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Full Length Research Paper

Relationship between renal ultrasonographic, CD4 cell count and proteinuria findings in HIV infected adult patients in Jos, Nigeria

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This study intends to describe renal sonographic outcomes in matured patients with HIV/AIDS in Plateau State capital Jos and relate the outcome with proteinuria and the subjects' CD4 cell count. The 504 subjects were evaluated for renal ultrasound scan, CD4 cell counts and dipstick proteinuria (using Combi 10 Urinalysis strips) were obtained, and their findings recorded. Twenty-four (4.8%) of the patients had abnormal renal sonographic findings while 480 (95.2%) had normal findings. Sixteen (3.2%) out of the 24 patients with abnormal renal ultrasound had enlarged kidneys, 24 (4.8%) patients had increased renal echogenicity while 19 (3.8%) had loss of corticomedullary differentiation. One (0.2%) patient was observed to have a globular kidney. Significant proteinuria was observed in all the 24 (4.8%) patients with abnormal renal sonographic findings to suggest HIV-associated nephropathy (HIVAN) while the remaining 480 (95.2%) patients had no significant proteinuria. Hence, the prevalence of HIVAN in this study was 4.8%. Kidney size, renal echogenicity and corticomedullary differentiation correlated significantly with proteinuria count with P-values of <0.05 and r-values of 0.531, 0.610 and 0.487, respectively. Similarly, renal echogenicity correlated significantly with CD4 cellular number (p < 0.05, r =-0.540). No substantial correlation was observed between renal size and CD4 tissue quantity (p > 0.05, r = 0.084) and between corticomedullary differentiation and CD4 cell amount (p > 0.05, r = 0.049). Sonographically based determination of renal size and echogenicity/echopattern combined with proteinuria are good determinants of renal parenchymal disease and may have diagnostic usefulness as a non-invasive procedure in the identification of HIVAN in HIV-positive subjects with renal disease.

Key words: HIV, AIDS, HIV-associated nephropathy, kidneys, ultrasonography, proteinuria.

INTRODUCTION

Human immune deficiency virus (HIV) is a retroviridae which reserves their genetic substance as RNA. Two

types of HIV are recognized to attack man, HIV-1 and HIV-2. HIV-1 is mostly popular and devastating disease

globally (Krogstad, 2003; Apetrei et al., 2004).

In 2014, it was projected that persons having HIV world round is about 36.9 million; 68% of these individuals are in sub-Saharan Africa with East and Southern Africa being the most hit regions having an HIV incidence of about 7.2% (UNAIDS, 2014). The incidence of HIV attack in Plateau State capital Jos is 4.9% while middle belt Nigeria has the most occurrence of HIV effect which is put at 6.1%; the national prevalence is put at 4.4% (FMOH, 2005; World Bank, 2014).

The first reported connection between HIV and kidney disease was in 1984 in subjects showing HIV symptoms with renal spread of proteinuria and extending to end-stage renal disease (ESRD) under 8 to 16 weeks (Pardo et al., 1984; Rao et al., 1984; Mazbar et al., 1990). An extended clinical spread of renal defects attack persons presenting HIV signs.

These issues include severe renal attack, electrolyte and acid-base alterations, HIV-related glomerular infection, mild-on chronic kidney illness and detrimental effects associated with management of HIV. Many reports that use different indices for investigation of severe renal issue have reported a number of occurrence between 6 and 48% of these sickness in individuals with HIV in sub-Saharan Africa; 6% in South Africa, 38-51.8% in Nigeria, 26% in Cote d'Ivoire, 28% in Tanzania, 25% in Kenya, 20 to 48.5% in Uganda and 33.5% in Zambia (Agaba et al., 2003; Emem et al., 2008; Fabian and Naicker, 2009). The causes of renal involvement in HIV are mainly drug toxicity as well as release of cytokines during HIV infection (Szczech et al., 2004).

HIVAN is the most common kind of severe kidney illness as a result of HIV attack (Herman and Klotman. 2003) with investigative characteristics expressed by gradual kidney collapse, followed by mild to severe renal spectrum of proteinuria, bland urinary sediment, and ultrasound results of large, and highly echogenic kidneys. Proteinuria serves as its first sign (Cachat et al., 1998). The major occurrence of HIVAN in Africa is relatively not known presently, mainly owing to inadequate investigation and proper report of kidney illness in HIV positive subjects (Ikpeme et al., 2012). In the USA, Atta et al. (2004) published HIVAN incidence of 39% in adult HIV-infected patients. Reports have it that in Southern and Western Nigeria, wide spread incidence of proteinuria among children suffering from HIV to be 18.8 to 31.6% (Anochie et al., 2008; Esezobor et al., 2010; Ikpeme et al., 2012). Emem et al. (2008) however reported the prevalence of HIVAN in Nigeria to be 38%.

Despite that thorough investigation of HIVAN requires histological analysis of the nephron, sonography is a crucial aspect in the examination of human immunodeficiency virus (HIV)-infected persons with

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kidney issues. It is non-invasive, rapid to perform, and relatively inexpensive (Atta et al., 2004; Adeyekun et al., 2011). Definite renal sonographic evidence which has been documented in these individuals, include expanded renal tissue and elevated cortical echogenicity (Cecconi et al., 1994; Di Fiori et al., 1998; Atta et al., 2004; Ikpeme et al., 2012).

CD4 cell numbering is one of the basic investigative methods and a vital index for assessing and following up AIDS sufferers. CD4 cell reduction in HIV-infected persons arises from continuous viral cell division. HIVAN was previously known to be related with modified immunosuppression (CD4 cell counts < 200 cells/mm³), but it was now realized to connote lesions connected with the illness occurring at certain stages of HIV-1 attack prior to antibody seroconversion (Cachat et al., 1998; Agaba et al., 2003; Herman and Klotman, 2003; Emem et al., 2008; Ikpeme et al., 2012).

Quick identification of HIVAN could be helpful in administering therapy promptly and thus avoiding future gradual disease spread to final stage kidney infection, needing renal transplant regime (Atta et al., 2004; Adeyekun et al., 2011; Ikpeme et al., 2012).

The connection between renal sonographic outcomes with CD4 cell numbering and proteinuria has not been duly investigated. The goal and mission of this study is to evaluate the prevalence of HIVAN in Jos with significant proteinuria and renal ultrasound alteration as the criteria for investigation. Connection of renal ultrasound findings with CD4 cell count and proteinuria will also be examined.

METHODOLOGY

Study design

This is a clinic based cross-sectional investigation carried out for a time frame of 10 months from May 2014 to February 2015. Subjects were recruited consecutively based on the inclusion criteria stated until the sample size was reached.

