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Prevalence of osteoporosis and osteopenia in a cohort of HIV positive women with a history of treated neoplasms

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Abstract

Introduction: Higher prevalence of osteopenia and osteoporosis in HIV positive patients compared to non-infected population has been recognized. However, cancer patients have a higher risk of bone loss and fractures that is multifactorial. The aim of the study was to describe the prevalence of osteopenia and osteoporosis in HIV positive women with history of treated cancer.

Methods: Between January 2018 and December 2019, women aged >40 years, HIV+ with a history of cancer diagnosis, who attended the AIDS Cancer Clinic at Instituto Nacional de Cancerología, Mexico City, and who had a dual X-ray absorptiometry performed during the study period were included. Two control groups (CG)—HIV negative women with history of cancer (CG1) and non-HIV, non-cancer women (CG2)—were matched by age 1:1.

Results: Forty-eight patients in each group were included; the mean age was 51.1 ± 8.1 years. Osteopenia was found in femoral neck in 54.2% (HIV+), 37.5% (CG1), and 27.1% (CG2), $p = 0.02$; in spine was 35.7%, 47.9%, and 31.2%, respectively, $p = 0.442$. Osteoporosis in femoral neck was documented in 12.5%, 2.1%, and 0% in HIV+, CG1, and CG2 ($p = 0.03$), and in the spine was 47.9%, 16.7%, and 14.6%, respectively ($p = 0.002$).

Conclusion: HIV patients with a history of treated cancer have a much higher prevalence of osteoporosis when compared with same-aged HIV-uninfected women with and without cancer. It is necessary to monitor Bone Mineral Density periodically, and all patients should be encouraged to make lifestyle changes, such as avoid tobacco and alcohol, and to increase exercising.

Keywords

Osteoporosis, osteopenia, cancer, HIV, antiretroviral therapy

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Introduction

Osteoporosis is defined as a systemic skeletal disorder is characterized by low Bone Mineral Density (BMD) and microarchitectural deterioration of bone tissue, with a consequent increase in bone fragility and pathological fractures, which has a negative impact on morbidity, quality of life, and mortality.[1] BMD decreases after the age of 30 years in both women and men, with a much more rapid rate of bone loss in women during perimenopause and early menopause.[2] Smoking and low body mass index (BMI) have been associated with lower BMD.[3]

The prevalence of osteopenia and osteoporosis in HIV positive patients has been associated with different factors, including different combined antiretroviral therapy (cART) regimes, and chronic immune activation.[1] Over the past two decades, there has been an increase in life expectancy in HIV population with access to cART, which has led to an increase in chronic degenerative diseases, such as osteoporosis.[4]

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However, the patients with a diagnosis of cancer have also a higher risk of bone loss and fractures, due to a combination of factors including their underlying malignancy and therapeutic regimens, particularly chemotherapies, which directly or indirectly affect bone cells.[5,6] The aim of this study was to describe the prevalence of osteopenia and osteoporosis in HIV positive women with a history of treated neoplasms and to compare this population with two control groups: women with cancer non-HIV infected, and women non-HIV and non-cancer diagnosis.

Methods

This was a retrospective study performed from January 2018 to December 2019, in HIV-infected women who attended the HIV/AIDS Cancer Clinic at the Instituto Nacional de Cancerología in Mexico City, Mexico.

The HIV/Cancer Clinic was founded in 1990, date from which 1365 patients have been treated. During the study period, there were 455 patients seen annually, distributed in 364 men (80%) and 91 (20%) women. There was not performed a power calculation for estimation of sample size; it was determined by convenience.

Inclusion criteria

HIV-infected women over the age of 40 years, with history of treated cancer, in whom a dual X-ray absorptiometry (DXA) was performed within the last 12 months. A DXA is performed routinely as part of follow-up visits in patients older than 40 years or in those who have received more than 3 years of cART.

Exclusion criteria

Patients with an anti-osteoporotic treatment drug (e.g. bisphosphonates), hip prosthesis, and/or therapy with drugs that cause low BMD (e.g. prednisone), and patients who received pelvic radiotherapy were excluded. Two control groups (CG) were matched 1:1 by age: CG1 included women with a history of cancer, non-HIV infected and CG2 included volunteers' women ⩾40 years old non-HIV infected without cancer, with a DXA performed during the study period.

