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KIDNEY FUNCTION PREDICTORS AND ASSOCIATED SERUM ELECTROLYTES CHANGES IN HIV OUT PATIENTS ATTENDING JARAMOGI OGINGA ODINGA TEACHING AND REFERRAL HOSPITAL, KISUMU COUNTY, KENYA
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W. O. OPIYO, A. G. M. NG'WENA and A. V. O. OFULLA

ABSTRACT

***Background*: Kidney disease has been recognised as one of the health challenges complicating HIV infection, prognosis and management. Early recognition, diagnosis and treatment are therefore key to ameliorating the deleterious impacts of kidney disease in HIV infected patients.**
***Objectives*: To assess serum kidney function markers in order to highlight the state of kidney health and the impact of its functional impairment on other serum electrolytes and body fluids parameters**
***Design*: A case-control study.**
***Setting*: At Jaramogi Oginga Odinga Teaching and Referral Hospital's Patient support centre.**
Subjects: **Eight Hundred HIV positive and 406 HIV negative participants above 18 years of age.**
***Results*: Mean serum creatinine and urea levels was significantly elevated in HIV – infected individuals than the healthy control group (95.2µmol/l v/s 86.2µmol/l, p<0.0001 and 4.6mmol/l v/s 4.1mmol/l, p<0.0001 respectively). The prevalence of pathological levels of serum creatinine, urea and sodium was higher in HIV-infected patients than HIV-negative participants (26.1% v/s 11.8%, p<0.0001; 4.4% v/s 0.5%, P<0.0001and 26.1% v/s 10.6%, p=0.001 respectively). Females experienced more serum creatinine disorders than their male counterparts (31.8% v/s 18.1%, p<0.0001). Age and anti-retroviral treatment were not predictors of aberrations in levels of kidney function markers in HIV infected patients. AIDS defining CD4 depletion was associated with enhanced deterioration of kidney function. However, kidney function anomalies were not sufficient explanation for co-existing electrolyte anomalies as clinically altered creatinine states only correlated and co-varied with urea states (r =0.715) while sodium states co-varied with chloride levels (r = 0.296).**
***Conclusion*: Kidney function disorders are not infrequent in HIV infected individuals. Serum sodium aberration is observed more frequently in seropositive than in sero-negative individuals. Routine review of kidney health status in local HIV infected individuals ought to be adopted for comprehensive management of HIV patients, more so among the female gender.**

INTRODUCTION

Since the inception of HAART, in the mid 1990s, life expectancy has improved in HIV infected persons but new health challenges have arisen out of both prolonged drug use, and effects of sustained viral presence in the body (1). For instance, studies have shown that the virus causes renal injury via various pathogenetic mechanisms. It has been reported that HIV-1 infects lymphocytes and macrophages which upon entry into the kidney might release inflammatory lymphokines or cytokines thereby causing renal injury (2). Furthermore, the HIV-1 proteins may also directly injure renal parenchymal cells. Available evidence has shown direct HIV-1 infection of kidney parenchymal cells which is a potential cause of cytopathic effects such as proliferation or apoptosis (3). Both HIV-1 protein and nucleic acid have been detected in podocytes and tubular epithelium by the use of sensitive in situ hybridisation techniques (4,5). Therefore HIV-associated kidney diseases have emerged as major outcomes of direct viral

infection and / or anti-retroviral as well as non-ARVs drug toxicities (6). There are mainly three types of chronic kidney diseases caused by HIV infection; HIV-associated thrombotic microangiopathies, HIV immune-mediated renal diseases, and classic HIV-associated nephropathy (7).

Kidney diseases complicate prognosis of HIV infection and treatment outcomes. In the western world extensive research has delineated the determinant factors, pathogenesis and management of renal diseases in HIV with mixed results. It has also been recognised that research needs to address and delineate pathogenesis of individual renal diseases that complicate HIV infection, understanding the relationship of kidney diseases to HIV infection, and devising appropriate treatments for kidney diseases co-existing with HIV infection (8). However, in middle and low income countries where the burden of HIV infection is immense, information is sparsely available concerning kidney health in HIV infection despite the negative impact that kidney diseases have on HIV treatment and prognosis.

In Kenya routine assessment of status of kidney health does not fall within the main bracket of continuing care of HIV patients. Inhibiting cost, insufficient locally directed research and information are among factors that have hindered adequate attention to renal diseases in HIV population in Kenya. This research therefore addressed knowledge gaps related to the state of serum markers of kidney function, their association with co-existing plasma fluids and electrolytes disorders and determinants that influence their distribution, in HIV infected patients attending the Patient Support Center (PSC) at Jaramogi Oginga Odinga Teaching and Referral Hospital (JOOTRH), in Kisumu, western Kenya. Serum creatinine and urea analysed as markers of kidney function.

MATERIALS AND METHODS

Population: Between November 2012 and April 2013, a case-control study was conducted involving 800 HIV sero-positive patients from PSC of JOOTRH and 406 HIV negative controls, over 18 years, able and willing to give verbal or written consent and of both gender. Enrolled participants in the control group were enlisted from among consenting ministry of health staff who in the period of the study were due for and underwent routine laboratory profiling at JOOTRH, as a pre-condition for vaccination against hepatitis B and C vaccinations. Patients were excluded if they had known co-infections or co-morbid conditions such as hypertension, diabetes or alcohol abuse or history of jaundice. Control participants were excluded if they were seropositive for HIV. Ethical approval was provided by the ethical committee of JOOTRH.

Specimen handling: Personnel in the PSC undertook routine medical review and care of HIV patient enrolled in the department and introduced them to study. Volunteers were then informed of consent provisions and consenting patients sent to the hospital's laboratory where assigned hospital laboratory personnel collected blood samples for analysis. Collected blood samples were prepared and assayed for the levels of the following analytes; urea, creatinine, sodium, potassium and chloride.

Common attributes recorded for both groups included age, gender, random blood sugar, Serum kidney and liver functions parameters. Additional attributes obtained for the HIV infected individuals were ARV use and CD4 levels.