The research work was undertaken at the Department of Radiology, Jos University Teaching Hospital (JUTH), a tertiary medical and health organization located in the heart of Jos. JUTH is one of the three teaching facilities in the middle belt region of Nigeria. It is used as a referral centre for the near-by like of Bauchi, Gombe, Benue, Kogi, Nasarawa, Taraba, Adamawa and parts of Kaduna State. Plateau State has over 30 several ethnic groups. The 2006 Nigerian provisional census estimated the number of persons in Plateau State to be 3,178,712 with 1,585,679 females.

Plateau State is found between latitude 7° and 11° North and longitude 70° and 250° East. The city centre is a spherical-like uphill popularly called Jos Plateau, which extends for about 104 km from north to south, and 80 km from east to west, covering an area of about 8,600 km². This region has a height of 1,200 m above sea level.

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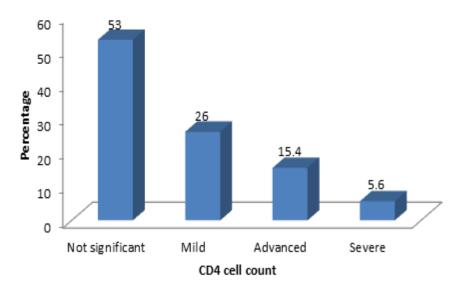


Figure 1. Percentage distribution of HIV/AIDS patients according to the WHO classification of CD_4 immunological profile.

Ethical approval and consent to participants

The official clearance to commence the work was gotten from the Research and Ethical group of the Jos University Teaching Hospital dated 19th June 2014 (JUTH/DCS/ADM/127/XIX/5949). Approval letter of consent was also gotten from the subjects to be used in the study. The individuals were intimated on the safety protocols of ultrasound scan and may wish to pull out from the study at any time without being penalized. The information gotten from the subjects was recorded chronologically and handled privately.

Study population

The study population comprised patients aged 18 years and above confirmed with HIV/AIDS who were referred for abdominal ultrasound scan from clinic II located within the Jos University Teaching Hospital complex. Clinic II is a specialized clinic exclusively for the management of HIV/AIDS patients.

Study inclusion criteria

Persons used in the study were individuals aged 18 years who are HIV sufferers ascertained by Western Blot.

Study exclusion criteria

Subjects that are below 18 years of age, HIV positive patients with confirmed cases of severe kidney issue, HIV positive subjects with ascertained co-infection with Hepatitis A, B or C Virus infection and HIV positive persons with co-existing clinical situations such as hypertension, sickle cell disorder and diabetes mellitus as well as pregnant patients were removed from the research work.

Data collection procedure

The examination was explained to each subject and bio data

including age, sex, height and weight were obtained. Blood pressure was determined in the left arm with individual in seated posture, using a mercury sphygmomanometer and correct cuff size. Measurement was taken at one visit, when patient had rested for 5 min. Three (3) different measurements were obtained a minute apart, and the average determined systolic and diastolic blood pressure values were recorded. This was aimed at exempting individuals with hypertension from the research work.

Western Blot and CD4 cell count outcomes were gotten from subject' case record. Mid-stream urine was taken from each person and tested for proteinuria via dipstick employing Combi 10 (Urinalysis strips). Proteinuria of at least 2+ was considered significant. The ultrasound screening was conducted via a fastresolution real time ultrasound scanning device (ALOKA SSD-3500; Aloka Co. Ltd, Tokyo, Japan, 2007) packaged with a curvilinear transducer of 3.5 MHz. The patients were starved for a period of 6 to 8 h before the start of the procedure to lower bowel air. The kidneys were examined sonographically using standard ultrasound scanning procedures. The ultrasound examinations and determinations were carried out by the laboratory technologists only to reduce inter observer mistakes. All determinations were carried out two times and the mean were computed to get intra observer mistakes. Kidney echogenicity was scored following a standardized score with four different variables (Di Fiori et al., 1998; Atta et al., 2004). 0, shows that the kidney cortex is a little echogenic compared to the liver (Figure 1). I, the kidney cortex and hepatocyte are seemly echogenic (Figure 2). II, the kidney cortex is more echogenic when matched with the hepatocyte (Figure 3). III, the kidney cortex and renal sinus are seemly echogenic (Figure 4). Corticomedullary differentiation was also assessed bilaterally.

Data preparation and analysis

The findings obtained from the abdominal ultrasound scan were inputted into a laptop to create an automated result data base for more determination and evaluation to get significant difference and association between different variables by analysis of variance and Duncan's Multiple Range Test, respectively using Statistical Package for Social Sciences (SPSS) 23 version. P value of 0.05 or

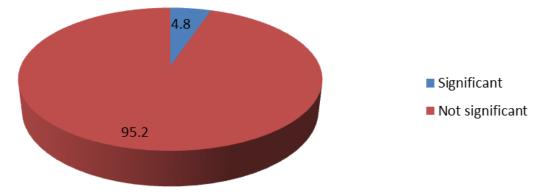


Figure 2. Percentage classification of individuals based on proteinuria. *Significant proteinuria \geq 2+.



Figure 3. Longitudinal photomicrogram of the right renal showing grade I kidney echogenicity in a 26 year old male person infected with HIVAN.

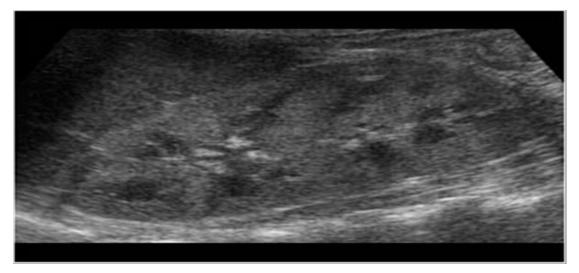


Figure 4. Longitudinal microgram of the right kidney showing grade II renal echogenicity in a 37 year-old female individual having HIVAN.

	Sex					
Age (years)	Male [Freq. (%)]	Female [Freq. (%)]	Total [Freq. (%)]			
≤25	12 (2.4)	19 (3.8)	31 (6.1)			
26-30	9 (1.8)	50 (10.0)	59 (11.7)			
31-35	16 (3.2)	65 (12.9)	81 (16.1)			
36-40	22 (4.4)	77 (15.3)	99 (19.6)			
41-45	21 (4.2)	65 (12.9)	86 (17.1)			
46-50	27 (5.4)	34 (6.7)	61 (12.1)			
>50	33 (6.5)	54 (10.8)	87 (17.3)			
Total	140 (27.8)	364 (72.2)	504 (100)			

Table 1. Age and sex arrangement of HIV/AIDS individuals in Jos.

Table 2. Connection involving age range and CD4 cell count of HIV/AIDS subjects.

