Early initiation of antiretroviral therapy for people newly diagnosed with HIV infection in Australia: trends and predictors, 2004–2015

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The known Treatment of people infected with human immunodeficiency virus (HIV) soon after diagnosis reduces the risk of transmission and HIV-related morbidity. The proportion and characteristics of people who receive early treatment are not well described.

The new The proportion of patients attending major Australian sexual health clinics who started treatment within 6 months of diagnosis increased during 2004–2015 from 17% to 53%. The rise reflected changes in CD4$^+$ cell count thresholds in treatment guidelines.

The implications Our findings provide a baseline for assessing the impact of early treatment for all newly diagnosed patients, regardless of CD4$^+$ cell count.

Abstract

Objectives: To determine trends in and predictors of early treatment for people newly diagnosed with human immunodeficiency virus (HIV) infection in Australia.

Design, setting: Retrospective cohort analysis of routinely collected longitudinal data from 44 sexual health clinics participating in the Australian Collaboration for Coordinated Enhanced Sentinel Surveillance (ACCESS) program.

Participants: Patients diagnosed with HIV infections, January 2004 – June 2015.

Main outcome measures: Commencement of antiretroviral therapy within 6 months of HIV diagnosis (early treatment); demographic, clinical, and risk group characteristics of patients associated with early treatment; trends in early treatment, by CD4$^+$ cell count at diagnosis.

Results: 917 people were diagnosed with HIV infections, their median age was 34 years (interquartile range [IQR]: 27–43 years), and 841 (92%) were men; the median CD4$^+$ cell count at diagnosis was 510 cells/μL (IQR, 350–674 cells/μL). The proportion of patients who received early treatment increased from 17% (15 patients) in 2004–06 to 20% (34 patients) in 2007–09, 34% (95 patients) in 2010–12, and 53% (197 patients) in 2013–15 (trend, $P < 0.001$). The probability of early treatment, which increased with time, was higher for patients with lower CD4$^+$ cell counts and higher viral loads at diagnosis.

Conclusions: The proportion of people newly diagnosed with HIV in sexual health clinics in Australia who received treatment within 6 months of diagnosis increased from 17% to 53% during 2004–2015, reflecting changes in the CD4$^+$ cell count threshold in treatment guidelines. Nevertheless, further strategies are needed to maximise the benefits of treatment to prevent viral transmission and morbidity.

The United Nations has set itself the ambitious goal of eliminating the epidemic of acquired immunodeficiency syndrome (AIDS) by 2030. Many countries are aiming to achieve this by striving for the "90–90–90" treatment target: 90% of people living with the human immunodeficiency virus (HIV) should know their status, 90% of people diagnosed with HIV infection should receive treatment, and 90% of treated people should achieve virological suppression.[1] The evidence base for this major shift in the focus of prevention was provided by the 2011 HPTN 052 multicentre randomised controlled trial, in which the risk of HIV transmission to partners of heterosexual people who started treatment early (at CD4$^+$ cell counts of 350–550 cells/μL) was 96% lower than for people for whom treatment was delayed (CD4$^+$ count below 250 cells/μL).[2] Further, observational studies of cohorts of same and opposite sex couples have found that HIV was not transmitted by people with sustained viral suppression,[2,3] and community-level studies have reported that greater treatment coverage is associated with lower incidence of HIV infections.[4]

Starting treatment during the early stages of infection was also found to be beneficial in the international Strategic Timing of Antiretroviral Treatment (START) randomised clinical trial.[5] The 2016 Australian HIV management guidelines[6] recommend initiating treatment as soon as possible after diagnosis, regardless of CD4$^+$ cell count, in order to reduce the risk of morbidity (including *Pneumocystis jiroveci* pneumonia, cerebral toxoplasmosis, pulmonary tuberculosis, Kaposi sarcoma, and HIV wasting syndrome), and some local recommendations (eg, in New South Wales[7]) encourage initiating therapy within 6 weeks of diagnosis.

