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LONG-TERM RENAL FUNCTION FOLLOWING EXPOSURE TO PETROLEUM ENVIRONMENTAL POLLUTANTS IN THE POPULATION OF OGONI WOMEN,NIGER DELTA: A POSSIBLE CELLULAR MECHANISMS OF ENVIRONMENTAL POLLUTANTS-INDUCED NEPHROTOXICITY

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ABSTRACT

Environmental toxic pollutants are of environmental concern because of their diversity of toxic effects on the human body. In this study, randomly selected 184 female volunteers,94 from Ogoni, Rivers State, Niger Delta and 90 from Ogoja Cross River State, consistently living in the petroleum exploration or gas and oil flaring region and non-petroleum production environments respectively, Nigeria, were used to estimate the contents of renal function indices using standard procedures. Volunteers' age ranged from 18 to 50 years. When compared to control, this study indicated significantly high levels of urea, creatinine, sodium, and potassium with the ratio of urea to creatinine of 3:1 for the population of Ogoni women. Correlation coefficient analysis revealed a significant positive relationship between heavy metals (lead, cadmium, and vanadium) and renal function indices (urea and creatinine). An indication that environmental toxic pollutants can cause direct damage to the kidneysplausibly mediated by the combination of the high content of the exposed environmental pollutants and the induced high level of the renal toxins, specifically urea, which possibly fragmented blood cells without heat leading to nephrotoxicity. Additionally, the inference is that the population in the petroleum exploitation and exploration or oil and gas flaring environments are predisposed to renal dysfunction and are unaware.

KEYWORDS

Urea, nephrotoxicity, environmental toxicity, heavy metals, Ogoni, women, mechanisms of toxicity.

Introduction:

Environmental pollution resulting from petroleum exploration or oil and gas flaring presents widespread of toxic pollutants [1-4]that pose serious health threats in humans [5-11], and detrimental impact on social-economic status [12-15]. In a previous study it was reported a high level of exposure to petroleum environmental contaminants from air, portable drinking water and farm soil, in the population of Ogoni women, specifically for heavy metals - cadmium , lead and vanadium[16].This led to the postulation that the cellular mechanisms of environmental toxicity associated with heavy metals could possibly due to the potency of the heavy metal pollutants negatively modulate plasma membrane diffusion potentials [17-19]with resultant increase in cellular uptake of pollutants which could adversely affect the functions in the human body[16]. Of interest is the renal system which is known to play important roles in the homeostatic system of the body, and as an excretory organ [20-22],exposure to petroleum products [23-26] has been reported to cause impairment of renal functions evident by the derangement of serum electrolytes including waste products and toxins such as urea and creatinine [22-25].According to the National Institutes of Health, the overall prevalence of chronic kidney disease (CKD) is approximately 14% worldwide, the most common causes of CKD are hypertension and diabetes [16],yet the contribution that chronic exposure to environmental pollutants make to renal dysfunctions are not clearly understood. Hence assessment of renal functions of exposed individuals to petroleum environmental pollutants is important in the pathologies of renal functions.

This study was designed to establish, if any, possibly nephrotoxic potential of exposure of the population of randomly selected Ogoni women volunteers, Rivers State, aged 18 to 50 years, to petroleum exploration and exploitation or oil and gas flaring environments, principally to heavy metals [16], compared to women in non-petroleum production environments of Ogoja, Cross River State.

MATERIALS AND METHODS

Specimen Collection and analysis,

A randomly selected one hundred and eighty four female volunteers who have consistently lived in the order of ten years and above in the petroleum and non-petroleum exploitation and exploration environments enrolled for the study. The participants were predominately local farmers whose portable drinking water supplies and farmlands were contaminated with exposed environmental pollutants [16].The women were informed about the study merits and were submitted to assist self-reporting questionnaire to record their age, occupation, biometrics, and duration of exposure or resident in the study environment. Blood samples were collected from 94 subjects from Ogoni (test group) and 90 from Ogoja (control group), with ages ranging from 18 to 50 years. Blood samples for toxicological studies were as described previously [16].

Renal function parameters - electrolytes (sodium and potassium), urea and creatinine were analysed. Modified Jaffe method was used to determine the level of creatinine in the samples using Randox test kits (United Kingdom). Urease-glutamate Dehydrogenase -UV method was used to determine the level of urea in the samples. Sodium particle and potassium ion concentrations were analyzed with sodium reagent (Teco Diagnostics) kits and potassium reagent (Teco Diagnostics) kit respectively, in accordance with the manual instructions. This study was approved by the Government health research ethical committees of Rivers state, and Cross River State and the Institutional ethical research committee, University of Port Harcourt, and carried out in strict compliance to the guidelines of the National Committee for Research Ethics in Science and Technology.