The informed consent for patients was waived because the studies carried out were part of those usually requested within the screening tests in case group and CG1. Informed consent for CG2 was obtained. The study was approved by Ethics Committee (REF/INCAN/CI/0835/2019).

Demographic and clinical data were recorded from clinical records including age, high risk of falls, history of low impact fracture, comorbidities (including renal disease and diabetes mellitus), date of HIV diagnosis, date of cART initiation, type of cART (classified in: Non-Nucleoside Reverse Transcriptase Inhibitors—NNRTIs, Protease Inhibitors—PIs, and Tenofovir Disoproxil Fumarate—TDF), CD4+ cell count and HIV viral load (VL) at nadir and at DXA study, co-infections including hepatitis B virus (HBV) and hepatitis C virus (HCV) status, date of cancer diagnosis, type of neoplasm, and treatment received for cancer (chemotherapy, radiotherapy, surgery and/or biological treatment). Risk factors included were: smoking habits, alcohol use, drug abuse, BMI, physical activity (⩾150 min of moderate-intensity aerobic physical activity or ⩾75 min of vigorous-intensity aerobic physical activity throughout the week, in the past month),[7] menopausal status and time since last menses, hormone replacement therapy, number of pregnancies, and a history of fractures.

Menopause was defined in women at least 50 years with an intact uterus, who has either: at least 6 months of spontaneous amenorrhea with follicle-stimulating hormone (FSH) of ⩾40 mIU/L, or women aged ⩾55 years who have 12 consecutive months of amenorrhea. It was defined in women <50 years old, with chemical menopause (induced by chemotherapy) or post-hysterectomy who have the FSH ⩾40 mIU/L.

BMD was measured by DXA (Hologic X-ray Bone Densitometer Discovery™ QDR™; Hologic, Inc., Waltham, MA, USA). (The APEX version 4.5.3 software programmed for Hispanic race was used.) Quantitative measurement of bone mass referring to the amount of mineral matter (calcium) in grams per square centimeter of bones was conducted at the following sites: lumbar spine (L1 to L4), total hip, and both femoral neck.

According to the World Health Organization (WHO), BMD is expressed in relation to a mean reference for young adult female Caucasians (*T*-score) and grouped accordingly as either normal, osteopenia, or osteoporosis. Criteria established by the WHO were employed as follows:[8]

- Osteoporosis: BMD >−2.5 standard deviations (SDs) of the *T*-score.
- Osteopenia: an intermediate category of bone loss defined as a *T*-score between −1 and −2.5 SD.
- Normal BMD: *T*-score between 1.0 and −0.9 SD at all three sites.

Statistical analysis

Student's *t*-test, analysis of variance (ANOVA), or the chi-square test was utilized to compare mean values for continuous and categorical variables, respectively, among the groups. Variables with a *p*-value of <0.1 were included in the multivariate logistic regression analysis. Exposure to different antiretroviral drugs (ARVs) was analyzed in study participants who received ⩾3 years. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated. *p*-values of ⩽0.05 were considered statistically significant. Data were analyzed employing STATA (version 14) software.

Results

During the study period, there were 91 HIV-infected women, attending periodically at hospital: 29 were younger than

Table 1. Baseline demographic and clinical characteristics in HIV positive patients with cancer, HIV-uninfected patients with cancer, and non-HIV non-cancer women.

