

ORIGINAL ARTICLE

Hodgkin and non-Hodgkin lymphomas in the post-antiretroviral therapy era according to HIV virological suppression

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Abstract

Objectives: Lymphomas remain among the most frequent HIV-associated malignancies, with risk persisting despite effective virological control.

This study describes the clinical, epidemiological and prognostic characteristics of lymphomas (both Hodgkin [HL] and non-Hodgkin [NHL]) in people living with HIV (PLWH), according to virological suppression at diagnosis and assesses lymphoma-related mortality at 1 year and overall survival at 5 years.

Methods: We conducted a multicentre retrospective study including PLWH from the Spanish CoRIS cohort, who were diagnosed with lymphoma between 2004 and 2022 and had HIV viral load data at diagnosis. Virological suppression was defined as HIV-1 RNA <100 copies/mL within the 6 months prior to and 1 month following lymphoma diagnosis.

Results: Among 18 573 PLWH included in CoRIS, 291 developed lymphoma, with virological data available for 245 individuals, of whom 49 (20%) had virological suppression. People with detectable HIV viral load had lower CD4

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counts (median 180 cells/mm³ [IQR 80–330] vs. 371 cells/mm³ [IQR 202–583], $p < 0.001$) and more advanced Ann Arbor stage (stage III–IV: 82.0% vs. 64.1%, $p = 0.027$) compared with those with virological suppression. Despite this, they showed similar treatment response (complete remission: 67.1% vs. 76.2%) and relapse rates (16.3% vs. 18.4%, $p = 0.779$). Furthermore, virological suppression was not independently associated with 1-year lymphoma-specific or 5-year overall survival.

Conclusions: In PLWH diagnosed with lymphoma, prognosis is independent of HIV virological status at diagnosis. These findings support that lymphoma management in PLWH should be guided by standard oncological criteria, alongside antiretroviral therapy, regardless of HIV viral suppression status.

KEYWORDS

HIV-associated malignancies, lymphoma, survival, virological suppression

INTRODUCTION

The association between HIV infection and an increased risk of lymphoma has been recognized since the early years of the AIDS pandemic [1–3]. Much of the current knowledge regarding lymphoma in people living with HIV (PLWH) is derived from studies conducted before the widespread use of combination antiretroviral therapy (ART) [4, 5]. Compared with lymphomas occurring in the general population, HIV-associated lymphomas have historically been characterized by more aggressive clinical behaviour, a higher frequency of extranodal involvement, poorer prognosis and a stronger association with oncogenic viral co-infections, particularly Epstein–Barr virus (EBV) [6].

The introduction of ART in 1996, and the subsequent control of HIV replication, has profoundly modified the natural history of HIV infection. Nevertheless, the occurrence of lymphomas has not been eliminated, and cases continue to be diagnosed even among individuals with sustained virological suppression. At present, cancer represents one of the leading causes of morbidity and mortality in PLWH, with lymphoproliferative disorders remaining among the most frequent malignancies [7, 8].

Regarding non-Hodgkin lymphoma (NHL), its incidence markedly declined during the initial years following ART introduction and appears to have stabilized thereafter. Notably, certain lymphoma subtypes not strictly associated with advanced immunosuppression, such as Burkitt lymphoma, have shown a relative increase in incidence [9–12]. In parallel, Hodgkin lymphoma (HL), which was rarely observed in PLWH during the pre-ART era, has experienced a substantial rise in incidence since the advent of combination ART [13, 14].

Understanding the mechanisms underlying lymphomagenesis in PLWH with effective virological control remains a major challenge. In individuals with

suppressed HIV viral load, it is plausible that persistent immune dysregulation, chronic immune activation, systemic inflammation and residual immunosuppression play a central role [15, 16]. However, the precise pathophysiological pathways involved have not yet been fully elucidated. Although survival and response to oncological treatment in PLWH with lymphoma have improved considerably in recent years, outcomes remain inferior to those observed in the HIV-negative population [17, 18].

Consequently, there is a need to further characterize lymphomas in PLWH, including their epidemiology, clinical presentation, response to treatment and survival, beyond the sole consideration of HIV virological control. In this context, the aim of the present study was to describe and analyse the characteristics of HL and NHLs in PLWH according to HIV viral load status during the 6 months preceding lymphoma diagnosis. In addition, we aimed to explore overall and lymphoma-specific survival in relation to both HIV virological status and malignancy-related factors.

METHODS

Study design

This was a multicentre, retrospective, non-interventional study nested within the Spanish HIV Cohort (CoRIS). The CoRIS cohort is an open, prospective, multicentre cohort of adult subjects with confirmed HIV infection, naïve to ART at study entry, recruited in 48 centres from 14 to 17 Autonomous Regions in Spain from 2004 onwards. Data are organized and standardized following the HIV Cohorts Data Exchange Protocol (HICDEP) for data collection (details at <https://hicdep.org/>) and adhere to internal strict annual quality controls. The CoRIS database collects baseline and follow-up socio-demographic,

immunological and clinical data. Participants are followed periodically according to routine clinical practice. The CoRIS cohort has been described in detail elsewhere [19].

Study population and data collection

The study population comprised all CoRIS participants aged ≥ 18 years who were diagnosed with lymphoma—HL or NHL—between 1 January 2004 and 30 November 2022, and who had available HIV viral load data at the time of lymphoma diagnosis.

Participants were considered to have achieved virological suppression if they were on ART and had an HIV-1 RNA viral load < 100 copies/mL during the 6 months preceding lymphoma diagnosis and up to 1 month after diagnosis in all available measurements during the same period. Only participants who clearly fulfilled this definition based on the available data—including viral load determinations and information on initiation or continuation of ART—were included in the group with virological suppression. The threshold of 100 copies/mL was selected because lower limits of detection were not consistently available during the earlier years of the study period for technical reasons.

Clinical data were collected during the 6 months preceding lymphoma diagnosis and up to 1 month after diagnosis. People with unknown lymphoma histology were excluded from all survival analyses.

Overall survival was defined as the time from lymphoma diagnosis to death from any cause. Lymphoma-specific survival was defined as time from diagnosis to death attributable to lymphoma; individuals who died from other causes were censored at the time of death. For lymphoma-specific survival, participants were administratively censored at 12 months (1-year analyses) and for overall survival at 60 months (5-year analyses).

Statistical analysis

Continuous variables are presented as medians with interquartile ranges (25th–75th percentiles), while categorical variables are reported as frequencies and percentages. Data distribution was assessed using frequency histograms and the Kolmogorov–Smirnov test.

