Research Articles

# Prevalence and predictors of Kaposi's sarcoma among HIV patients receiving care at a tertiary hospital in Jos, Nigeria

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## Background

Kaposi's sarcoma became prevalent with the appearance of human immune deficiency virus (HIV) in the 1980s. However, the widespread use of the highly active antiretroviral therapy (HAART) reduced its prevalence in communities with good access to the antiretroviral drugs. The objective of this study was to determine the prevalence and predictors of Kaposi's sarcoma among persons receiving HIV care at a tertiary hospital in Jos, Nigeria.

## Methods

The study used a cross-sectional study design, based on secondary data related to patients who had received HIV care between January 2004 and December 2017. Logistic regression was then used to determine the variables that were predictors of Kaposi's sarcoma risk.

## Results

The prevalence of Kaposi's sarcoma among the patients was 1.2% (95% confidence interval, CI=1.06-1.34). Patients whose baseline viral loads were higher than 10,000 copies/mm$^3$ were three times more likely to develop Kaposi's sarcoma than those with lesser viral load (OR: 3.13, CI: 2.19-4.47). Gender, duration of HAART and education had modifying effect on the Kaposi's sarcoma risk.

## Conclusions

Kaposi's sarcoma is a substantial public health problem among the HIV population in Jos. Universal access to HAART by the Federal Government of Nigeria and its partners is recommended to reduce its prevalence. In addition, education, skill acquisition and income generating programs should be targeted at girls and women by governments and other stakeholders in order to reduce the inequality that worsens their vulnerability to HIV infection and Kaposi's sarcoma.

Kaposi's sarcoma (KS) is an angio-proliferative malignancy that affects the endothelium of blood vessels. It predominantly affects the skin at different sites and can progress and regress depending on the immunity of the host.[1] KS is one of the most common malignancies in the world and the most important AIDS defining cancer (ADC).[2] The Human Herpes Virus type 8 (HHV-8) is a necessary, but not sufficient cause of KS.[3] The prevalence of KS is higher among the human immunodeficiency virus (HIV) infected population than the general population and it varies widely among different HIV infected populations.[4–9]

It is not certain what factors predict the development of KS among the HIV infected persons. In the late stages of the HIV infection, KS runs a very aggressive course, suggesting the modifying role of lowered immunity in its development and progression.[9] However, some HIV patients treated with the highly active antiretroviral therapy (HAART), and who experience a substantial restoration of the CD4+ cell count have also been reported to develop KS, even at this stage, suggesting that there may be other factors at play.[2] The advent of HAART has reduced the incidence of KS in Europe and North America, but disaggregated data even in such societies have provided evidence of stagnant or even worsening incidence of KS among certain population subgroups.[9] This is often attributed to the inequality in access to HAART in those populations. In low- and middle-income countries (LMICs), access to HAART has been suboptimal, while the adherence to the prescribed regime is partial even in those patients who receive it.[10] There appears to be a complex interplay of factors that interact among themselves to deter-

mine the development of KS among those infected with the HIV. Besides the background HIV and HHV-8 infections, it is not known what other factors predict the development of KS among the HIV patients.

The objective of this study was to determine the prevalence of KS among HIV infected patients, who were receiving care at the tertiary hospital and the factors that predict the development of this malignancy among them.

## METHODS

The study was conducted in the AIDS Prevention Initiative in Nigeria, APIN-supported HIV treatment and care centre of the Jos University Teaching Hospital in Jos, Nigeria. The Centre serves patients from all over central Nigeria. The Centre routinely collects the data from patients using standardized forms at three monthly interval visits. At first contact, all patients have their HIV infection status confirmed using double HIV rapid testing. They are then registered for care and the information recorded on each patient includes demographic characteristics and history of AIDS related conditions. Clinical investigations routinely done for the patients at baseline include full blood count, viral load, CD4+ count and chest X-rays. During follow up visits, CD4+ cell count and viral load estimation are repeated and recorded along with history of HAART over the follow up period. Any adverse events such as drug reactions, opportunistic infections, cancer development and any other illness are recorded. Diagnosis of Kaposi's sarcoma is based on the attending physician's diagnosis, supported by a histology report.

