Effect of Cement Pollution on Creatinine and Blood Urea in Hamam Al-Alil Factory Workers

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Abstract:

The present study investigate the effects on 55 workers which were divided into groups according to the departments of Hamam AL-Alil Cement Factory-Iraq. The parameters included in the study are: potassium, sodium, blood urea, BUN, and creatinine.

The results showed that there was a significant increase in sodium, blood urea and BUN in workers serum which increased with increasing exposure time. The results also showed a significant decrease in potassium in workers serum which increased with increasing exposure time in all factory department. Whereas the creatinine illustrate no significant decrease with increasing exposure time.

أظهرت النتائج بأن هنالك زيادة معنوية في كل من مستوى ايونات الصوديوم ويوريا الدم ونتروجين يوريا الدم مع انخفاض معنوي في مستوى ايونات البوتاسيوم في حين أعطى الكرياتنين انخفاض غير معنوي في مصل دم العاملين مع ازدياد فترة التعرض.

Introduction:

Portland cement is composed of tri-calcium silicate, di-calcium silicate, tri-calcium aluminate, tetra-calcium aluminoferrate and gypsum, with trace constituents like potassium oxide, sodium oxide, chromium compound and nickel compound (Adamu and Iloba, 2008).

Cement industry is one of the most pollutant industries due to large quantities of its gas emissions and dust (Lioy *et al.*, 2002). The major gaseous emissions are nitrogen oxides (NO_x) and sulfur oxides (SO₂). Other emissions of less significance are carbon dioxide (CO₂), thallium, ammonia and heavy metals (Carrasco *et al.*, 2002). And the composition of dust can be greatly diversified due to a great variety of materials used at the different stages that lead to cement production, like raw material grinding, coal mills, rotary kilns, clinker cooling, finished grinding, silos and packaging (Somma *et al.*, 2006).

All process involving potential exposure to hazardous substances may cause harm as a result of intake of the substances into the body, by inhalation through the respiratory tract, by ingestion, or through the skin. The effect may appear on a number of organs and systems of the human body (Robert *et al.*, 2001).

Any variation in minerals and the enzymes activity is an indication of the effect of the pollution. Therefore, this study is to evaluate the cement dust exposure and other pollutants in workers employed in different departments of cement factory by measuring potassium and sodium ions levels, blood urea, blood urea nitrogen and creatinine which may lead to know the effects on kidney's functions.

Materials and Methods Specimen Collection and Serum Preparation

The blood specimen collected from 55 workers of different departments of Hamam AL-Alil Cement Factory and from students and employees of the University of Mosul as the control group. 4-5 ml of the drawn blood were put in plain tube, left to complete clot formation, the serum was then separated from the clotted blood by centrifugation at 3000 rpm for 15 min., then decanted into clean and sterile plain tubes and stored at (-20) C^o.

Estimation of Potassium and Sodium levels in Serum

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Atomic absorption spectroscopy (AAS) technique was used to determine K and Na levels using the standard curve to convert the optical density into concentration (Zhang and Zhang, 2003).

Estimation of Blood Urea Activity and Blood Urea Nitrogen in Serum

The blood urea activity and blood urea nitrogen (BUN) was determined using a BIOLABO-Reagent/France kit (Tietz, 1999).

Estimation of Creatinine Activity in Serum

The serum creatinine was determined using a Biomaghreb/France kit (Henry, 1984).

Statistical Analysis

The statistical program (SPSS) was used to analyze the data (Indrayan and Sarkaddam, 2001).

Results and Discussion

K and Na levels :

The result showed that there was a significant decrease in potassium ion level in the serum of cement workers especially at Cement mill and Kiln departments (Table, 1). The result also indicated that there was a significant decrease in potassium ion with increasing exposure time to cement pollution (Table, 2).

The increase might be due to thallium is a mainly associated with potassium and rubidium, the source of thallium are gaseous emission of cement factories (EPA, 2006 ; HSDB, 1998). Also thallium and potassium ions have similar prosperities and on biological handling, both ions transverse cell membranes in similar way (Kazantzis, 1980). Or might be due to sulfur dioxides is observed by (Asha and Nirupma, 2004) who found a significant decrease in potassium level in the serum specimen of albino rats exposed to 120 ppm of sulfur dioxides after 4 weeks.

The results also showed a significant increase in sodium ion level in workers serum of all departments of the factory especially in Raw Mill and Kiln departments (Table, 1). A significant increase with increasing exposure time was also observed (Table, 2).

The increase might be due to the fact that potassium levels often change

with sodium level, when sodium level go up potassium level go down and reversed, or might be due to kidney disease or injury (Caroline, 2008).