Until 2012, US guidelines recommended delaying the initiation of therapy, balancing immune preservation and virological suppression on the one hand and treatment-related morbidity and mortality on the other. Until late 2007, antiretroviral treatment was recommended when the CD4$^+$ cell count fell below 200 cells/μL.[8] This threshold was raised when concerns about the toxicity of treatment eased, supported by large cohort studies that found clinical outcomes were improved when treatment commenced earlier (2008, fewer than 350 cells/μL in asymptomatic people;[9] after 2009, fewer than 500 cells/μL[10]).

To monitor progress towards the 90–90–90 target at the population level, most countries measure coverage of HIV testing, treatment, and viral suppression with the "HIV care

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cascade" model, in which data from several cross-sectional administrative data sources are compiled.[11] In countries such as Australia, where HIV treatment coverage for people diagnosed with HIV infection is good (84%),[12] such cascades do not assess the uptake of recommendations for initiating HIV treatment as soon as possible after diagnosis.[13] Cascades do not provide detail on determinants of the rate of progression through the continuum of care, nor on predictors of the early initiation of treatment.

Few countries have established national systems for monitoring early initiation of treatment for people newly diagnosed with HIV infection.[11] We analysed data from a national network of publicly funded sexual health clinics to determine trends in and predictors of early treatment of people newly diagnosed with HIV infection.

Methods

Setting and data source

Australia has a concentrated HIV epidemic: 70% of the approximately 1000 HIV diagnoses each year are in homosexually or bisexually active men (men who have sex with men, MSM).[12,14] Annual community survey data indicate that HIV prevalence among gay men exceeds 7%.[12,15] About half the infections in MSM newly diagnosed with HIV in NSW are detected by testing in sexual health clinics.[16]

We analysed data from sexual health clinics participating in the Australian Collaboration for Coordinated Enhanced Sentinel Surveillance of Sexually Transmissible Infections and Bloodborne Viruses (ACCESS) program.[17] ACCESS includes 44 of the largest sexual health clinics in Australia, with centres in all states apart from South Australia.[18] The clinics collect data in computerised medical record systems as part of routine care; de-identified line-listed data are regularly transferred to the Kirby Institute in Sydney.

Patient cohort

Patients were included if the ACCESS program recorded that they were newly diagnosed with HIV infection between 1 January 2004 and 30 June 2015 (cohort censoring date: 31 December 2015). Patients were followed for a maximum of 183 days (6 months) after diagnosis. Patients were excluded from the primary analysis if their initial $CD4^+$ cell count was not recorded, there was a record of HIV management at another service, or their $CD4^+$ cell count was not monitored further and antiretroviral therapy was not commenced at the service within 390 days (one year, consistent with the international definition of "retention in care",[19] plus one month).

Study outcome

The primary outcome was early treatment, defined as antiretroviral therapy initiated within 183 days (6 months) of diagnosis with HIV infection. Treatment initiated after 6 months was defined as late treatment.

Variables included in the analysis

- *Demographic*: Age, sex, remoteness of residence according to standard classifications (major cities, regional or remote areas),[20] and country of birth (Australia/New Zealand, other).
- *Clinical*:
  - $CD4^+$ cell counts were analysed in two ways. First, the parameters $CD4^+$ cell count at diagnosis (first $CD4^+$ count after

and within 90 days of diagnosis but before treatment) and nadir $CD4^+$ cell count prior to treatment (lowest $CD4^+$ cell count after diagnosis and before cohort censoring date or commencement of treatment, whichever was earlier) enabled us to measure the stage of disease at presentation and the extent of immunologic decline before treatment. Second, $CD4^+$ cell counts were categorised as < 350 cells/μL, 350–499 cells/μL, ≥ 500 cells/μL.
  - Viral load at diagnosis was based on the $\log_{10}$ of viral load between 30 days before and after diagnosis (< 3.70, 3.70–4.59, 4.60–4.99, ≥ 5.00 $\log_{10}$ cells/mL). The viral load categories were defined so as to include approximately equal numbers of patients; missing values were assumed to be missing at random and imputed by multivariate ordinal logistic regression.
  - Other clinical variables were diagnoses at any time with hepatitis B (positive hepatitis B virus surface antigen test result) or with hepatitis C (positive hepatitis C virus antibody test result).