The study design was cross-sectional, based on the secondary data on adult HIV patients registered at the Centre between 1st January 2004 and 31st December 2017, irrespective of whether or not they received HAART. Inclusion criteria were adults aged 18 years and above and confirmed HIV infection status. Patients whose socio-demographic, HAART and HIV clinical/laboratory data were incomplete were excluded from the study.

The minimum sample size was estimated to be 139 using the formula for sample size estimation in cross sectional studies ($n = Z^2pq/d^2$),[11] and the proportion of HIV patients who developed KS from a previous study (10%).[12] However, a total sample of all the records that met the inclusion criteria were included in the analysis. Data were imported into the IBM Statistical Package for Social Sciences (SPSS) version 21 for statistical analysis. Patients who developed KS were compared with those who did not develop the cancer using age groups, sex, baseline CD4+ cell count, end line CD4+ cell count, baseline viral load and end line viral load. Chi square test was used to test the association between socio-demographic factors and other clinical conditions on one hand and the development of KS on the other. Logistic regression was used to determine the factors that predict the development of KS in the patients using their odds ratios and 95% confidence intervals. The independent variables in this model were the patients' age, sex, education, marital status, CD4+ cell count, and viral load while the dependent variable was the development of Kaposi's sarcoma.

Permission for the use of secondary data was obtained from the management of Treatment and Care Centre, APIN Public Health Initiatives, and Harvard T. H. Chan School of Public Health, Boston, USA. In addition, the Centre had obtained an ethical approval from the institution's Health Research Ethics Committee for all research work involving data collected in the Centre which is renewed regularly. All data exported for analysis carried no personal identifying information on the individual patients and strict confidentiality was maintained.

## RESULTS

The majority of the patients were aged between 31 and 50 years (70.3%). A total of 16 235 (65.4%) of the patients were female and 15 214 (63.0%) had completed at least secondary school. Most of patients (54.9%) were married at the time of registration for care (Table 1).

There were 281 patients with the KS diagnosis among 24 430 HIV infected patients, yielding an overall KS prevalence of 1.2% (95% C.I: 1.06 – 1.34; Table 2). Two hundred and fifty two (89.6%) of the 281 KS cases were aged between 30 and 59 years while 193 (67.5%) of the 286 patients with KS were female, giving a male: female ratio of KS of 1:2. Most; 171 (61.1%) of the KS cases were married.

Out of 22 243 patients with baseline CD4+ counts of 500 cells/ml or less, 255 (1.2%) developed KS, while 26 (1.3%) of the 2071 patients with baseline CD4+ counts greater than 500 cells/ml developed KS (Table 3). At end of the study period, 187 (1.1%) of the 16 679 patients with CD4+ counts of 500 cells/ml or less developed KS and 90 (1.3%) of the 7168 patients with CD4 count greater than 500cell/ml developed KS. There was no statistically significant relationship between level of CD4+ count and development of KS. Thirty five (0.5%) of 6 932 patients with baseline viral load of 1000 copies/ml or less developed KS whereas 230 (1.6%) of the 14712 patients with baseline viral load greater than 1000 copies/ml developed KS. There was a statistically significant association between the baseline viral load and development of KS ($p < 0.001$). At the end of the study period, 152 (1.6%) of the 9 557 patients with viral load of 1000 copies/ml or less developed KS while 93 (1.7%) of 5 484 with viral load greater than 1000 developed KS. There was no statistically significant association between end of study viral load and development of KS ($p = 0.62$).

Out of the 21149 patients who had received HAART for less than 10 years, 217 (1.1%) developed KS compared to the 48 (3.5%) of 1390 who had HAART for 10 or more years. There was a statistically significant association between duration on HAART and development of KS ($P < 0.001$; Table 4). Eighty of the 24825 HIV infected patients also had tuberculosis infection giving a prevalence of 0.3% and 75 (93.7%) out of the 80 patients with TB also developed KS. There was a statistically significant association between tuberculosis co-infection and development of KS ($P < 0.001$; Table 5)

Gender, secondary school education, baseline viral load and duration on HAART were found to be independent predictors of KS). The odds ratios show that being male and attaining secondary level of education half the risk of development of KS while having been on HAART for ten years or more increases the risk of KS by 70% (Table 6).