Donortmonto	K	%	Na	%
Departments	Mean ± SE	Decrease	Mean ± SE	Increase
Control	$5.02\pm0.15^{\ b}$	-	139.9 ± 1.54 ^a	-
Maintenance	3.92 ± 0.33 ^a	22.0	142.0 ± 4.97 $^{\rm a}$	1.5
Crusher	4.24 ± 0.19^{ab}	15.5	147.6 ± 2.60 ^{ab}	5.5
Raw mill	4.13 ± 0.20^{a}	17.7	163.3 ± 6.98 bc	16.7
Cement mill	3.90 ± 0.19^{a}	22.3	142.9 ± 1.82 ^a	2.1
Kiln	3.84 ± 0.21 ^a	23.5	166.9 ± 6.56 ^c	19.2
Packing	4.27 ± 0.23 ^{ab}	14.9	159.9 ± 5.01^{bc}	14.3

Table1: K and Na levels in s	serum of ceme	ent workers	depending or
	departments (1	mmol/L).	

* Different letter vertically refers to presence of significant differences between treatment at $P \le 0.05$, according to Duncan - test.

Table 2 : K and Na levels in serum of cement workers depending on exposure time (mmol/L).

Variables	K	0/0	Na	0/0
Exposure time	Mean ± SE	Decrease	Mean ± SE	Increase
Control	$5.02\pm0.15^{\text{ b}}$	-	139.9 ± 1.54 ^a	-
1-3	4.19 ± 0.14 ^a	16.5	151.2 ± 4.93 ^b	9.7
4 - 6	3.94 ± 0.13 ^a	21.5	155.8 ± 4.02 ^b	12.3
7 – 9 >	3.88 ± 0.22 ^a	22.7	157.5 ± 3.28 ^b	13.5

* Different letter vertically refers to presence of significant differences between treatment at $P \le 0.05$, according to Duncan - test.

Blood urea and Creatinine:

The result indicated that there was a significant increase in blood urea in the serum of cement factory workers, the highest activity was in the Kiln and Crusher departments (Table, 3). The urea also increased with increasing exposure time (Table, 4).

The increase might be due to the tendency of urea enhancing protein catabolism together with accelerated amino acid deamination for gluconeogensis, on the other hand, the elevated levels of urea might be due to the destruction of red blood cells (Adamu and Audu, 2008; Calistus Jude *et al.*, 2002). And any malfunctioning in glomerular filtration results in the retention of substances including urea (Kamis *et al.*, 2003).

The results agree with publication of (Adamu and Kori-Siakpere, 2008) who found a significant increase in urea in serum, liver and kidney of the African catfish (*Clarias gariepinus*) when exposed to Portland cement powder in solution for 15 days.

	Blood Urea		Creatinine
Departments	Mean ± SE	% Increase	Mean ± SE
Control	$22.7\pm0.44^{\text{ a}}$	-	0.71 ± 0.01 a
Maintenance	27.0 ± 1.74 ^{ab}	18.9	0.71 ± 0.03 a
Crusher	32.4 ± 2.89 ^b	42.7	0.69 ± 0.06 a
Raw mill	26.5 ± 2.81 ^{ab}	16.7	0.72 ± 0.04 a
Cement mill	28.0 ± 2.67 ^{ab}	23.3	0.71 ± 0.03 a
Kiln	29.5 ± 3.08 ^{ab}	30.0	0.71 ± 0.04 a
Packing	24.0 ± 1.99 ^{ab}	5.7	0.69 ± 0.03 ^a

Table 3 : Effect of cement pollution on blood urea and creatinine in serum of
cement workers depending on departments (mg/100 ml).

* Different letter vertically refers to presence of significant differences between treatment at $P \le 0.05$, according to Duncan - test.

The result demonstrated that there was no effects on serum creatinine in cement workers (Table, 3). Also the results showed non significant decreased between creatinine and exposure time to cement pollution(Table, 4).

This might be due to that the decrease not occur in the first time of work and with respect to the slightly decrease with exposure might be due to that cement adversely interfered with the metabolism of creatinine leading to its observed reduction, an indication of partial loss of its functional capacity of tubular excretion (Zilva *et al.*, 1991).

Variables	Blood Urea		Creatinine	
Exposure time	Mean ± SE	% Increase	Mean ± SE	% Decrease
Control	$22.7\pm0.44^{\text{ a}}$	-	0.71 ± 0.01 a	-
1-3	27.1 ± 3.69^{ab}	19.3	0.69 ± 0.04 a	2.8
4 - 6	28.7 ± 1.14 ^b	26.4	0.67 ± 0.03 $^{\rm a}$	5.6

Table 4 : Effect of cement pollution on blood urea, BUN and creatinine inserum of cement workers depending on exposure time (mg/100 ml).