A		CD ₄ ⁺ classification							
Age group (years)	Not significant [Freq. (%)]	Mild [Freq. (%)]	Advanced [Freq. (%)]	Severe [Freq. (%)]	Total [Freq. (%)]				
≤25	20 (64.5)	5 (16.1)	4 (12.9)	2 (6.5)	31 (100.0)				
26-30	31 (52.5)	15 (25.4)	9 (15.3)	4 (6.8)	59 (100.0)				
31-35	46 (56.8)	19 (23.5)	15 (18.5)	1 (1.2)	81 (100.0)				
36-40	46 (46.5)	30 (30.3)	12 (12.1)	11 (11.1)	99 (100.0)				
41-45	51 (59.3)	24 (27.9)	6 (6.9)	5 (5.9)	86 (100.0)				
46-50	34 (55.7)	12 (19.7)	13 (21.3)	2 (3.3)	61 (100.0)				
>50	39 (44.8)	26 (29.9)	19 (21.8)	3 (3.4)	87 (100.0)				
Total	267 (53.0)	131 (26.0)	78 (15.5)	28 (5.5)	504 (100)				

 $\chi^2 = 18.903$, df = 12, p = 0.091.

less was taken to be exceptionally different. The results were presented in the form of tables, graphs and charts as appropriate.

RESULTS

A known figure of 504 HIV positive persons, who fulfilled the guidelines for eligibility of being added to the study were evaluated. This involves 140 males (27.8%) and 364 females (72.2%) with men to women range of about 1:3. These patients have been on Highly Active Antiretroviral Therapy (HAART) for a median session of 23.6 months. The ages of the patients ranged between 19 and 80 years with a mean and standard deviation of 40.70±10.5 years. The number of year span for boys and girls ranged from 19 to 77 years and 20 to 80 years with an average and deviation of standard (SD) with 43.2±11.9 and 39.8±8.7 years, respectively. The major age category was 36-40 years (19.6%) and next to 41-45 years (17.1%) (Table 1).

The mean CD_4 cell count was 551.40±301.0 cells/mm³. The mean CD_4 cell count for males and females were 495.10 and 573.00 cells/mm³, respectively. The minimum CD_4 cell count for males and females were 98 and 41 cells/mm³, respectively while the maximum CD_4 cell count was 1440 and 1660 cells/mm³ for males and females, respectively. A reasonable difference exists when considering the two groups (p=0.001).

The age group 36-40 years had the highest frequency of 99 patients (19.7%) with not significant, mild, advanced and severe categories constituting 46 (46.5%), 30 (30.3%), 12 (12.1%) and 11 (11.1%) patients, respectively. The less than 25 years age group had the least frequency of 31 patients (6.2%) with 20 (64.5%), 5 (16.1%), 4 (12.9%) and 2 (6.5%) patients for the not substantive, moderate, advanced and severe groupings, respectively. Appreciable difference was not observed when age was set side by side with CD₄ cell count in the present research activity (p=0.091) (Table 2).

Forty-eight (9.5%) males and 219 (43.50%) females were in the "Not significant" category and 49 (9.7%) males and 82 (16.3%) females were in the "Mild" class. Similarly, 36 (7.1%) males and 42 (8.3%) females and 7 (1.4%) males and 21 (4.2%) females were in the advanced and severe categories, respectively. Notable difference occurs in the correlation when sex is weighed with patient's CD_4^+ numbering having a p value of 0.001 (Table 3).

CD ₄ classification —		Sex	
	Male [Freq. (%)]	Female [Freq. (%)]	Total [Freq. (%)]
Not significant (≥500)	48 (9.5)	219 (43.5)	267 (53.0)
Mild (350-499)	49 (9.7)	82 (16.3)	131 (26.0)
Advanced (200-349)	36 (7.1)	42 (8.3)	78 (15.4)
Severe (<200)	7 (1.4)	21 (4.2)	28 (5.6)
Total	140 (27.8)	364 (72.2)	504 (100.0)

 Table 3. Sex distribution of CD4 cell count in HIV/AIDS patients.

 $\chi^2 = 11.381$, df = 3, p = 0.001.

Table 4. Relationship of proteinuria with sex and CD4 cell count of HIV/AIDS patients.

M. 1.1.1	Pr		Duralius	
Variable	Significant [Frequency (%)] Not significant [Frequency (%)]		 Total (%) 	P-value
Sex				
Male	11 (7.9)	129 (92.1)	140 (100)	0 000
Female	13 (3.6)	351(96.4)	364 (100)	0.009
CD4 cell count				
Not significant	4 (1.5)	263 (98.5)	267 (100)	
Mild	5 (3.8)	126 (96.2)	131 (100)	0.004
Advanced	8 (10.3)	70 (89.7)	78 (100)	0.001
Severe	7 (25.0)	21 (75.0)	28 (100)	

Dipstick proteinuria was negative in 352 (69.8%) patients, 1+ in 128 (25.4%) patients, 2+ in 1 (0.2%) patient and 3+ in 23 (4.6%) patients. Proteinuria of at least 2+ was considered significant. Thus, 24 (4.8%) patients had significant proteinuria (Table 4), out of whom 11 (7.9%) were males and 13 (3.6%) were females. A substantial alliance occurs when proteinuria is placed side by side with sex (p = 0.009) (Table 4).

For the patients with significant proteinuria, 4 (1.5%) subjects were in the 'Not comparable' CD4 cell count group, 5 (3.8%) patients in the 'mild' CD4 cell count category, 8 (10.3%) patients in the 'Advanced' CD4+ count category and 7 (25.0%) patients in the 'Severe' CD4 cell count category. A remarkable relation occurs in the interconnection involving proteinuria with CD4 cell count (p = 0.001) (Table 4).

Renal sonographic findings

The average length of the right kidney was 10.3 ± 1.1 cm while the left kidney was 10.5 ± 1.0 cm. Sixteen (3.2%) patients had enlarged kidneys, consisting 9 (1.8%) males and 7 (1.4%) females while 488 (96.8%) patients had normal sized kidneys consisting 131 (26.0%) males and 357 (70.8%) females. The relationship between renal size and sex of patients studied was appreciably different (p =

0.010) (Table 5).

In the "not significant" WHO CD4 category, 1 (0.2%) patient and 267 (53.0%) patients had enlarged and normal sized kidneys, respectively. While in the "severe" CD4 category, 5 (1.0%) patients and 23 (4.6%) patients had enlarged and normal renal sizes, respectively. This relationship was found to be statistically significant (p = 0.001) (Table 5).

Predominant age groups for renal size abnormalities are 31-35, 36-40 and 41-45 years with 3 (3.7%), 3 (3.0%) and 4 (4.7%), respectively. However, the relationship between renal size and age was not statistically significant (p = 0.932) (Table 6).

Similarly, predominant age groups for abnormal renal echogenicity are 31-35, 36-40, 41-45 and 46-50 years with 6 (7.4%), 5 (5.0%), 4 (4.7%) and 5 (5.5%), respectively. However, the relationship between renal echogenicity and age was not statistically significant (p = 0.983) (Table 6).

