


African Journal of Tropical Medicine and Biomedical Research (AJTMBR)



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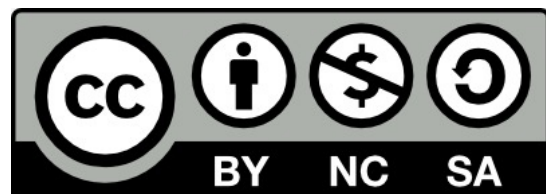


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Biochemical Impact Of Occupational Cement Dust Exposure On Block Industry Workers In Rivers State, Nigeria

¹John, GC ²Anosike IK

ABSTRACT

Introduction: Occupational exposure to cement dust is a recognized health hazard linked to systemic complications. This study evaluated the impact of chronic cement dust exposure on the biochemical parameters of cement block moulders in Khana Local Government Area (LGA), Rivers State, Nigeria, to assess potential renal and electrolytic alterations.

Materials and Methods: cross-sectional study was conducted involving 70 male participants, categorized into cement block moulders (subjects, n=35) and non-exposed individuals (controls, n=35). Five millilitres (5ml) of venous blood were collected from each participant into heparinized bottles. Samples were analyzed for urea, creatinine, sodium, and potassium using standard spectrophotometric methods. Statistical analysis was performed using Mean and Standard Deviation, with significance set at $P < 0.05$.

Results: The study revealed a statistically significant increase ($P < 0.05$) in serum creatinine and potassium levels among the cement block moulders compared to the control group. Conversely, no significant differences ($P > 0.05$) were observed in serum sodium and urea levels between the two groups.

Conclusion: Chronic exposure to cement dust is associated with significant elevations in serum creatinine and potassium, indicating early signs of altered renal filtration and electrolyte imbalance. While urea and sodium levels remained stable, the elevation in specific markers suggests that block moulders are at an increased risk of developing renal impairment. Regular health screenings and the consistent use of personal protective equipment (PPE) are recommended to mitigate these occupational risks

Keywords: Cement dust, Creatinine, Potassium, Renal function, Occupational health, Khana LGA.

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Introduction

Cement dust is generated during the production, packaging, loading, and offloading of cement. Everyone in the industry—from directors and managers to staff, customers, and bricklayers—is exposed to the inhalation of this dust. Furthermore, individuals living in the vicinity of cement plants or those passing by are also at risk.

Consistent exposure to cement dust over long periods can cause systemic toxicity due to the accumulation of toxic constituents⁹. The primary chemical components of cement include calcium, silica, alumina, and iron. Calcium is derived from limestone or chalk, while silica, alumina, and iron are sourced from sand and clay¹. Different organs, tissues, and cells are affected in varying ways and

degrees when exposed to these toxic elements. The results for creatinine and potassium in this study align with findings by ², which showed a significant increase in the creatinine levels of exposed subjects compared to the control group, but no significant increase in sodium levels. This research aims to assess the effects of cement dust on specific biochemical parameters among cement block moulders in the Khana Local Government Area of Rivers State, Nigeria. Several studies have demonstrated links between cement dust exposure and the chronic impairment of lung function and respiratory symptoms. Cement dust irritates the skin, the mucous membranes of the eyes, and the respiratory system. Its deposition in the respiratory tract triggers an alkaline reaction, leading to increased pH values that irritate exposed membranes³. Occupational exposure has also been associated with an increased risk of liver abnormalities, pulmonary disorders, and carcinogenesis. Decreased antioxidant capacity and increased plasma lipid peroxidation have been proposed as potential causal mechanisms for these diseases⁴.

Additionally, evidence suggests that cement dust exposure acts as an independent risk factor—separate from tobacco, alcohol, and asbestos—for laryngeal carcinoma⁵. Cement dust contains heavy metals such as nickel, cobalt, lead, and chromium, which are pollutants hazardous to the biotic environment, adversely impacting vegetation, human and animal health, and entire ecosystems⁶. Inhalable dust concentrations in production plants, especially during cleaning tasks, are considerably higher than those found at construction sites⁷. People living in high-exposure zones are severely affected by respiratory problems and gastrointestinal diseases⁸. Studies indicate that these adverse respiratory effects—including increased frequency of symptoms and decreased ventilatory function—cannot be explained by age, BMI, or smoking, and are thus

likely caused by cement dust. Symptoms such as chest pain, cough, and eye irritation are prevalent in villages near cement plants. A relative risk ratio assessment indicates that exposed subjects are 7.5 to 22.5 times more likely to develop these diseases compared to unexposed individuals¹⁰.

Materials and Methods

Sample Collection

This study was carried out in Khana Local government area, Rivers State Port Harcourt. 70 (seventy) adult men were involved in this study, 50 (fifty) were cement block moulders (subject) and (twenty) 20 were non-cement block moulders.

Ethical Considerations

The study followed the ethical principles guiding the use of human participants in research. Ethical approval was sought from University of Port Harcourt Ethical Committee. All procedures were followed in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2000. (World Medical Association declaration of Helsinki, 2000). Informed consent were obtained from all the research participants. With respect to confidentiality, no identifiers like name of respondents were required.

Serum Preparation

5ml of blood specimen drawn from 50 cement block moulders and 20 drawn from non-cement block moulders and were put into oxalate bottle, and then the serum was then separated from the blood by centrifugation of 3000rpm at 15 minute and was analysed.

Principle: Urea in serum is hydrolyzed to ammonia in the presence of urease. The ammonia is then measured in spectrophotometer. The solution in each of the tubes was mixed and incubated at 37⁰c for 10 minutes.

I mixed the solution in each of the test tube and incubated at 37^oc for 15 minutes after which I zeroed the spectrophotometer using distilled water at a wavelength of 546nm. Then I read absorbance of standard and test and then recorded my result.