Comparisons between groups were performed using parametric tests (Student's *t*-test or analysis of variance) or non-parametric tests (Mann–Whitney *U*-test or Kruskal–Wallis test), as appropriate, based on data distribution and sample size. Associations between categorical variables were evaluated using Pearson's chi-square test or Fisher's exact test, as appropriate.

Overall and lymphoma-specific survival were estimated using the Kaplan–Meier method and compared between groups using the log-rank test. Multivariable Cox proportional hazards models were fitted to estimate adjusted hazard ratios, including virological suppression status, age (per 10-year increase), CD4 cell count at diagnosis, lymphoma type and duration of HIV infection (per 10-year increase) as covariates. Results are presented as hazard ratios (HRs) with corresponding 95% confidence intervals (CIs).

All statistical analyses were performed using IBM SPSS Statistics for Windows, version 25.0 (IBM Corp., Armonk, NY, USA) and R, version 4.5.3. All tests were two-sided, and *p*-values < 0.05 were considered statistically significant.

Sensitivity analysis

Sensitivity analyses were performed to evaluate the robustness of the results. First, the definition of virological suppression to viral load measurements was obtained prior to lymphoma diagnosis. Additionally, analyses were repeated excluding participants with unknown lymphoma histology.

Ethics statement

The study protocol (code MICRO.HGUGM.2022-017) was approved by the Ethics Committee of Hospital General Universitario Gregorio Marañón on 12 September 2022 (meeting record 17/2022).

Each CoRIS participant provided their written informed consent prior to enrolling in this study. The CoRIS cohort was approved by the Research Ethic Committee of the Gregorio Marañón Hospital.

RESULTS

Study population

Among the 18 573 PLWH included in the CoRIS cohort up to 2022, 291 (1.57%) were diagnosed with lymphoma, either HL, NHL, or unknown. HIV viral load data at the time of lymphoma diagnosis were available for 245 individuals, who constituted the study population. Baseline characteristics were comparable between included and excluded participants (Supplementary Table S1).

Of the overall included population, 49 (20.0%) had virological suppression at diagnosis.

Among the people diagnosed with lymphoma, 63 had HL, of whom 25 (39.7%) had virological suppression. A

total of 157 persons were diagnosed with NHL, and 21 (13.4%) of them had suppressed HIV viral load at the time of diagnosis. For the remaining 25 individuals included, no histological data was available. (See Flow-chart in Figure 1). Table 1 summarizes the characteristics of the included population according to histological type.

Epidemiological trends

Between 1 January 2004 and 31 December 2009, 62 lymphomas were diagnosed in people with detectable HIV viral load and seven in people with virological suppression; 47 of the former and four of the latter corresponded to NHL.

From 1 January 2010 to 31 December 2014, 72 lymphoma cases were diagnosed among people with detectable HIV viral load, of which 48 were NHL, whereas 12 cases occurred in individuals with virological suppression, including five NHL cases.

Between 1 January 2015 and 31 December 2019, 53 lymphomas were diagnosed in people with detectable HIV viral load and 26 in individuals with virological

suppression. Among those with detectable HIV viral load, 35 cases were NHL, while nine NHL cases were identified among people with virological suppression.

Finally, from 1 January 2020 to 31 December 2022, nine lymphoma cases were diagnosed in people with detectable HIV viral load, six of which were NHL, and four cases were diagnosed in individuals with virological suppression, including three NHL cases. (Figure 2).

In Figure 3, from the total of lymphoma cases recorded in CoRIS each year, we reflect the proportion of those occurring in people with undetectable HIV viral load.

Baseline characteristics according to virological suppression

Baseline demographic, clinical, immunological and oncological characteristics of the study population, stratified by virological suppression, are summarized in Table 2.

No significant differences were observed in sex, age, or HIV transmission category when comparing people with detectable versus suppressed HIV viral load. These

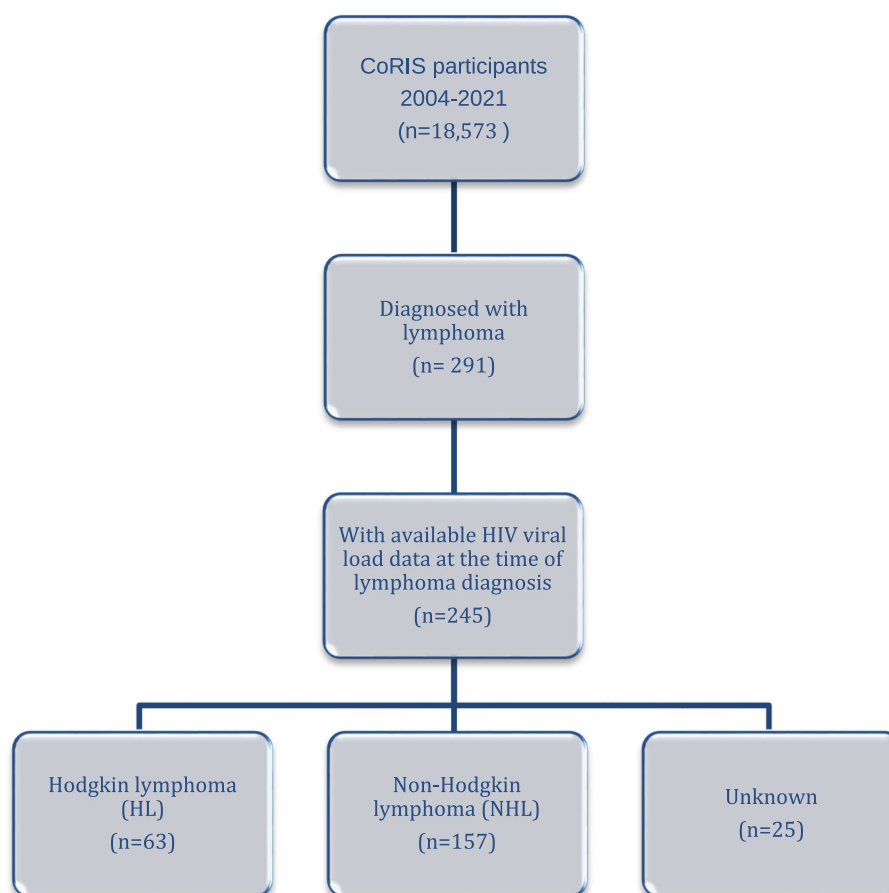


FIGURE 1 Flow chart

TABLE 1 Clinical characteristics by histological type.