Serum creatinine and urea, were assayed using colorimetric method with wavelength range of 340-670 nm (Bio systems BTS – 330 Photometer, Biosystems, SA). Sodium, potassium and chloride were measured using combined photometric-ion exchange and turbidimetry techniques (Eurolyser, Eurolyser diagnostic, and Austria). HIV status of PSC patients were confirmed from the patients' records while for the consenting control participants were confirmed by ELISA and Western Blot. CD4 + cell level was also determined (FACSCalibur instrument, Becton Dickinson, San Jose, CA).

Comparison of means was done using student t-test while comparison of proportions was done using $\chi 2$ and One –way analysis of variance where applicable. Linear correlation was used to explore association between quantitative variables and regression statistics was used to elucidate co-variation. SPSS version 21 was used for statistical analysis.

RESULTS

Demographics of the 800 HIV positive and 406 HIV negative participants and the distribution of twelve analytes assayed are provided in Table 1 and 2 respectively.

Table 1
Characersitics of participants in the study population

	Percent	
	HIV Positive	HIV negative
Males	41.4	32
Females	58.6	68
Old (≥50yrs)	15	16.5
Young (<50yrs)	85	83.5
Using ARV	79.9	
ARV naïve	20.1	
CD4+ <500	59.1	
CD4+≥500	40.9	

Key: CD4+-lymphocyte sub-group bearing CD4 membrane markers; ARV- anti-retroviral drugs

Table 2
Serum electrolytes distribution by HIV status

HIV status									
electrolytes	units	HIV Positive	HIV Negative						
		Mean	Std Dev.	Min.	Max.	Mean	Std Dev.	Min	Max.
Creatinine	μmol/l	95.2	35.6	11.5	410.57	86.2	20.4	40	173.1
Potassium	mmol/l	4.2	0.7	2.8	6.4	4.2	0.7	2.9	6
Sodium	mmol/l	138.9	6	127	160	139.1	4.9	130	153
Chloride	mmol/l	100.2	3.9	90	139	99	3.2	90	108
Urea	mmol/l	4.6	2.1	1.5	36.7	4.1	1.0	1.9	8.8

Key: ALT- alanine-aminotransferase; AST- aspartate –aminotransferase

Creatinine: Mean serum creatinine levels in HIV positive participants was significantly raised than that in the seronegative group (95.2μmol/l v/s 86.2μmol/l, p<0.0001) see table 3. Similarly, a higher proportion of HIV infected patients (26.1%) had serum creatinine disorder (defined as < 40μmol/l or > 120μmol/l) than the control population (11.8%), χ^2=32.6, p<0.0001). Gender and age related disparities were also noticed in physiological and clinical creatinine states across the two study groups. Mean creatinine level in seropositive females was significantly raised than in uninfected females (95.4μmol/l v/s 84.6μmol/l, p<0.0001), as was the prevalence of abnormal serum creatinine states (31.8% v/s 12.7%, p<0.001). Similarly, prevalence of serum creatinine disorders in HIV-infected males (18.1%) was significantly more than in HIV negative males (10%), p = 0.031. This was also the case with younger participants (<50 years) among whom mean creatinine level was significantly higher in those with HIV infection than uninfected counterparts (95.1μmol/l v/s 85.8μmol/l, p<0.0001) as well as the prevalence of anomalous creatinine levels (27.1% v/s 11.2%, χ^2 =33.4, p<0.0001).

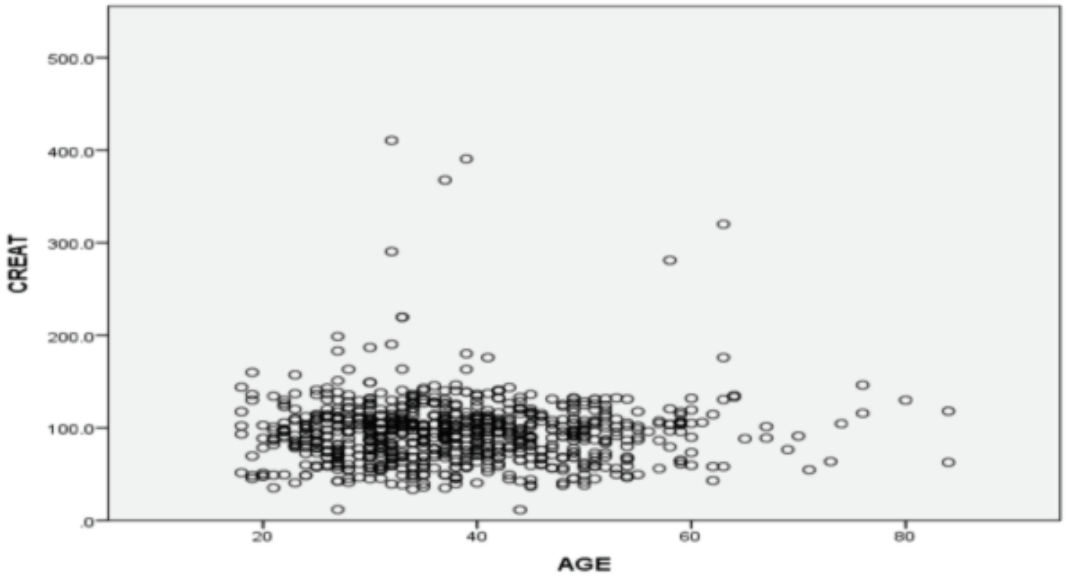
Table 3
Comparison of serum creatinine states between various HIV population attributes

	Mean creatinine (μmol/l)	T	p-value	Deranged creatinine (percent)	χ^2	p-value
HIV+	95.2	5.6	<0.0001	26.1	32.6	<0.0001
HIV-	86.2			11.8		
Male HIV+	94.9	1.8	0.07	18.1	4.6	0.031
Male HIV-						
	89.8			10		
Female HIV+						
	95.4	5.6	<0.0001	31.8	34	<0.0001
Female HIV-	84.6			12.7		
Male HIV+	94.9	0.2	0.850	18.1	18.7	<0.0001
Female HIV+						
	95.4			31.8		
Arv-yes	94.7	0.8	0.438	24.9	2.5	0.111
Arv-no	97.1			31.1		
≥50yrs HIV+	94.9	1.7	0.07	20.8	0.986	0.321
≥50yrs HIV-	89.8			14.9		
<50yrs HIV+	95.1	5.3	<0.0001	27.1	33.4	<0.0001
<50yrs HIV-	85.8			11.2		