Statistical Analysis

Analysis was carried out using Statistical Packaging for Social Sciences (SPSS) version 22.0. Data analysis were expressed as mean ± SEM. Data were analysed using Independent Samples Test. Percentages for independent variables were calculated; p value of <0.05 was considered statistically significant.

RESULTS AND DISCUSSION

The present study reports that the high level of exposed environmental pollutants from air, soil and water [16] significantly ($p<0.05$) cause an increase in urea, creatinine, sodium and potassium of 207.35%, 72.13%, 4.81% and 123.12 % respectively for the population of Ogoni women compared to the Ogoja (control group). This is an indication of a direct damage to the kidneys-acute renal impairment or acute kidney injury (AKI). The sudden onset of kidney injury or damage to the kidneys by nephrotoxicity may plausibly be mediated by the

combination of the high content of the exposed environmental pollutants and the induced high level of the renal function indices, specifically urea, which possibly fragmented blood cells without heat [27] in the kidneys. Notwithstanding, serum creatinine is a more accurate assessment of renal function than urea; however, urea increases earlier in renal disease. Indeed urea may increase in other conditions not related to renal diseases such as dehydration, catabolic states [20]. The ratio of urea to creatinine found in this study was 3:1, suggesting that the participants may not be suffering renal diseases. However, it has been suggested that the ratio of urea to creatinine can be useful to differentiate pre-renal from renal causes when the urea level is increased. In pre-renal disease, the ratio is close to 20:1, while in intrinsic renal disease, it is closer to 10:1. Upper gastrointestinal bleeding can be associated with a very high urea to creatinine ratio (sometimes >30:1) [16]. However, the observation that exposure to petroleum toxic pollutants can modify renal function indices is in agreement with previous reports [27].

Table 2 showed that the relationship between urea, creatinine and potassium decreased with age while the opposite was the situation for sodium (column 6; table 2). Table 3 describes the duration of exposure to environmental pollutants to to kidney function indices and showed a decrease in urea, creatinine and potassium but an increase in sodium (column 6; table 3).

Table 1: Heavy metal exposure effect on renal function indices (range in parenthesis)

Renal function indices	Heavy metal exposure effect on the Study population		Percentage difference	P- value
	Control (Ogoja)	Test (Ogoni)		
Urea (mg/dl)	16.58±.52 (0-36.26)	50.96±1.50 (0-108)	207.35	.001
Creatinine (µmol/L)	43.28±.90 (27.13-62.62)	74.50±1.29 (41.73-99.86)	72.13	.001
Sodium (mEqu/L	136.25±.77 (0-148.38)	142.81±.51 (134.01-149.74)	4.81	.001
Potassium (mEqu/L)	2.94±.16 (0.56-7.1)	6.56±.37 (1.06-19.49)	123.12	.001

Table 2: Relationship between renal function indices and age dependent effect of exposure to heavy metals

Renal function indices	Study population	Age (years)			
		<25	25-34	35-44	45-50
Urea (mg/dl)	Control	16.64±1.04 (10.13-28.71)	15.44±.63 (10.72-22.11)	15.65±.90 (9.50-23.07)	20.53±1.71 (12.73-30.77)
	Test	44.07±7.32 (10-62.64)	51.19±2.67 (33.20-81.56)	51.19±2.14 (29.51-80.78)	53.11±3.27 (27.67-82.41)
	% dif.	164.84	231.54	227.09	158.69
	P value	0.001	0.001	0.001	0.001
Creatinine (µmol/L)	Control	44.23±1.98 (29.40-61.68)	39.38±1.10 (28.79-51.85)	44.59±1.68 (32.90-61.68)	48.24±2.35 (30.84-62.62)
	Test	69.78±3.21 (58.07-84.98)	73.13±2.59 (44.76-91.32)	76.27±1.96 (54.45-99.86)	75.27±2.86 (41.78-98.16)
	% dif.	57.76	85.70	71.04	56.03
	P value	0.001	0.001	0.001	0.001
Sodium (mEqu/L)	Control	135.21±1.42	138.82±1.23	137.56±1.48	130.48±1.87