Characteristics, *n* (%)	Total (*N* = 144)	HIV positive (*n* = 48)	Patients with cancer, non-HIV (*n* = 48)	*p*[a]	Non-HIV, non-cancer group (*n* = 48)	*p*[b]
Age[c] (years)	51.1 ± 8.1	51.5 ± 7.8	49.9 ± 8.1	0.331	51.9 ± 8.5	0.658
BMI[d,e] (kg/m^2)	26.4 (23.4–29.3)	23.9 (22.9–36.3)	27.5 (25.3–30.9)	<0.001	27.5 (25.3–30.9)	<0.001
Diabetes	22 (15.3)	9 (18.8)	3 (6.25)	0.120	10 (20.8)	0.326
High blood pressure (HBP)	20 (15.3)	8 (16.7)	7 (14.6)	1	5 (10.4)	0.609
Hypothyroidism	18 (12.5)	5 (10.4)	6 (12.5)	1	7 (14.6)	0.790
Physical activity[f]	35 (24.3)	5 (10.4)	2 (4.2)	0.435	28 (58.3)	0.006
Smoking	5 (3.5)	1 (2.1)	2 (4.2)	1	2 (4.2)	0.519
Median number of pregnancies[e]	2 (1–3)	3 (2–4)	2 (0–3)	0.203	2 (1–3)	0.05
Life history of non-pathologic fractures	17 (11.8)	5 (10.4)	4 (8.3)	1	8 (16.6)	0.791
Pathologic fractures	1 (0.7)	0	0	–	1 (2.1)	–
Menopause	108 (75)	38 (79.2)	45 (93.8)	0.07	25 (52.1)	0.54
Time since last menses (years)[c]	9.5 ± 7.7	9.6 ± 6.9	7.2 ± 5.1	0.08	14.7 ± 11.2	<0.0001
Time of cancer diagnosis (years)[e]	6.4 (3.2–14.7)	12.6 (3.6–16.4)	5.3 (2.3–8.5)	0.0006	–	–
Hormone replacement therapy	7 (21.9)	4 (8.3)	0	n/a	3 (6.3)	0.686
Radiotherapy (non-pelvic)	38 (26.4)	10 (20.9)	28 (58.3)	0.0003	0	0.697

n/a: not available.
[a]*p*-value comparing HIV positive patients versus HIV negative patients with cancer.
[b]*p*-value comparing non-infected patients with cancer versus non-infected patients, without cancer.
[c]Mean (standard deviation (SD)).
[d]BMI: body mass index.
[e]Median (interquartile range).
[f]Physical activity was documented if patients performed ⩾150 min of moderate-intensity aerobic physical activity or ⩾75 min of vigorous-intensity aerobic physical activity throughout the week, in the past month.

40 years, 10 had received zoledronic acid during the previous year, 1 had a hip prosthesis, and 3 were receiving chronic steroids: 48 women were included in HIV-infected group and 48 subjects in each CG1 and CG2.

Mean age for the whole group was 51.1 ± 8.1 years; there were no statistically significant differences between groups. On analyzing risk factors for osteoporosis, patients in both control groups had higher median BMI when compared with HIV positive patients 23.8 (interquartile range (IQR) = 22.8–26.2) versus CG1 27.5 (IQR = 25.3–30.9) versus CG2 27.6 (IQR = 24.1–29.7), $p < 0.0001$. Physical activity was higher in CG2 ($n = 29$), but rare in CG1 ($n = 2$) and in patients with HIV ($n = 5$) ($p < 0.001$). Smoking and alcohol abuse were scarce in all patients (Table 1).

Menopause was not different in the three groups: 38 women in HIV+ (79.2%); 45 in CG1 (93.8%), and 24 in CG2 (52.1%), $p = 0.928$. Four patients in CG1 (8.3%) were on hormone replacement therapy, none in HIV group, and three (6.3%) in CG2. Other clinical and demographic characteristics are shown in Table 1.

On analyzing HIV-infected patients, the most frequent neoplasms were: cervical ($n = 23$, 47.9%), non-Hodgkin lymphoma ($n = 7$, 14.6%), and breast cancer ($n = 6$, 12.5%). Median time from cancer diagnosis was 12.6 years (IQR = 3.6–16.4). There were 11 patients (22.9%) with two different neoplasms (genital cancer—vulvar, vagina, cervix, or anus in at least two sites ($n = 4$), genital cancer combined with lymphoma ($n = 5$), with thyroids cancer ($n = 1$), and with basocellular cancer ($n = 1$)). Two patients (4.2%) had three different neoplasms (one with breast, vulva, and lymphoma, and the other with vulva, cervix, and anus).

In CG1, the most frequent neoplasms were: breast ($n = 28$, 58.3%), ovarian ($n = 8$, 16.7%), and cervical ($n = 3$, 6.3%). Median years from cancer diagnosis were 5.3 years (IQR = 2.3–8.5). Five patients (10.4%) had two different neoplasms (breast and thyroid, breast and colon, breast and lymphoma, breast and hypophysis tumor, and ovarian with endometrium).