Characteristic	Overall N = 245 ^a	HL N = 63 ^a	NHL N = 157 ^a	Unknown N = 25 ^a
Sex				
Male	210 (85.7%)	57 (90.5%)	133 (84.7%)	20 (80.0%)
Female	35 (14.3%)	6 (9.5%)	24 (15.3%)	5 (20.0%)
Age at diagnosis, years	45.4 (36.1–53.5)	43.8 (36.3–51.0)	45.0 (35.8–53.5)	48.8 (40.1–59.0)
Transmission route				
MSM	131 (53.5%)	39 (61.9%)	83 (52.9%)	9 (36.0%)
IDU	25 (10.2%)	7 (11.1%)	17 (10.8%)	1 (4.0%)
Heterosexual	77 (31.4%)	15 (23.8%)	50 (31.8%)	12 (48.0%)
Other/Unknown	12 (4.9%)	2 (3.2%)	7 (4.5%)	3 (12.0%)
Region of origin				
Spain	156 (63.7%)	39 (61.9%)	97 (61.8%)	20 (80.0%)
Europe	40 (16.3%)	12 (19.0%)	27 (17.2%)	1 (4.0%)
Sub-Saharan Africa	6 (2.4%)	1 (1.6%)	4 (2.5%)	1 (4.0%)
Latin America	36 (14.7%)	8 (12.7%)	25 (15.9%)	3 (12.0%)
Other/Unknown	7 (2.9%)	3 (4.8%)	4 (2.5%)	0 (0.0%)
Educational level				
None or primary education	40 (16.3%)	10 (15.9%)	27 (17.2%)	3 (12.0%)
Upper secondary or university	113 (46.1%)	35 (55.6%)	72 (45.9%)	6 (24.0%)
University or higher	45 (18.4%)	14 (22.2%)	25 (15.9%)	6 (24.0%)
Other/Unknown	47 (19.2%)	4 (6.3%)	33 (21.0%)	10 (40.0%)
Smoking status				
Never smoker	36 (14.7%)	9 (14.3%)	24 (15.3%)	3 (12.0%)
Smoker	41 (16.7%)	19 (30.2%)	20 (12.7%)	2 (8.0%)
Ex-Smoker	16 (6.5%)	5 (7.9%)	10 (6.4%)	1 (4.0%)
Unknown	152 (62.0%)	30 (47.6%)	103 (65.6%)	19 (76.0%)
Period of diagnosis				
2004–2014	167 (68.2%)	35 (55.6%)	113 (72.0%)	19 (76.0%)
2015–2021	78 (31.8%)	28 (44.4%)	44 (28.0%)	6 (24.0%)
HIV duration, years	0.7 (0.1–4.7)	3.5 (0.5–8.0)	0.4 (0.0–3.0)	0.0 (0.0–1.0)
CD4 count, cells/ μ L	207.0 (94.0–381.0)	210.0 (137.5–355.0)	210.0 (84.0–387.0)	165.5 (77.5–402.0)
Unknown	10	3	6	1
CD4 count category				
<200	112 (45.7%)	27 (42.9%)	71 (45.2%)	14 (56.0%)
$\geq$ 200	123 (50.2%)	33 (52.4%)	80 (51.0%)	10 (40.0%)
Unknown	10 (4.1%)	3 (4.8%)	6 (3.8%)	1 (4.0%)
ART status				
ART at diagnosis	150 (61.2%)	54 (85.7%)	87 (55.4%)	9 (36.0%)
No ART prior to diagnosis	95 (38.8%)	9 (14.3%)	70 (44.6%)	16 (64.0%)
Type of HL				
Nodular sclerosis		11 (17.4%)		
Mixed cellularity		16 (25.4%)		
Lymphocyte-depleted		1 (1.6%)		
Lymphocyte-rich		3 (4.8%)		

TABLE 1 (Continued)

Characteristic	Overall N = 245 ^a	HL N = 63 ^a	NHL N = 157 ^a	Unknown N = 25 ^a
Unknown		32 (50.8%)		
Type of LNH				
DLBCL			65 (41.4%)	
BL			40 (25.5%)	
PCNSL			7 (4.4%)	
PL			10 (6.4%)	
PEL			5 (3.2%)	
Unknown			30 (19.1%)	
IPI score				
0–2	NA	NA	38 (24.2%)	0 (0.0%)
3–4	NA	NA	37 (23.6%)	0 (0.0%)
Unknown	NA	NA	82 (52.2%)	25 (100.0%)
Ann Arbor stage				
I-II	37 (15.1%)	15 (23.8%)	22 (14.0%)	0 (0.0%)
III-IV	130 (53.1%)	39 (61.9%)	90 (57.3%)	1 (4.0%)
Unknown	78 (31.8%)	9 (14.3%)	45 (28.7%)	24 (96.0%)
Chemotherapy				
No	15 (6.1%)	5 (7.9%)	10 (6.4%)	0 (0.0%)
Yes	182 (74.3%)	50 (79.4%)	130 (82.8%)	2 (8.0%)
Unknown	48 (19.6%)	8 (12.7%)	17 (10.8%)	23 (92.0%)
Radiotherapy				
No	159 (64.9%)	38 (60.3%)	119 (75.8%)	2 (8.0%)
Yes	36 (14.7%)	16 (25.4%)	20 (12.7%)	0 (0.0%)
Unknown	50 (20.4%)	9 (14.3%)	18 (11.5%)	23 (92.0%)
Autologous transplant				
No	174 (71.0%)	52 (82.5%)	120 (76.4%)	2 (8.0%)
Yes	21 (8.6%)	2 (3.2%)	19 (12.1%)	0 (0.0%)
Unknown	50 (20.4%)	9 (14.3%)	18 (11.5%)	23 (92.0%)
Allogeneic transplant				
No	191 (78.0%)	53 (84.1%)	136 (86.6%)	2 (8.0%)
Yes	3 (1.2%)	1 (1.6%)	2 (1.3%)	0 (0.0%)
Unknown	51 (20.8%)	9 (14.3%)	19 (12.1%)	23 (92.0%)
No treatment				
No	154 (62.9%)	40 (63.5%)	112 (71.3%)	2 (8.0%)
Yes	8 (3.3%)	2 (3.2%)	6 (3.8%)	0 (0.0%)
Unknown	83 (33.9%)	21 (33.3%)	39 (24.8%)	23 (92.0%)
Treatment outcome				
Complete remission	136 (55.5%)	48 (76.2%)	86 (54.8%)	2 (8.0%)
Partial remission	8 (3.3%)	1 (1.6%)	7 (4.5%)	0 (0.0%)
1 year Lymphoma-related Mortality	53 (21.6%)	5 (7.9%)	47 (29.9%)	1 (4.0%)
Unknown	48 (19.6%)	9 (14.3%)	17 (10.8%)	22 (88.0%)
Relapse				
No	139 (56.7%)	44 (69.8%)	94 (59.9%)	1 (4.0%)

(Continues)

TABLE 1 (Continued)

Characteristic	Overall <i>N</i> = 245 ^a	HL <i>N</i> = 63 ^a	NHL <i>N</i> = 157 ^a	Unknown <i>N</i> = 25 ^a
Yes	20 (8.2%)	8 (12.7%)	11 (7.0%)	1 (4.0%)
Unknown	86 (35.1%)	11 (17.5%)	52 (33.1%)	23 (92.0%)
Time to relapse, years	1.6 (0.6–3.2)	3.2 (2.2–4.0)	0.8 (0.4–1.3)	9.3 (9.3–9.3)

^a*n* (%); Median (Q1–Q3).