ARV yes- using anti-retro viral drugs; ARV no: Not on anti-retro viral treatment

Among HIV infected individuals, 26.1% of participants had abnormal levels of creatinine out of which majority were hypercreatininemia (91%) while 9% were hypocreatininemia. HIV afflicted females had a higher prevalence of serum creatinine disorders than their male counterparts (31.8% v/s 18.1%, χ^2 = 18.7, p<0.0001). However no correlation between age and changes in serum creatinine states occurred (see figure 1). HIV afflicted participants using ARVs did not exhibit different mean creatinine levels from those not using ARVs (94.7μmol/l v/s 97.1μmol/l, p=0.438) and neither was the prevalence of abnormal creatinine states between them disparate (24.9% v/s 31.1%, p = 0.111).

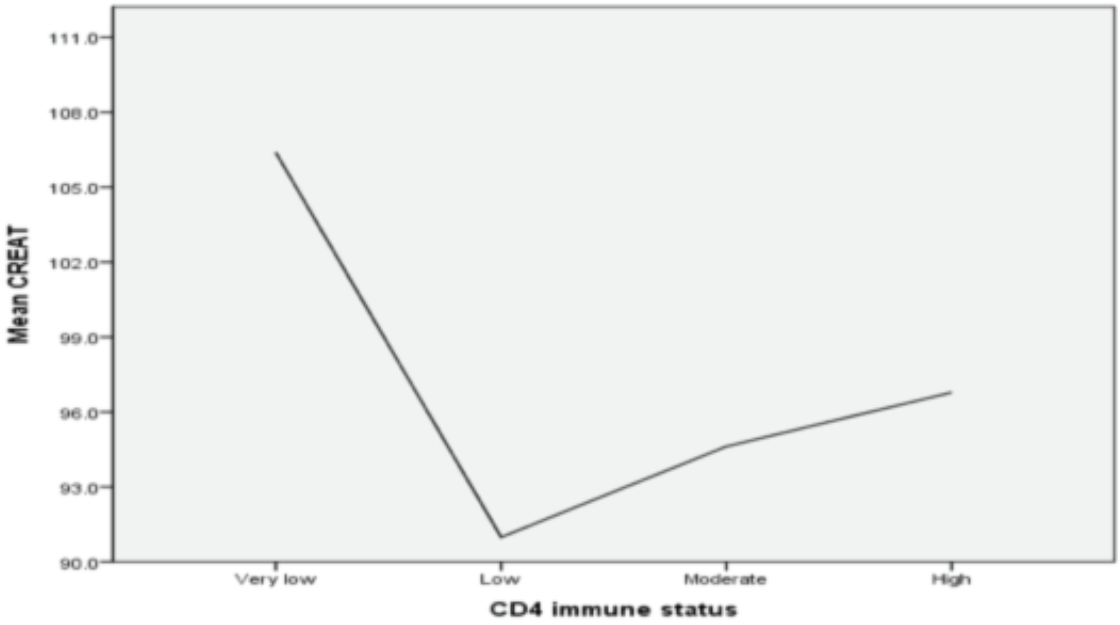
Figure 1
Correlation of creatinine level to age in HIV infected patients (r = 0.018)



Key; Very low-CD4 <200; Low-201-500; Moderate-501-800; High->800cells/μl

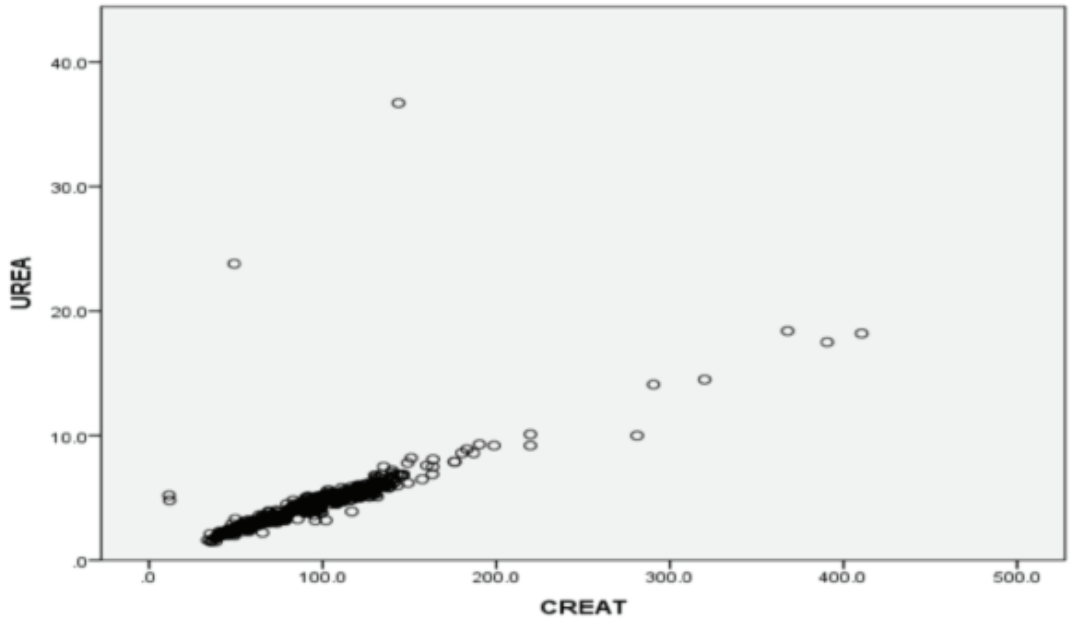
Initial decline in CD4 cell count in patients with robust immunity was accompanied with decline in serum creatinine level. However, below 500 cells/mm^3, further decline in CD4 cell counts was associated with a steady rise in serum creatinine levels surpassing levels at commencement of immune depletion (see figure 2).