- *Risk*:
  - Mode of HIV exposure: male-to-male sex, injecting drug use without male-to-male sex, heterosexual sex, other/unknown.
  - Risk group: recent male-to-male sex (in preceding 12 months), recent injecting drug use (in preceding 12 months), frequent male-to-male sex (more than 20 partners in 12 months or more than five partners in any 3 months of preceding year). The partner number criteria were consistent with Australian MSM sexually transmitted infection testing guidelines.[21]
  - *Time period*: The date of HIV infection diagnosis was grouped into four periods — 2004–2006, 2007–2009, 2010–2012, 2013–2015 — corresponding to changes in clinical guidelines, and ensuring that there were adequate numbers of diagnoses in each period for our analyses.

Statistical methods

Patient characteristics were tabulated by period of diagnosis. Differences in categorical covariates were assessed in χ^2 tests; trends in median values were assessed with the Cuzick method.[22]

The probability of early treatment was assessed in mixed effects logistic regression models with robust variance calculations; "clinic" was included as a random effect because of the clustering of patient data by clinic. Univariate models for the variables listed above were fitted. Potential interactions between $CD4^+$ cell count at diagnosis and demographic, clinical, or risk characteristics with respect to time to treatment were assessed in sensitivity analyses. The associations of covariates with the primary outcome were assessed in Wald tests.

The multivariate model included variables significant in univariate analyses ($P < 0.05$). Interaction terms were included to evaluate time period-specific variations in outcomes associated with $CD4^+$ cell count. Goodness of fit was assessed by computing Anscombe residuals by ordered covariate category (not reported). A reduced interaction model including $CD4^+$ cell count and period of diagnosis assessed the effect of changes in guidelines. Predicted probability of early treatment was plotted by $CD4^+$ cell count at diagnosis and time period. In a sensitivity analysis assessing bias associated with excluding

patients lost to follow-up, these patients were re-classified as receiving "late treatment". Analyses were conducted in Stata 14.2 (StataCorp).

Ethics approval

Ethics approval for ACCESS was granted by the human research ethics committees of St Vincent's Hospital, Sydney (reference, 08/51) and the University of New South Wales (reference, 8076); site-specific assessment approvals were obtained from local research governance offices as required.

Results

Patient characteristics

During 2004–15, 917 people were newly diagnosed with HIV infection; their median age was 34 years (interquartile range [IQR], 27–43 years), 841 (91.7%) were men, 732 (81.9%) lived in major cities, and 520 (56.8%) were born in Australia or New Zealand. Thirty-nine (4.3%) had prior hepatitis B diagnoses, and the proportions were similar across time periods ($P = 0.62$); 22 people