Sonographic evaluation of the kidneys of 504 patients reveals increased echogenicity in 24 (4.8%) patients while 480 (95.2%) are normal (Table 7).

Of the patients observed to have echogenic kidneys, 11 (2.2%) were males while 13 (2.6%) were females. Six (1.2%) patients have CD4 cell count in the 'Not significant' category, 5 (1%) in the 'Mild' category, 6(1.2%) in the 'Advanced' category and 7 (1.4%) in the 'Severe'

Verieble	Kidn	ey size	Total	Duralius	
Variable -	Enlarged [Freq. (%)]	Not enlarged [Freq. (%)]	[Freq. (%)]	P-value	
Sex					
Male	9 (1.8)	131 (26.0)	140 (27.8)	0.010	
Female	7 (1.4)	357 (68.6)	364 (72.2)		
CD4 count					
Not significant	1 (0.2)	266 (52.8)	267 (53.0)		
Mild	6 (1.2)	125 (24.4)	131 (26.0)	0.001	
Advanced	4 (0.8)	74 (14.6)	78 (15.4)	0.001	
Severe	5 (1.0)	23 (4.6)	28 (5.6)		

Table 5. Interrelation among kidney size, sex as well as CD4 class in HIV/AIDS patients.

Table 6. Association involving Renal Sonographic findings with age of patients.

	Age group (years)							
Sonographic finding	≤25	26-30	31-35	36-40	41-45	46-50	>50	*P-value
	[Freq. (%)]	[Freq. (%)]	[Freq. (%)]	[Freq. (%)]	[Freq. (%)]	[Freq. (%)]	[Freq. (%)]	
Echogenicity								
Cortex < Liver	30 (96.8)	56 (94.9)	75 (92.6)	94 (94.9)	82 (95.3)	59 (96.7)	84 (96.6)	
Cortex = Liver	1 (3.2)	1 (1.7)	2 (2.5)	2 (2.0)	1 (1.2)	1 (1.6)	0 (0.0)	0.983
Cortex > Liver	0 (0.0)	1 (1.7)	3 (3.7)	1 (1.0)	1 (1.2)	1 (1.6)	2 (2.3)	0.963
Cortex = Renal sinus	0 (0.0)	1 (1.7)	1 (1.2)	2 (2.0)	2 (2.3)	2 (2.3)	1 (1.1)	
Renal size								
Enlarged	1 (3.2)	2 (3.4)	3 (3.7)	3 (3.0)	4 (4.7)	2 (3.3)	1 (1.1)	0.000
Not enlarged	30 (96.8)	57 (96.6)	78 (96.3)	96 (97.0)	82 (95.3)	59 (96.7)	86 (98.9)	0.932
Corticomedullary different	iation							
Prominent pyramids	1 (3.2)	0 (0.0)	2 (2.5)	1 (1.0)	1 (1.2)	1 (1.6)	0 (0.0)	
Normal relationship	30 (96.0)	57 (96.6)	76 (93.8)	96 (97.0)	83 (96.5)	59 (96.7)	84 (96.6)	0.912
Decreased conspicuity	0 (0.0)	2 (3.4)	3 (3.7)	2 (2.0)	2 (2.3)	1 (1.6)	3 (3.4)	

*Fisher's exact test

category. In addition, a sufficient connection exist amongst renal echogenicity as well as CD4 cell count (p = 0.001) (Table 8).

Abnormal corticomedullary differentiation (CMD) was observed in 19 (3.8%) consisting of 8 (1.6%) and 11 (2.2%) males and females, respectively. One hundred and thirty-two (26.2%) males and 353 (70.0%) females had normal CMD. No significant statistical relationship between CMD and sex of patient was observed (p =0.308) (Table 9).

Two hundred and sixty-two (52.0%) patients in the "not significant" CD4 cell count had normal CMD and 5 (1.0%) patients in the same CD4 category had loss of CMD. Twenty-three (4.6%) patients had normal CMD and 5 (1.0%) patients had abnormal CMD in the 'severe' CD4 cell count category. This was appreciably different (p =

0.002) (Table 9).

Features suggestive of HIVAN and correlations

Proteinuria and distinct renal sonographic findings to suggest HIVAN were found in 24 (4.8%) patients. Significant proteinuria was observed in 24 (4.8%) patients with 11 (45.8%) being males and 13 (54.2%) being females. This was meaningfully different (p = 0.013) (Table 10).

Twenty-four (4.8%) patients had increased renal echogenicity with 11 (45.8%) males and 13 (54.2%) females. This was statistically not significant (p = 0.059). Nineteen (3.8%) patients had abnormal corticomedullary differentiation, 8 (42.1%) males and 11 (57.9%) females.

Total

Denal ashegonisity		Sex		P
Renal echogenicity	Male [Freq. (%)]	Female [Freq. (%)]	—— Total (%)	Г
Normal	129 (25.6)	351 (69.6)	480 (95.2)	
Increased	11 (2.2)	13 (2.6)	24 (4.8)	0.240

Table 7. Relationship between renal echogenicity and sex of patients studied.

140 (27.8)

Table 8. Connection of renal echogenicity and CD4cell counts.

	CD4 cell counts				_	
Renal echogenicity	Not significant [Freq. (%)]	Mild [Freq. (%)]	Advanced [Freq. (%)]	Severe [Freq. (%)]	Total (%)	*P- value
Normal	261 (51.8)	126 (25.0)	72 (14.3)	21 (4.2)	480 (95.2)	
Increased	6 (1.2)	5 (1.0)	6 (1.2)	7 (1.4)	24 (4.8)	0.000
Total (%)	267 (53.0)	131 (26.0)	78 (15.4)	28 (5.6)	504(100.0)	

364 (72.2)

This was not statistically significant (p = 0.190) (Table10).

Sixteen (3.2%) patients were found to have enlarged kidneys consisting of 9 (56.3%) males and 7 (43.7%) females, respectively. This was found to be statistically significant (p = 0.014, Table 11). The result also showed exceptional relationship between renal size as well as proteinuria (p = 0.003, r = 0.531) (Table 11).

All 24 (4.8%) patients with increased echogenicity had significant proteinuria while 480 patients did not have significant proteinuria. More so, it revealed a notable connection between renal echogenicity with proteinuria (p = 0.003, r = 0.610) (Table 11).

Four hundred and eighty (98.6%) patients with normal CMD had 'not significant' proteinuria while 7 (1.4%) patients with normal CMD had significant proteinuria. All the 17 patients with abnormal CMD had significant proteinuria. A statistical difference in relationship was observed when corticomedullary differentiation was liken with proteinuria (p = 0.001, r = 0.487) (Table 11).

A negative correlation was observed between renal sonographic findings and CD4+ cell count; this being statistically significant among kidney echogenicity as well as CD4 cell number (p = 0.002, r = -0.540). On the other hand, without any substantial connection when matching kidney size with CD4 cell count (p = 0.059, r = -0.084) as well as between corticomedullary differentiation and CD4 cell quantity (p = 0.268, r = -0.049) (Table 12).