Comparing DXA, there were significantly lower parameters in patients with HIV+ versus both control groups. Median BMD in femoral necks was in HIV-infected women: −1.4, CG1: −0.5, and CG2: −0.5 ($p = 0.0002$). Median BMD in spine was −2.4 in HIV-infected group, −1.3 in CG1, and −0.75 in CG2 ($p < 0.0001$). On analyzing osteoporosis in the three groups, there were significantly more cases in HIV patients ($n = 23$, 47.9%) compared with both control groups ($n = 15$, 15.6%; $p < 0.001$). Osteopenia was documented in 17 patients in the HIV+ group (35.4%), in 23 patients from CG1 (47.9%), and in 15 patients (31.3%) from CG2 (p = ns). Only eight HIV patients (16.6%) had a normal DXA, compared to 17 (35.4%) in CG1% and 22 (45.8%) in CG2. Data are presented in Table 2.

Mean time from HIV diagnosis was 16.8 ± 7.9 years. The median count at baseline of CD4+ cell count and HIV VL was 160 cells/mm^3 (IQR = 52–300) and 23,935 copies/mL

Table 2. DXA measurements in femoral neck and spine, classified as osteopenia or osteoporosis in HIV+ patients, HIV− patients with cancer, and in HIV− patients' non-cancer.

Measuring site	DXA result	HIV positive patients with cancer ($n=48$)	HIV negative with cancer diagnosis ($n=48$)	p[a]	HIV negative without cancer ($n=48$)	p[b]
Femoral neck	DXA value[c]	−1.4 (−1.9 to −0.7)	−0.5 (−1.4 to 0.17)	0.001	−0.5 (−1.25 to 0.15)	0.597
	Osteopenia[d]	26 (54.2)	18 (37.5)	0.02	13 (27.1%)	0.243
	Osteoporosis[d]	6 (12.5)	1 (2.1)	0.03	0	n/a
Spine	DXA value[c]	−2.4 (−2.9 to −1.55)	−1.3 (−2.2 to −0.6)	<0.001	−0.75 (−0.15 to −2)	0.119
	Osteopenia[d]	17 (35.4)	23 (47.9)	0.442	15 (31.25)	0.06
	Osteoporosis[d]	23 (47.9)	8 (16.7)	0.002	7 (14.6)	0.380

DXA: dual X-ray absorptiometry; n/a: not available.
[a]p-value comparing HIV positive patients versus HIV negative patients with cancer.
[b]p-value comparing non-infected patients with cancer versus non-infected patients, without cancer.
[c]Median (interquartile range).
[d]N (%).

Table 3. HIV positive patients and related factors associated with low Bone Mineral Density (BMD).

Characteristics	Total HIV positive patients ($N=48$)	Normal BMD $n=8$ (%)	Low BMD $n=40$ (%)	p
Time since HIV diagnosis, years[a]	16.7 ± 8	16.1 ± 7.5	16.9 ± 8.1	0.58
Baseline CD4+ count, cells/mL[b]	160 (52–300)	338.5 (241–397)	123 (55–290)	0.442
Current CD4+count, cells/mL[a]	573.7 ± 317.1	541 ± 233.1	579.2 ± 331.3	0.61
Baseline HIV viral load, copies/mL[b,h]	23,935 (1076–235,301)	10,272 (463–23,493)	40,766 (1690–252,162)	0.39
Current undetectable HIV-RNA	46 (96)	7 (87.5)	40 (97.5)	0.27
History of AIDS-defining event	11 (22.3)	2 (25)	9 (22.5)	0.65
Duration of antiretroviral therapy, years[a]	12.5 ± 6.07	12.1 ± 5.5	12.7 ± 6.4	0.8
TDF[c]	42 (87.5)	7 (87.5)	35 (87.5)	0.56
TDF exposure, years[a,c]	5.9 ± 3.9	6.8 ± 4.3	5.9 ± 3.9	0.81
PI[d]	26 (54)	6 (75)	20 (50)	0.36
PI exposure, years[a,d]	12.3 ± 5.6	14.8 ± 2.2	11.6 ± 6.1	0.45
NNRTI[e]	35 (73)	2 (25)	33 (82.5)	0.003
NNRTI exposure, years[a,e]	7.31 ± 4.6	6.4 ± 4.8	7.5 ± 4.6	0.46
NRTI[f]	48 (100)	8 (100)	40 (100)	–
NRTI exposure, years[a,f]	11.7 ± 5.7	10.4 ± 5	12.2 ± 6	0.65
INSTI[g]	18 (38)	4 (50)	14 (35)	0.42
INSTI exposure, years[a,g]	3.1 ± 2.3	3.5 ± 3.8	3 ± 1.8	0.73