**Table 1. Socio-demographic characteristics of patients in care in the Centre 2004-2017**

| Characteristics | N | Percentage |
|---|---|---|
| **Age group (years)** | | |
| ≤ 20 | 93 | 0.4 |
| 21-30 | 1276 | 5.2 |
| 31-40 | 8250 | 33.8 |
| 41-50 | 8921 | 36.5 |
| 51-60 | 4381 | 17.9 |
| >60 | 1507 | 6.2 |
| **Total** | **24428** | **100.0** |
| **Mean age ± SD** | **43.8 ± 9.2** | |
| **Sex** | | |
| Male | 8605 | 34.6 |
| Female | 16235 | 65.4 |
| **Total** | **24840** | **100.0** |
| **Marital status** | | |
| Single | 5340 | 22.1 |
| Married | 13260 | 54.9 |
| Widowed | 3624 | 15.0 |
| Divorced | 1936 | 8.0 |
| **Total** | **24160** | **100.0** |
| **Highest education** | | |
| No formal | 4125 | 17.0 |
| Primary | 4825 | 20.0 |
| Secondary | 7639 | 31.6 |
| Tertiary | 7575 | 31.4 |
| **Total** | **24164** | **100.0** |

## DISCUSSION

The prevalence of Kaposi's sarcoma among the HIV patients attending the treatment Centre was 1.2 %. The female gender, high baseline viral load and having been on HAART for 10 or more years were significant predictors of KS development, whereas having attained secondary level of education significantly reduced the risk of KS development.

The prevalence of KS in this study is lower than what was documented earlier in 2009 from the same Centre.[13] The earlier study was conducted at a time when HAART just became available in Nigeria, therefore the true impact of HAART was largely unknown. In addition, the earlier study used a smaller sample size compared to the current study which could account for the difference in prevalence found in the two studies. The finding is similar to the 1.2% earlier found in Lagos,[14] but lower than what was found in other studies.[15,16] In Zaria, a city located in northern Nigeria, a KS prevalence of 1.4% was documented.[17] All the findings above are, however, higher than the 0.6 %[8] and 0.8%[4] found earlier in Kano and Abuja respectively.

As early as 1996, the prevalence of KS among HIV positive clients was said to be 0.52% in Lagos.[18] It would appear that, although the prevalence of KS is still low among people living with HIV in Nigeria, it is showing an increasing trend. The figures obtained in this study are much lower than that from Sao Paulo, Brazil, where the prevalence of KS among persons on HAART was reported to be 6%.[5] In North America, the cumulative incidence of KS was recorded to be 4.4 % in 2015.[19] In LMICs, access to HAART is suboptimal and sero-prevalence of HHV-8 is reported to be as high as 20-30%.[1] These factors are likely to continue to drive the prevalence of KS in directions dictated by the balance of force between access to HAART and other protective factors against KS on one hand and HHV-8 and other KS risk factors on the other. Since the prevalence of KS is lower in Sub Saharan Africa (SSA) than in Europe and USA despite poorer access and adherence to HAART, other factors such as genetics, melanin content of the black skin and other environmental factors are probably protective against KS among the HIV infected population in SSA.

The male to female ratio of KS in this study was 1:2. This agrees with earlier findings of 1:1.4[13] and 1:2[20] in Jos but lower than that in Zaria.[17] In Abuja and Kano, the M: F ratio was as high as 2:1[4] and 2.4:1[8] respectively. Before the advent of HIV, KS was about four times more common among the males than the females globally.[1] However, the higher prevalence of HIV among women has tilted the balance to a higher prevalence of KS among the females than males in SSA. This position is supported by the data in this study