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7 – 9 >	30.7 ± 3.07 ^b	35.2	0.66 ± 0.02 $^{\rm a}$	7.0

* Different letter vertically refers to presence of significant differences between treatment at $P \le 0.05$, according to Duncan - test.

BUN and BUN/Creatinine ratio:

The result in (Table, 5) showed that there was a significant increase in BUN in cement workers serum in all factory departments, the increase was proportional to exposure time (Table, 6).

The increase might be due to kidney or liver damage when exposure to thallium (Conerly and Blain, 2008). The results are in agreement with (Leung and Ooi, 2000; Fleck and Appenroth, 1996) who stated that the elevate of clinical chemistry parameters such as ALT, AST, BUN, blood glucose and sodium levels, indicating liver and kidney damage with thallium exposed.

Results also showed that there were increase in BUN/Creatinine ratio in workers serum of all cement factory departments (Table, 5), the highest in Kiln and Crusher. Also the increase was proportional with exposure time (Table, 6).

The increase might be due to the fact that ammonia formed from the oxidative deamination of amino acids is converted to urea by enzymes in the liver and then excreted in the urine by the kidneys, thus any change occurred in these enzymes (ALT, AST, LDH, ALP, ...ect.) (AL-Hayali, 2009; Adamu and Iloba, 2008) may accelerate the delivery of amino acids to the liver which can enhance urea nitrogen formation and increase the BUN/Creatinine ratio (Brian and Robert, 1999).

	BUN (mg/100 ml)		BUN/Creatinine	
Departments	Mean ± SE	% Increase	Ratio	% Increase
Control	10.6 ± 0.20 a	-	14.9	-
Maintenance	$12.6\pm0.81~^{ab}$	18.8	17.7	18.7
Crusher	15.1 ± 1.36 ^b	42.4	21.8	46.3
Raw mill	12.3 ± 1.29 ^{ab}	16.0	17.0	14.1
Cement mill	13.1 ± 1.25 ^{ab}	23.5	18.4	23.4
Kiln	13.7 ± 1.44 ^{ab}	29.2	19.3	29.5
Packing	11.1 ± 0.92 ^{ab}	4.7	16.0	7.3

Table 5 : Effect of cement pollution on BUN and creatinine ratio in serum of cement workers depending on departments.

* Different letter vertically refers to presence of significant differences between treatment at $P \le 0.05$, according to Duncan - test.

Variables	BUN (mg/100 ml)		BUN/Creatinine	
Exposure time	Mean ± SE	% Increase	Ratio	% Increase
Control	10.6 ± 0.20 $^{\rm a}$	-	14.9	-
1-3	12.6 ± 1.72 ^{ab}	18.8	18.2	22.1
4-6	13.1 ± 0.54 ^b	23.5	19.5	30.8
7 – 9 >	13.6 ± 1.28 ^b	28.3	20.6	38.2

Table 6 : Effect of cement pollution on BUN and creatinine ratio in serum of cement workers depending on exposure time.

* Different letter vertically refers to presence of significant differences between treatment at $P \le 0.05$, according to Duncan - test.

Conclusion

The changes may occurred in blood urea and creatinine can be considered suitable prognostic indicators of renal function in biomonitoring studies on workers exposed to occupational hazards.

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References

- Adamu, K. M. and Audu, B. S. 2008. Haematological assessment of the Nile tilapia Oreochromis niloticus exposed to sublethal concentrations of Portland cement powder in solution. International Journal of Zoological Research 4(1): pp. 48-52.
- Adamu, K. M. and Iloba, I. K. 2008. Effects of sublethal concentrations of Portland cement powder in solution on the tissue aminotransferases of the African catfish (Clarias gariepinus) (Burchell, 1822). Acta Zoologica Lituanica, 18(1) : pp. 50-54.
- Adamu, K. M. and Kori-Siakpere, O. 2008. Effects of sublethal concentrations of Portland cement powder in solution on nitrogenous waste products of the African catfish (Clarias gariepinus) (Burchell, 1822). Acta Zoologica Lituanica, 18(1) : pp. 55-60.

