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High Rates of Sexually transmitted infections in HIV-positive patients in the Australian HIV Observational Database - a prospective cohort study

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Abstract

Background—Sexually transmitted infections (STI) may directly increase the risk of HIV infection, or may indicate sexual practices that increase the risk of HIV infection. In persons with HIV they probably also increase the infectiousness of HIV, even in the context of treatment with antiretroviral drugs (ARV). Estimating STI in this group has proved problematic, and there are few longitudinal studies able to accurately measure incidence.

Methods—In 2010, we established a cohort of individuals from ten widely dispersed sexual health clinics that were already enrolled in the Australian HIV Observational Database (AHOD). We calculated retrospective diagnosis rates for four STI (chlamydia, gonorrhoea, infectious syphilis, anogenital warts) from 2005-2010, and prospective incidence rates from 2010-2011.

Results—At baseline (2010), the patient characteristics (n=554) were similar to the rest of AHOD (n=1767), namely they were predominantly male, homosexual, middle-aged, and pre-treated with ARV. Overall, the incidence of any STI was 12.5/100 person years (py). There was a gradual increase in chlamydial infections, from 3.4/100 py (95% CI 1.9-5.7) in 2005 to 6.7/100py (95% CI 4.5-9.5) in 2011, with a substantial peak of 8.1/100py (95% CI 5.6-11.2) in 2010. The cases were distributed between rectal (61.9%), urethral (34%), and pharyngeal (6.3%) sites. Similarly, gonococcal infections increased, with a peak in 2010 of 4.7/100py (95% CI 5.6-11.2), (p value for trend=0.0099), distributed between rectal (63.9%), urethral (27.9%), and pharyngeal (14.8%) sites. Infectious syphilis showed several peaks, the largest in 2008 (5.3/100py, 95% CI 3.3-8.0), but the overall trend was not significant (p=0.113). Diagnoses of genital warts declined from 7.5/100py (95% CI 4.8-11.3) in 2005 (95% CI 4.8-11.3) to 2.4/100py (95% CI 1.1-4.5) in 2011 (p value for trend=0.0016).

Conclusions—The incidence of chlamydial and gonococcal infections in this cohort was higher than previous estimates in Australia among HIV-infected men who have sex with men (MSM), and increased during the 2005-2011 study period. Rectal infections greatly outnumbered infections at other sites. The incidence of infectious syphilis remained high, but did not increase, and the incidence of genital warts was lower, and decreased. These are the first measurements of STI incidence among persons being treated for HIV in Australia. They may underestimate the true incidence of these infections in the HIV-infected population.

Introduction

Over the last 20 years a wealth of evidence has accumulated to support the strong amplification effect of STI on the acquisition and infectiousness of HIV, despite the apparent failure of intervention trials (1-4). Recently, it has been shown that treatment with ARV can reduce HIV transmission in certain circumstances (5-14), but some doubt exists whether this efficacy persists in the presence of concurrent STI (15-18), and possible associations between ARV treatment and STI incidence have not been explored.

In Australia, new HIV diagnoses occur predominantly among MSM, a population in whom STI rates have been increasing for several years, most dramatically with infectious syphilis, particularly among those who are HIV-positive (19-25).

Here, we describe the incidence of STI in HIV-infected patients by using data from individuals from sexual health clinics who had previously enrolled in the Australian HIV Observational Database (AHOD). This is a well-studied prospective cohort, and thus accurate for measuring trends in STI incidence. In this preliminary analysis, we describe patient characteristics at recruitment (baseline), and present temporal STI rates for 2005-2011.

Methods

AHOD has been described elsewhere in detail (26-28). Briefly, AHOD data collection commenced in 1999 and currently 27 hospitals, sexual health clinics, and general medical practices throughout Australia contribute data every 6 months. At March 2011, over 3000 patients had been recruited to AHOD, and over 2000 were under active follow-up. Data are

collected on a core set of variables including sex, age, HIV exposure category, hepatitis B virus surface antigen (HBV), hepatitis C antibody status (HCV), CD4 and CD8 cell counts, viral load, ARV history, AIDS illnesses, date and cause of death. Additional ethics approval was sought for the current sub-study and approved by all local Human Research Ethics Committees.

Sexual health clinics within AHOD were invited to provide retrospective data for 2005-2010, and prospective data thereafter. Besides the core AHOD data above, extra STI-specific data variables were extracted from each clinic database and sent electronically to the Kirby Institute twice annually. They included confirmed diagnoses of STI, (infectious syphilis, chlamydia, gonorrhoea, and genital warts), site of infection, STI treatment, and injecting drug use. Specifically, specimens were taken according to symptoms, and/or adherence to guidelines for routine screening in MSM (29), that included blood, urine, and swabs from ano-genital and pharyngeal sites. They were tested using nucleic acid amplification tests for chlamydia and gonorrhoea, and in the latter case, included supplemental testing, and/or culture, as per current guidelines (30,31).

