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### Renal Impairment in HIV Patients

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**Abstract:** Renal impairment in HIV patients on drugs and those not on antiretroviral drugs was assessed using biochemical markers such as Uric acid, Urea, Creatinine and Phosphate. A total of one hundred individuals were used for the study and they were within the age of  $35 \pm 7.1$  years consisting of 50 HIV positive individuals on antiretroviral therapy, 30 HIV positive individuals not on antiretroviral therapy and 20 HIV sero negative individuals (control) all within Aba Metropolitan. HIV patients were on antiretroviral drugs (triviro-LNS-Lamivudine, Nevirapine, Stavudine) 1-2 pills daily depending on the CD4 count. Moreso, have been on the drug for the duration of 2- 3 years. The investigations were done with serum. The biochemical parameters were assayed using fortress diagnostic kit based on calorimetric method. The results were subjected to statistical analysis. The results of the study revealed that there was a significant increase in the serum levels of Uric Acid and Urea in HIV positive individuals not on drugs compared to HIV sero negative subjects( Uric acid level control (mg/dL).  $5.61 \pm 2.31$  vs  $7.16 \pm 2.42$ , Urea -control (Mmol/L)  $4.7 \pm 1.58$  vs  $7.82 \pm 3.8$  ( $P < 0.05$ ). In addition, there was remarkable decrease in Phosphate levels in HIV patients not on drugs compared to control and HIV infected subjects on antiretroviral drugs (phosphate level Mmol/L Control  $1.07 \pm 0.27$  vs  $0.79 \pm 0.19$ ). Equally indicated was that HIV positive subjects not on drugs exhibited slight increase in Creatinine levels compared to the other two groups (control mg/L;  $0.90 \pm 0.50$  vs  $1.90 \pm 1.20$  ( $P < 0.05$ ). The use of antiretroviral drugs is helping in alleviating the HIV associated metabolic abnormalities. This notwithstanding, attention needs to be paid to hyperuricaemia which is common with patients on antiretroviral drugs. Prevention of hypophosphataemia is also vital.

**Key words:** Uric acid, Urea, Phosphate, Bicarbonate, HIV, AIDS.

## Introduction

HIV/AIDS is a major health problem in many parts of the world, and is considered a pandemic. Incidentally, Nigeria is not left out. [1,2]. As of 2010, approximately 34 million people have HIV globally, of these, approximately 16.8 million are women and 3.4 million are less than 15 years old. HIV/AIDS resulted in about 1.8 million deaths in 2010, down from 3.1 million in 2001 [18]. Unfortunately, approximately 220,000 people died from AIDS in Nigeria in 2009 [19]. More awareness concerning this deadly disease is still necessary, moreso when, 80-95 percent of HIV infections in Nigeria are as a result of heterosexual sex and some youths till this moment do not believe in its existence [3, 4, 5, 6].

The advent of highly active antiretroviral therapy in conjunction with improved standard antiviral and antibiotic regimens has dramatically changed the clinical course of HIV infection, resulting in prolonged survival [8] Hyperuricemia has been associated with HIV disease progression especially those with low CD4<sup>+</sup> and co-infection with either hepatitis B or C virus. [9,10]. But when considering the potentials pathophysiological mechanism of the frequent occurrence of Hyperuricemia in HIV infected patients, it was suggested that it may result from multiple metabolic, immunologic and pathological abnormalities which characterized the disease progression from asymptomatic infection to terminal illness. Infact, abnormal serum urate levels have been observed in a broad spectrum of pathologic situations often complicating the course of HIV disease such as prolong fever due to infections, neoplastic or autoimmune disorders, hypercatabolic states associated with fasting or cathexia, viremia and possibly HIV related loss of mononuclear cells [9, 10]. Hypouricemia has also be attributed to increased renotubular loss [11], 1990).Aside from HIV infection itself, serum urate levels could potentially be altered by antiretroviral drugs. However the use of Didanosine and Stavudine or the combination of the two drugs was associated with hyperuricemia [12].Respiratory chain failure causes ATP depletion which increases urate production in the purine nucleotide cycle. Mitochondrial dysfunction may increase the formation of lactate which competes with urate for tubular excretion in the kidney. This mechanism is the basis for the hyperuricemia and other metabolic myopathies. And, may also provide an explanation, for the association between dideoxynucleoside analogues (Stavudine and Didanosine) and elevated urate. Therefore hyperuricemia is multi factorial origin in HIV patients.

