HIV-2 in West Africa. Epidemiological studies
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Contrasts in plasma viral load, CD4% and survival in a community-based cohort of HIV-1 and HIV-2 infected women in The Gambia

Submitted

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Contrasts in plasma viral load, CD4% and survival in a community-based cohort of HIV-1 and HIV-2 infected women in The Gambia

ABSTRACT

Objectives: To estimate and compare all-cause mortality rates of HIV-1, HIV-2 and uninfected women; to assess the predictive value of baseline plasma viral load (PVL) and CD4% for mortality.

Methods: On presentation to antenatal clinics in The Gambia in 1993-5, pregnant women were screened for HIV-1 and HIV-2 antibodies. Seropositive subjects and a similar number of seronegative controls were enrolled and baseline PVL and CD4% measured. Participants were visited regularly by field workers until 18 months post-delivery and again 4-7 years later.

Findings: Thirty-two of 101 women infected with HIV-1, 23/243 with HIV-2 and 9/468 seronegative women died during a median follow-up period of 6.9 years. The mortality rate was 55 per 1000 person years-of-observation (pyo) for HIV-1 infected, 16 for HIV-2 infected, and 3.1 for HIV uninfected women. In a multivariate analysis, one log increase of PVL was associated with a 1.8 fold higher mortality rate in HIV-1 (95% Confidence Interval (CI) 0.9-3.4). In HIV-2 infection women with a high PVL (>10,000 copies/ml) had an 8.7 (95%CI 2.8-28) higher mortality than those with a low PVL (<1,000 copies/ml). A 10% decrease in CD4% was associated with higher mortality rates in HIV-1 (1.6-fold; 95%CI 1.1-2.3) and HIV-2 infected subjects (1.5-fold; 95%CI 1.0-2.3).

Interpretation: Survival of HIV-1 infected women in The Gambia is similar to that in industrialised countries prior to the introduction of anti-retroviral treatment. Survival of HIV-2 infected women is much better. However, those with high PVL die as quickly as HIV-1 infected women.
HIV-2 in West Africa

Introduction

In Sub-Saharan Africa two types of HIV are prevalent and cause disease. HIV-1 is epidemic across the continent with 29 million Africans infected, but HIV-2 is mainly limited to West Africa with an estimated 1 million people infected. In general, HIV-2 infection has been associated with substantially lower rates of transmission, disease progression, and mortality compared to HIV-1.

In industrialised countries there are good data on natural history and survival of HIV-1 infection in men prior to the use of highly active antiretroviral treatment (HAART). Median survival is between 9 and 13 years depending on age of infection.

Longitudinal data on survival with HIV-1 infection in Africa are rare, and there are very few incident cohort studies. In a sero-incident cohort in rural Uganda, Morgan et al. estimated a median survival time from seroconversion of 9.8 years. A community-based seroprevalent cohort study in Malawi recently reported a median survival time of 8 years.

Although the natural history in African adults infected with HIV-2 is less well described, studies to date indicate disease progression is much lower compared to those infected with HIV-1. Two community-based studies and one occupational cohort study from Guinea-Bissau, have reported 2.3 to 6.6 times higher mortality rates in HIV-2 infected adults compared to uninfected adults. These rates contrast with an 11-fold higher mortality rate in HIV-1 infected compared to seronegative adults in a rural community-based study in East Africa and a 9-fold higher mortality in a community-based study in women in Rwanda.

Plasma viral load (PVL) and CD4 count are independent predictors of mortality in HIV-1 infection, and a reduction of PVL through HAART is strongly associated with improved survival. There are no community-based studies of HIV-1 in sub-Saharan Africa that have assessed the predictive value of PVL and CD4 count. A community-based study of HIV-2 in Guinea-Bissau showed that PVL and CD4 count are clinically important independent predictors of mortality in HIV-2 infection.
HIV-2 IN WEST AFRICA

This study compares the mortality rates in HIV-1 infected, HIV-2 infected and uninfected adult women in The Gambia and assesses the predictive value of PVL and CD4% for mortality in both infections.