For syphilis, a positive screening enzyme immunoassay (EIA) was confirmed with the *Treponema pallidum* particle agglutination assay and/or the fluorescent treponemal antibody absorption test. The rapid plasma reagin (RPR) test was used to assist clinical staging and to detect re-infection. Infectious syphilis was defined using conventional criteria (32).

We tabulated patient characteristics and evaluated differences between AHOD-STI participants and the remaining AHOD population. The extra variables had been collected by some clinics from 1999, and from others since that time. Inspection of eight of the ten databases that used the same data capture mechanism showed that all were consistently recording these STI data by 2005. Therefore, it was possible to calculate diagnosis rates for all STIs and selected STIs, from 2005-2011. We calculated temporal STI diagnosis rates 2005-2011 by summing the total number of patients diagnosed with an STI in a given calendar year divided by the total number of patient years observed in the same period. We used Poisson confidence intervals to evaluate the uncertainty of the calculated rate. Rates are expressed per 100 person-years, and approximate very closely true incidence rates, which by convention are usually restricted in cohort studies to prospective data from baseline, (in this case, 2010), to reflect potentially more uniformly collected data of better quality. We used the Cochran-Armitage statistic to test for a p-trend over the same period.

Results

At baseline (Jan 2010), ten sexual health clinics that had patients already enrolled in AHOD were recruited (four metropolitan, four regional, and two rural). The ten clinics had been part of AHOD since 1999 (total number of patients ever enrolled=773), and the current study population consisted of 554 patients for the AHOD-STI substudy. The demographics and other characteristics of the sub-cohort were compared with the rest of the AHOD cohort (n=1767), and were similar, namely broadly representative of the parent cohort (Tables 1 and 2). Most were male, (94%), homosexual (75%), middle-aged (median 42.4yrs, q1 27.9-q3 49.7yrs), and with an average of 6.5 years of follow up. Just under half had a CD4 count

of >500 cells per microliter, and 60% had an undetectable viral load (lower limit of detection <400 copies/ml, latterly, the assay was more sensitive, <50 copies/ml). The times since HIV diagnosis were <10yrs (30%), 10-20yrs (40%), and 20-30yrs (20%). 85% had been pre-treated with ARV.

Combined retrospective and prospective data showed that at March 2012, there had been 143 diagnoses of STI in 87 persons, of whom thirty had more than one STI diagnosis during the observed period. Anatomical information on the site of infection was available for 56% of chlamydial diagnoses and 92% of gonorrhoea diagnoses.

Figure 1 shows a gradual increase in chlamydial infections, from 3.4/100 person-years (py) (95% CI 1.9-5.7) in 2005, to 6.7/100py (95% CI 4.5-9.5) in 2011, with a substantial peak in 2010, 8.1/100py (95% CI 5.6-11.2) (p value for trend=0.0001). The cases were distributed between rectal (61.9%), urethral (34%), and pharyngeal (6.3%) sites. Similarly, gonococcal infections increased, with a peak in 2010 (4.7/100py, 95% CI 5.6-11.2), with rectal (63.9%), urethral (27.9%), and pharyngeal (14.8%) infections (p value for trend=0.0099). Infectious syphilis showed several peaks, the largest in 2008 (5.3/100py, 95% CI 3.3-8.0). Although this was the highest rate ever recorded in Australia, the trend over the six-year period was not significant, p=0.113). The incidence of genital warts declined from 7.5/100py in 2005 (95% CI 4.8-11.3) to 2.4/100py in 2011 (95% CI 1.1-4.5), p value for downward trend=0.0016)

Discussion

We found new infections with chlamydia and gonorrhoea to be frequent and increasing over the time period 2005-2011 in a representative sample of patients enrolled in AHOD. Rectal infections greatly outnumbered infections at other sites. The incidence of syphilis was high, but did not increase, whilst new infections with genital warts decreased.

The strengths of our study, compared with previous studies, lie chiefly in its carefully defined characteristics. The prospective data (from 2010) are amenable to precise calculations of STI incidence, and lend themselves to subsequent studies of predictive factors for STI incidence, as well as possible bi-directional effects. The retrospective data are also unusually useful, because patients were already part of the parent AHOD cohort, with attendant checks on quality control.

There are limitations to our study design, which was added to the parent cohort, which is primarily a study of patterns of treatment change.

First, although our sample is representative of the parent cohort (AHOD), the latter may not be representative of the general population with HIV infection. However, the trends in incidence are likely to be indicative.