		(122.02-148.38)	(122.02-147.36)	(119.27-144.36)	(122.02-140.18)
	Test	143.70±1.81 (134.09-148.24)	143.23±.92 (134.22-149.71)	141.87±.78 (134.15-149.06)	143.52±1.20 (134.01-149.74)
	% dif.	6.27	3.17	3.13	9.99
	P value	0.001	0.006	0.009	0.001
Potassium (mEqu/L)	Control	3.21±.36 (.74-7.10)	2.73±.25 (1.13-5.68)	2.64±.26 (.56-4.27)	3.41±.46 (1.07-5.68)
	Test	7.87±1.75 (2.15-15.33)	7.11±.60 (2.15-15.33)	5.52±.48 (1.78-12.07)	7.09±.96 (1.06-15.59)
	% dif.	145.17	160.43	109.09	107.91
	P value	0.001	0.001	0.001	0.001

Table 3: Duration of exposure to petroleum contaminants and renal function indices

Renal function indices	Exposed group	Duration of exposure (years)				
		<11	11-20	21-30	31-40	41-50
Urea (mg/dl)	Control	16.24±.85 (10.13-25.27)	16.55±1.02 (10.05-28.71)	16.85±1.06 (11.40-27.47)	14.74±1.26 (11.40-23.07)	19.24±2.46 (9.50-30.77)
	Test	51.29±3.18 (31.35-80.78)	50.11±5.08 (29.51-81.56)	46.22±4.11 (20.10-66.98)	53.07±2.38 (27.67-72.82)	52.65±3.17 (33.20-82.41)
	% dif.	215.82	202.77	174.30	260.04	173.64
	P value	0.001	0.001	0.001	0.001	0.001
Creatinine (µmol/L)	Control	42.91±1.70 (28.79-61.68)	43.59±1.63 (29.40-60.75)	42.49±1.86 (30.84-58.88)	43.10±2.83 (32.90-61.68)	45.66±3.72 (30.84-62.62)
	Test	74.16±2.86 (55.440-92.51)	76.43±3.79 (54.719-99.86)	76.45±2.79 (60.12-91.32)	74.31±2.38 (44.76-95.23)	71.81±3.27 (41.78-98.16)
	% dif.	72.82	75.33	79.92	72.41	57.27
	P value	0.001	0.001	0.001	0.001	0.001
Sodium (mEqu/L	Control	137.52±1.31 (122.02-146.34)	134.42±1.49 (122.02-148.38)	137.23±1.61 (124.77-147.36)	137.33±3.22 (119.27-144.36)	133.62±1.96 (122.94-138.15)
	Test	144.88±.88 (137.21-149.71)	142.35±1.51 (134.09-149.74)	141.61±1.31 (134.68-149.31)	142.78±.90 (134.22-148.70)	141.96±1.32 (134.01-149.53)
	% dif.	5.35	5.89	3.19	3.96	6.24
	P value	0.001	0.001	0.001	0.001	0.001
Potassium (mEqu/L)	Control	2.81±.30 (.56-7.10)	3.13±.35 (1.13-7.10)	3.01±.33 (1.07-5.68)	3.06±.42 (1.13-5.68)	2.61±.49 (1.50-5.34)
	Test	6.23±.83 (2.15-15.33)	4.71±.79 (2.15-9.66)	7.36±.87 (2.15-12.25)	6.76±.61 (2.15-15.33)	7.22±1.08 (1.06-15.59)
	% dif.	121.70	50.47	144.51	120.91	176.62
	P value	0.001	0.001	0.001	0.001	0.001

Correlation coefficient analysis between heavy metals and renal function indices revealed moderate positive correlation between vanadium and creatinine(r-value =0.469; P-value =0.00) but strong correlation with urea (r-value =0.701; P-value =0.00) while lead showed strong positive correlation with urea (r-value =0.710; P-value =0.00) and creatinine(r-value =0.681; P-value =0.00). Similarly, cadmium showed strong positive correlation with urea (r-value =0.743; P-value =0.00) and creatinine(r-value =0.692; P-value =0.00), an indication that toxic heavy metals and renal waste products and toxins can fragment blood cells in the systemic circulations differentially [27] to induce nephrotoxicity

CONCLUSION

The findings of this study have shown that high levels of exposed environmental pollutants with increase in renal function indices can induce nephrotoxicity. Additionally, the present study revealed that the population in the petroleum exploitation and exploration or oil and gas flaring environments are predisposed to renal dysfunction unaware.

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