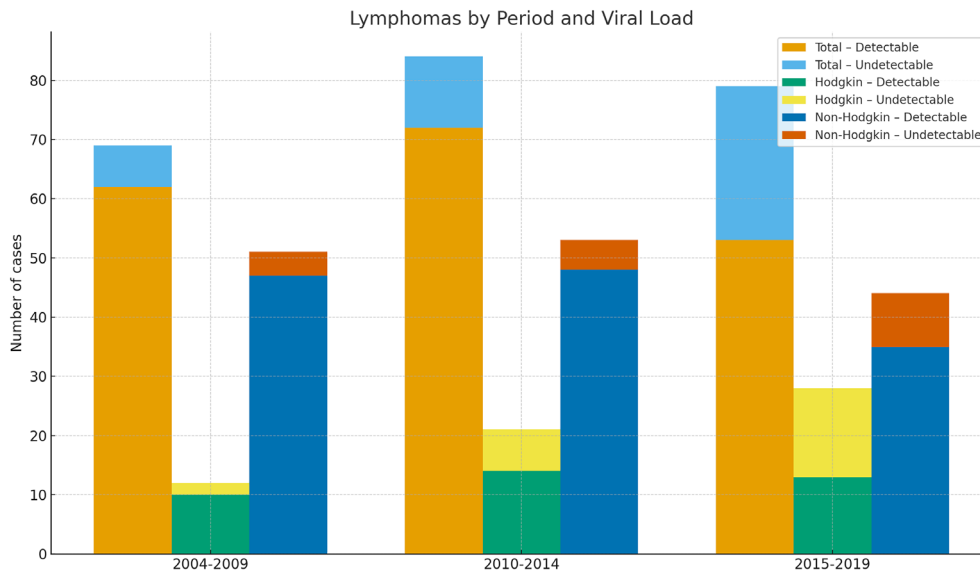


FIGURE 2 Number of lymphomas according to **HIV** virological status. Number of lymphoma cases among people living with HIV, overall and stratified by Hodgkin lymphoma (HL) and non-Hodgkin lymphoma (NHL), according to HIV virological status at diagnosis (HIV-1 RNA <100 vs. ≥100 copies/mL).

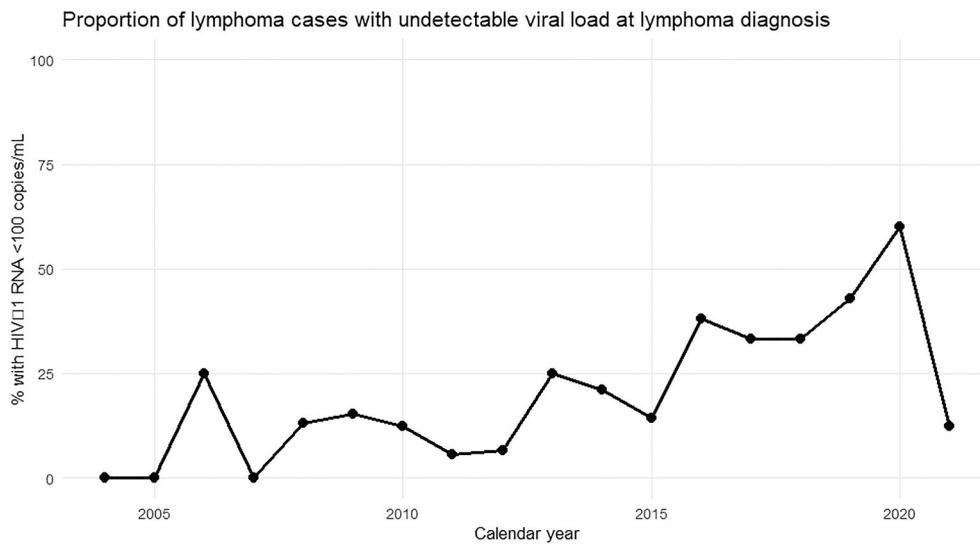


FIGURE 3 Proportion of lymphoma cases diagnosed in people living with HIV with virological suppression over time. Proportion of Hodgkin and non-Hodgkin lymphoma cases diagnosed in people living with HIV with HIV-1 RNA < 100 copies/mL at the time of lymphoma diagnosis, by calendar year.

TABLE 2 Clinical characteristics by HIV-1 viral load status at lymphoma diagnosis.

Characteristic	HIV-1 RNA at diagnosis		p-value ^a
	HIV-1 detectable <i>N</i> = 196 ^b	HIV-1 undetectable <i>N</i> = 49 ^b	
Sex			1
Male	168 (85.7%)	42 (85.7%)	
Female	28 (14.3%)	7 (14.3%)	
Age at diagnosis, years	45.3 (35.4–53.0)	45.5 (39.1–55.1)	0.392
Transmission route			0.37
MSM	103 (52.6%)	28 (57.1%)	
IDU	20 (10.2%)	5 (10.2%)	
Other/Unknown	12 (6.1%)	0 (0.0%)	
Heterosexual	61 (31.1%)	16 (32.7%)	
Region of origin			0.616
Spain	127 (64.8%)	29 (59.2%)	
Europe	31 (15.8%)	9 (18.4%)	
Sub-Saharan Africa	6 (3.1%)	0 (0.0%)	
Other/Unknown	5 (2.6%)	2 (4.1%)	
Latin America	27 (13.8%)	9 (18.4%)	
Educational level			0.327
None or primary education	31 (15.8%)	9 (18.4%)	
Upper secondary or university	87 (44.4%)	26 (53.1%)	
University or higher	36 (18.4%)	9 (18.4%)	
Other/Unknown	42 (21.4%)	5 (10.2%)	
Smoking status			0.564
Never smoker	29 (42.0%)	7 (29.2%)	
Smoker	29 (42.0%)	12 (50.0%)	
Ex-Smoker	11 (15.9%)	5 (20.8%)	
Unknown	127	25	
Period of diagnosis			5.49e-05
2004–2014	146 (74.5%)	21 (42.9%)	
2015–2021	50 (25.5%)	28 (57.1%)	
HIV duration, years	0.3 (0.0–2.8)	5.2 (2.1–8.9)	3.57e-11
CD4 count, cells/μL	180.0 (80.0–330.0)	370.5 (202.0–583.0)	1.46e-06
Unknown	7	3	
CD4 count category			4.56e-04
<200	101 (53.4%)	11 (23.9%)	
≥200	88 (46.6%)	35 (76.1%)	
Unknown	7	3	
ART status			1.57e-12
ART at diagnosis	101 (51.5%)	49 (100.0%)	
No ART prior to diagnosis	95 (48.5%)	0 (0.0%)	
IPI score			0.262
0–2	32 (47.8%)	6 (75.0%)	
3–4	35 (52.2%)	2 (25.0%)	
Unknown	69	13	

