Quest Journals
Journal of Medical and Dental Science Research
Volume 9~ Issue 4 (2022) pp: 59-63
ISSN(Online) : 2394-076X ISSN (Print):2394-0751
www.questjournals.org

Research Paper

Nephrotoxicity Among Adult Male Residents Exposed To Constant Gas Flares In Bayelsa State Nigeria

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The present study determines the renal profile and blood pressure as biomarkers to nephrotoxicity among adult male subjects exposed to gas flares in Bayelsa state Nigeria. A total of two hundred and fifty male subjects were recruited for this study. The body mass index of the study population was (27.02kg/m²) and were categorized into short (<10 yrs) andlong (>10yrs) duration of exposure to gas flares, age range and blood pressure in relation to renal function. Blood samples were collected through the cubital vein into heparinized bottles to assess the renal profile of the participants at the chemical pathology laboratory, Niger Delta university teaching hospital while an Acuson sphygmomanometer, calibrated meter rule and a bathroom scale balance was used to determine the blood pressure and body mass index. There was a significant increase in serum uric acid (6.81mg/dl), urea (5.92mmol/L) among subjects exposed to gas flares above ten years while serum creatinine in particular reach a peak reference range of (114µmol/l).Similar significant increase in serum creatinine was also observed among male subjects aged (45-50yrs) = (106µmol/L), (51-60yrs) = (103µmol/L) and (107µmol/L) in hypertensive subjects. Furthermore, the blood pressure of the sample population was (141mmHg))/ (86mmHg) using the WHO standard for blood pressure categorization. This study have shown that serum creatinine increase in blood and gradual elevation of other renal indices are road map to the genesis leading to nephrotoxicity among adult males exposed to gas flares and therefor advise regular renal check up at the teaching hospital in Bayelsa state.

Keywords: *Hyperuricemia, urea, creatinine, kidneys, toxicity, Bayelsa*

***R**eceived 10Apr, 2022; **R**evised25 Apr, 2022; **A**ccepted 27 Apr, 2022 © The author(s) 2022.*
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I. INTRODUCTION

Nephrotoxicity is a progressive deterioration in kidney Physiology due to effect from chemicals or environmental pollutants and certain medications. Certain poisonous chemicals such as lead (pb), cadmium (cd), vanadium (vd) and particulates can thus result in adverse effect on kidney function due to their accumulation in the blood of residents exposed to gas flares over a prolonged period of time. Other harmful chemicals released from gas flares include carbon monoxide, sulphur dioxide, methane ,hydrogen sulphide, dioxins, particulate matters such as soot and black carbon with resultant nephrotoxic effect and other organs and system such as immune ,nervous, and respiratory leading to marked increase in serum urea, creatinine, uric acid, sodium and potassium among men chronically exposed to gas flares [1 2].Sustained gas flares is a predicament nationally due to incomplete combustion during the process, accompany by many toxic gasses emission such as black carbon or soot into the air and in turn inhaled into the inhabitants circulatory system through the respiratory air- ways, water and nutrition to influence renal changes. The heat and carbon emission from gas flares increases the carbon dioxide composition in the environment as it reverberate in all direction to release accumulated heat in the surrounding that cause dehydration in body fluid among residents and subsequent retention, decrease in glomerular filtration rate and increase in renal indices [3 4] .The flaring of gases remain the most predominant source of air emission from gas installations into the environment. Toxic chemicals such as benzene and heavy

metals are harmful to both humans, animals and plants usually consumed by residents with increase in ambient temperature that cause persistent dehydration with subsequent serum urea increase due to reduction in renal perfusion and dysfunction [5] [6]. Exposure to harmful chemicals, emissions and pollutants associated with oil and gas production is likely more among those residents dwelling close to oil and gas facilities. Flaring of gases is a practice by the oil and gas industries to emit unwanted gases into the environment that result in global warming [7].Heavy metals from gas flares usually target the calcium channels in the renal tubules, accumulate to alters the membranes electrical charges to produce polyuria and proteinuria .These alterations in addition to fluid depletion due to dehydration from gas flares are sensed by the macula densa in the proximal tubules which then triggers the renin angiotensinogen aldosterone system to increase the blood pressures. Renal tubules are the driving forces in the progression of kidney disease in response to injury. The tubular epithelial cells can synthesize and secrete bioactive molecules that drive interstitial inflammation and fibrosis [8].Processes leading to decrease renal function result in a severe reduction in glomerular filtration rate. Several mechanisms have been proposed in the alteration of glomerular filtration rate such as the back-leak theory, the tubular obstruction theory and the primary filtration failure theory. The back –leak theory of increase permeability proposes that the lack of urine output is caused by disruption of tubular epithelium rather glomerular filtration rate. Therefore substances are reabsorbed from the tubular lumen and interstitium into the peritubular circulation. Tubular damaged demonstrated anatomically using experimental animals after the induction of nephrotoxicity shows tubular changes are inconsistent and may be minor in acute tubular necrosis [9].Hypertension is the most commonest cause of sudden natural death and ranked first as the medical illness most frequently diagnosed among male elderly subjects in Nigeria. The prevalence of hypertension was 23.3% among adult males in 2005 compared to adult females 16.4%.However 42% prevalence was reported in south-south Nigeria in 2011[10].