[a]Mean (standard deviation (SD)).
[b]Median (interquartile range (IQR)).
[c]TDF: Tenofovir Disoproxil Fumarate.
[d]PI: Protease Inhibitor.
[e]NNRTI: Non-Nucleoside Reverse Transcriptase Inhibitor.
[f]NRTI: Nucleoside Reverse Transcriptase Inhibitor.
[g]INSTI: Integrase Strand Transfer Inhibitor.
[h]Viral load was documented in 24 patients: 5 with normal BMD and 19 with low BMD.

(IQR = 1076–235,301), respectively. At the time of DXA, all patients were receiving cART; median CD4 count was 544 cells (IQR = 371–648) and the HIV-VL was undetectable in 46 patients (95.8%); the remaining two patients had virological failure related with poor cART adherence, with HIV VL of 1084 and 24,623 copies/mL. Other clinical characteristics including cART are shown in Table 3. A subanalysis comparing different ARVs directly related with low BMD (NNRTIs, PIs, and TDF) did not show statistically differences with the DXA measurements. Data are presented in Table 4.

The univariate analysis showed as risk factors for osteoporosis were BMI $<24\,kg/m^2$, longer time from menopause, non-hormonal therapy replacement, having received radiotherapy, history of cancer, and HIV infection. In the multivariate analysis, BMI $<24\,kg/m^2$ (OR = 6.03, 95% CI = 2.07–17.58, $p=0.001$), longer time from menopause (OR = 1.98, 95% CI = 1.01–1.16, $p=0.03$), and HIV infection

Table 4. Patients with HIV+ who received ⩾3 years of the antiretroviral drug.

Measurement site	DXA result	HIV with NNRTI[a] (*n* = 28)	HIV with PI[b] (*n* = 23)	HIV with TDF[c] (*n* = 33)
Femoral neck	DXA value[d]	−1.52 (−2.2 to −1.1)	−1.4 (−1.7 to −0.35)	−1.3 (−2 to −0.45)
	Osteopenia	15 (53.6)	12 (52.2)	17 (51.5)
	Osteoporosis	5 (17.9)	2 (8.7)	4 (12.1)
Spine	DXA value[d]	−2.85 (−3.4 to −1.7)	−2.3 (−2.8 to −0.8)	−2.1 (−3.1 to −1.2)
	Osteopenia	12 (42.8)	7 (30.4)	12 (36.4)
	Osteoporosis	14 (50)	10 (43.5)	14 (42.4)

DXA: dual X-ray absorptiometry.
[a]NNRTI: Non-Nucleoside Reverse Transcriptase Inhibitor.
[b]PI: Protease Inhibitor.
[c]TDF: Tenofovir Disoproxil Fumarate.
[d]Median (interquartile range).

(OR = 4.35, 95% CI = 1.14–16.64, $p = 0.03$) were associated factors.

Discussion

This study describes the prevalence of osteopenia and osteoporosis in a cohort of HIV-infected women with a history of cancer and compared with two different groups, one of women with cancer without HIV, and the other, women without cancer who were HIV negative. We found marked discordances in BMD scores at lumbar spine and femoral neck in these three groups, being much lower measurements in HIV women.

