Significant

NS- Not Significant

Glu- Glucose

TC- Total cholesterol

TG- Triglycerides

HDL- High density lipoprotein

LDL- Low density lipoprotein

AC- Atherogenic coefficient

AIP- Atherogenic index of plasma

SBP- Systolic blood pressure

DSP- Diastolic blood pressure

CRI-I- Castelli risk index I

CRI-II- Castelli risk index II

Table represents comparison of mean ± standard deviation of plasma glucose, lipid profile, anthropometric indices, blood pressure and atherogenic ratios in cement dust exposed workers and controls. The total cholesterol, HDL-cholesterol, LDL-cholesterol,triglycerides, fasting plasma glucose , and systolic blood pressure were significantly higher in cement workers than in controls. There was, however, no significant difference (P>0.05) in the mean values of anthropometric indices, atherogenic coefficient, castelli risk index I and II, atherogenic index of plasma and diastolic blood pressure.

These elevations can be attributed to some of the toxicants present in the cement dust. When heavy metals in cement is inhaled it induces oxidative stress which further causes lipid peroxidation, and consequently, dyslipidemia. Other toxic substances such as dioxins interfere with lipid metabolism. 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) which is a type of dioxin present in cement inhibits lipoprotein lipase, which results in decreased clearance of VLDL and chylomicrons resulting in increased levels of triglycerides (Bassey et al (2017).).

Lead increases the activity of HMG CoA reductase and reduces the number and affinity of LDL receptors for cholesterol. This may be responsible for the higher total cholesterol and LDL-cholesterol observed in the cement workers. These findings conform with those of other studies (Aydin et al., 2010; Modhir et al., 2014; Dier et al., 2014 and Bassey et al., 2017) who reported increases in total cholesterol, LDL-cholesterol and triglycerides in cement workers.

The increased HDL-cholesterol found in the cement workers may be attributed to the physical activity greatly engaged by them resulting from the nature of their work (Ademuyiwa et al., 2005). This finding is conformitywith another study whichobserved increased HDL-cholesterol in the cement workers (Bassey et al., 2017).

Elevated serum LDL cholesterol and triglyceride concentrations may be risk factors to atherosclerosis which causes thickening of the walls of blood vessels (Guttman, 1999; Ochei and Kolhatker, 2007).

There was no significant difference in the mean of the anthropometric indices assessed among the cement workers and controls. This finding is consistent with the studyreported byBassey et al (2017).

A significant increase in the mean of the plasma glucose was found in the cement workers, when compared with the controls. This is line with the study of Zober et al., (1994) but did not conform with Calvert et al., (1999).Cement dust contains the most toxic form of dioxin, known as 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD). This dioxin induces hyperglycemia by lowering the insulin secretion by beta cells which reduces glucose up take resulting fromdecrease in Glucose Transporter 2 (GLUT2) (Matsumura, 1995).

Dioxin also activates the Aryl hydrocarbon receptor AhR and then suppress the function of peroxisome proliferator activated receptor (PPAR) γ, which may lead to insulin resistance (Nebert et al., 2004). PPARs are ligand-activated transcription factors that control lipid metabolism and homeostasis, and (PPAR) γ promotes differentiation of adipocytes and translation of the glucose transporter protein GLUT4 (Remillard et al., 2002). Thus, dioxin exposure progressively lower the translation of GLUT4 and cause insulin resistance and hyperglycemia.

The prevalence of dyslipidemia among cement dust exposed workers are high. This calls for a parallel measure to curtail this mammoth problem. Cement surrounds us every day and therefore, its toxicity cannot be overemphasized and neglected.

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REFERENCES

Ademuyiwa, O., Ugbaja, R. N., Idumebor, F. and Adebawo, O. (2005). Plasma lipid profiles and risk of cardiovascular disease in occupational lead exposure in Abeokuta. *Lipids in Health and Disease*,**4**: 19.

Anne K. And Fell M. (2010). Airway inflammation in cement production workers. *Occupational Environmental Medicine.* 67: 6

Aydin, S., Croteau, G. A., Sahin I. and Citil, C. (2010). Ghrelin, nitrite and paraoxonase/arylesterase concentrations in cement plant workers. *Journal of Medical Biochemistry*,**29**(2): 78–83.

Aziz, M. and Yadav, K. S. (2016). Pathogenesis of atherosclerosis. *Medical and Clinical Journal*, **2**: 3.