Following the World Health Organization (WHO) distribution of CD4 immunological information in matured HIV-infected persons, 267 (53.0%) patients had CD₄ cell count \geq 500 cells/mm³ (Not significant), 131 (26.0%) patients were having CD₄ cellular number between 350 and 499 cells/mm³ (Mild), 78 (15.4%) patients had CD₄ cell amount ranging from 200 to 349 cells/mm³ (Advanced), while 28 (5.6%) patients were in the severe category with CD_4 cell amount <200 cells/mm³ (Figure 1).

504 (100)

Sonographic evaluation of the kidneys of 504 patients reveals increased echogenicity in 24 (4.8%) patients while 480 (95.2%) are normal (Figures 3 to 5).

One (0.2%) patient had kidneys that were globular in appearance in addition to being enlarged and echogenic (Figure 6).

DISCUSSION

About 504 matured persons living with HIV/AIDS were evaluated with many of them as females (72.2%) while the male to female ratio was 1:3. Nearly 71% of these persons were under the age bracket of 18 to 46 years, which represents the age range mostly predisposed to HIV/AIDS and the financially viable section of the community (Adeoye, 2005). Igbinedion et al. (2009) in Benin-Nigeria reported that about 89% of the sufferers to be aged 18 to 47 years and a male to female range of 1: 2.5. In this study, the average age for females and males was observed to be 39.8±8.7 and 43.2±11.9 years, respectively. A research work published in South Western Nigeria by Obajimi et al. (2008) also stressed that female dominance (66.5%) and female average age of 38.02 years. This was different from the information handed down following the research work in North Western Nigeria (Saidu et al., 2005) and a few regions of the universe (Rao et al., 1984; N'zi et al., 1999; Tshibwabwa et al., 2000) that revealed an increase in male prevalence. The finding of female majority in this work may imply that females are more susceptible to the disease, visit health care facility more and enjoy awareness at the antenatal period for HIV diagnosis resulting in the confirmation of their HIV state (Herman

Corticomedull	ary differentiation (CMD)	Normal relationship	Abnormal CMD	Total	P - value	
	Male [Freq. (%)]	132 (26.2)	8 (1.6)	140 (27.8)		
Sex	Female [Freq. (%)]	353 (70.0)	11 (2.2)	364 (72.21)	0.308	$\chi^2 = 21.0$
Total (%) 485 (92.6)	19 (3.8)	504 (100.0)				
	Not significant [Freq. (%)]	262 (52.0)	5 (1.0)	267 (53.0)		
	Mild [Freq. (%)]	127 (25.2)	4 (0.8)	131 (26.0)		2 01 100
CD4 counts	Advanced [Freq. (%)]	73 (14.4)	5 (1.0)	78 (15.4)	0.002	$\chi^2 = 21.102$
	Severe [Freq. (%)]	23 (4.6)	5 (1.0)	28 (5.6)		Df = 6
	Total (%)	485 (96.2)	19 (3.8)	504 (100)		

 Table 9. Corticomedullary differentiation of patients by sex and CD4 cell count.

 Table 10. HIVAN features by sex of patients.

F = - (Gei	nder	Tatal	Duralius	
Feature	Male Female		Total	P-value	
Kidney size					
Normal	131 (26.8)	357 (73.2)	488 (100)	0.014	
Enlarged	9 (56.3)	7 (43.7)	16 (100)	0.014	
Echogenicity					
Normal	129 (26.9)	351 (73.1)	480 (100)	0.059	
Increased	11 (45.8)	13 (54.2)	24 (100)	0.059	
Corticomedullary differentiation					
Normal	132 (27.2)	353 (72.8)	485 (100)	0.400	
Abnormal	8 (42.1)	11 (57.9)	19 (100)	0.190	
Proteinuria					
Not significant	129 (26.9)	351 (73.1)	480 (100)	0.040	
Significant	11 (45.8)	13 (54.2)	24 (100)	0.013	

and Klotman, 2003; Garko et al., 2015).

Following the World Health Organization (WHO) analysis of CD₄ immunological status in mature HIV sufferers (WHO, 2006), 87.0% of these persons got CD4 cell count of 350/µl and above resulting in 47.2 and 39.8% for the "Not significant" and "Moderate" groups, respectively while about 8.2% were in the "Chronic" group (that is, CD4 cell count <200/µl). This was different from the reports given by Igbinedion et al. (2009) who discovered that about half (46.3%) of the individuals to be in the "Extreme Case" CD4 range. The increased CD4 cell count of this work may indicate early detection and close monitoring as well as accessibility of anti-retroviral treatment regimen contrary to the past where most persons hardly get these anti-retroviral treatments. This may be adduced to enhanced action by government mediation as per medical guidance and presence of treatment regimens.

In this research, the frequency of renal disease as decided by distinct renal sonographic findings and proteinuria in matured HIV positive subjects in Jos was 4.8% (24 out of 504 patients). This is small compared to findings in prior published research in Nigeria (Emem et al., 2008; Obajimi et al., 2008; Adeyekun et al., 2011) and other parts of the world (O'Neill, 1997; Carol, 1992). This little occurrence could mean that most of the victims while carrying the research work had since been placed on HAART regimen for an average duration of 23.6 months.

The patients involved in this study are black Africans and studies (O'Neill, 1997; Lucas et al., 2004; Adeyekun et al., 2011) have shown that human immunodeficiency virus-associated nephropathy (HIVAN) is of high incidence and a major cause of terminal point kidney infection in black HIV-sufferers (Winston et al., 1999; Atta et al., 2004; Han et al., 2006; Adeyekun et al., 2011). According to some of the earlier researchers, this could

Table 11. Correlation between renal ultrasonographic findings and proteinuria.

	Prote	inuria			
Sonographic feature	Not significant [Frequency (%)]	Significant [Frequency (%)] Total (%) P-value 8 (1.6) 16 (100) 488 (100) 16 (100) 0.003 0 (0.0) 24 (100) 480 (100) 24 (100) 0.001 7 (1.4) 17 (100) 487 (100) 17 (100) 0.001	*r		
Kidney size					
Not enlarged	480 (98.4)	8 (1.6)	488 (100)	0.000	0.531
Enlarged	0	16 (100)	16 (100)	0.003	0.531
Echogenicity					
Normal	480 (100)	0 (0.0)	480 (100)	0.004	0.010
Increased	0 (0.0)	24 (100)	24 (100)	0.001	0.610
Corticomedullary different	tiation				
Normal relationship	480 (98.6)	7 (1.4)	487 (100)	0.004	0.400
Abnormal	0 (0.0)	17 (100)	17 (100)	0.001	0.482

*Pearson's correlation coefficient.