Figure 2
CD4 count associated creatinine level changes



Correlation of changes in serum creatinine with other serum analytes: A strong positive correlation existed between creatinine levels and urea (r = 0.953) with over 90% of changes in urea associated with changes in creatinine (r2 = 0.908), among seronegative individuals. Among HIV-infected individuals, a strong positive correlation persisted between creatinine and urea in those with normal creatinine states (r = 0.656) and those with abnormally altered creatinine levels (r = 0.715, see figure 3). There was also 43% and 51.1% co-variation respectively between creatinine and urea in the two HIV infected categories which was not displayed between creatinine and the measured electrolytes.

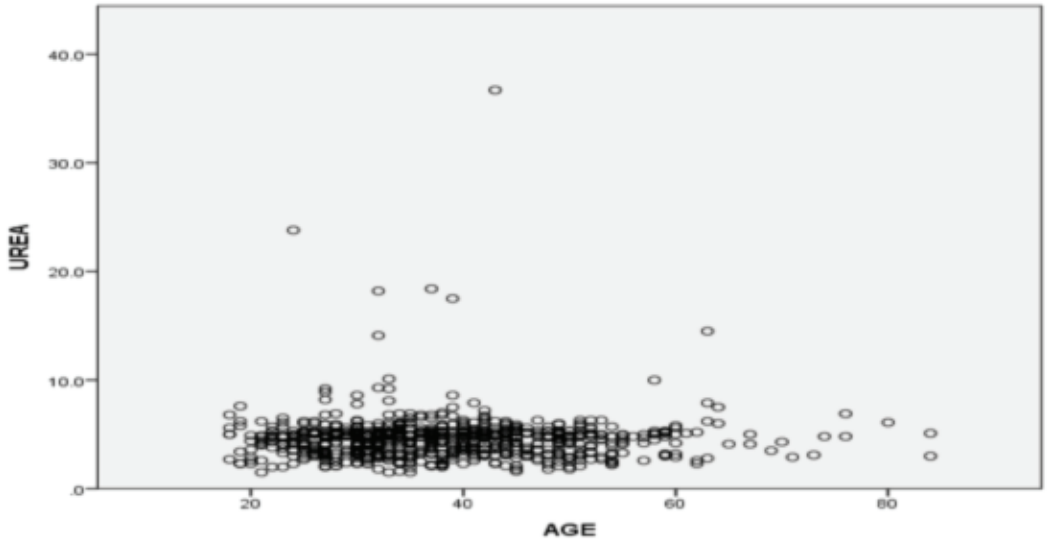
Figure 3
Correlation of urea to creatinine levels in HIV infected (r=0.715)



Correlation and co-variation between creatinine and urea was also co-directional with regard to development of pathological clinical states. Thus whereas the proportion with abnormal urea states was 0.5% in HIV infected patients with normal creatinine levels, this significantly increased in HIV infected patients who had abnormal creatinine states (15.3%, χ^2 = 80.9, p<0.0001). Moreover, the type of urea anomaly found in the later group matched the type of creatinine disorder existing in the patient. As such, concomitant urea disorders found in patients with creatinine retention disorders were also only urea retention disorders. Conversely concomitant urea disorders found in patients with creatinine depletion disorders were only urea depletion disorders. Alternately, no patient with creatinine retention disorders had urea depletion disorders and no patient with creatinine depletion disorders had urea retention disorders. Such co-directional shifts between urea and creatinine, were not exhibited between creatinine and any of the electrolytes with or without marginal correlation between them. This shows that co-existing electrolytes anomalies in HIV infected patients with abnormal creatinine levels are not tied to causes responsible for creatinine anomalies other than for urea related anomalies.

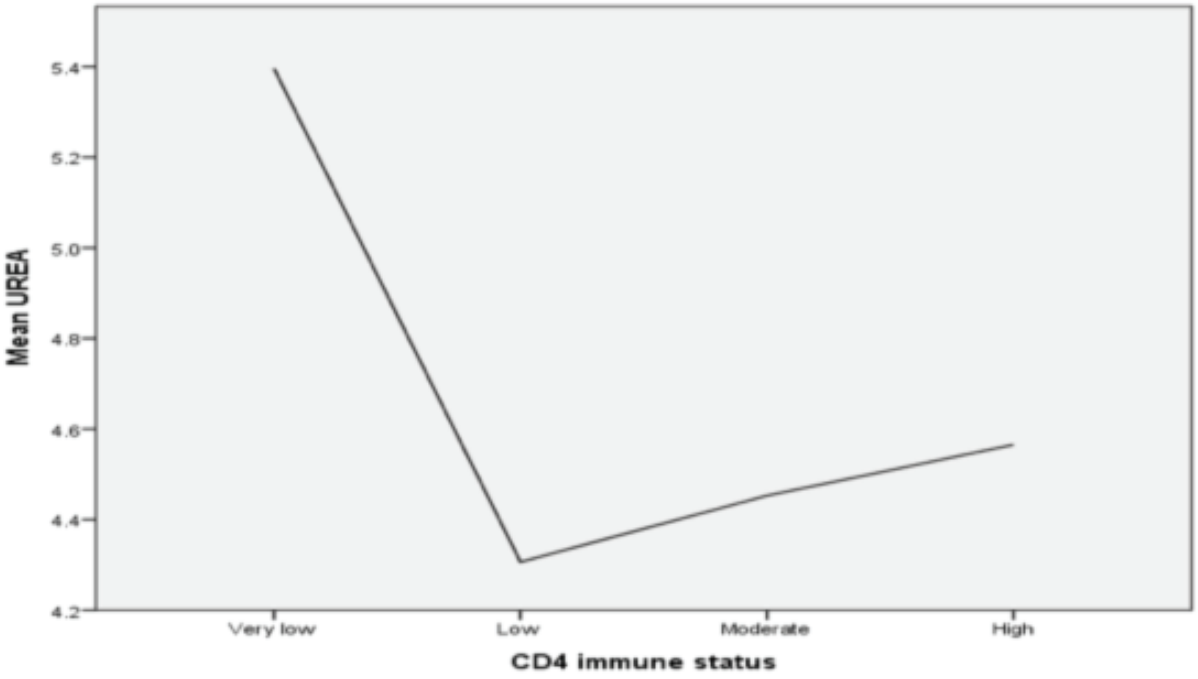
Urea: Four point four per cent (4.4%) of HIV infected participants had serum urea disorders (defined as <2mmol/l or >7.5mmol/l). Majority of the urea anomalies (65.7%) were serum urea retention disorders while a few were (34.3%) hypouremia. Mean urea values in HIV infected participants (4.6mmol/l) was significantly raised than in HIV negative control (4.1mmol/l) p<0.0001. Similarly the proportion with abnormal urea levels was higher in the seropositive than seronegative control group (4.4% V/S 0.5%, χ^2= 13.7, P<0.0001). Prevalence of abnormal urea levels was higher in HIV infected males than uninfected males (6% v/s o%, χ^2 = 8.2, p = 0.004). On the other hand infected females presented significant differences in both mean urea levels (4.6mmol/l v/s 4.1mmol/l, p<0.0001) and prevalence of abnormal urea levels (3.2% v/s 0.7%, χ^2= 4.8, p =0.029) from uninfected females. Similar outcomes were observed in younger infected participants (<50 years) among whom mean urea level was significantly higher than in uninfected counterparts (4.6mmol/l v/s 4.1mmol/l p<0.0001) as well as the prevalence of abnormal creatinine states, (4.6% v/s 0.6%, χ^2 = 11.4, p= 0.001). Gender and age were not determinants of changes in urea levels in HIV infected individuals (see figure 4). Initial decline in CD4 cell lymphocyte levels was accompanied with corresponding decline in serum urea levels but CD4 cells within AIDS defining range were associated with steady rise in serum urea levels attaining levels higher than pre-immune depletion states (see figure 5). Patients using ARVs did not have significance difference in physiological and pathological serum urea level from those not using ARVs.