**Table 2. Socio-demographic characteristics of patients with KS and without KS in the Centre, 2004-2017.**

| Characteristics | Presence of Kaposi's Sarcoma | | | | |
|---|---|---|---|---|---|
| | No (%) | Yes (%) | Total (%) | $X^2$ | P |
| **Age group (years)** | | | | | |
| <20 | 93 (0.4) | 0 (0.0) | | 93 (0.4) | |
| 20-29 | 1268 (5.3) | 8 (2.9) | 1276 (5.2) | | |
| 30-39 | 8155 (33.8) | 95 (33.8) | 8250 (33.8) | | |
| 40-49 | 8822 (36.5) | 99 (35.2) | 8921 (36.5) | | |
| 50-59 | 4325 (17.9) | 58 (20.6) | 4383 (17.9) | | |
| ≥60 | 1486 (6.1) | 21 (7.5) | 1507 (6.2) | | |
| **Total** | **24149(98.8)** | **281(1.2)** | **24430(100.0)** | **6.223** | **0.285** |
| **Sex** | | | | | |
| Male | 8512 (34.7) | 93 (32.5) | 8605(34.6) | | |
| Female | 16042 (65.3) | 193 (67.5) | 16235(65.4) | | |
| **Total** | **24554 (98.8)** | **286 (1.2)** | **24840(100.0)** | **0.577** | **0.448** |
| **Education** | | | | | |
| No Formal Education | 4079 (17.1) | 46 (16.4) | 4125 (17.0) | | |
| Primary | 4772 (20.0) | 53 (18.9) | 4825 (24.1) | | |
| Secondary | 7552 (31.6) | 87 (31.1) | 7639 (31.6) | | |
| Tertiary | 7481 (31.3) | 94 (33.6) | 7575 (31.3) | | |
| **Total** | **23884 (98.8)** | **280 (1.2)** | **24164 (100.0)** | **0.695** | **0.874** |
| **Marital Status** | | | | | |
| Single | 5281 (22.1) | 57 (20.3) | 5338 (22.1) | | |
| Married | 13091(54.8) | 171 (61.1) | 13262 (54.9) | | |
| Widowed | 3593 (15.1) | 31 (11.1) | 3624 (15.0) | | |
| Divorced/separated | 1915 (8.0) | 21 (7.5) | 1936 (8.0) | | |
| **Total** | **23880 (98.8)** | **280 (1.2)** | **24160 (100.0)** | **4.690** | **0.196** |

where majority of the HIV infected persons in care were women.

Majority of those who developed KS in this study were young. This compares well with what was obtained in Abuja.[4] A similar study in Lagos found the modal age group for HIV patients with KS to be 40-49 years. In the era of HIV, KS tends to occur at a younger age and this is even more so for the female gender.[4] This pattern is distinct from the classical KS which occurs typically in the elderly. It is possible that other factors acting indirectly may also play a role in influencing KS outcome since, elsewhere, age had no effect on the risk of development of KS.[21]

The prevalence of KS appears to increase with increasing level of education. Although it was not statistically significant in this study, other studies found elevated prevalence of KS among those who have attained higher education, in occupations that are well paid and who travel widely.[22] It is believed that this increased prevalence is related to increased risk of exposure to sexually transmitted agents. Unexpectedly, KS was more commonly found among men married to one wife than men married to multiple spouses in that study.[22] It is possible that the men's marital status masked their actual sexual practices as many men in monogamous marriages have many other sexual partners outside marriage. In this study almost two thirds of the KS patients were currently married but this could be due to the fact that more than half of the study population were currently married. There was however no statistically significant relationship between marital status and development of KS.