1 Demographic characteristics of 917 patients newly diagnosed with HIV infections, 2004–2015, by period of diagnosis*

Characteristic	2004–2006	2007–2009	2010–2012	2013–2015	Total
Number of patients	88	174	283	372	917
Demographic characteristic					
Age (years), median (IQR)	37 (30–44)	37 (29–44)	33 (27–41)	33 (26–42)	34 (27–43)
< 30	18 (20%)	46 (26%)	94 (33%)	136 (37%)	294 (32.1%)
30–39	34 (39%)	58 (33%)	107 (38%)	117 (32%)	316 (34.5%)
40–49	23 (26%)	47 (27%)	49 (17%)	68 (18%)	187 (20.4%)
≥ 50	13 (15%)	23 (13%)	33 (12%)	51 (14%)	120 (13.1%)
Sex (men)	83 (94%)	156 (89.7%)	257 (90.8%)	345 (92.7%)	841 (91.7%)
Remoteness					
Major cities	63 (72%)	118 (69%)	234 (84%)	317 (88%)	732 (81.9%)
Regional/remote	24 (28%)	53 (31%)	43 (16%)	42 (12%)	162 (18.1%)
Missing data	1	3	6	13	23
Place of birth					
Australia or New Zealand	67 (76%)	106 (61%)	157 (55.5%)	190 (51.2%)	520 (56.8%)
Other	21 (24%)	68 (39%)	126 (44.5%)	181 (48.8%)	396 (43.2%)
Missing data	0	0	0	1	1
Hepatitis B (ever; HBV surface antigen test)					
No	12 (14%)	28 (16%)	54 (19%)	90 (24%)	184 (20.1%)
Yes	4 (5%)	8 (5%)	15 (5.3%)	12 (3.2%)	39 (4.3%)
No record	72 (82%)	138 (79%)	214 (76%)	270 (73%)	694 (75.7%)
Hepatitis C (ever; HCV antibody test)					
No	13 (15%)	32 (18%)	60 (21%)	96 (26%)	201 (21.9%)
Yes	3 (3%)	4 (2%)	9 (3%)	6 (2%)	22 (2.4%)
No record	72 (82%)	138 (79%)	214 (76%)	270 (73%)	694 (75.7%)
Mode of exposure					
Male-to-male sex	48 (54%)	75 (43%)	82 (29%)	102 (27%)	307 (33.5%)
Intravenous drug user	14 (16%)	25 (14%)	31 (11%)	45 (12%)	115 (12.5%)
Heterosexual	9 (10%)	26 (15%)	38 (13%)	44 (12%)	117 (12.8%)
Blood	1 (1%)	0	1 (0.4%)	0	2 (0.2%)
Missing data/other/unknown	16 (18%)	48 (28%)	131 (46%)	181 (49%)	376 (41.0%)
Risk group					
Recent male-to-male sex	66 (75%)	131 (75%)	227 (80%)	301 (81%)	725 (79.1%)
Recent injecting drug use	15 (17%)	27 (16%)	31 (11%)	46 (12%)	119 (13.0%)
High risk status[†]	4 (4%)	40 (23%)	91 (32%)	116 (31%)	251 (27.4%)

HBV = hepatitis B virus; HCV = hepatitis C virus; IQR = interquartile range. * Year category is based on year of first treatment or 183 days after diagnosis (whichever was earlier). † Defined as more than 20 partners over 12 months, or more than five partners over 3 months at any stage during preceding year. ◆

2 Clinical characteristics of 917 patients newly diagnosed with HIV infections, 2004–2015, by period of diagnosis*

Characteristic	2004–2006	2007–2009	2010–2012	2013–2015	Total
Number of patients	88	174	283	372	917
CD4$^+$ cell count at diagnosis (cells/μL), median (IQR)†	520 (370–710)	500 (370–694)	510 (324–666)	510 (350–659)	510 (350–674)
≥ 500	49 (56%)	88 (51%)	148 (52%)	196 (53%)	481 (52%)
350–499	20 (23%)	51 (29%)	58 (20%)	84 (23%)	213 (23%)
< 350	19 (22%)	35 (20%)	77 (27%)	92 (25%)	223 (24%)
CD4$^+$ cell count nadir (cells/μL), median (IQR)	417 (295–571)	440 (285–610)	401 (270–550)	430 (300–578)	420 (290–570)
Viral load at diagnosis (log$_{10}$ cells/mL), median (IQR)	4.93 (4.25–5.03)	4.58 (4.01–5)	4.45 (3.93–5.09)	4.58 (3.78–5.04)	4.58 (3.92–5.00)
< 3.70	13 (18%)	27 (19%)	40 (17%)	77 (23%)	157 (20.1%)
3.70–4.59	12 (16%)	42 (29%)	86 (37%)	85 (25%)	225 (28.8%)
4.60–4.99	19 (26%)	36 (25%)	38 (16%)	81 (25%)	174 (22.3%)
≥ 5.00	30 (40%)	39 (27%)	70 (30%)	85 (26%)	224 (28.7%)
Missing data	14	30	49	44	137
Time to treatment following diagnosis (days), median (IQR)	816 (238–1642)	592 (153–1243)	266 (76–651)	82 (35–266)	216 (54–608)
Within 6 months	15 (17%)	34 (20%)	95 (34%)	197 (53%)	341 (37.2%)
After 6 months but within study period	19 (22%)	48 (28%)	61 (22%)	68 (18%)	196 (21.4%)
Not started within study period	54 (61%)	92 (53%)	127 (45%)	107 (29%)	380 (41.4%)