Figure 4
Correlation between urea and age in HIV infected participants



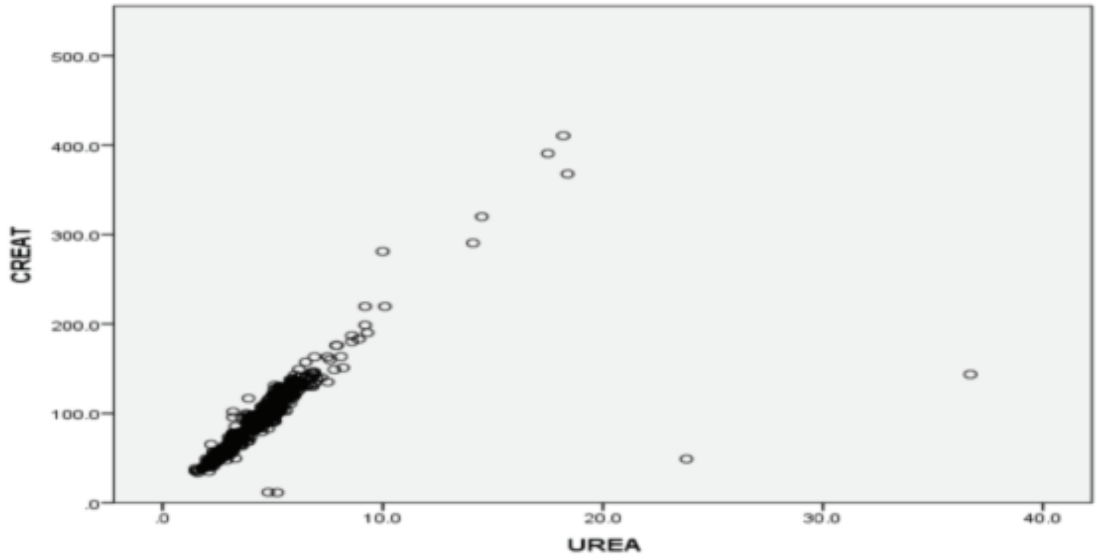
Key; Very low-CD4 <200; Low-201-500; Moderate-501-800; High->800cells/μl

Figure 5
CD4 cell count associated serum urea levels



Serum electrolytes changes and their correlation with urea: Urea was correlated to creatinine levels in both HIV negative and HIV positive participants. In HIV negative individuals urea had a strong positive correlation to creatinine (r = 0.953) with 90.7% co-variation between them (r2= 0.907). Among HIV infected individuals with normal urea states, a strong correlation was retained between urea and creatinine (r = 0.943) with 88.9% co-variation between them (r2=0.889 see figure 6.). In this category urea also exhibited minimal correlation with potassium but without co-variation between them (r2 = 0.004). In HIV infected individuals with abnormal urea states, a significant positive correlation only existed between urea and creatinine (r =0.576) with 31.2% co-variation between them.