II. MATERIALS AND METHODS

This is a cross-sectional descriptive qualitative clinical research study that investigated renal profile and blood pressure of adult male subjects exposed to gas flares in Bayelsa state.

Location of study: Obunagha, Polaku and Immiringi.

Study Population: The study involve 250 adult males (20-60 years) residing in Bayelsa state of Nigeria

ETHICAL CONSIDERATION

The methodology of this study was approved by the ethical committee of the University of Port Harcourt River State.

Inclusion criteria: Adult male subjects resident only between seven years and above in Bayelsa state.

Exclusion criteria: Subjects on antacid, non-residents, females, obvious disease condition etc.

Statistical analysis was done using SPSS version 23.0

Consent was obtained from each participant before they could participate in the study. They freely decided to participate without being pressured.

Random sampling method was used to select the participants and all samples collected were taken to the Niger Delta University teaching Hospital for onward determination of the following:

The renal parameters were determined using standard laboratory test kits (Randox, United Kingdom).

Creatinine (µmol/L); Agappe Diagnostics (Switzerland GMBH)

Method: Kinetic method. The principle states that creatinine in alkaline solution react with picric acid to form a colored complex. The amount of the complex formed is proportional to the creatinine concentration. Creatinine concentration in blood illustrates the filtration capacity of the glomerulus. Creatinine is formed in the liver and transported to muscles where its phosphorylation form creatine phosphate that act as a storage deport for muscular energy. The enzyme creatine phosphokinase catalyze the reaction between creatine and creatine phosphate and a cyclic anhydride of creatine is the metabolic end product of creatine metabolism (Thorir 1979).

Serum urea: Randox urea test kit (United Kingdom)

Method: Urease Berthelot method in (mmol/L).This principle state that urea in serum/plasma is hydrolyzed to ammonia in the presence of urease. The ammonia is then measured photochemicaly by Berthelot reaction. Urea $+H2O \rightarrow 2NH_3 + CO_2$.

NH_2+Hypochlorite + phenol → Indophenol (blue compound).Urea is a product of protein metabolism and the concentration of urea is dependent on protein intake, the body's capacity to catabolize protein and effectively excrete urea through the kidneys.

Uric Acid (mg/dl): Randox (United Kingdom) uric acid test kits.

Method: Enzymatic colorimetric method.

The principle state that uric acid is converted by uricase to allantoin and hydrogen peroxide, which under the catalytic of peroxidase ,oxidizes 3,5-2-hydroxybenzenesulfonic acid and 4-aminophenazone to form a violet quinoneimine compound.

The blood pressures of the participants were measured in a sitting position with an Acuson (England) mercurial sphygmomanometer and a stethoscope twice from the left arm while the average was taken using auscultatory method. The pulse rate was taken at the anatomical snuff box

Body mass index: A bathroom scale balance and a calibrated meter rule. The BMI was determined using the square of the height to divide the weight of the subjects.

III. RESULTS

The results are presented as mean± standard deviation and a p-value of <0.05 isconsidered statistically significant.

Table 1: Renal characteristics of the study population

Parameters	**Mean values**	**SD**
Age (yrs)	44.31	±10.64
Uric acid (mg/dl)	3.94	±2.84
Urea (mmol/dl)	3.75	±1.92
Creatinine (µmol/L)	104.71	±19.50
SBP (mmHg)	141.23	±12.42
DBP (mmHg)	87.24	±14.31
Pulse (bpm)	86.31	±9.24
BMI (kg/m^2)	27.02	±3.24

Table 2: Duration of exposure among male subjects

Indices	<10yrs n=145	>10yrs n=105	P-value	%diff.
Uric acid (mg/dl)	4.32±1.15	6.81±2.10	0.00	44.74
Urea (mmol/L)	3.42±0.92	5.92±1.22	0.01	53.53
Creatinine (µmol/L)	91.21±17.13	114.01±17.20	0.01	22.33

Table 3: Comparison of male renal function with age (yrs)

Parameters	**21-30**	**31-40**	**41-50**	**51-60**	**p-value**
Urea (mmol/L)	**3.41**	**4.46**	**5.40**	**4.56**	**0.04**
Creatinine (µmol/L)	**97.19±13.22**	**101.14±17.25**	**106.17±15.74**	**103.32±21.89**	**0.01**
Uric acid(mg/dl)	**3.81±1.27**	**3.94±1.25**	**4.25±0.26**	**4.90±1.44**	**0.00**