Reduced spine and hip bone density (BMD) in HIV-infected individuals have been demonstrated in a number of cross-sectional studies. A threefold increase in the prevalence of osteoporosis was reported in a meta-analysis, with a significantly higher prevalence in infected patients receiving ART than in those not treated.[9] A study performed in 33 sites on 6 continents reported 1.9% of osteoporosis and 35.1% of osteopenia in naïve HIV patients.[10] In Belgium, a study analyzed the prevalence of osteopenia/osteoporosis in naïve patients and reported 13.5% at the spine and 21.6% at the femoral neck; in patients with cART (without PI), it was reported in 16% and 32% respectively, and in those who received PI, it was 25.9% and 33.3% in both measurements.[11] In Brazil, the prevalence of osteoporosis in HIV-infected patients versus without HIV was 14.6% and 4.6% in spine, and 5.6% versus 3.3% in the femoral neck measurements.[5] In this study, the prevalence of osteoporosis was (12.5%) in femoral neck and 47.9% in spine in patients with HIV, compared with non-HIV-infected patients (1% in femoral neck and 15.6% in spine).

Likewise, the follow-up of our patients was double that of other studies (16.9 compared range 2–10 years), and also the patients were older in our series (51 years), compared with other studies (range 34–48 years). It is also an important finding in this study, the higher percentage for osteoporosis in spine compared with femoral neck for the three groups (Δ 35.4% in the HIV group, Δ 14.6% in the non-HIV, cancer group, and Δ 14.6% in the non-HIV, non-cancer group). Previous studies have reported a difference between those two measures.[11] Other report that included middle-aged, HIV positive, and HIV negative women did not find these differences on comparing these two measurements.[12]

There are scarce reports describing the prevalence of osteoporosis in cancer patients. In studies performed in women with breast cancer, the prevalence of osteopenia and osteoporosis was 60.1% and 22.2%, respectively.[10] In this series, women non-HIV with history of cancer, we found osteopenia in 47.9% and osteoporosis in 16.7%.

In patients with HIV, many factors are related in the pathogenesis of the decrease in BDM: (1) increased levels of inflammatory cytokines present in chronic HIV infection that may increase bone turnover through osteoclast stimulation; (2) other pathologies can contribute to chronic inflammation, such as co-infection with HCV or HBV, renal disease, and diabetes; (3) factors related with HIV, such as low body weight, estrogen depletion, malabsorption, tobacco use, low CD4+ T cell count, duration of HIV infection, lipodystrophy, low levels of vitamin D, insulin resistance, and hyperlactatemia; and (4) antiretroviral treatment, which is generally associated with bone loss (2%–6% over the first 2 years of therapy).[3,4,7,13–18] Mexican HIV-infected women are facing missing opportunities in medical care such as inadequate screening strategies, in conjunction with fewer opportunities to receive preventive or therapeutic intervention for BMD loss.[19]

Considering ARV drugs, TDF and PIs, particularly Lopinavir, have been linked to low BMD.[3,16,18] In this study, we did not found a significant relation with low BMD in patients who received more than 3 years of NNRTI (58.3%), PI (47.9%), and TDF (68.8%).

The main goal of preventing osteoporosis is to decrease the risk of fractures (particularly, hip fractures).[13] In this study, no differences were found between the number of pathological and non-pathological fractures in the three groups.

The limitations in this study are related to the observational studies, especially a significant selection bias. Also,

control patients with history of cancer could not be matched by neoplasms, because the cancer types of the HIV-infected population are different from that occurring in the general population.[18] Finally, there was not performed a power calculation for estimation of sample size.

In patients with HIV and cancer, the mean time since cancer diagnosis was 12.6 years compared with non-HIV cancer women (5.3 years), indicating a clear survival bias. This may be a bias, but it can also be an advantage, since HIV-infected women are group of women, who have been followed since HIV diagnosis for several years and all of them on cART for a long period. The main strength of this study lies the comparison of HIV patients with the two control groups which allows us to have a view of the prevalence of low bone loss in these three populations.

Conclusion

HIV-infected women with a history of treated cancer have a higher prevalence of osteoporosis when compared with same-aged HIV-uninfected women with and without cancer. It is important to perform a BMD in HIV-infected women older than 40 years and/or who have received cART for ⩾3 years.

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Author contributions

P.C.-J. contributed to the study concept and design. P.C.-J. and J.L.V. contributed to the analysis and interpretation. C.E.R.-D. contributed to the drafting of the manuscript. P.V.-F. contributed to the critical revision of the manuscript for important intellectual content.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical approval

This study was approved by the Ethics Committee (REF/INCAN/CI/0835/2019).