Methods

Subjects
The women in this cohort were recruited between 1993 and 1995 during a study estimating the mother-to-child-transmission (MTCT) of HIV-1 and HIV-2 in The Gambia (O'Donovan 2000). After delivery, mothers and children were visited at 2, 6, 9, 12, 15, and 18 months by a field worker or clinician.4,20

Follow-up
Between July 9th and November 1st 2001 three experienced and well trained counsellors / field workers from the genito-urinary clinic (GU) at the MRC in Fajara, the national referral centre for HIV care, revisited all women in their homes. The counsellors were blinded to the HIV status of the women. Study participants were asked for verbal informed consent, which was documented. If a woman was known to be alive but not seen at her compound, the date she was last seen alive by a close relative was recorded. If the woman was temporarily absent, she was revisited. Women who had moved within The Gambia, were visited at their new address. For women who could not be traced, or on whom no information could be obtained in the follow-up in 2001, the last date she was known to be alive was obtained from earlier records. Where available, data from these follow-up and clinic visits were included in our analysis. To check the quality of the collected data, 5.3% of the women were visited twice in 2001 by two different field workers.

Ethics
In the initial screening 1993-5, all women were counselled and only those agreeing to an HIV test, were enrolled. The test results were available two weeks later at the same clinic from an HIV counsellor for women who wanted to know their HIV status, in accordance with the National
AIDS Policy. Very few women took advantage of this opportunity. In the follow-up in 2001 the results of the old HIV test were not provided, since the woman's sero-status might have changed in the meantime. Instead, a new test was offered to all women. The study team offered free medical treatment and fares to the next health centre to any women or other person found to be sick in the household. Women who wanted to be re-tested and who were found to be HIV infected were offered free care at the MRC GU clinic. HIV-1 infected women who were pregnant were offered short course nevirapine to prevent mother-to-child transmission of HIV-1. In The Gambia anti-retroviral treatment is not yet available. The study was approved by the joint Gambian Government-MRC Ethics Committee (Project Number 868/825).

**Laboratory methods**

Serology, CD4 cell counting and plasma viral load assays were performed as described previously. Briefly, screening for HIV infection was done in pools of ten, using a combined enzyme immunoassay (EIA) for HIV-1 and HIV-2 (Wellcozyme HIV Recombinant, Murex Diagnostics Ltd, Dartford, UK). Individual sera from a positive pool were re-tested in the same assay. Those found to be positive were tested with the Wellcozyme HIV-1 and HIV-2 specific EIAs (Murex Diagnostics Ltd). Seropositivity was confirmed using a combined HIV-1 and HIV-2 peptide-based EIA (Pepti-LAV 1-2, Sanofi Diagnostic Pasteur SA, Marnes la Coquette, France). To determine CD4 cell percentages, whole blood fluorescent antibody cell sorting (FACS) analysis (FACScan, Becton Dickinson, Oxford, UK) was used, adapted for field use. Virus loads were assayed in heparin-plasma samples stored at −80°C. RNA was extracted by the method of Boom et al., reverse-transcribed and PCR-amplified using primers targeted to the LTR region of HIV-1 or HIV-2, and PCR products quantified in a microtitre-format hybridisation assay. The lower limit of detection of these assays was 500 copies per ml.

**Statistical methods**

The data were double entered. Analysis was done with Stata version 6 (Stata Corporation, College Station, TX, USA). Person years of observation (pyo) were calculated from the time people were enrolled until 1st July 2001 or until the date of death or until the last date known to be alive, whichever came first. Mortality rates were calculated as deaths per 1000 pyo, with 95%
Confidence Intervals (CI). Data on 10 HIV-1 and HIV-2 dually infected women were excluded from this analysis.

Time to death was examined by Kaplan-Meier graphs. Mortality rates were compared by strata of CD4% and of PVL, and Mantel-Haenszel mortality rate ratios calculated to adjust for these. Poisson regression analysis was used for multivariate analysis of mortality rates. Because mortality rates increased with increasing time since recruitment, the observation period of 8.4 years was split in three time periods (0-2.99 years; 3-5.99 years; and 6-8.4 years) and time period was one of the factors adjusted for in the Poisson regression.

Role of the funding sources
The funding sources had no role in the data collection, analysis, or interpretation, nor in the writing of the report or the decision to publish it.