In this study, CD4+ cell counts did not affect the KS outcome even though the median CD4+ cell counts both at baseline and at the end of the study period were higher for those without KS compared to those with KS. Patients who had baseline viral load greater than 1000 copies/ml were three times more likely to develop KS than those with baseline viral load of 1000 copies/ml or less. The median baseline viral load was also higher among those with KS compared with those without KS. These finding are in agreement with those from a number of studies which have linked the immunologic and virologic status of patients to their risk of development of KS.[15,16] In an earlier study in Jos Nigeria, the median CD4+ cell count was significantly higher among those who did not have KS compared with those who had. However against expectation, the median viral load was higher among those without KS compared to those with KS in that study.[21] It has been reported that as part of the immune reconstitution syndrome, some patients with low viral load and high CD4+ cell count could develop KS.[2] In Lagos Nigeria, HAART and high CD4+ cell count

**Table 3. Kaposi's sarcoma and HIV characteristics of patients in care in the Centre 2004-2017**

| HIV Characteristics | Kaposi's sarcoma | | | |
|---|---|---|---|---|
| | No (%) | Yes (%) | Total | P |
| **CD4 cells/ml** | | | | |
| **Baseline** | | | | |
| ≤500 | 21988 (98.8) | 255 (1.2) | 22243 | |
| >500 | 2045 (98.7) | 26 (1.3) | 2071 | 0.683 |
| **Total** | **24033 (98.8)** | **281 (1.2)** | **24314** | |
| Median | 171(79-308) | 98 (50-200) | | |
| **End of study** | | | | |
| ≤500 | 16492 (98.9) | 187 (1.1) | 16679 | |
| >500 | 7078 (98.7) | 90 (1.3) | 7168 | 0.371 |
| **Total** | **23570 (98.8)** | **277 (1.2)** | **23847** | |
| Median | 351 (168-545) | 217 (95-468) | | |
| **Viral load copies/ml** | | | | |
| **Baseline** | | | | |
| ≤1000 | 6897 (99.5) | 35 (0.5) | 6932 | |
| >1000 | 14482 (98.4) | 230 (1.6) | 14712 | < 0.001 |
| **Total** | **21379 (98.8)** | **265 (1.2)** | **21644** | |
| Median | 35977 (4736-155877) | 55825 (8493-176775) | | |
| **End of study** | | | | |
| ≤1000 | 9405 (98.4) | 152 (1.6) | 9557 | |
| >1000 | 5391 (98.3) | 93 (1.7) | 5484 | 0.624 |
| **Total** | **14796 (98.4)** | **245 (1.6)** | **15041** | |
| Median | 200 (0-9878) | 200 (0-24809) | | |

**Table 4. Duration on HAART and development of Kaposi sarcoma among patients in care in the Centre 2004-2017**

| | Kaposi's sarcoma | | |
|---|---|---|---|
| **Duration on HAART (years)** | **Yes (%)** | **No (%)** | **Total** |
| <10 | 217 (1.1) | 20932 (98.9) | 21149 (100.0) |
| ≥10 | 48 (3.5) | 1342 (96.5) | 1390 (100.0) |
| **Total** | **265 (1.2)** | **22274 (98.8)** | **22539 (100.0)** |

$X^2 = 66.13$, df =1, P < 0.001

were found to selectively reduce the risk of malignancy especially KS and Non-Hodgkin's Lymphoma (NHL).[14] Also in Kenya, those HIV infected persons with CD4+ cell counts less than 350 cells/ml had 7 times increased risk of KS compared to those with higher CD4+ cell counts.[23] In the USA and South America the risk of KS was found to be inversely related to the CD4+ cell count.[5,24] In another study, low CD4+ cell count, not being on HAART and non-cancer AIDS diagnosis like tuberculosis were predictors of ADCs.[25]

The use of HAART as such did not influence the KS outcome in this study contrary to the finding elsewhere in which the use of HAART was protective against KS.[26] However, patients who had been on HAART for less than ten years were less likely to develop KS than those who had received HAART for ten years or more. The duration on HAART was probably a proxy indicator of the duration of HIV infection. In those reported to be on HAART for longer periods, the long duration of immunosuppression associated with poor adherence to HAART probably increased their risk of development of KS. This is more so that the optimum benefits of HAART are only obtained with adherence of at least 80%,[27,28] and patients with imperfect adherence to HAART are 20 times more likely to develop KS than those with perfect adherence.[23] Unfortunately, it is not known at what point in their illness the patients developed KS; before or after the commencement of HAART. Therefore, the rela-