Second, we did not collect data on the presence or absence of symptoms, nor testing rates. In a previous cross-sectional Australian study of STI in HIV, all diagnoses of chlamydial or gonococcal infection were symptomatic (25), although the results may also have indicated low testing rates in asymptomatic individuals presenting only for ARV repeat prescriptions.

It is well known that testing rates influence the true incidence of STI infections in any population, (the more you test the more you find). Our subset is a special population, drawn exclusively from specialized sexual health clinics, in which clinicians adhere closely to their own national guidelines for STI screening in MSM, that recommend testing at least once a year, regardless of symptoms (29). The importance of testing rates and patterns are probably restricted mainly to infections that are usually asymptomatic, such as rectal chlamydial infection, and pharyngeal gonorrhoea. The dates of testing were not collected in the present study, and the proportion of our sample actually tested for chlamydia can only be inferred from previous studies on very similar populations. For example, the ten clinics in the current analysis were part of a different study of eighteen sexual health clinics, which showed a modest increase 2004-2008 in the proportion of new MSM attendees who had been tested for chlamydia, from 71% to 79% (33). Another investigation in one of the present clinics supported the assertion that increased notification rates in MSM may be due to increased screening, at least for chlamydial rectal infections (34)

A third limitation is our lack of information on incident genital herpes infections. There is strong evidence of the interaction between incident herpes infection and raised plasma HIV viral loads, although trials of treatment interventions have not been successful in preventing HIV transmission. (4). Our initial enquiries revealed potential inconsistencies and misclassification bias in collection of herpes data across sites, and we hope to address this deficiency in future analyses.

Finally, our study lacks data on sexual behaviour.

Previous studies of STI in HIV-infected patients have generally used estimates of *prevalence*, and have been hampered by non-standardised procedures. A review of 37 studies found a 12% median point-prevalence of confirmed STI, and the most common were those that cause HIV shedding (35). There have been very few studies that measured *incidence* of STI, or in patients on ARV. In Baltimore , (36) , HIV-infected patients had an incidence of 7/100py for any STI, compared with a mean of 12.5/100py in the current study (data not shown). Infectious syphilis had an incidence of 6.2/100py in New York (37) and 3.6/100py in Sydney (23);, and in our study, it ranged from 0.9-5.3/100py (see Figure 1). Incidence data on chlamydia and gonorrhoea in HIV-infected patients are unavailable, but our estimates are higher than those in non-HIV infected Australian MSM (21), or inferred from chlamydia positivity rates in MSM who were first-time attendees at 25 Australian sexual health clinics (33). Similarly, there are no incidence data on anogenital warts in HIV-infected patients, but our estimates are lower (for reasons that are unclear) than those measured in non-HIV infected Australian MSM (38) . Very recent data show that there was a modest decline (24%) in the proportion of new MSM attendees who presented with genital warts between 2007-2011 (39). This was not seen in bi-sexual men and therefore the authors considered it more likely not due to herd immunity, reflecting instead the increased numbers of asymptomatic MSM successfully triaged for screening under national guidelines.

We believe there is consensus that the increased incidence of some STI is due to the increased prevalence of unprotected anal intercourse (UAI) in both HIV-negative, and HIV-positive MSM (40, 41). Why this increased prevalence of UAI has not translated into a

similar increase in HIV incidence is unclear, though it should be noted that HIV diagnoses in Australia have continued to increase over the last decade (19). Variations in sexual behaviour strategies in MSM such as “sero-sorting”, and “strategic positioning”, which aim to minimize HIV transmission risk, may account for the differential effects upon HIV- and STI- transmissions. It is quite possible that these behavior strategies are effective at reducing HIV transmission risk but have no effect on STI transmission, as has been suggested (42,43). The increased rate of UAI would therefore tend to push up STI transmission among MSM. Finally, the increased use of recreational drugs in certain groups of MSM may be an important factor. Drug use seems linked to dis-inhibiting behaviours and pushing boundaries to seek new sexual experiences, including fisting and scat play. Ecstasy, cocaine, ketamine, speed, mephedrone, and particularly methylamphetamine (the latter in pill, or powdered form for intravenous use-“slamming”) increase the likelihood of chaotic and ill-defined sexual behaviours, especially when used for prolonged periods and in group situations (44,45), with the potential for conventional and other STI and/or HIV transmission (40,46,47).

However, we do not have information on drug use, and proving any of these hypotheses is beyond the scope of the current study.