* Year category is based on year of first treatment or 183 days after diagnosis (whichever was earlier). † 139 patients were lost to follow-up and therefore excluded from the analysis (median cell count at diagnosis, 510 cells/μL; IQR, 355–680 cells/μL). ◆

(2.4%) had prior hepatitis C diagnoses, and the proportions were also similar across time ($P = 0.55$). Most patients (725, 79.1%) reported recent male-to-male sex (251 [27.4%] reported high risk status), and 119 (13.0%) reported injecting drug use (Box 1).

CD4$^+$ cell count and viral load

The median overall CD4$^+$ cell count at diagnosis was 510 cells/μL (IQR, 350–674 cells/μL), and was consistent across the study period ($P = 0.93$) (Box 2). The median count at diagnosis for patients excluded because they were lost to follow-up was 510 cells/μL (IQR, 355–680 cells/μL). The proportion of people with CD4$^+$ cell counts under 350 cells/μL (late diagnosis) was 22% in 2004–06, 27% in 2010–12, and 25% in 2013–15 ($P = 0.34$); the proportion with counts of 350–499 cells/μL was 23% in 2004–06, 29% in 2007–09, and 23% in 2013–15 ($P = 0.18$). The median nadir CD4$^+$ cell count was 420 cells/μL (IQR, 290–570 cells/μL) and was consistent across periods ($P = 0.56$). The proportion of patients with log$_{10}$ viral loads of 5 or more was 40% in 2004–06 and 26% in 2013–15 ($P = 0.08$) (Box 2).

Time to treatment

The proportion of people who commenced treatment within 6 months of diagnosis increased from 17% (15 patients) in 2004–06 to 53% (197 patients) in 2013–15 ($P < 0.001$); the median time to treatment fell from 816 days (IQR, 238–1642 days) in 2004–06 to 82 days (IQR, 35–266 days) in 2013–15. The proportion of patients who commenced treatment later than 6 months but during the study period was 22% in 2004–06 and 18% in 2013–15 ($P = 0.48$) (Box 2).

Predictors of early treatment

The likelihood of early treatment was significantly higher for people with lower CD4$^+$ cell counts or higher log$_{10}$ viral loads at diagnosis, and for those diagnosed during later time periods. Recent injecting drug use was associated with lower likelihood of early treatment (Box 3). In sensitivity analyses, there were no significant interactions between demographic characteristics or risk factors and CD4$^+$ cell count at diagnosis (data not shown). In multivariate analyses, CD4$^+$ cell count, viral load, and period of diagnosis were significantly associated with early treatment (Box 3).

Probability of early treatment

The probability of early treatment increased across time for all categories of CD4$^+$ cell count at diagnosis: from the outset for people with counts below 350 cells/μL, from 2007–2009 for those with counts of 350–500 cells/μL, and from 2010–2012 for people with counts of at least 500 cells/μL. (Box 4). The results of a sensitivity analysis in which patients lost to follow-up were classified as having started treatment late were similar (data not shown).