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Informed consent

The informed consent for patients was waived because the studies carried out were part of those usually requested within the screening tests in this group of patients. Informed consent of the control group without HIV or cancer was obtained.

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References

1. Brown TT and Qaqish RB. Antiretroviral therapy and the prevalence of osteopenia and osteoporosis: a meta-analytic review. *AIDS* 2006; 20: 2165–2174.
2. Carvalho EH, Gelenske T, Bandeira F, et al. Bone mineral density in HIV-infected women taking antiretroviral therapy: a systematic review. *Arq Bras Endocrinol Metabol* 2010; 54(2): 133–142.
3. Hamill MM, Pettifor JM, Ward KA, et al. Changes in bone mineral density, body composition, Vitamin D status, and mineral metabolism in urban HIV-positive South African women over 12 months. *J Bone Miner Res* 2017; 32(8): 1615–1624.
4. Mata-Marín JA, Arroyo-Anduiza CI, Berrospe-Silva MLÁ, et al. Mexican patients with HIV have a high prevalence of vertebral fractures. *Infect Dis Rep* 2018; 10: 7409.
5. Gomes DC, Valadares ALR, Amaral E, et al. Association between HIV infection and bone mineral density in climacteric women. *Arch Osteoporos* 2015; 10: 33.
6. Anastos K, Lu D, Shi O, et al. The association of bone mineral density with HIV infection and antiretroviral treatment in women. *Antivir Ther* 2007; 12(7): 1049–1058.
7. OMS definition: Physical activity, 2020, https://www.who.int/news-room/fact-sheets/detail/physical-activity
8. Kruger MJ and Nell TA. Bone mineral density in people living with HIV: a narrative review of the literature. *AIDS Res Ther* 2017; 14: 35.
9. Compston J. HIV infection and bone disease. *J Intern Med* 2016; 280: 350–358.
10. Carr A, Grund B, Neuhaus J, et al. Prevalence of and risk factors for low bone mineral density in untreated HIV infection: a substudy of the INSIGHT Strategic Timing of AntiRetroviral Treatment (START) trial. *HIV Med* 2015; 16(Suppl. 1): 137–146.
11. Libois A, Clumeck N, Kabeya K, et al. Risk factors of osteopenia in HIV-infected women: no role of antiretroviral therapy. *Maturitas* 2010; 65(1): 51–54.
12. Finnerty F, Walker-Bone K and Tariq S. Osteoporosis in postmenopausal women living with HIV. *Maturitas* 2017; 95: 50–54.
13. Sharma A, Cohen HW, Freeman R, et al. Prospective evaluation of bone mineral density among middle-aged HIV-infected and uninfected women: association between methadone use and bone loss. *Maturitas* 2011; 70(3): 295–301.
14. Bruyère O, Bergmann P, Cavalier E, et al. Skeletal health in breast cancer survivors. *Maturitas* 2017; 105: 78–82.
15. D'Oronzo S, Stucci S, Tucci M, et al. Cancer Treatment Induced Bone Loss (CTIBL): pathogenesis and clinical implications. *Cancer Treat Rev* 2015; 41(9): 798–808.
16. Drake MT. Osteoporosis and cancer. *Curr Osteoporos Rep* 2013; 11: 163–170.
17. Johansson H, Kanis JA, Odén A, et al. Impact of femoral neck and lumbar spine BMD discordances on FRAX probabilities in women: a meta-analysis of international cohorts. *Calcif Tissue Int* 2014; 95(5): 428–435.
18. Cornejo-Juárez P, Cavildo-Jerónimo D and Volkow-Fernández P. Non-AIDS defining cancer (NADC) among HIV-infected patients at an oncology tertiary-care center in Mexico. *AIDS Res Ther* 2018; 15: 16.
19. Martin-Onraët A, Volkow-Fernández P, Álvarez-Wyssmann V, et al. Late diagnosis due to missed opportunities and inadequate screening strategies in HIV infected Mexican women. *AIDS Behav* 2017; 21(2): 505–514.