Table 12. Correlation between renal ultrasonographic findings and CD4 cell counts.

		CD4 counts					
Sonographic feature	Not significant [Freq. (%)]	Mild [Freq. (%)]	Advanced [Freq. (%)]	Severe [Freq. (%)]	Total (%)	P-value	* r
Kidney size							
Not enlarged	266 (54.5)	125 (25.6)	74 (15.2)	23 (4.7)	488 (100)	0.050	0.004
Enlarged	1 (6.3)	6 (37.5)	4 (25.0)	5 (31.2)	16 (100)	0.059	-0.084
Echogenicity							
Cortex < Liver	261 (54.4)	126 (26.3)	72 (15.0)	21 (4.3)	480 (100)		
Cortex = Liver	3 (37.5)	0 (0.0)	2 (25.0)	3 (37.5)	8 (100)	0.002	-0.540
Cortex > Liver	2 (22.2)	2 (22.2)	4 (44.4)	1 (11.2)	9 (100)	0.002	-0.540
Cortex = Renal sinus	1 (14.2)	3 (42.9)	0 (0.0)	3 (42.9)	7 (100)		
Corticomedullary different	entiation						
Prominent pyramids	2 (33.3)	1 (16.7)	1 (16.7)	2 (33.3)	6 (100)		
Normal relationship	262 (54.0)	127 (26.2)	73 (15.1)	23 (4.7)	485 (100)	0.268	-0.049
Decreased conspicuity	3 (23.1)	3 (23.1)	4 (30.7)	3 (23.1)	13 (100)		

*Pearson's correlation coefficient.

be attributable to a genetic predisposition to the disease in patients of African descent. This linkage is also backed by the findings and work of other scientists (Ibinaiye et al., 2014; Garko et al., 2015).

Most studies have shown that bilateral echogenic and mostly expanded kidneys are usual ultrasound outcomes in HIVAN (Di Fiori et al., 1998; Hamper et al., 1988; Saidu et al., 2005). Renal abnormalities associated with HIV were first described by Rao et al. (1984). These researchers described associated sonographic findings of enlarged kidneys and increased echogenicity. Hamper et al. (1988) described the kidneys of patients with HIVAN as enlarged and echogenic, measuring greater than 13 cm in their bipolar length. Garko et al. (2015) in Borno, Nigeria reported enlarged kidneys in HIV/AIDS patients with HIVAN. Similar findings had earlier been reported by Saidu et al. (2005) in Sokoto. Glassock et al. (1990) postulated the renal enlargement in HIVAN to occur following inadequate time for general sclerosis and fibrosis, owing to the fast development of the kidney disease; pronounced expansion of the tubules with countless microcysts, in disparity to the regular tubular fall observed in other kinds of severe kidney wound; and musculus dropsy. The finding in this study is in agreement



Figure 5. Longitudinal representation of the right renal tissue revealing grade III kidney echogenicity in a subject suffering from HIVAN.

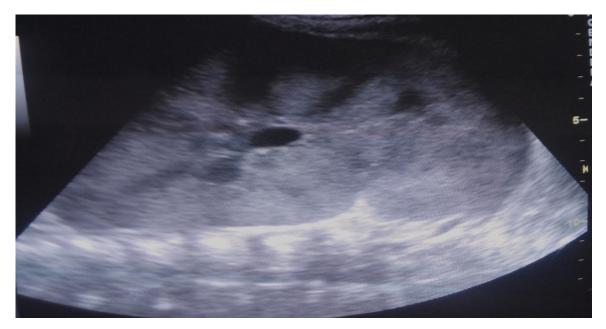


Figure 6. Longitudinal sonogram of a globular and echogenic left kidney in a 33-year-old female candidate with HIVAN.

with the observations of the aforementioned work because 16 (67%) out of the 24 patients with echogenic kidneys, had enlarged kidneys. The reasons for the renal enlargement are probably the same as those postulated by Glassock et al. (1990).

Nonetheless, some studies have reported normal sized echogenic kidneys in HIVAN (Atta et al., 2004; Anochie et al., 2008). Atta et al. (2004) in a research work that evaluated 87 subjects suffering from HIV infection using renal sonography and kidney biopsy showed that many persons suffering from kidney parenchymal disease showed usual renal shape despite the HIVAN state. They also postulated that patients with HIVAN develop overt renal failure as evidenced by rapid increase in creatinine levels without shrinking of the kidneys evidenced in more renal attack. This characteristic may result in the concept stating that HIVAN renal are big taken that they looked bigger than anticipated, owing to the extent of kidney collapse.

Obajimi et al. (2008) of Ibadan, Nigeria studied abdominal ultrasound on 391 HIV/AIDS persons, and documented elevated kidney cortical/medullary echotexture in 8.4% of patients.

N'zi et al. (1999) in Cote d'Ivoire identified hyperechoic renal tissues indicative of HIVAN in 13.7% patients of the 146 with severe immunosuppression on whom abdominal ultrasound was carried out. Garko et al. (2015) conducting renal ultrasound on 100 confirmed HIV-positive patients in Borno, Nigeria recorded a much higher percentage. They detected increased renal echogenicity with loss of corticomedullary differentiation in about 80 to 95% of the patients. The high percentage was however explained by the fact that renal ultrasound was conducted in subjects suffering from clinical and laboratory characteristics of HIVAN many of which appear later as the illness progresses. In the research work, the increased renal echogenicity was as observed in the aforementioned studies but a smaller percentage (4.8%) was found. As stated earlier, this may arise because majority of the sufferers during the research period, were since being administered HAART for a median period of about 23.6 months.

The works of Di Fiori et al. (1998), Ibinaiye et al. (2014) and Garko et al. (2015) on HIVAN patients, described increased renal echogenicity, loss of corticomedullary definition and decreased renal sinus fat respectively on ultrasound. Other findings include parenchymal heterogeneity with or without echogenic striations and globular renal configuration. These findings were most remarkable in advanced stages of HIVAN. According to a study by Wachsberg et al. (1995), increase in renal echogenicity on ultrasound in HIVAN patients may manifest as patchy or spotty echogenicity. They added that advanced stages typically demonstrate a diffuse increase in renal echogenicity and pelvi-calyceal thickening. Similar findings were observed in this study.

In studies that employed a patterned classifying approach of assessing the sonographic renal echogenicity, an extremely echogenic cortex was revealed to be the major disorder in patients suffering from HIV-related kidney disorders and the extent of rising echogenicity was seen to be exact proportion to the harshness of the infection (Paivansalo et al., 1985; Hamper et al., 1988; Page et al., 1994; Di Fiori et al., 1998; Atta et al., 2004). The pathophysiology of acute echogenic kidneys in persons suffering from HIVAN is uncertain but it is believed to be further recurrence in tubulointerstitial illness compared to the glomerular own (Paivansalo et al., 1985; Hamper et al., 1988; Page et al., 1994). It is agreed that, despite that sonography created a great anticipating worth for kidney parenchymal illness, no particular diagnosis could be prominent (Atta et al., 2004; Paivansalo et al., 1985; Page et al., 1994).