Results

Baseline characteristics
The baseline characteristics of the 812 women at the time of recruitment (Table 1) show that HIV-1 positive women were younger than HIV-2 infected women (mean age 23.4 years vs. 27.0 years (p<10^-4) and younger than HIV-negative women (mean age 25.8 years (p=0.0002)). HIV-1 infected women had lower median baseline CD4% (33%) compared to HIV-2 infected women (42%, p<10^-4) and uninfected women (48%, p<10^-4). The median PVL, assayed in 94 HIV-1 infected women, was 13,600 copies/ml (range 250-265,000). The median PVL of 228 HIV-2 infected women was 500 copies/ml (range 250-63,000), which was 27 fold lower (p<10^-4). Comparison of PVL distributions showed 6% and 61% of HIV-1 and HIV-2 infected subjects respectively had loads below 1000 RNA copies/ml and 9% and 0% respectively had loads above 100,000 copies/ml (Figure 1).
### Table 1. Baseline characteristics of 812 women at time of recruitment

<table>
<thead>
<tr>
<th>HIV status of woman</th>
<th>HIV-negative</th>
<th>HIV-1</th>
<th>HIV-2</th>
</tr>
</thead>
<tbody>
<tr>
<td>n = 468</td>
<td>n = 101</td>
<td>n = 243</td>
<td></td>
</tr>
<tr>
<td>Mean age (SD)</td>
<td>25.8 (5.7)</td>
<td>23.4 (5.5)</td>
<td>27.0 (5.5)</td>
</tr>
<tr>
<td>Median CD4% (range)</td>
<td>48 (8-68) b</td>
<td>33 (1-57) c</td>
<td>42 (5-66) d</td>
</tr>
<tr>
<td>Number (%) of women with:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CD4 &lt; 14%</td>
<td>3 (3%)</td>
<td>6 (7%)</td>
<td>10 (5%)</td>
</tr>
<tr>
<td>CD4 14 – 28%</td>
<td>4 (3%)</td>
<td>26 (30%)</td>
<td>18 (8%)</td>
</tr>
<tr>
<td>CD4 &gt; 28%</td>
<td>112 (94%)</td>
<td>56 (64%)</td>
<td>188 (87%)</td>
</tr>
<tr>
<td>Plasma viral load:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>median RNA copies / ml (range)</td>
<td>---</td>
<td>13,600 e</td>
<td>500 f</td>
</tr>
<tr>
<td>Number (%) of women with:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 1,000 RNA copies/ml</td>
<td>---</td>
<td>6 (6%)</td>
<td>140 (61%)</td>
</tr>
<tr>
<td>1,000 – 9,999 RNA copies/ml</td>
<td>---</td>
<td>26 (28%)</td>
<td>60 (26%)</td>
</tr>
<tr>
<td>10,000 – 99,999 RNA copies/ml</td>
<td>---</td>
<td>54 (57%)</td>
<td>29 (13%)</td>
</tr>
<tr>
<td>≥ 100,000 RNA copies/ml</td>
<td>---</td>
<td>8 (9%)</td>
<td>0 (0%)</td>
</tr>
</tbody>
</table>

Notes: a. age not available for one subject; b. available for 119 subjects; c. available for 88 subjects; d. available for 216 subjects; e. available for 94 subjects; f. available for 229 subjects. SD = standard deviation. Seronegative women were selected matched for age to HIV-infected women. Percentages do not add always up to 100% due to rounding. All differences between the three groups P < 0.01.

### Follow-up

During the follow-up exercise between July and November 2001, 655 of the 812 women (81%) were found to be alive, 64 (8%) had died, 8 (1%) had refused further follow-up prior to 2001 and 85 (10%) were lost during follow-up (Table 2). The rate of loss during follow-up was 17 (95%CI 14-21) per 1000 pyo. The median follow-up time was 6.9 years (range: 3 months to 8.4 years). No significant differences were found in HIV status, age, CD4%, PVL, or parity between women who refused or who were lost during follow-up and women with known survival status,
but those lost had had less education, were more often of the Jola ethnic group, were more often from the coastal area, and tended to have a poorer water supply (data available upon request).