Table 4: Relationship between adult male blood pressure and renal function

Parameters	Low(mmHg) <90/60 n=-(0%)	Normal(mmHg) 90-119/69-79 n=120(48%)	Pre-hyperten (mmHg)120-139/80-89 n=79(31.6%)	Hyperten(mmHg) >140/90 n=51(20.4%)	p-value
Uric acid (mg/dL)	-	3.98±1.12	4.59±0.11	5.20±2.22	0.03
Urea (mmol/L)	-	4.40±1.33	4.91±0.96	4.62±1.27	0.06
Creatinine (µmol/L)	-	92.24±19.03	100.32±17.01	107±7.11	0.02

IV. DISCUSSION

Serum **uric acid** concentration of the study subjects was (3.94mg/dl) and a mean age of (44.34) years, urea level was (3.75mmol/L) while the serum creatinine was at (104.74µmol/L).However the systolic blood pressure significantly increase to (141mmHg) with the diastolic pressure at (86mmHg), pulse (86.31bpm) and body mass index ($27kg/m^2$) respectively. Significant uric acid values obtained from this study among adult male

subjects exposed to gas flares over a prolonged period of over ten years was (6.81mg/dL) compared with subjects exposed in less than ten years (4.32 mg/dL). The serum uric acid was (5.20mg/dL) among adult males in hypertensive state compared with those having normal blood pressure (3.98mg/dL).The correlation of serum uric acid with blood pressure has been previously observed but there are sparse evidence regarding the association among adult males consistently residing in gas flaring communities in Bayelsa state. The prevalence of hypertension and Hyperuricemia has been previously observed to be 28.4% and 17.02% in a study conducted by [11].In China compared to our result of (20.4%). The potential mechanism of action regarding gas flares in the elevation of blood pressure could be from the induction of oxidative stressie imbalance between free acid radicals and antioxidants, inhibition of nitric oxide effect on blood vessels, activation of the RAAS mechanism due to dehydration, inflammation (gout) due to increase uric acid concentration in blood serum and endothelial cell dysfunction. Uric acid act as a potent antioxidant compound in the extracellular fluid but has pro-inflammatory effect in the intracellular fluid compartment that can leads to micro vascular injury. This can cause afferent arteriolopathy and hypertension that may become unresponsive to Hyperuricemia lowering therapies over time. [12]. In a long chronicity of Hyperuricemia, deposit of urate in and around joint may lead to extensive destruction of the articular surfaces and sometimes to permanent loss of joint function. Regarding serum **creatinine**, there was a significant increase (114µmol/L) among residents that have consistently spent more than ten years in gas flaring communities within Bayelsa state. Observation from this study also indicate that the serum creatinine level also significantly increase with age especially among adult males aged 41-50 and 51-60 year compared with adults within the ages of 21-30 and 31-40 years. Aging, decrease in lean body mass, dehydration and consistent exposure to particulate matters from gas flares reduce renal tissues, number of nephron and thus decrease renal perfusion rate which in turn increase serum creatinine concentration. The kidney can be significantly damaged before the loss of major function to modify the normal clinical indications of renal disease before elevated creatinine level may be detected. Increase in creatinine level associated with degeneration and necrosis of the glomerulus have also been observed in rats exposed to crude oil from gas flares[13] [14] Hence if GFR is decreased, creatinine clearance through the renal system could be compromised and the decrease GFR will then result to an increase creatinine concentration in the plasma as observed from this study. This is widely used as an index of renal function[15].

Sustained elevated blood pressure can cause kidney dysfunction by constricting and narrowing renal blood vessels that eventually damages, Haden, weaken them and reduce blood flow. The nephrons responsible for blood filtration will be prevented from receiving sufficient oxygen and nutrients. This process will result to the inability of the kidneys to regulate blood pressure as they slowly short down[16] [17] [18] [19].

The result of the study population further reveals a (3.75mmol/L) in serum **urea**. However the serum urea level significantly increase to (6.81mmol/L) among residents exposed to gas flares for over ten years compared with participants of less than ten years exposure (3.42mmol/L).We also observed an increase in urea among subjects in prehypertensive stage (5.40mmol/L) compared to normal and those aged (41-50) years. Though other factors may have contributed to this hike but is most likely due to the effect of gas flares on body fluid depletion and poor renal perfusion from regular heat emission from the flares as the dominant factor.

V. CONCLUSION

This study have shown that evaluation of serum urea, creatinine and uric acid alongside blood pressure increase can be more reliable biomarkers to diagnose nephrotoxicity in adult male residents exposed to gas flares in their respective communities in Bayelsa state and the Niger delta region of Nigeria were toxic gases are emitted for decades. We therefore encourage all in gas flaring communities to avail themselves for regular renal function check in hospitals within the state.

Conflicting interest: There is no conflicting interest among the authors

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