Garko et al. (2015) reported that HIVAN normally take place lastly in the event of HIV plague and the harmful point for its progression which involves a reduced CD4 cell quantity and a high viral burden. These observations have been previously reported (O'Neill, 1997, 2000).

Other authors have demonstrated HIVAN to be an early manifestation of HIV infection (Hricak et al., 1982; Paivansalo et al., 1985; Anochie et al., 2008). Findings in this study agree with the former reports because thirteen of the 24 patients with features suggestive of HIVAN

presented in the later stages of the disease with six in the 'Advanced' category (CD4 cell count between 200 and 349 cells/mm³) and seven in the 'Severe' category (CD4 cell count < 200 cells/mm³).

This study found a statistically significant negative correlation between renal echogenicity and CD4 cell count. This agrees with the works of Garko et al. (2015) and Ibinaiye et al. (2014). However, no significant correlation was observed between renal size and CD4 cell count, likewise between corticomedullary differentiation and CD4 cell count.

Studies have found that most patients with HIVAN have advanced renal failure at the time of diagnosis with high serum creatinine and/or proteinuria. Emem et al. (2008) studied 400 HIV-positive patients using proteinuria and serum creatinine and found significant proteinuria in 21.9% patients and elevated serum creatinine in 52% of the patients. This is in agreement with works of Atta et al. (2004). Di Fiori et al. (1998) and Garko et al. (2015). These authors further explained that elevated serum creatinine and/or proteinuria are indicators of declining renal function in HIVAN. According to the findings of Cachat et al. (1998), proteinuria may be the first sign of HIVAN. Ikpeme et al. (2012) and Atta et al. (2004) observed that patients with HIVAN have nephrotic range proteinuria at presentation and many will develop full blown nephrotic syndrome. The index study reports significant proteinuria in 4.8% of the patients studied. This low percentage may be explained by the fact that most of these patients are already on Highly Active Antiretroviral Therapy (HAART), which has been associated with a substantial reduction in the incidence of HIVAN.

From findings of the work, a notable relationship occurred among increased renal echogenicity and proteinuria. Similarly, kidney enlargement and loss of corticomedullary differentiation correlated significantly with proteinuria. This is in accordance with the research findings of Garko et al. (2015) who reported the extent to which renal echogenicity correlates positively with the severity of HIVAN as indicated by raised serum creatinine level and proteinuria. They further stated that prognosis worsens with higher serum creatinine and proteinuria.

This study's limitation is the lack of pathologic confirmation. However, Di Fiori et al. (1998) have stated that renal biopsy is rarely performed in HIV-infected patients in most major centres because of the success of clinical and laboratory findings and that biopsy adds to morbidity, mortality and cost without significantly improving patient outcome or treatment. Furthermore, Atta et al. (2004) opined that a patterned procedure in assessing renal echogenicity possess the capacity to facilitate the determination of HIVAN in HIV-positive individuals with kidney issues, especially in subjects whose renal biopsy is comparatively or realistically counter positioned. This, according to them, might to a great extent account for a predominantly harmful

echogenicity, permit a presumptive diagnosis of HIVAN.

Conclusion

As observed in this study, the degree of renal echogenicity/echopattern correlated with the severity of possible HIVAN as measured by proteinuria. There may be exclusion of HIVAN diagnosis in very low level of echogenicity/echopattern with no proteinuria.

RECOMMENDATIONS

Renal ultrasound scan should be carried out on all patients with suspected HIVAN because it is a good determinant of renal parenchymal disease (echogenicity/ echopattern).

CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

REFERENCES

- Adeoye S (2005). Sexual behaviour, perception of HIV/AIDS and Condom use Among Commercial Motorcyclists in Benin City. Nigerian Postgraduate Medical Journal 12(4):262-265.
- Adeyekun AA, Unuigbe EI, Azubuike CO (2011). Renal Sonographic Parameters in Human Immunodeficiency Virus –Infected Subjects and Relationship to CD4 Cell count. Saudi Journal of Kidney Disease and Transplantation 22(6):1164-1168.
- Agaba EI, Agaba PA, Sirisena ND, Anteyi EA, Idoko JA (2003). Renal disease in the acquired immunodeficiency syndrome in North Central Nigeria. Nigerian Journal of Medicine 12(3):120-125.
- Anochie IC, Eke FU, Okpere AN (2008). Human immunodeficiency virus-associated nephropathy (HIVAN) in Nigerian children. Pediatr Nephrol, 23(1):117-122.
- Apetrei C, Marx PA, Smith, SM (2004). The evolution of HIV and its consequences. Infectious Disease Clinics of North America 18:369-394.
- Atta MG, Longenecker JC, Fine DM, Nagajothi N, Grover DD, Wu J (2004). Sonography as a predictor of human immunodeficiency virusassociated nephropathy. Journal of Ultrasound in Medicine 23:603-610.
- Cachat F, Cheseaux JJ, Guignard JP (1998). HIV-associated nephropathy in children. Archives de pediatrie 5(12):1353-1358.
- Carol AM (1992). Kidney. In: Carol AM (ED). General Ultrasound. Churchill, Livingstone New York P 846.
- Cecconi L, Schinina V, BusiRizzi E, Mazzuoli G (1994). Increased renal cortical echogenicity in HIV positive subjects. Rays 19:235-243.
- Di Fiori JL, Rodrigue D, Kaptein EM, Ralls PW (1998). Diagnostic sonography of HIV-associated nephropathy: new observations and clinical correlation. American Journal of Roentgenology 171(3):713-716.
- Emem CP, Arogundade F, Sanusi A, Adelusola K, Wokoma F, Akinsola A (2008). Renal disease in HIV-seropositive patients in Nigeria: An assessment of prevalence, clinical features and risk factors. Nephrology Dialysis Transplantation 23(2):741-746.
- Esezobor CI, Iroha E, Onifade E, Akinsulie AO, Temiye EO, Ezeaka C (2010). Prevalence of proteinuria among HIV infected children attending a tertiary hospital in Lagos, Nigeria. Journal of Tropical Pediatrics 56(3):187-190.

Fabian J, Naicker S (2009). HIV and kidney disease in sub-Saharan

Africa. Review Nephrology 5(10):591-598.