(Continues)

TABLE 2 (Continued)

Characteristic	HIV-1 RNA at diagnosis		p-value ^a
	HIV-1 detectable <i>N</i> = 196 ^b	HIV-1 undetectable <i>N</i> = 49 ^b	
Ann Arbor stage			0.027
I–II	23 (18.0%)	14 (35.9%)	
III–IV	105 (82.0%)	25 (64.1%)	
Unknown	68	10	
Chemotherapy	143 (92.3%)	39 (92.9%)	1
Unknown	41	7	
Radiotherapy	23 (15.0%)	13 (31.0%)	0.025
Unknown	43	7	
Autologous transplant	20 (13.1%)	1 (2.4%)	0.051
Unknown	43	7	
Allogeneic transplant	2 (1.3%)	1 (2.4%)	0.521
Unknown	44	7	
No treatment	7 (5.2%)	1 (3.6%)	1
Unknown	62	21	
Treatment outcome			0.291
Complete remission	104 (67.1%)	32 (76.2%)	
Partial remission	8 (5.2%)	0 (0.0%)	
1 year Lymphoma-related Mortality	43 (27.7%)	10 (23.8%)	
Unknown	41	7	
Relapse	15 (12.2%)	5 (13.9%)	0.779
Unknown	73	13	
Time to relapse, years	1.3 (0.4–2.9)	3.1 (1.2–4.0)	0.095

^aFisher's exact test; Kruskal–Wallis rank sum test.^b*n* (%); Median (Q1–Q3).

findings remained consistent when analyses were restricted to HL or NHL subgroups (Table 3). HL was more frequent among individuals with undetectable HIV viral load and, within subtypes, those with undetectable viral load more often had the nodular sclerosis histological subtype; however, the sample size is very small. Nevertheless, no significant differences were observed in the distribution of histological subtypes for NHL according to HIV virological status at diagnosis.

In bivariate analyses, people with detectable HIV viral load at the time of lymphoma diagnosis had significantly lower CD4 cell counts compared with people with virological suppression (median 180 cells/mm³ [IQR 80–330] vs. 370.5 cells/mm³ [IQR 202–583], $p < 0.001$). As expected, they also had a shorter duration of known HIV infection (median 0.3 years [IQR 0.0–2.8] vs. 5.2 years [IQR 2.1–8.9], $p < 0.001$).

People with detectable HIV viral load were more frequently diagnosed at advanced Ann Arbor stages

(stage III–IV: 82% vs. 64.1%, $p = 0.03$), indicating a greater extent of disease at presentation. Consistent with this finding, differences were observed in treatment approaches: people with detectable HIV viral load more often underwent autologous stem cell transplantation (13.1% vs. 2.4%, $p = 0.05$), whereas people with virological suppression more frequently received radiotherapy (31% vs. 15%, $p = 0.02$), which is typically indicated in less advanced disease.

Despite these differences in clinical presentation and treatment intensity, no significant differences were observed between people with detectable HIV viral load and those with virological suppression in terms of treatment response, mortality, or relapse rates. Complete remission was achieved in 67% of people with detectable HIV viral load and 76% of people with virological suppression. Lymphoma-related mortality at 1 year was 27.7% and 23.8%, respectively ($p = 0.65$), and relapse occurred in 13.9% and 12.2% of individuals, respectively

TABLE 3 Clinical characteristics by HIV-1 viral load status at lymphoma diagnosis stratified by lymphoma type.

Characteristic	HL		<i>p</i> -value ^b	NHL		<i>p</i> -value ^b
	HIV-1 detectable <i>N</i> = 38 ^a	HIV-1 undetectable <i>N</i> = 25 ^a		HIV-1 detectable <i>N</i> = 136 ^a	HIV-1 undetectable <i>N</i> = 21 ^a	
Sex			1			1
Male	34 (89.5%)	23 (92.0%)		115 (84.6%)	18 (85.7%)	
Female	4 (10.5%)	2 (8.0%)		21 (15.4%)	3 (14.3%)	
Age at diagnosis, years	44.1 (36.3–51.0)	43.8 (37.3–49.9)	0.779	44.6 (35.1–53.0)	49.2 (41.8–55.6)	0.137
Transmission route			0.217			0.231
MSM	20 (52.6%)	19 (76.0%)		75 (55.1%)	8 (38.1%)	
IDU	6 (15.8%)	1 (4.0%)		13 (9.6%)	4 (19.0%)	
Other/Unknown	2 (5.3%)	0 (0.0%)		7 (5.1%)	0 (0.0%)	
Heterosexual	10 (26.3%)	5 (20.0%)		41 (30.1%)	9 (42.9%)	
Region of origin			0.099			0.917
Spain	27 (71.1%)	12 (48.0%)		82 (60.3%)	15 (71.4%)	
Europe	7 (18.4%)	5 (20.0%)		23 (16.9%)	4 (19.0%)	
Sub-Saharan Africa	1 (2.6%)	0 (0.0%)		4 (2.9%)	0 (0.0%)	
Other/Unknown	1 (2.6%)	2 (8.0%)		4 (2.9%)	0 (0.0%)	
Latin America	2 (5.3%)	6 (24.0%)		23 (16.9%)	2 (9.5%)	
Educational level			0.546			0.259
None or primary education	5 (13.2%)	5 (20.0%)		24 (17.6%)	3 (14.3%)	
Upper secondary or university	24 (63.2%)	11 (44.0%)		58 (42.6%)	14 (66.7%)	
University or higher	7 (18.4%)	7 (28.0%)		23 (16.9%)	2 (9.5%)	
Other/Unknown	2 (5.3%)	2 (8.0%)		31 (22.8%)	2 (9.5%)	
Smoking status			0.157			0.109
Never smoker	3 (16.7%)	6 (40.0%)		23 (50.0%)	1 (12.5%)	
Smoker	13 (72.2%)	6 (40.0%)		15 (32.6%)	5 (62.5%)	
Ex-Smoker	2 (11.1%)	3 (20.0%)		8 (17.4%)	2 (25.0%)	
Unknown	20	10		90	13	
Period of diagnosis			0.069			0.003
2004–2014	25 (65.8%)	10 (40.0%)		104 (76.5%)	9 (42.9%)	
2015–2021	13 (34.2%)	15 (60.0%)		32 (23.5%)	12 (57.1%)	
HIV duration, years	1.1 (0.2–7.6)	5.3 (3.5–8.9)	0.003	0.2 (0.0–2.2)	3.1 (1.8–8.9)	4.87e-06
CD4 count, cells/μL	157.0 (91.0–276.0)	336.0 (190.0–608.0)	0.002	189.5 (76.0–356.0)	440.0 (323.0–540.0)	1.27e-04
Unknown	1	2		6	0	
CD4 count category			0.032			0.008
<200	21 (56.8%)	6 (26.1%)		67 (51.5%)	4 (19.0%)	
≥200	16 (43.2%)	17 (73.9%)		63 (48.5%)	17 (81.0%)	
Unknown	1	2		6	0	
ART status			0.009			1.44e-06
ART at diagnosis	29 (76.3%)	25 (100.0%)		66 (48.5%)	21 (100.0%)	
No ART prior to diagnosis	9 (23.7%)	0 (0.0%)		70 (51.5%)	0 (0.0%)	