Figure 6
Correlation of urea and creatinine in HIV infected individuals
r= 0.889

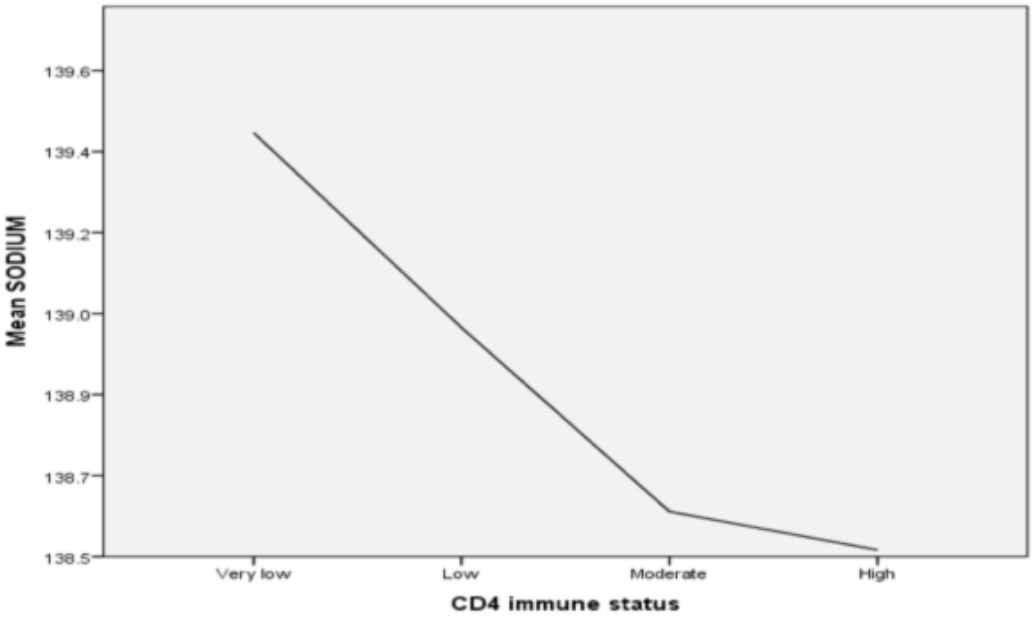


Evidence of co-variation was also seen in the distribution of creatinine disorders in different urea states. Virtually every participant with urea disorder also had abnormal creatinine states. Twenty two out of 23 urea retention disorders (hyperuremia) also had creatinine retention disorders and 10 out of 12 urea depletion disorders (hypouremia) also had creatinine depletion disorders.

Sodium: HIV infected participants, had a higher proportion with abnormal sodium levels (26.1%) than uninfected counterparts (10.6%), $\chi^2 = 10.6$, p = 0.001. Comparison of gender by HIV status, revealed different physiological and clinical sodium states with the proportion with sodium disorders in infected females (27.9%) being more than in uninfected counterparts (18%, p= 0.003). In participants below 50 years, HIV infection was also associated with significantly higher prevalence of abnormal sodium levels compared to those without HIV infection (26.6% v/s 17.1%, χ^2= 11.4, p=0.001).

Two hundred and seven of the 209 participants found with serum sodium disorders presented with hyponatremia (99.0%) while two cases were due to hypernatremia. Gender and age were not associated with significant changes in sodium outcomes among HIV infected participants. Generally HIV infected participants not using ARVs had a higher prevalence of serum sodium disorders (32.3%) than those using ARVs (24.6%), $\chi^2 = 3.98$, p = 0.046. However, even though ARV use was not associated with significant changes in sodium outcomes in infected males, in females those using ARVs had a higher mean sodium level (139.2mmol/l v/s 137.5mmol/l, p = 0.007) and a lower prevalence of sodium anomalies (24.3% v/s 38.2%, χ^2= 8.8, p = 0.003), than those not using ARVs. Similarly whereas in HIV infected patients over 50 years, ARV use had no significant impact on sodium states, in younger HIV infected patients (<50 years) those using ARVs had a higher mean sodium level 139mmol/l v/s 137.6mmol/l, p = 0.013) and a lower prevalence of sodium anomalies (24.3% v/s 36%, χ^2= 7.7 p = 0.005) than those not using ARVs. Immune depletion was accompanied with rise in mean sodium levels that constituted two phases; a modest rise associated with initial CD4 lymphocyte decline up to 800 cells/ mm^3 and a steep rise associated with fall in CD4 cell count below 800 cells/mm^3 (see figure 7). However, mean sodium did not fall out of the reference range (135 – 155mmol/l).

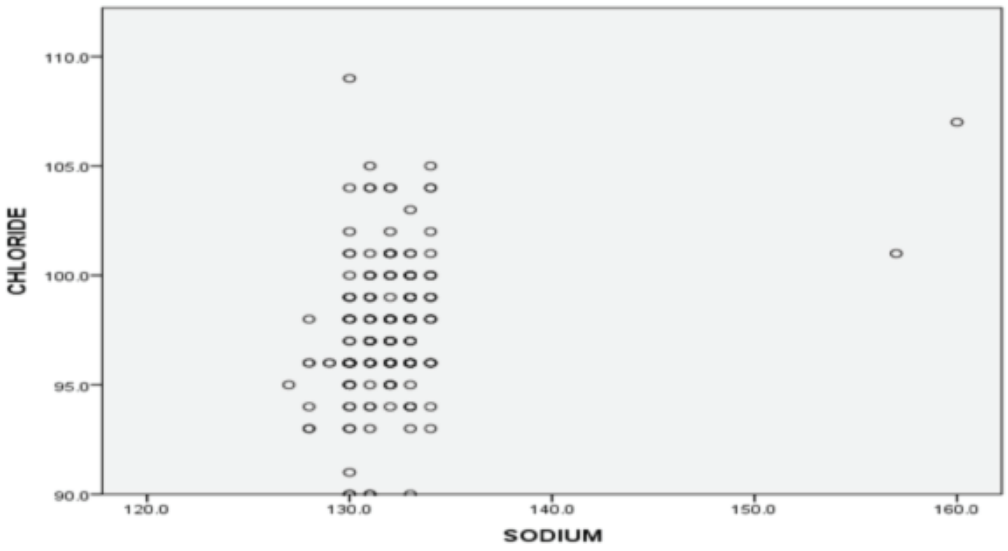
Figure 7
Mean serum sodium against CD4 count



Key; Very low-CD4 <200; Low-201-500; Moderate-501-800; High->800cells/μl

Serum electrolytes' correlation with changes in sodium level: Sodium levels correlated mainly with potassium and chloride levels in all categories of the participants. In HIV negative participants sodium levels had modest positive correlation with chloride levels (r = 0.446, p<0.0001) with 19.7% co-variation in their levels. There was also a noticeable correlation between sodium and potassium levels (r = 0.308, p<0.0001) but with minimal co-variation between their level (r2 = 0.092). In HIV infected participants with normal sodium levels a similar trend was observed with sodium retaining a noticeable correlation with chloride levels (r = 0.427) and 18.1% co-variation between them (see figure 8). In this group, correlation also existed between sodium and potassium (r= 0.244, p<0.0001) but with diminished co-variation between their levels (r2 = 0.058). However in HIV infected individuals with abnormal sodium there was correlation between sodium and three electrolytes i.e. chloride, potassium and glucose. However co-variation diminished between sodium and chloride levels (r2= 0.088), potassium levels (r2=0.022) and glucose levels (r2=0.043) in infected patients with sodium disorders.