More recently, tubular disorders have been related to ARV drug toxicity .Acyclic nucleotide reverse transcriptase inhibitor and more particularly tenofovir despoil fumarate (TDF) has been involved in tubulopathy leading to Fanconi or Fanconi-like syndrome with or without acute renal failure .An impairment of renal proximal tubular function is characterized by decreased tubular handling of phosphate leading to hypophosphatemia and their relation to tubular reabsorption disorder in tenofovir treated patients remain uncertain [14]. In this study, we aimed at determining the Serum levels of Uric acid, urea, creatinine and Phosphate in HIV positive individuals and those on Antiretroviral Drugs to enable us assess possible variation in biochemical parameters among the groups studied.

## Material and Methods

Renal impairment in HIV patients on drugs and those not on antiretroviral drugs was assessed using biochemical markers such as Uric acid, Urea, Creatinine and Phosphate. A total of one hundred individuals were used for the study and they were within the age of 35±7.1 years consisting of 50 HIV positive individuals on antiretroviral therapy, 30 HIV positive individuals not on antiretroviral therapy and 20 HIV sero negative individuals (control) all within Aba Metropolitan. HIV patients are on antiretroviral drugs (triviro-LNS-Lamivudine, Nevirapine and Starvudine) 1-2 pills daily depending on the CD4 count. Moreso, have been on the drug for the duration of 2- 3 years. The investigations were done with serum. The biochemical parameters were assayed using fortress diagnostic kit based on calorimetric method. The results were subjected to statistical analysis of one-way analysis of variance ANOVA. [15].

## Result and Discussion

**Table-1** Serum Levels Of Uric Acid, Urea Creatinine and Phosphate of Hiv Negative, Hiv Positive Not On Drugs And Hiv Positive Subjects On Drugs.

| Parameters                         | Phosphate (mmol/L) | Uric Acid (mg/dL) | Urea (mmol/L). | Creatinine (mg/dL) |
|------------------------------------|--------------------|-------------------|----------------|--------------------|
| HIV Negative Subjects (control)    | 1.07 ± 0.27        | 5.61 ± 2. 31      | 4.75 +1.58     | 0.90 ± 0.50        |
| HIV Positive Subjects not On drugs | 0.79 ± 0.19**      | 9.60± 3.60**      | 7.82. ±3.84*   | 1.90±1.20**        |
| HIV Positive On drug               | 0.75 ± 0.15*       | 7.16 ± 2.42*      | 6.54+2.10*     | 1.50± 0.80*        |

\* Significant difference at P < 0.05

The results of the study revealed that there was a significant increase in the serum levels of Uric Acid and Urea in HIV positive individuals not on drugs compared to HIV sero negative subjects ( Uric acid level control (mg/dL).  $5.61 \pm 2.31$  vs  $7.16 \pm 2.42$ , Urea -control (Mmol/L)  $4.7 \pm 1.58$  vs  $7.82 \pm 3.8$  (  $P < 0.05$  ) (Table-1). In addition, there was remarkable decrease in Phosphate levels in HIV patients not on drugs compared to control and HIV infected subjects on antiretroviral drugs (phosphate level Mmol/L Control  $1.07 \pm 0.27$  vs  $0.79 \pm 0.19$ ). Equally indicated was that HIV positive subjects not on drugs exhibited slight increase in Creatinine levels compared to the other two groups (control mg/L;  $0.90 \pm 0.50$  vs  $1.90 \pm 1.20$   $P < 0.05$ ). Table.

The higher uric acid level observed in this study may be due to the oxidative damage to cells causing an increase in cell turnover and muscle wasting. This shows that HIV infection is associated with increase cell turnover as suggested by [13]. Again, most antiviral/retroviral drugs cause mitochondrial toxicity leading to increase lactate formation, which competes with urate for tubular secretion in the kidneys leading to hyperuricaemia. The variation may also be due to the degree of dehydration as suggested by [16] because the climate condition in this part of the country is extremely hot which might increase cell turnover resulting to hyperuricaemia. The urea levels obtained showed that there was no significant difference among those on drugs and HIV positive patients not on antiretroviral drugs. The decrease in phosphate levels observed in patients used for this study showed that hypophosphataemia is found in HIV infection independent of the use of antiretroviral drugs and this agrees with the study of Badiou [17]. Conversely, increase in creatinine level particularly in patients not on antiretroviral drugs may be due to muscle wasting observed in HIV patients. From the result of the study, the use of antiretroviral drugs is therefore helping in alleviating the HIV associated metabolic abnormalities but attention needs to be paid to hyperuricaemia common with antiretroviral drugs. Prevention of hypophosphataemia is also vital.

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