Discussion

The proportion of patients newly diagnosed at major Australian sexual health clinics with HIV infection for whom treatment

3 Predictors of early treatment: mixed effects logistic regression models, with clinic as random effects covariate

Characteristic	Univariate odds ratio (95% CI)	P*	P†	Multivariate odds ratio (95% CI)	P*	P†
Age (v under 30 years)			0.18			
30–39 years	0.93 (0.64–1.36)	0.73				
40–49 years	0.81 (0.60–1.08)	0.15				
≥ 50 years	1.01 (0.76–1.33)	0.96				
Sex (women)	1.56 (0.97–2.50)	0.06	0.06			
Remoteness (v Major cities)			0.16			
Regional/remote	0.93 (0.60–1.44)	0.76				
Missing	0.52 (0.26–1.02)	0.06				
Born outside Australia or New Zealand	1.34 (0.94–1.92)	0.10	0.10			
CD4$^+$ cell count at diagnosis (v ≥ 500 cells/μL)			< 0.001			< 0.001
350–499 cells/μL	3.18 (2.29–4.41)	< 0.001		2.30 (1.56–3.39)	< 0.001	
< 350 cells/μL	14.2 (9.00–22.3)	< 0.001		9.40 (3.81–23.2)	< 0.001	
Viral load at diagnosis (v log$_{10}$ cells/mL < 3.7)			< 0.001			0.007
3.70–4.59	1.17 (0.71–1.92)	0.54		1.20 (0.69–2.08)	0.52	
4.60–4.99	1.49 (0.92–2.40)	0.10		1.34 (0.96–1.87)	0.09	
≥ 5.00	2.36 (1.65–3.37)	< 0.001		1.80 (1.23–2.65)	0.003	
Hepatitis B (ever)‡	1.27 (0.66–2.43)	0.48	0.48			
Hepatitis C (ever)§	0.98 (0.47–2.02)	0.95	0.95			
Mode of exposure (v male-to-male sex)			0.11			
Intravenous drug use	0.63 (0.37–1.07)	0.09				
Heterosexual sex	1.09 (0.64–1.86)	0.76				
Other	1.50 (0.08–27.5)	0.79				
Missing data	1.02 (0.70–1.49)	0.91				
Recent male-to-male sex	0.71 (0.48–1.05)	0.09	0.09			
Recent intravenous drug use	0.64 (0.45–0.92)	0.017	0.017			
High risk status¶	1.05 (0.84–1.30)	0.69	0.69			
Year of diagnosis (v 2004–06)						< 0.001
2007–09	1.16 (0.83–1.61)	0.40		0.29 (0.07–1.20)	0.09	
2010–12	2.46 (1.79–3.39)	< 0.001		1.36 (0.78–2.37)	0.27	
2013–15	5.51 (3.86–7.88)	< 0.001		8.01 (4.85–13.2)	< 0.001	
Interaction: year of diagnosis × CD4$^+$ cell count at diagnosis						0.001
2007–09 × 350–499 cells/μL				9.34 (2.62–33.3)	0.001	
2007–09 × < 350 cells/μL				3.85 (0.91–16.3)	0.07	
2010–12 × 350–499 cells/μL				3.66 (1.74–7.68)	0.001	
2010–12 × < 350 cells/μL				2.94 (1.37–6.31)	0.006	

CI = confidence interval. * Wald test. † Wald test for homogeneity of categorical covariates. ‡ Based on hepatitis B surface antigen test or current diagnosis. § Based on hepatitis C virus antibody test or current diagnosis. ¶ Defined as more than 20 partners over 12 months, or more than five partners over 3 months at any stage during preceding year. Initial model included "missing data" as a category level (odds ratio, 0.82; 95% CI, 0.78–1.34), but was combined with "not high risk" category in final models. ◆

commenced within 6 months of diagnosis increased during 2004–2015 from 17% to 53%. The probability of early treatment for patients presenting with CD4$^+$ cell counts of 350–499 cells/μL increased steadily from 2007–2009, and the probability for patients with counts above 500 cells/μL increased rapidly during 2013–2015. These patterns are consistent with guideline changes during the 12-year study period. In multivariate analyses, lower CD4$^+$ cell count and higher viral load were associated with early treatment, as was being diagnosed more recently.