For quality assurance purposes 43 women (5.3%) were visited on two occasions in 2001 by different fieldworkers. No discrepancies in recorded survival status occurred. There were four discrepancies in the date the woman was last known to be alive (median difference 10 months); this was caused by interviewing different informants. Despite intensive counselling by the fieldworkers, only twelve women opted to have a new HIV test.
<table>
<thead>
<tr>
<th></th>
<th>HIV status</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>seronegative</td>
</tr>
<tr>
<td>Women enrolled in 1993-5</td>
<td>468</td>
</tr>
<tr>
<td>Refusals since enrolment (%)</td>
<td>1 (0.2%)</td>
</tr>
<tr>
<td>Died (%)</td>
<td>9 (2%)</td>
</tr>
<tr>
<td>Lost to follow-up (%)</td>
<td>56 (12%)</td>
</tr>
<tr>
<td>Alive and traced (%)</td>
<td>402 (86%)</td>
</tr>
<tr>
<td>Observation time in years</td>
<td>2882</td>
</tr>
<tr>
<td>Mortality rate per 1000 pyo (95% CI)</td>
<td>3.1 (1.6-6.0)</td>
</tr>
<tr>
<td>Mortality rates per 1000 pyo (95% CI) by agegroup</td>
<td></td>
</tr>
<tr>
<td>age &lt; 25 years</td>
<td>2.8 (0.89-8.6)</td>
</tr>
<tr>
<td>age 25 - 29 years</td>
<td>2.0 (0.51-8.1)</td>
</tr>
<tr>
<td>age ≥ 30 years</td>
<td>4.9 (1.8-13)</td>
</tr>
</tbody>
</table>

Percentages do not always add to 100% because of rounding. pyo = person-years of observation; CI = confidence interval.
Mortality

Thirty-two out of 101 HIV-1, 23 of 243 HIV-2 and 9 of 468 seronegative women died. The mortality rates were 56 per 1000 pyo in HIV-1 (95%CI: 39-79), 16 in HIV-2 (95%CI: 10-23) and 3.1 in sero-negative women (95%CI: 1.6-6.0). Table 2 and Figure 2 compare the mortality rate and survival probability of the three groups. The mortality rate of women with HIV-1 infection was 18-fold higher (95%CI 8.5-37; p<10^-4) than that of HIV uninfected women. The mortality rate of women with HIV-2 was 5.0 fold higher (95%CI 2.3-11; p<10^-4) than that of HIV uninfected women and 3.6 fold lower (95%CI 2.1-6.1; p<10^-4) than of HIV-1 infected women. There were no significant differences in mortality rate by age group in HIV-1 infected (p=0.6), HIV-2 infected (p=0.11) or seronegative women (p=0.5; see Table 2). The mortality rate increased over time. This trend was significant overall (p=0.001), in HIV-1 infected women (p=0.02), in HIV-2 infected women (p=0.005), but not in HIV-negative women (p=0.5).

Mortality by PVL and CD4 %

Among HIV-1 infected study subjects the mortality rate increased with the log of PVL, though not significantly so (p=0.09) (Table 3); among HIV-2 infected women the mortality rate increased significantly with PVL (p=0.002) (Table 3). When comparing the mortality rates between HIV-1 and HIV-2 infected women within the same PVL category, the rates of HIV-2 were lower, but this was only significant in one category. A Mantel-Haenszel rate ratio, adjusted for PVL category showed no significant difference between the two infections (p=0.4; table 3).
Among HIV-1 infected subjects the mortality rate increased with decreasing CD4 % (p=0.008) (Table 3). In HIV-2 infected women the mortality rates also increased with decreasing CD4% (p<0.0005). Among women with CD4% > 28%, the mortality rate was significantly lower in HIV-2 than in HIV-1 infected subjects (p<10^-4), but in the two lower CD4% bands there were no significant differences in mortality rates between the two infections (p=0.4 each).