Figure 8
Correlation of chloride to sodium level in HIV patients
r=0.427



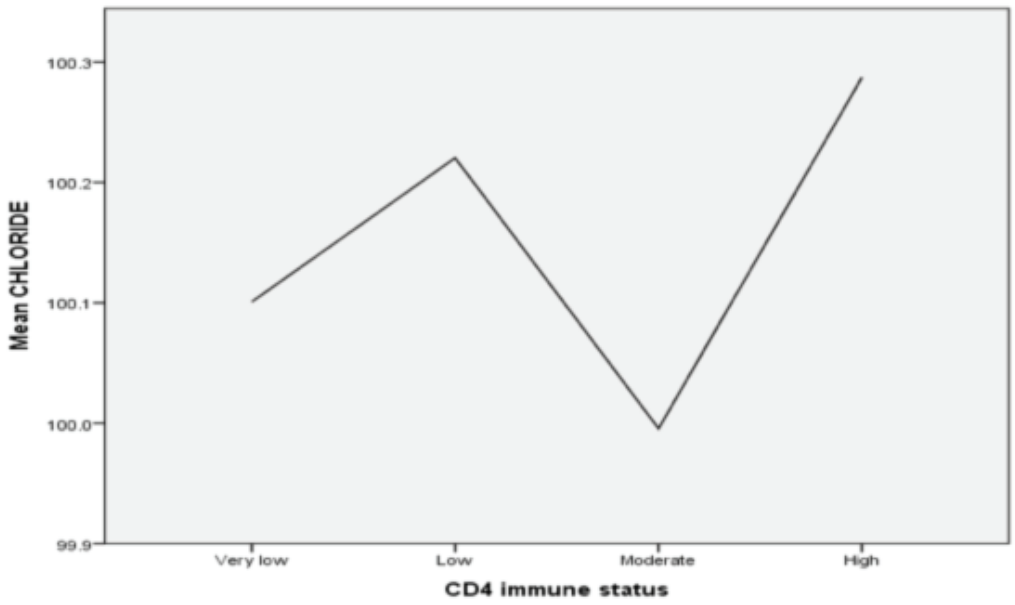
Chloride co-variance with sodium levels tended to show general co-directional anomalous shift as HIV infected patients with normal sodium levels had significantly less concurrent chloride disorders (16.2%) compared to HIV infected individuals with abnormal sodium levels (58.9%, $\chi 2 = 141$, $p<0.0001$). However potassium levels had no co-directional shift with sodium, as the proportion with abnormal potassium levels in HIV afflicted participants with abnormal and normal sodium levels were not distinct (16.7% v/s 17.4%, p=0.823).

Chloride: The proportion with Serum chloride disorders (<98 or >106mml/l) in the study population, was 27.4% with 87.2% due to hypochloremia and 12.8% hyperchloremia. Mean chloride in HIV+ participants (100.2mmol/l) was not significantly altered from that of HIV negative participants (99.9, p=0.252) and neither was the prevalence of serum chloride disorders (27.4% v/s 25.9%, p=0.575). However among males, HIV infection was associated with significantly different mean chloride levels compared to uninfected males (100.2mmol/l v/s 99.5mmol/l, p=0.047).

Gender, age and ARV use were not determinants of altered serum chloride states in HIV afflicted participants. Initial decline in CD4 cell level was accompanied with rapid decline in serum chloride levels up to moderate levels of CD4. Between this level and low CD4 levels there was a modest rise in mean serum chloride levels but beyond low CD4 cell count there was another decline in mean serum chloride levels (see figure 9).

Figure 9

Serum chloride association with CD4 depeltion in HIV ingfectd patients



Key; Very low-CD4 <200; Low-201-500; Moderate-501-800; High->800cells/μl

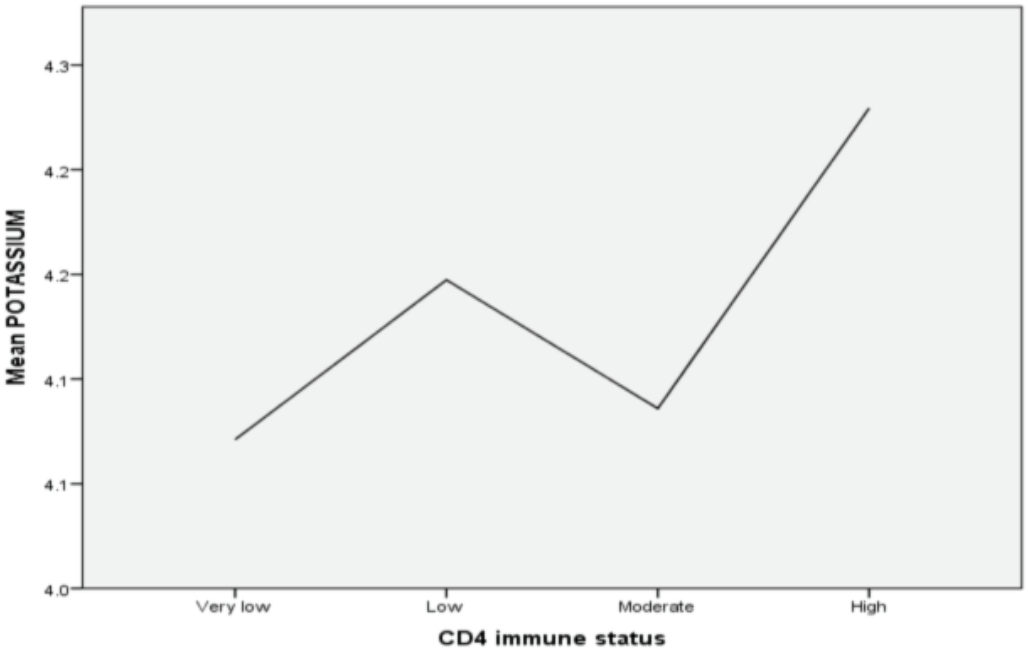
Serum electrolytes' correlation with changes in serum chloride level: Serum chloride levels had a significant positive correlation with sodium levels in uninfected participants (r = 0.446, p<0.0001) but with 19.7% co-variation between their levels. Correlation between chlorides and sodium persisted in HIV infected participants with normal chloride levels (r= 0.483, p<0.0001) and with pathologically altered chloride levels (r = 0.561, p<0.0001) with 23.2% and 31.1% co-variation respectively among them. Correlation was also seen between chloride and potassium levels in these three categories of participants. In uninfected individuals chloride correlated with potassium (r = 0.186, p<0.0001) but with minimal co-variation (r2 = 0.032). In HIV infected participants with normal chloride levels potassium chloride correlated with potassium (r = 0.173, p<0.0001) with more diminished co-variation between them (r2= 0.028) but in HIV infected individuals with abnormal chloride levels, chloride correlated with potassium (r = 0.372) but with a stronger co-variation (r2= 0.135).