(Continues)

TABLE 3 (Continued)

Characteristic	HL			NHL		
	HIV-1 detectable N = 38 ^a	HIV-1 undetectable N = 25 ^a	p-value ^b	HIV-1 detectable N = 136 ^a	HIV-1 undetectable N = 21 ^a	p-value ^b
Type of HL			0.006			
Nodular sclerosis	3 (14.3%)	8 (80.0%)				
Mixed cellularity	15 (71.4%)	1 (10.0%)				
Lymphocyte-depleted	1 (4.8%)	0 (0.0%)				
Lymphocyte-rich	2 (9.5%)	1 (10.0%)				
Unknown	17	15				
Type of NHL						0.67
DLBCL				60 (52.2%)	5 (41.7%)	
BL				36 (31.3%)	4 (33.3%)	
PCNSL				7 (6.1%)	0 (0.0%)	
PL				8 (7.0%)	2 (16.7%)	
PEL				4 (3.5%)	1 (8.3%)	
Unknown				21	9	
IPI score						0.262
0–2	NA	NA		32 (47.8%)	6 (75%)	
3–4	NA	NA		35 (52.2%)	2 (25%)	
Unknown				136	21	
Ann Arbor stage			0.369			0.073
I–II	7 (22.6%)	8 (34.8%)		16 (16.5%)	6 (40.0%)	
III–IV	24 (77.4%)	15 (65.2%)		81 (83.5%)	9 (60.0%)	
Unknown	7	2		39	6	
Chemotherapy	29 (90.6%)	21 (91.3%)	1	113 (92.6%)	17 (94.4%)	1
Unknown	6	2		14	3	
Radiotherapy	7 (22.6%)	9 (39.1%)	0.235	16 (13.2%)	4 (22.2%)	0.294
Unknown	7	2		15	3	
Autologous transplant	1 (3.2%)	1 (4.3%)	1	19 (15.7%)	0 (0.0%)	0.133
Unknown	7	2		15	3	
Allogeneic transplant	0 (0.0%)	1 (4.3%)	0.426	2 (1.7%)	0 (0.0%)	1
Unknown	7	2		16	3	
No treatment	2 (7.4%)	0 (0.0%)	0.53	5 (4.7%)	1 (8.3%)	0.482
Unknown	11	10		30	9	
Treatment outcome			1			0.559
Complete remission	27 (87.1%)	21 (91.3%)		76 (62.3%)	10 (55.6%)	
Partial remission	1 (3.2%)	0 (0.0%)		7 (5.7%)	0 (0.0%)	
1 year Lymphoma-related Mortality	3 (9.7%)	2 (8.7%)		39 (32.0%)	8 (44.4%)	
Unknown	7	2		14	3	
Relapse	4 (13.3%)	4 (18.2%)	0.708	10 (10.9%)	1 (7.7%)	1
Unknown	8	3		44	8	

TABLE 3 (Continued)

Characteristic	HL			NHL		
	HIV-1 detectable N = 38 ^a	HIV-1 undetectable N = 25 ^a	p-value ^b	HIV-1 detectable N = 136 ^a	HIV-1 undetectable N = 21 ^a	p-value ^b
Time to relapse, years	2.9 (2.1–3.3)	3.7 (3.1–4.0)	0.251	0.6 (0.3–1.4)	1.0 (0.8–1.2)	0.655

Abbreviations: BL, burkitt lymphoma; DLBCL, diffuse large B-cell lymphoma; HIV, human immunodeficiency virus; HL, Hodgkin lymphoma; IDU, injecting drug use; IQR, interquartile range; MSM, men who have sex with men; NHL, non-Hodgkin lymphoma; PCNSL, primary central nervous system lymphoma; PEL, primary effusion lymphoma; PL, plasmablastic lymphoma.

^an (%); Median (Q1–Q3).

^bFisher's exact test; Kruskal–Wallis rank sum test; Fisher's Exact Test for Count Data with simulated p-value (based on 10000 replicates).

($p = 0.779$). These findings were consistent in the overall cohort and when analyses were stratified by HL and NHL (Table 3).

Survival analysis

Among the 220 participants diagnosed with either NHL or HL and followed for up to 5 years, the overall mortality rate was 0.96 per 100 person-years (95% CI 0.76–1.21) (Figure 4). In multivariable analyses, HIV virological suppression at the time of lymphoma diagnosis was not independently associated with 5-year mortality. The hazard of death increased by 28% for each 10-year increase in age at diagnosis and by 35% for each additional 10 years of known HIV duration. Year of lymphoma diagnosis was not associated with overall survival in the fully adjusted model.

A CD4 cell count ≥ 200 cells/ μ L was associated with a 44% lower hazard of death over the 5-year follow-up period. Additionally, 5-year mortality was 70% lower among individuals with HL compared with those with NHL (Table 4).

Lymphoma-specific mortality was evaluated exclusively during the first year of follow-up. The lymphoma-specific mortality rate during this period was 2.00 per 100 person-years (95% CI 1.44–2.71) (Figure 5). In multivariable analyses of 1-year lymphoma-specific survival, HIV virological suppression at the time of lymphoma diagnosis was not independently associated with mortality. The hazard of lymphoma-specific death increased by 36% for each 10-year increase in age at diagnosis, whereas a CD4 cell count ≥ 200 cells/ μ L was associated with a 37% reduction in the hazard of death. Consistent with findings for overall survival, lymphoma-specific mortality within the first year was lower among individuals with HL than among those with NHL (Table 4).