Potassium Determination

Potassium was assayed using the sodium tetraphenylboron method. In this reaction, potassium ions react with sodium tetraphenylboron in a specifically prepared mixture to produce a colloidal suspension. The resulting turbidity is directly proportional to the potassium concentration. After incubation for 3 minutes at room temperature, the absorbance was measured at 500 nm against a reagent blank using a spectrophotometer.

Creatinine Determination

Serum creatinine was assayed by the Jaffé method, as previously described by ¹¹. The

principle involves the reaction of creatinine with picric acid in an alkaline medium to form a red-colored complex, which is then measured spectrophotometrically

Data were subjected to statistical analysis using Statistical Package for Social Sciences (SPSS) version 20.0. Data were expressed as mean and standard error of mean. Student's t-test was adopted for comparison. *P* values of < 0.05 were considered statistically significant

Results

Reject Ho at $P < 0.05$ i.e. $t_{cal} > t_{critical}$

There is a significant difference in creatinine subject when compare with control.

Ho: – No significant difference between the subject and control of urea and sodium.

H₁: There is significant difference between the subject and control of creatinine and potassium respectively.

Table 1: Biochemical parameters of creatinine and urea

Parameter/ Group	Creatinine ($\mu\text{mol/L}$)	Urea (mmol/l)
Subject	126.46 \pm 2.49	3.99 \pm 1.10
Control	89.22 \pm 10.44	4.12 \pm 0.79
P-value	$P < 0.05$	$P > 0.05$

The mean and standard deviation of Biochemical parameters of creatinine and urea concentration in the tested subject are 126.46 \pm 2.49 and 3.99 \pm 1.10 all in ($\mu\text{mol/L}$) while control are 89.22 \pm 10.44 and 4.12 \pm 0.79 all in (mmol/l) are shown above in table 1.

Table 2 Biochemical parameters of potassium and sodium

Parameter/ Group	Potassium (mmol/l)	Sodium (mmol/l)
Subject	3.79 \pm 0.64	143.49 \pm 6.54
Control	4.53 \pm 0.53	143.90 \pm 5.37
P-value	$P < 0.05$	$P > 0.05$

The mean and standard deviation of Biochemical parameters of potassium and sodium concentration in the tested subject are 3.79 \pm 0.64 and 143 \pm 6.54 all in (mmol/l) while control are 4.53 \pm 0.53 and 143.90 \pm 5.37 all in (mmol/l) are shown above in table 2.

DISCUSSION

The observed significant increase in serum creatinine and potassium ($P < 0.05$) among cement block moulders indicates a profound compromise in renal functional integrity. These results align with findings by ², which also reported significant creatinine elevation. The nephrotoxicity observed is likely driven by the inhalation and systemic absorption of heavy metals found in cement, specifically Lead (Pb) and Hexavalent Chromium (Cr VI). These metals are filtered by the glomerulus and subsequently reabsorbed by the proximal convoluted tubules. Once inside the tubular cells, they trigger the production of Reactive Oxygen Species (ROS), leading to oxidative stress and lipid peroxidation of the renal cellular membranes. This biochemical assault results in Renal Tubular Necrosis, effectively reducing the kidney's ability to filter waste. The concurrent rise in creatinine and potassium is a classic hallmark of a declining Glomerular Filtration Rate (GFR): Creatinine: As a byproduct of muscle metabolism usually filtered freely by the kidney, its accumulation in the blood is a direct "proxy" for reduced filtration capacity. Potassium (Hyperkalemia): The kidneys are responsible for excreting 90% of dietary potassium. When the tubular cells are damaged—specifically the distal tubule and collecting ducts—the sodium-potassium exchange pump (Na^+/K^+ -ATPase) fails. Consequently, potassium is not secreted into the urine but is instead retained in the blood. The rejection of the H_1 hypothesis for sodium and urea suggests a "differential sensitivity" in renal markers. Urea levels are highly influenced by non-renal factors such as dietary protein intake and hydration status, making it less specific than creatinine. Similarly, the body employs aggressive homeostatic mechanisms (such as aldosterone regulation) to maintain sodium levels within a narrow range, even during the early stages of renal distress. This indicates that

creatinine and potassium are more sensitive early-warning biomarkers for cement-induced nephrotoxicity in this population. The renal impairment seen here likely represents only one facet of a broader multi-organ toxidrome. The heavy metals identified in cement dust, particularly lead, are known to inhibit delta-aminolevulinic acid dehydratase (ALAD), an enzyme critical for heme synthesis. This suggests that while these workers are currently presenting with renal distress, they are also at high risk for occupational anemia and hepatotoxicity, as evidenced by literature linking chronic exposure to elevated liver enzymes (ALT/AST).

CONCLUSION

In conclusion, this study confirms that occupational exposure to cement dust among block moulders in Khana LGA leads to significant alterations in critical renal biomarkers, specifically creatinine and potassium. The significant elevation of these parameters ($P < 0.05$) in comparison to the control group indicates early-stage renal impairment, likely resulting from the systemic absorption of toxic cement constituents. While sodium and urea levels remained relatively stable ($P < 0.05$), the overall biochemical profile suggests that chronic inhalation and dermal contact with cement dust pose a substantial threat to the long-term health of these workers. To prevent irreversible organ damage, it is imperative that regulatory bodies and health educators implement mandatory annual workshops. These programs must focus on the consistent use of Personal Protective Equipment (PPE), improved dust-suppression techniques, and regular medical surveillance. Ultimately, mitigating these health risks requires a shift in workplace culture where safety protocols are as fundamental to the block-moulding process as the production itself.

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