- Federal Ministry of Health (FMOH) (2005). HIV seroprevalence and sentinel survey 2005. National AIDS/STDs Control Programme. Abuja: Federal Ministry of Health.
- Garko SS, Ibinaye PO, Abba SM, Ahmed A, Tanimu SS, Okere PC (2015). The utilization of diagnostic ultrasound in the evaluation of the kidneys in HIV-associated nephropathy. West African Journal of Radiology 22(1):20-26.
- Glassock RJ, Cohen AH, Danovitch G, Parsa P (1990). Human immunodeficiency virus (HIV) infection and the kidney. Annals of internal medicine, 112(1):35-49.
- Hamper UM, Goldblum LE, Hutchins GM, Sheth S, Dahnert WF, Bartlett JG (1988). Renal involvement in AIDS: sonographic-pathologic correlation. American Journal of Roentgenology 150(6):1321-1325.
- Han TM, Naicker S, Randial RK, Assounga AG (2006). A cross sectional study of the HIV seropositive patients with varying degrees of proteinuria in South Africa. Kidney International 69(12):2243-2250.
- Herman ES, Klotman PE (2003). HIV–associated: epidemiology, pathogenesis, and treatment. Seminars in Nephrology 23:200-208.
- Hricak H, Cruz C, Romanski R, Uniewski MH, Levin NW, Madrazo BL (1982). Renal parenchymal disease: sonographic-histologic correlation. Radiology 144(1):141-147.
- Ibinaiye PO, Garko SS, Ahmed A, Tanimu SS, Tahir NM (2014). Relationship of ultrasound renal echogenicity, serum creatinine level and CD4+ cell count in patients with human immunodeficiencyassociated nephropathy. Sub-Saharan Africa Journal of Medicine 1(4):191.
- Igbinedion BO, Marchie TT, Ogbeide, E (2009). Trans-abdominal ultrasound findings correlated with CD_4^+ counts in adults HIV-infected patients in Benin Nigeria. South Africa Journal of Radiology 13(2):34-40.
- Ikpeme EE, Ekrikpo UE, Akpan MU, Ekaidem SI (2012). Determining the prevalence of Human Immunodeficiency Virus–Associated nephropathy (HIVAN) using proteinuria and ultrasound findings in a Nigerian paediatric HIV population. The Pan African Medical Journal 11(1).
- Krogstad P (2003). Molecular biology of the human immunodeficiency virus: current and future targets for intervention. Seminars in Pediatric Infectious Diseases 14(4):258-268.
- Lucas GM, Eustace JA, Sozio S, Mentari EK, Appiah KA, Moore RD (2004). Highly active antiretroviral therapy and the incidence of HIV-1-associated nephropathy: a 12-year cohort study. AIDS 18:541-546.
- Mazbar SA, Schoenfeld PY, Humphreys MH (2004). Renal involvement in patients infected with HIV: experience at San Francisco General Hospital. Kidney International 37(5):1325-1332.
- N'zi PK, Coulibaly A, N'Dri K, Quattara ND, Diabate SA, Zunon-Kipre E (1999). Ultrasound aspects of abdominal involvement in adults with HIV infections in the Ivory Coast: A propos of 146 cases. Santé 9(2):85-88.
- O'Neill WC (1997). Renal ultrasonography: a procedure for nephrologists. American Journal of Kidney Diseases 30:579-585.
- O'Neill WC (2000). Sonographic evaluation of renal failure. American Journal of Kidney Disorder 35(6):1021-1038.
- Obajimi MO, Atalabi MO, Ogbole GI, Adenke TA, Agunloye AM, Adekanmi AJ (2008). Abdominal ultrasonography in HIV/AIDS patients in south-western Nigeria. BMC Medical Imaging 8(5).
- Page JE, Morgan SH, Eastwood JB, Smith SA, Webb DJ, Dilly SA (1994). Ultrasound findings in renal parenchymal disease: comparison with histological appearances. Clinical Radiology 49(12):867-870.
- Paivansalo M, Huttunen K, Suramo I (1985). Ultrasonographic findings in renal parenchymal diseases. Scandinavian Journal of Urology and Nephrology 19(2):119-123.
- Pardo V, Aldana M, Colton RM, Fischl MA, Jaffe D, Moskowitz L (1984). Glomerular lesions in the acquired immunodeficiency syndrome. Annals of Internal Medicine 101(4):429-434.
- Rao TKS, Filippone EJ, Nicastri AD, Landesman SH, Frank E, Chen CK (1984). Associated focal and segmental glomerulosclerosis in the acquired immunodeficiency syndrome. The New England. Journal of Medicine 310(11):669-673.
- Saidu SA, Makusidi MA, Isyaku K, Ma'aji SM (2005). Abdominal

ultrasound findings in AIDS patients in Sokoto: A preliminary report. Borno Medical Journal 2:11-13.

- Szczech LA, Hoover DR, Feldman JG (2004). Association between renal disease and outcomes among HIV-infected women receiving or not receiving antiretroviral therapy. Clinical Infectious Diseases 39(8):1199-1206.
- Tshibwabwa ET, Mwaba P, Bogle-Taylor J, Zumla A (2000). Four-year Study of Abdominal Ultrasound in 900 Central African Adults with AIDS Referred for Diagnostic Imaging. Abdominal Imaging 25(3):290-296.
- UNAIDS (2014). The Joint United Nations Programme on HIV/AIDS. UNAIDS/WHO. AIDS Epidemic Update: December 2014. https://www.google.com/search?q=UNAIDS+%282014%29.+The+Joi nt+United+Nations+Programme+on+HIV%2FAIDS.+UNAIDS%2FWH O.+AIDS+Epidemic+Update%3A+December+2014.&ei=1s7mYNPCE oyKlwTfkZ-

ADQ&oq=UNAIDS+%282014%29.+The+Joint+United+Nations+Prog ramme+on+HIV%2FAIDS.+UNAIDS%2FWHO.+AIDS+Epidemic+Up date%3A+December+2014.&gs_lcp=Cgdnd3Mtd2l6EAM6BQgAELA DOgIIAEoECEEYAVCjsYEGWLPsjAZg5o2NBmgDcAB4BIABjQKIAe cLkgEFMC4xLjWYAQGgAQGgAQKqAQdnd3Mtd2l6sAEAyAEBwAE B&sclient=gws-

wiz&ved=0ahUKEwjThdbMndPxAhUMxYUKHd_IB9AQ4dUDCA4&ua ct=5

Wachsberg RH, Obolevich AT, Lasker N (1995). Pelvicalyceal thickening in HIV-Associated Nephropathy. Abdom Imaging 20(4):371-375.

- Winston JA, Klotman ME, Klotman PE (1999). HIV–associated nephropathy is a late, not early manifestation of HIV-1 infection. Kidney International 55(3):1036-1040.
- World Bank 2014. Nigeria Economic report No. 2. Washington, DC: World Bank Group. Available from: http://documents.worldbank.org/curated/en/2014/07/198832 economic-report-no-2.
- World Health Organization (WHO) (2006). WHO case definitions of HIV for surveillance and revised clinical staging and immunological classification of HIV-related disease in adults and children. Geneva: World Health Organization 2006:2016.