Bassey, I. E., Akpan, U. O., Nehemiah, E. D., Arekong, R., Okonkwo, O. L. and Udoh, A. E. (2017). Cardiovascular disease risk factors and cardiac markers among male cement workers in Calabar, Nigeria. *Journal of Chemical Health Risks*.**7**(2), 85–94.

Bravo, E., Napolitano, M. and Botham, K. (2012). Post prandial lipid metabolism: The missing link between life-style habits and the increasing incidence of metabolic diseases in western countries. *Journal of Translational Medicine*, **2**: 1–13.

Calvert, G. M., Sweeney, M. H. and Deddens, J. (1999). Evaluation of diabetes mellitus, serum glucose, and thyroid function among United States workers exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Occupational and Environmental Medicine*, 56: 270–276.

Dier, M. S., Sirwan, M. M. and Longman, O. H. (2014). Some biochemical and Hematological parameters among petroleum and cement factory workers in Sulaimaniyah city/Kurdistan/ Iraq. *Chemistry and Materials Research*, **6**(8): 29–33.

Falola, Toyin, Heaton and Matthew. (2008). A History of Nigeria. Available at https://assets.cambridge.org/97805218/62943/frontmatter/9780521862943_frontmatter.pdf

Fogarty R, Halpin K, Hyatt AD, Daszak P, and Mungall BA. (2008). Henipavirus susceptibility to environmental variables. *Virus Research*. 2008; **132**(1–2): 140–144.

Friedewald, W. T., Levy, R. I. and Fredrickson, D. S. (1972). Estimation of the concentration of low-density lipoprotein cholesterol in plasma without use of preparative ultra- centrifuge. *Clinical Chemistry*,**18**: 499–502.

Fryar C D, Chen T C, and Li X. (2019). Prevalence of uncontrolled risk factors for cardiovascular disease. *National Center for Health Statistics*,**19**: 10-12

Guttman, G. S. (1999). Biology. *McGraw Hill*, **1**: 964–965.

Indrayan, A. and Kumar, R. M. (2017). Medical Biostatics. *Chemical Rubber Company Press*, 4: 800–994.

Khan, S., Cao, Q., Zheng, Y. M., Huang, Y. Z. and Zhu Y. G. (2008). "Health risks of heavy metals in contaminated soils and food crops irrigated with wastewater in Beijing, China," *Environmental Pollution*,**152** (3): 686–692, 2008.

Lopez-virella, M. F., Stone, P., Eltis, S. and Colwell, J. A. (1977). Cholesterol determination in HDL separated by three different methods. *Clinical Chemistry*,**23**: 822–884.

Matsumura, F. (1995). Mechanism of action of dioxin-type chemicals, pesticides, and other xenobiotics affecting nutritional indexes. *American Journal of Clinical Nutrition*, **61**: 695–701.

Modhir, N.A., Mohamed, A.R., and Rawaa. J. (2014). Study of some Biochemical changes in the blood serum of Sadet-Al-Hindiah cement factory workers, Babylon, Iraq. *International Journal of Interdisciplinary and Multidisciplinary Studies*, **2**: 700–702.

Nebert, D. W., Dalton, T. P. and Okey, A. B. (2004). Role of aryl hydrocarbon receptor- mediated induction of the Cytochromes P450-1 enzymes in environmental toxicity and cancer. *Journal of Biological Chemistry*, **279**: 23847–23850.

Ochei, J. and Kolhatker, A. (2007). Medical Laboratory Science (Theory and Practice) *Tata McGraw Hill*, **1**:196.

Remillard, R. B. and Bunce, N. J. (2002). Linking dioxin to diabetes: epidemiology and biologic plausibility. *Environmental Health Perspectives*, **110**: 853–858.

Tietz, N. W. (1995). Clinical guide to laboratory tests. *Saunders Company*, **2**: 1–178.

Trinder, P. (1969). The principle of assay for enzymatic determination of cholesterol. *Annual Clinical Biochemistry*, **6**: 24–25.

Trinder, P. (1969). The principle of assay for enzymatic determination of cholesterol. *Annual Clinical Biochemistry*,**6**: 24–25.

Valko, M., Rhodes, C. J., Moncol, J., Izakovic, M. and Mazur, M. (2006). Free radicals, metals and antioxidants in oxidative stress-induced cancer. *Chemico-Biological Interactions*, **160**: 1–40.

Zober, A., Ott, M. G. and Messerer, P. (1994). Morbidity follow up study of BASF employees exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) after a 1953 chemical reactor incident. *Occupational and Environmental Medicine*, **51**:479–486.