Scientific uncertainty surrounds STI/HIV interactions and the transmission of HIV. Randomised controlled trials, considered the gold standard of proof of efficacy for STI interventions to prevent HIV, have failed to provide that proof, possibly due to methodological issues, and in any case, could never satisfactorily separate HIV acquisition from onward HIV transmission. Moreover, it is apparent that some STIs are not only markers of risky sexual behaviour, but also considerably increase genital tract shedding of HIV. This increase in HIV-shedding could potentially exceed the inoculum needed for HIV transmission, even in the context of suppression of HIV suppression by ARV. In general terms, these concepts of infectiousness despite ARV treatment were foreseen in the original “Swiss statement” (48), and have more recently been repeated in a “British statement” (49). It is clear that the role of STIs in onward transmission from HIV-infected individuals, as well as their relationships with other parameters such as ARV treatment, viral load, CD4 count, and other factors, will be the subject of close scrutiny for the foreseeable future.

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AHOD-STI participating sites:

New South Wales: Holden Street Sexual Health Clinic, Gosford; Lismore Sexual Health & AIDS Services, Lismore; RPA Sexual Health, Camperdown; Blue Mountains Sexual Health and HIV Clinic, Katoomba; Tamworth Sexual Health Service, Tamworth; Nepean Sexual Health and HIV Clinic, Penrith; Illawarra Sexual Health Service, Warrawong; Sydney Sexual Health Centre, Sydney;

Queensland: Cairns Sexual Health Service, Cairns;

Victoria: Melbourne Sexual Health Centre, Melbourne

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Figure 1.
Rates of STI Diagnoses, 2005 – 2011.
(Rates are expressed per 100 person-years, with 95% Poisson confidence intervals)

Table 1

Characteristics of patients not participating and participating in the AHOD STI sub-study, at enrolment (2010)

	AHOD	
	Non-participating patients, N (%)	STI sub study patients, N (%)
Sex		
Male	1668 (94.4)	520 (93.9)
Female	99 (5.6)	34 (6.1)
Age		
mean	42.9	43
median(q1,q3)	41.8 (35.8 - 49.3)	42.4 (35.1 - 49.7)
Age at HIV diagnosis		
mean	35.6	35.6
median(q1,q3)	34.2 (28.1 - 41.7)	33.9 (27.9 - 41.71)
HIV exposure		
Homosexual	1342 (75.9)	417 (75.3)
Heterosexual	176 (10)	62 (11.2)
Injecting Drug Use	66 (3.7)	35 (6.3)
Other	159 (9)	34 (6.1)
Unknown	24 (1.4)	6 (1.1)
Hepatitis B (sAg +ve)		
Yes	58 (3.3)	20 (3.6)
No	1487 (84.2)	411 (74.2)
Not tested	222 (12.6)	123 (22.2)
Hepatitis C (HCV Ab +ve)		
Yes	172 (9.7)	57 (10.3)
No	1424 (80.6)	469 (84.7)
Not tested	171 (9.7)	28 (5.1)
AIDS since enrolment		
Yes	153 (8.7)	49 (8.8)
No	1614 (91.3)	505 (91.2)
CD4 at enrolment		
0-199	161 (9.1)	48 (8.7)
200-349	273 (15.4)	123 (22.2)
350-499	424 (24)	145 (26.2)
>500	882 (49.9)	229 (41.3)
Unknown	26 (1.5)	9 (1.6)
Viral load at enrolment		
0-400	1150 (65.1)	308 (55.6)
400-10,000	284 (16.1)	100 (18.1)
10,000 - 100,000	245 (13.9)	111 (20)
>100,000	87 (4.9)	32 (5.8)
Unknown	0 (0)	2 (0.4)

Table 2

Characteristics of patients not participating and participating in the AHOD STI sub-study, at enrolment (2010)

	AHOD	
	Non-participating patients, N (%)	STI sub study patients, N (%)
Original enrolment		
1. 1999-2004	1019 (57.7)	290 (52.3)
2. 2005-2011	748 (42.3)	264 (47.7)
First HIV diagnosis		
1. 1980-1989	348 (19.7)	120 (21.7)
2. 1990-1999	730 (41.3)	209 (37.7)
3. 2000-2012	595 (33.7)	220 (39.7)
4. Unknown	94 (5.3)	5 (0.9)
Year ARV initiation		
0. Naïve/mono/duo	129 (7.3)	78 (14.1)
1. 1996-1999	844 (47.8)	222 (40.1)
2. 2000-2004	273 (15.4)	90 (16.2)
3. 2005-2012	521 (29.5)	164 (29.6)
Most recent CD4 count		
1. 0-199	84 (4.8)	28 (5.1)
2. 200-349	187 (10.6)	60 (10.8)
3. 350-499	351 (19.9)	128 (23.1)
4. >500	1144 (64.7)	336 (60.6)
5. Unknown	0 (0)	2 (0.4)
Most recent viral load		
1. 0-400	1601 (90.6)	458 (82.7)
2. 400-10K	65 (3.7)	33 (6)
3. 10K-100K	72 (4.1)	43 (7.8)
4. >100K	28 (1.6)	17 (3.1)
5. Unknown	0 (0)	2 (0.4)