4 Probability of early treatment (with 95% confidence intervals), by CD4$^+$ cell count at diagnosis and period of diagnosis
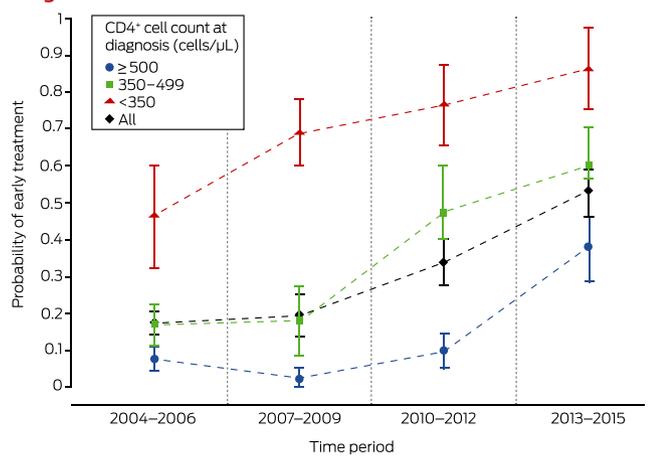
During 2004–2006, in keeping with then current treatment guidelines, patients with advanced infection (lower CD4$^+$ cell count at diagnosis) were treated early, but treatment was deferred for those with less advanced disease. By 2013–2015, an increasing proportion of patients with higher CD4$^+$ cell counts were receiving early treatment. Offering antiretroviral therapy to all patients with HIV infections has been recommended in Australia since June 2015.[23] While the strength of evidence ratings for the influential US guidelines were upgraded in January 2016[24] and some Australian physicians will have anticipated the changes, the final period in our study included patients diagnosed before they were published. We therefore expect further increases in the median CD4$^+$ cell count at diagnosis and the pre-treatment nadir.

No socio-demographic subgroups, risk factors, or comorbid condition included in our analysis were associated with early treatment after adjustment for CD4$^+$ cell count, viral load and period of diagnosis. In Australia, HIV treatment is subsidised by the Pharmaceutical Benefits Scheme (PBS), relieving financial impediments to treatment, although co-payments are required in some states. People who have recently migrated to Australia cannot routinely access the PBS; during the study period, an alternative access scheme that provided pharmaceutical company-supported antiretroviral treatment to such individuals was established at certain sites.[25] However, aspects not assessed by our study may have been associated with delayed treatment, such as stigma and other psychosocial factors that are associated with lower rates of treatment coverage and adherence.[26,27]

Limitations

First, the clinics in the ACCESS network may not be representative of all sexual health clinics in Australia, nor their clientele of all patients diagnosed in Australia with HIV infection. However, ACCESS includes most large Australian sexual health clinics, and therefore probably also most of the patients diagnosed at Australian sexual health clinics with HIV infections. Second, patients lost to follow-up soon after diagnosis were excluded from analysis because of the insufficient data on treatment uptake. While loss to follow-up can indicate disengagement from clinical care (and treatment not being initiated), some patients may have engaged with clinical care at other centres or returned to their home countries for care.[28] The median CD4$^+$ cell count of these patients was similar to that for the overall cohort, suggesting that their stage of disease at presentation was also similar. The results of a sensitivity analysis that regarded patients lost to follow-up as not having received early treatment early were similar to those of the primary analysis.

Conclusion

The proportion of people newly diagnosed in sexual health clinics in Australia with HIV infection who received treatment within 6 months of diagnosis increased from 17% to 53% during 2004–2015, reflecting changes in the CD4$^+$ cell count threshold in treatment guidelines. Our findings provide a baseline for assessing the impact of fully implementing the 2015 Australian guideline recommendations that all newly diagnosed patients should receive early treatment, regardless of CD4$^+$ cell count. They also provide information that will help understand gaps in the cascade of care, including in the response to changes in guidelines, which was independent of patient socio-demographic characteristics, risk factors, and clinical comorbidity.

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