Potassium: Mean potassium level in HIV infected participants was not significantly different from that of HIV negative individuals (4.2mmol/l v/s 4.2mmol/l, p = 0.895) and neither was the prevalence of serum potassium disorders (17.3% v/s 14.8%, p = 0.274).

Among the seropositive participants, 17.3% had potassium disorders with majority due to hypokalemia (77.5%) and 2.5% hyperkalemia. Gender, age and ARV use were not associated with altered mean potassium levels and disorders of serum potassium states. Initial decline in CD4 immune levels was associated with decline of serum potassium level up to moderate CD4 cell count. Beyond this CD4 level, serum potassium level rose up to low CD4 levels. In very low CD4 levels serum potassium declined again (see figure 10).

Figure 10

Potassium changes with CD4 cell depletion in HIV patients



Key; Very low-CD4 <200; Low-201-500; Moderate-501-800; High->800cells/μl

Correlation of potassium with serum analytes: Potassium levels exhibited consistent correlation with sodium and chloride levels in both study participants. Among HIV negative participants, potassium levels correlated with sodium levels ($r=0.308$, $p<0.0001$), and chloride levels ($r= 0.186$, $p<0.0001$). However there was minimal co-variation between these electrolytes in the seronegative individuals. Among HIV positive participants with normal potassium levels, correlation was observed between potassium levels and sodium ($r= 0.254$, $p<0.0001$), chloride ($r= 0.210$, $p<0.0001$), creatinine ($r = 0.102$, $p = 0.009$) and urea ($r = 0.084$, $p = 0.032$). Nonetheless there was also no co-variation between these electrolytes. On the other hand, in HIV infected participants with serum potassium disorders, correlation between potassium and sodium ($r= 0.491$, $p<0.0001$) and chloride ($r = 0.401$, $p<0.0001$), were accompanied with higher co-variation between them at 24.3% and 16% respectively.

DISCUSSION

Serum creatinine assessment is recommended as a component of the screening panel for monitoring kidney function, and a persistent rise in serum creatinine is indicative of reduced creatinine clearance resulting from impaired kidney function (9). Findings in the current study concur with reports that creatinine clearance decreases in HIV–infected individuals (10). This was further exemplified in HIV- infected females whose serum creatinine levels was higher than uninfected females. The former group also had a higher burden of creatinine disorders than the later. Younger HIV infected individuals manifested a similar trend with elevated serum creatinine levels and burden of creatinine disorders than uninfected younger individuals. Our study established that prevalence of impaired serum creatinine level was 26.1% which was higher than that of the HIV negative control participants. This is also relatively higher than the 4% to 17% prevalence of reduced kidney function which has been reported in diverse HIV-infected populations (11). Older age, and male, as well as protease inhibitors (indinavir) have been cited as risk factors for developing kidney function impairment which could impact on creatinine clearance (12). In our study however, whereas female had a higher rate of serum creatinine disorders than males, age and anti-retroviral drugs use were not risk factors of serum creatinine disorders. Co-existing serum electrolytes disorders could not be attributed to renal function impairment resulting in serum creatinine

disorders. The current study demonstrated that low CD4 cell count, is a determinant of elevated serum creatinine level. Assessment of CD4+ cell counts have served as markers of the progression of HIV infection, as a measure of the relative risk of developing opportunistic infections, and to estimate the impact of HIV and the use of antiretroviral drugs (13). Low CD4 cell count (<500 cells/mm3), constitutes depleted immunity which can predispose to development of opportunistic infection and even malignancies (14). The deterioration of creatinine levels in individuals with low CD4 level observed in the current study could therefore be attributable to opportunistic infections afflicting kidneys and other elements concerned with regulation of body serum creatinine content.

HIV+ individuals had a higher rate of impaired serum sodium balance than HIV negative individuals in the current study. It was also noted that HIV-infected patients not using ARVs were more likely to develop sodium anomalies than their counterparts using ARVs. However gender and age were not associated with sodium disorders in HIV-infected individuals. Hyponatremia was the most common form of sodium disorder in the study which concurred with findings by Tang *et al*, (15), who reported 39% hyponatremia in their study. Ugwuja and Eze (16) demonstrated that mean serum sodium, chloride and potassium levels were significantly lower in HIV positive patients compared to HIV negative controls contrasting our findings that mean serum levels of the three electrolytes were not significantly raised in HIV-infected individuals than in uninfected controls.

In conclusion, the study has demonstrated that kidney function impairment is a common complicating health challenge in the local HIV population. Therefore routine kidney function assessment ought to be one of the care components of HIV-infected individuals in local health institutions to eradicate impediments emanating from concurrent kidney diseases in HIV patients. Further research is needed to illuminate epidemiology, pathogenesis and management priorities to HIV related kidney diseases as they manifest in our health settings. Similarly the study has elucidated that hyponatremia is a common electrolyte disorder in HIV infection and that use of anti-retroviral drugs can help to alleviate its occurrence.

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