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- AL-Hayali H. L. 2009. Effect of Cement Pollution on some Biochemical Parameters in the Blood Serum of Hamam AL-Alil Cement Factory Workers. J. Raf. Sci., 20(2) : pp. 19-28.
- Asha, A. and Nirupma, Y. 2004. Effect of sulphur dioxide inhalation on serum Na and K ions concentration in albino rat. J. Environ. Bio., 25(3) : pp. 321-323.
- Calistus Jude, A. L. ; Sasikala, K. ; Ashok Kumar, R. ; Sudha S. and Raichel J. 2002. Haematological and cytogenetic studies in workers occupationally exposed to cement dust. Int. J. Hum. Genet., 2(2) : 95-99.
- Caroline, R. 2008. Potassium in blood. Test overview. University of Michigan. Health system. http://www.health.med.Umich.edu.
- Carrasco, F. ; Bredin, N. and Heitz, M. 2002. Atmospheric Pollutants and Trace Gases-Gaseous Contaminant Emissions as Affected by Burning Scrap Tires in Cement Manufacturing. Journal of Environmental Quality. 31: 1484-1490.
- Conerly, O. and Blain, R. B. 2008. Toxicological review of thallium and compounds. In Support of Summary Information on the Integrated Risk Information System (IRIS). U.S. Environmental Protection Agency Washington, DC. pp. 59.
- Brian, G. D. and Robert, J. A. 1999. Diagnostic evaluation of the patient with acute renal failure. Chapter 12. In Atlas of diseases of the kidney. Vol: 1 (edit by Robert W. Schrier). Denever. Colorado.
- EPA. 2006. Ground water and drinking water : Drinking water contaminants [online]. Office of Ground Water and Drinking Water (OGWDW), US Environmental Protection Agency, Washington, DC. Updated 2/28/2006. Availablefrom:http://www.epa.gov/OGWDW/hfacts.htl.
- Fleck, C. and Appenroth, D. 1996. Renal amino acid transport in immature and adult rats during thallium-induced nephrotoxicity. Toxicology, 106: pp. 229-236.
- HSDB. 1998. Hazardous Substances Data Bank. Thallium Micromedex, Inc. 36 : expires. April 30, 1998.
- Henry, J. B. 1984. Clinical diagnosis and management. 17th ed. Sauders publisher.
- Indrayan, A. and Sarmukaddam, S. B. 2001. Medical Biostatics. Morcel Dekker, Inc, USA. : pp. 299,303,405.
- Kazantzis, G. 1980. Thallium. In : Handbook on the toxicology of metals. 2nd ed. Elsevier, North Holland. pp. 599-612.
- Kamis, A. B. ; Modu, S. ; Zanna, H. and Oniyangi, T. A. 2003. Preliminary biochemical and haematological effects of aqueous suspension of pulp of Hyphaene thebaica (L.) mart in rats. Biochemistry. 13(1) : 1-7.

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- Leung, K. M and Ooi, V. E. C. 2000. Studies on thallium toxicity, its tissue distribution and histopathological effects in rats. Chemosphere, 41: pp. 155-159.
- Lioy, P. J. ; Weisel, C. P. ; Millette, J. R. ; Eisenreich, S. ; Vallero, D. and Offenberg, J. 2002. Characterization of the dust/smoke aerosol that settled east of the World Trade Center (WTC) in Lower Manhattan after the collapse of the WTC 11 September 2001. Environ Health Perspect 110: pp. 703-714.
- Robert, F. M. H. ; John, H. D. ; Jytte, M. Ch. ; Erik, O. and Milton, V. P. 2001. Risk assessment for occupational exposure to chemicals. a review of current methodology. Pure Appl. Chem., 73(6) : pp. 993-1031.
- Somma, G. ; Magrini, A. ; Romeo, E. ; Coppeta, L. ; Grana, M.; Vicentini, L. and Bergamaschi1, A. 2006. Exposure to cement dust and its particle size distribution measured with grimm laser dust monitor 1.108. G. Ital. Med. Lav. Erg., 28(3) : Suppl. pp 125-126.
- Tietz, N. W. 1999. "Text Book Of Clinical Chemistry ". 3rd ed. Philadelphia. W. B. Saunders Co. : pp. 668-672.
- Zhang, X. and Zhang, C. 2003. Atomic Absorption And Atomic Emission Spectrophotometry. In : Handbook Of Elemental Speciation. Techniques and methodology. [Ed. Comelis, R. ; Grews, H. ; Caruso, J. and Heumann, K. G.] Chichester John Wiley and Sons, : pp. 241-260.
- Zilva, J. F. ; Panmall, P. R. and Mayne, P. D. 1991. Clinical Chemistry in diagnosis and treatment. 5th ed. England Clays Ltd : St. Ives Plc.