**Table 5. Relationship between Tuberculosis Infection and Kaposi's sarcoma among patients in care in the Centre, 2004-2017**

| | Kaposi Sarcoma | | |
|---|---|---|---|
| Tuberculosis | Absent (%) | Present (%) | Total (%) |
| Absent | 24549(99.2) | 196(0.8) | 24745(100.0) |
| Present | 5(6.3) | 75(93.7) | 80(100.0) |
| **Total** | **24554(98.9)** | **271(1.1)** | **24825(100.0)** |

$X^2$= 6381.87, df = 1, p < 0.001

**Table 6. Predictors of Kaposi's sarcoma among HIV infected persons attending clinic in the Centre, 2004-2017**

| Parameter | p | Odds Ratio (OR) | 95 % *C.I of OR |
|---|---|---|---|
| **Age** | | | |
| 21-30 | | | |
| 30-39 | 0.876 | 1.10 | 0.32-3.78 |
| 40-49 | 0.847 | 0.92 | 0.38-2.24 |
| 50-59 | 0.712 | 0.85 | 0.36-2.01 |
| ≥50 | 0.408 | 1.44 | 0.61-3.42 |
| **Gender** | | | |
| Female | | 1 | |
| Male | 0.024 | 0.56 | 0.33-0.93 |
| **Education** | | | |
| No formal education | | 1 | |
| Primary | 0.368 | 0.75 | 0.39-1.41 |
| Secondary | 0.026 | 0.47 | 0.24-0.91 |
| Tertiary | 0.372 | 0.78 | 0.46-1.34 |
| **Alcohol use** | | | |
| No | | 1 | |
| Yes | 0.107 | 0.68 | 0.42-1.09 |
| **HBSAg** | | | |
| Negative | | 1 | |
| Positive | 0.561 | 0.87 | 0.55-1.38 |
| HCV antibody | | | |
| Negative | | 1 | |
| Positive | 0.631 | 0.88 | 0.51-1.51 |
| **Baseline viral load (copies/ml)** | | | |
| ≤1000 | | 1 | |
| >1000 | 0.004 | 3.13 | 2.19-4.47 |
| **On HAART** | | | |
| No | | 1 | |
| Yes | 0.841 | 1.05 | 0.65-1.69 |
| **Duration on HAART** | | | |
| ≥10 years | 0.000 | 1 | 0.19-0.50 |
| <10 years | | 0.31 | |

*C.I: Confidence interval

tionship between duration on HAART and development of KS should be interpreted in terms of the duration of HIV infection.

In a logistic regression model, this factor was found to

be a strong predictor of KS development as those who had been on HAART for ten years or more had their risk of development of KS increased by 70%. Prolonged exposure to HIV and poor adherence to HAART allow prolonged immunosuppression with increased risk of KS.[29–31] The public health and policy implication of this finding is that the current 'test and treat' policy of government is not sufficient until strict adherence is ensured among HIV infected patients on HAART. This will also minimize the potential for drug resistance and treatment failure.

One of the biggest problems in this study was the missing data. A possible way forward in this situation would be a cohort multicenter study, which could allow for the determination of incidence per person years of follow up.

## CONCLUSIONS

This study found a low prevalence of Kaposi's sarcoma among HIV infected adults receiving care at the Treatment and Care Centre in Jos, Nigeria. Patients on HAART for more than ten years had up to 70% higher risk of development of KS than those on HAART for less than ten years. Other factors found to be predictors of KS development in these patients included the female gender, low education and high baseline viral load. Measures to improve adherence to HAART among HIV patients as well as female education and empowerment are recommended.

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AUTHORSHIP CONTRIBUTIONS

Concept and study design - DJC, CMP and PFH. Data collection – DJC. Data analysis and interpretation – DJC, CMP and PFH. Drafting of article – DJC. Revising the article critically for important intellectual content – AO and ATJ. All the authors revised and approved the final version of the article for publication. DJC takes overall responsibility for questions and integrity of the work.

COMPETING INTERESTS

The authors completed the Unified Competing Interest form at www.icmje.org/coi_disclosure.pdf (available upon request from the corresponding author) and declare no conflicts of interest.

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