Sensitivity analyses restricting the definition of virological suppression to pre-diagnosis HIV-1 RNA

measurements yielded consistent results with the primary analysis. No significant association was observed between virological suppression and 5-year overall survival. The direction and magnitude of the associations for age, CD4 cell count and lymphoma subtype remained unchanged. Detailed results are provided in Supplementary Tables S2–S4 and Figures S1 and S2.

DISCUSSION

This study confirms that lymphoproliferative disorders remain a relevant clinical problem in PLWH despite effective ART and sustained virological suppression. Prior studies have demonstrated that the incidence of both HL and NHL remains higher than that observed in the general population [20]. In our work, we observed a progressive increase over time in the number and the proportion of lymphoma cases occurring in individuals with suppressed HIV viral load. As expected, lymphoma cases among individuals with suppressed HIV viral load have become more frequent in recent years, likely reflecting earlier initiation of ART. As the proportion of PLWH with virological suppression increases, lymphoma is progressively shifting from a complication of uncontrolled HIV to a malignancy occurring despite optimal HIV management. The increasing proportion of people with virological suppression among lymphoma cases over time likely reflects improvements in HIV care at the cohort level, rather than a direct association between viral suppression and lymphoma risk. In our study, this trend was evident until 2020, after which a potential change in epidemiological patterns was observed. Whether this reflects a true shift related to earlier initiation of ART or, alternatively, the impact of the SARS-CoV-2 pandemic on healthcare access and cancer diagnosis warrants further investigation.

Historically, immunosuppression and uncontrolled HIV replication have been considered the main drivers of

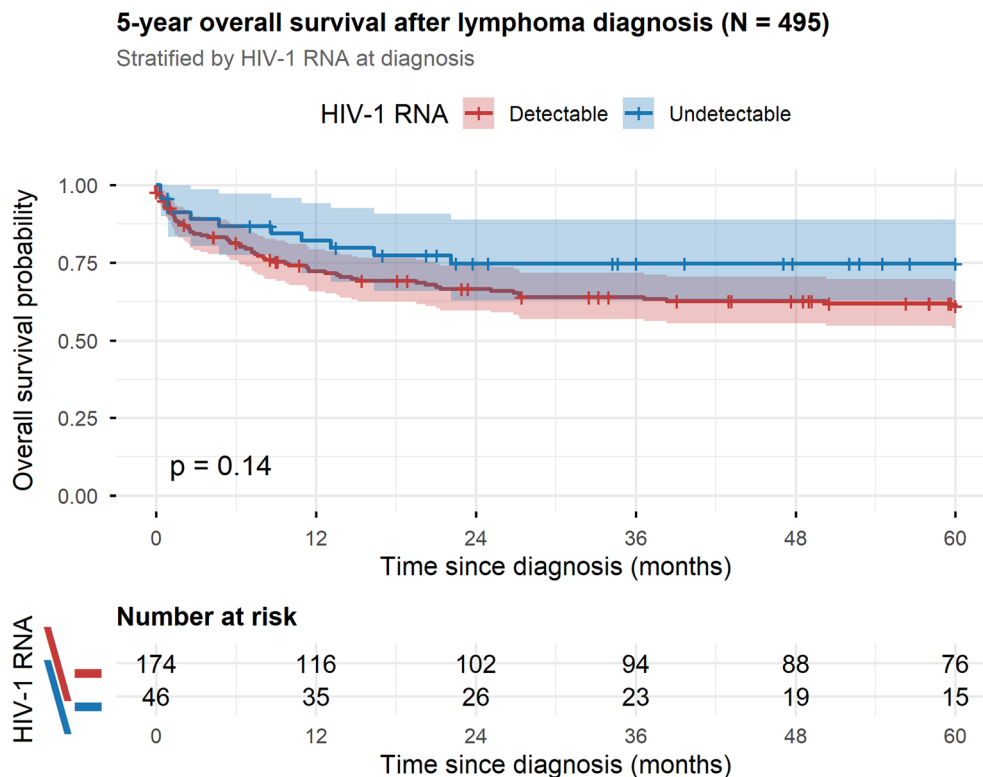


FIGURE 4 5-year overall survival.

lymphoma development in PLWH [21, 22]. However, despite the fact that currently most PLWH maintain an undetectable HIV viral load, cases still occur, as observed in our cohort, suggesting that additional mechanisms may contribute to the persistent risk of lymphomagenesis even under optimal immunovirological conditions. In our study, the predominant histology in individuals with suppressed viral load is HL, and within this, the nodular sclerosis subtype, as is typically observed [23]. But importantly, the distribution of the most prevalent histological subtypes of NHL in our study—distinct from that observed in the HIV-negative population—did not differ according to HIV detectability. This observation is consistent with findings from a recent cohort of PLWH with lymphoma in Malawi [24] and with earlier studies conducted in people with virological suppression [25]. These results suggest the existence of HIV-associated lymphomagenic mechanisms that persist beyond virological control.

When comparing lymphomas diagnosed in people with detectable versus suppressed HIV viral load, we found that individuals with ongoing viral replication presented with more advanced disease at diagnosis, as reflected by higher Ann Arbor stage. Nevertheless, despite differences in disease extent and treatment intensity, overall and lymphoma-specific survival and relapse rates were similar between both groups, and HIV

