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Variations in Nitrogenous Product Ratio in Control and Subjects with Renal Dysfunction

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ABSTRACT

Complications arising from Kidney damage and other renal pathologies have been a subject of major concern, which have been attributed to various sources. Concerns for patients with renal disease prompted the need for this study. We evaluated nitrogenous product levels on control (normal) subjects and those with dysfunction. Using spectrophotometric approach, we determined levels of creatinine, Urea, uric acid and some other parameters in four categories of patients. We observed a consistent elevated pattern of the parameters measured with severity of the status of the kidney dysfunction. A four-fold increase in concentration of the metabolites analyzed was observed in tandem with the respective disease condition. Values obtained necessitate the need for sustained laboratory investigation for early detection of those at risk to restore and mention homeostasis

Keywords
Nitrogenous Product, Creatinine, Urea, Renal dysfunction.

Introduction
The maintenance of water, hydrogen ion (pH) and salt balance are critical components for the sustenance of homeostatis. The kidney play a central role in this metabolic process and its integrity must not be compromised. The functionality of the nephrons, adequate blood supply to the kidney and its other functions of secretion and feedback are mandatory pre-requisite for the kidney to function effectively [1].

Previous findings have elucidated the fact that the measurement of creatinine can provide a clear reflection of the state of alteration in glomerular filtration rate (GFR). Its value is known to fall significantly as creatinine level increases [2]. Knowledge from earlier findings have shown that there is much more excretion of creatinine occurring at the tubules than the glomerulus which makes the clearance of some substance including creatinine greater [3].

Various substance such as diodone and para-aminohippuric acid have been applied in attempt to determine raised plasma clearance. However, non-have shown to elicit value above the renal plasma flow [4]. The production of creatinine in the body is enhanced by the non-enzymatic and irreversible conversion of creatine. The release of creatinine affects the level of creatinine as serum level of creatinine is measured by the creatine output, which is stable even with diet and exercise [5].

The non-protein nitrogen compounds also include urea, which approximately makes up about 50% of these compounds and is a major product of the catabolism of nitrogen containing compounds in man. There is scientific evidence to show that man is not able to catabolize the purine skeleton up to urea terminating at uric acid formation. Taking advantage of the Kreb's Henseleit cycle the nitrogen of other nitrogenous compounds can be inserted into urea [6].

Characteristically, urea is easily distributed in many tissues because of its easy diffusibility, possessing mild diuretic action almost wholly excreted by the kidney. Due to decreased clearance caused by renal disorders, urea levels are usually elevated. Inability of the heart to deliver adequate blood to the kidney will eventually lead to decrease clearance especially as it happens in heart failure or cardiac decompensation. These are not related to kidney disease [7].

The end-product of purine in man is uric acid. Human lack the enzyme uricase to metabolize it further to allantion. In normal condition where the kidney is functioning optimally the intake of nucleoprotein has little affect on uric acid plasma concentration. A unique feature of uric acid is that although it is easily filtered by the glomerulus almost all of it is wholly reabsorbed in the proximal tubule of the nephron.

Materials and Method

Study was carried out at three tertiary health institutions, Niger Delta University Teaching Hospital, Federal Medical Centre, and Diete Cooky Memorial Hospital all in Yenagoa, Bayelsa State, Nigeria. Blood and urine sample were collected from 30 patients from each of the Hospital renal clinic. Another 30 samples were collected from apparently healthy patients not attending clinic.

Blood samples obtained from the patients and controls were allowed to clot and was then spun to obtain serum. The serum was used for all the analysis creatinine, urea, uric acid, bicarbonate, calcium, phosphate and albumin. Urine collected was used for the estimated glomerular filtration rate (eGFR).

Creatinine was quantitated spectrophotometricaly by the modified Jaffe method were creatinine in serum reacts with picric acid to produce alkaline picrate that is measured at 546nm Reagent was product of Quinica clinica applicadia S. A. QCA.

Urea was determined spectrophotometrically by the urea modified Berthelot's method in which urea in serum sample react with salicylate and hypochlorite to form green complex measured at 600nm. Reagent was a product of Fortress diagnostics Autrim.

Uric acid was determined quantitatively by enzymatic colorimetric method Uricase PAP (a product of clinichem, Budapest).

Results

We determine the concentration of creatinine, urea, uric acid, pH and bicarbonate. Values are shown in table 1. In table 2, results for calcium, phosphate, eGFR and albumin are shown.

Comparison of values obtained for patients with kidney dysfunction and control shows significant difference at $P<0.05$) with a linearity that was time dependent. There was a generalized accumulation of nitrogenous products concomitant with duration of the diseases.

Table 1: Observed values of Nitrogenous product pH and bicarbonate for controls and subjects.

Analyte	Control	Mild	Moderate	Severe
Creatinine (µmol/L)	80±15.62	144±10.51	180±20.31	800±105.52
Urea (mmol/L)	4.0±1.33	15±3.51	25±5.04	35±3.02
Uric acid (mmol/L)	100±10.52	120±4.30	144±5.30	130±5.21
pH	7.32±0.02	7.41±0.01	7.31±0.11	7.21±0.12
$HC0_3$ (mmol/L)	23.0±3.5	24.±0.9	29.0±0.81	26±1.21

Values are means of Triplicate determinations

Table 2: Status of some Biochemical Parameters over time.
State of kidney dysfunction

Analyte	Control	Mild	Moderate	Severe
Ca^{2+}(mmol/L)	2.4±0.40	2.3±0.30	2.3±20.0.30	2.0±0.20
PO_4^3 (mmol/L)	1.5±0.40	1.4±0.20	2.4±0.50	3.0±0.40
eGFR (mL/min)	125±5.0	40±5.82	33±4.22	28.0±0.33
Albumin (g/L)	38±2.01	37±1.30	35±1.12	30±1.50

Values are mean of triplicate determination.

Discussion

We studied variation in Nitrogenous products in this research. Our observation show that creatinine and urea values rises in tandem with the degree of dysfunction of the kidney. We observed mild elevation of calcium and phosphate with marked reduction in the glomerular filtration rate. Several factors have been implicated to be responsible for kidney disease. These include metabolic and genetic disorders, autoimmune diseases, Ischaemia, toxins, infection and allograt rejection. Over time creatinine and urea has been used as biomarkers of renal function or dysfunction. These nitrogenous products have been variously applied to evaluate the severity and nature of kidney injury. This research result correlate with several other works done in the past relating kidney function with Nitrogenous product derangement.

Understanding the potential danger of kidney disease requires determination of biomarkers. Obtained values of biomarkers can be used to predict potentials risk disease progression, as well as being useful for assessing response to therapy [8]. Although there are several biomarkers available, creatinine stands unique as an indicator of glomerular filtration rate. Others include enzymes, up regulated proteins and cyclic arrest biomarkers that have been discovered which are now being used to serve as biomarker to enhance early and accurate diagnosis of Acute Kidney Injury (AKI), Chronic Kidney Diseases (CKD) and End-stage Renal Disease (ESRD) [9].

The relevance of creatinine measured in this research reflects its uniqueness since its value does not change until around 50% of the kidney is lost with limitation associated with age, muscles mass, sex, medication [10]. Other novel biomarkers of AKI include cystatin C, inter Leukin-18, and N-acetyl-β-d-glucosaminidase.

It is concluded that there is absolute need to introducing biomarker protocol or algorithm for the diagnosis of nitrogenous product, which will elucidate clinical relevance to enhance medical care.

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