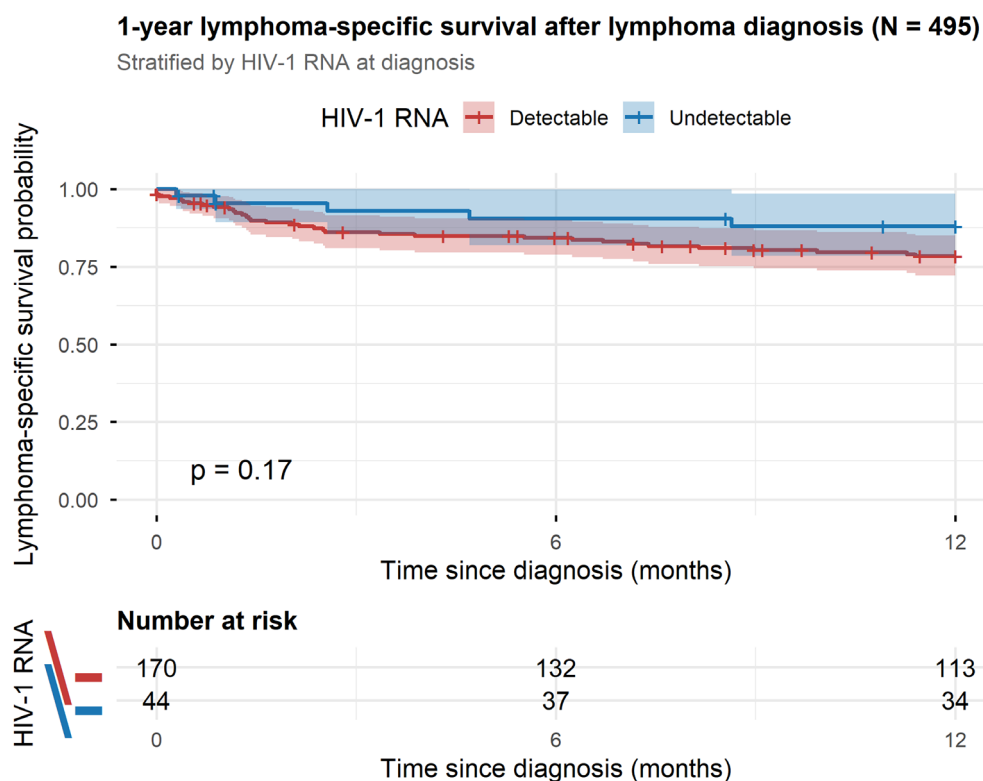
virological status was not an independent predictor of survival. For several years, PLWH have been treated for lymphoma using the same oncological regimens as HIV-negative persons, in combination with ART [26–28]. Our findings reinforce the current evidence indicating that, in the contemporary ART era, HIV-related factors—including ongoing viral replication—do not independently determine lymphoma prognosis. Rather, the excess risk associated with certain histological subtypes appears to be largely mediated by tumour burden at diagnosis. So, HIV replication may influence disease burden at presentation but not treatment efficacy or survival once standard oncological therapy is delivered.

With the application of standard oncological treatment strategies alongside effective ART, survival outcomes comparable to those observed in individuals with virological suppression can be achieved, even in people with detectable HIV viral load at diagnosis. This concept had already been suggested in earlier studies assessing chemotherapy response in PLWH, which demonstrated that prognosis depended predominantly on tumour-related factors rather than HIV-specific variables [29, 30]. Although subsequent research proposed prognostic models incorporating HIV-related parameters [31], our results—derived from a contemporary cohort—provide robust evidence supporting the primacy of standard oncological determinants in guiding treatment and prognosis.

TABLE 4 Multivariable Cox regression for 5-year overall survival and 1-year lymphoma-specific survival.

Characteristic	5-year overall survival			1-year lymphoma-specific survival		
	HR ^a	95% CI	p-value	HR ^a	95% CI	p-value
HIV-1 RNA (ref: Detectable)	0.85	0.42, 1.74	0.658	1.03	0.39, 2.73	0.959
Age (per 10-year increase)	1.28	1.03, 1.59	0.026	1.36	1.02, 1.81	0.038
CD4 count (ref: <200 cells/ μ L)						
<200	–	–	–	–	–	–
≥ 200	0.56	0.35, 0.91	0.019	0.63	0.34, 1.19	0.158
Unknown	0.41	0.10, 1.71	0.222	0.00	0.00, Inf	0.996
Lymphoma type (ref: NHL)						
NHL	–	–	–	–	–	–
HL	0.31	0.16, 0.62	7.49e-04	0.17	0.05, 0.56	0.004
Unknown	–	–	–	–	–	–
HIV duration (per 10-year increase)	1.35	0.96, 1.91	0.089	–	–	–
Year of lymphoma diagnosis						
2004–2014	–	–	–	0.90	0.45, 1.83	0.776
2015–2021	1.20	0.71, 2.01	0.497	1.09	0.65, 1.82	0.756

Abbreviations: CI, confidence interval; HR, hazard ratio.

^aModels adjusted for all variables shown.**FIGURE 5** 1-year lymphoma-specific survival

The favourable outcomes observed in people with detectable HIV viral load may, at least in part, be explained by the improved safety profile of modern antiretroviral agents, which have minimal

overlapping toxicity with antineoplastic therapies. This allows for safe coadministration without treatment interruptions or delays in chemotherapy regimens [32].

A study published by Gopal et al. in 2013 reported higher mortality among people with detectable HIV viraemia at lymphoma diagnosis, particularly among those with longer duration of HIV infection [33]. In contrast, in our cohort, HIV viral load was not associated with mortality, and people with detectable HIV viral load had, on average, a shorter duration of known HIV infection than individuals with virological suppression. This finding likely reflects current clinical practice, in which ART is initiated promptly after HIV diagnosis, in accordance with contemporary treatment guidelines. Moreover, a recent study from China similarly found that virological factors did not significantly influence lymphoma outcomes in PLWH [34].

In our survival analyses, immunosuppression—reflected by CD4 cell counts below 200 cells/mm³—emerged as a strong predictor for survival, in agreement with previous studies [34, 35]. Age and lymphoma type were also associated with worse outcomes, consistent with well-established prognostic factors in oncohaematological diseases, regardless of HIV status. These findings suggest that, in the current ART era, lymphoma prognosis in PLWH is primarily driven by host immune status and lymphoma-related factors, rather than HIV viral replication itself.

This study has several limitations. From a methodological point of view, first, missing virological data led to the exclusion of a proportion of cases, although no major differences were observed between included and excluded individuals. Second, lymphoma histology was unavailable for some participants, although sensitivity analyses yielded consistent results, and some important lymphoma-related information, such as Ann Arbor stage, was only available for a few patients and therefore could not be included in the multivariable analysis. Third, the definition of virological suppression included measurements shortly after diagnosis, which may introduce misclassification; however, sensitivity analyses confirmed the robustness of our findings. Also, we did not include a comparator population without HIV infection. We were unable to assess immunological and inflammatory biomarkers that may influence lymphomagenesis, as suggested by previous research [36, 37]. Additionally, we did not account for co-infection with oncogenic viruses such as EBV or human herpesvirus 8, which play an important pathophysiological role in HIV-associated lymphomas [35, 38]. Despite these limitations, our study includes a large number of cases and spans an extended time period, providing relevant clinical insight into lymphomas in PLWH in the contemporary ART era. From a purely clinical perspective, our findings do not support concerns that ART or virological suppression negatively influence lymphoma management or outcomes,

unlike emerging hypotheses suggesting potential interactions between prior ART exposure and tumour biology [39].

CONCLUSIONS

Although PLWH diagnosed with lymphoma who have detectable HIV viral load are more frequently present with advanced-stage disease, lymphoma prognosis is independent of HIV virological status at diagnosis. These findings support that the management of Hodgkin and NHLs in PLWH should be primarily guided by standard oncohaematological criteria, with concomitant ART, regardless of HIV viral suppression status.

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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