Multivariate analysis

In the multivariate analysis only PVL and CD4% were independent predictors of mortality. Age was a priori in the model and time period was a confounder and kept in the model as well. After adjusting for the other variables, each 10 percent decrease in CD4% was associated with a 1.6
<table>
<thead>
<tr>
<th>Baseline Value</th>
<th>HIV-1</th>
<th>HIV-2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>no. of subjects</td>
<td>no. (%) of deaths</td>
</tr>
<tr>
<td>Overall</td>
<td>101</td>
<td>32 (32%)</td>
</tr>
<tr>
<td>Plasma viral load (RNA copies/ml)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;1,000</td>
<td>6</td>
<td>1 (17%)</td>
</tr>
<tr>
<td>1,000-9,999</td>
<td>26</td>
<td>7 (27%)</td>
</tr>
<tr>
<td>10,000-99,999</td>
<td>54</td>
<td>17 (31%)</td>
</tr>
<tr>
<td>≥ 100,000</td>
<td>8</td>
<td>4 (50%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CD4%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 28%</td>
<td>56</td>
<td>14 (25%)</td>
</tr>
<tr>
<td>14 – 28%</td>
<td>26</td>
<td>11 (42%)</td>
</tr>
<tr>
<td>&lt; 14%</td>
<td>6</td>
<td>4 (67%)</td>
</tr>
</tbody>
</table>

Notes: a. Comparing HIV-2 with HIV-1; b. Mantel-Haenszel combined mortality rate ratio, controlling for plasma viral load; c. Mantel-Haenszel combined mortality rate ratio, controlling for CD4%. PVL = plasma viral load; pyo = person-years of observation; CI = confidence interval.
Figure 2. Kaplan-Meier graph showing probability of survival of 812 women by HIV status.

<table>
<thead>
<tr>
<th>years since recruitment</th>
<th>0</th>
<th>2</th>
<th>4</th>
<th>6</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>uninfected</td>
<td>468</td>
<td>403</td>
<td>375</td>
<td>357</td>
<td>57</td>
</tr>
<tr>
<td>HIV-1</td>
<td>101</td>
<td>88</td>
<td>75</td>
<td>60</td>
<td>14</td>
</tr>
<tr>
<td>HIV-2</td>
<td>243</td>
<td>210</td>
<td>197</td>
<td>174</td>
<td>38</td>
</tr>
</tbody>
</table>
fold increase in mortality rate (95%CI 1.1-2.3, p=0.02) in HIV-1 and a 1.5-fold increase (95%CI 1.0-2.3, p=0.046) in HIV-2. After adjusting for the other variables, an increase of one log in PVL was associated with a 1.8 fold increase in mortality rate (95%CI 0.9-3.4; p=0.08) in HIV-1. As 41% of HIV-2 infected women had a PVL below the detection limit, we could not calculate the effect of a one log increase in PVL in HIV-2. In a multivariate analysis using categories, the mortality rate was 1.3 fold (95%CI 0.36-4.5) higher in women with PVL between 1,000 and 9,999 copies per ml, and 8.7 fold (95%CI 2.8-28) higher in women with PVL ≥ 10,000 copies per ml, compared to women with PVL < 1,000 copies per ml. The mortality rate in HIV-2 infected women with normal CD4% and undetectable PVL was not significantly different from that among seronegative women (p=0.6).

In an analysis combining HIV-1 and HIV-2, each 10% decrease in CD4% was associated with a 1.7 fold increased risk of mortality (95%CI: 1.3-2.2, p<0.0005). PVL was also associated with mortality: rate ratios were 2.1 (95%CI 0.71-6.1) for those with PVL between 1,000 and 9,999 copies per ml and 3.9 (95%CI 1.3-12) for those with PVL ≥ 10,000 copies per ml, when compared to the baseline group (<1,000 copies per ml). When adjusted for CD4%, PVL, age group and time period, there was no significant difference in mortality rates between the two infections (p=0.7).

Discussion

Main findings

This is the first study that has been able to compare the mortality of both infections in persons from the same population, and it confirms that the excess mortality of HIV-2 is much less than that of HIV-1.7,10,12

The study confirms data from other community-based studies that showed that mortality rates in African HIV-1 patients are comparable to those of HIV-1 infected patients in industrialised countries before the introduction of HAART.7,8 Eight years after recruitment into this seroprevalent cohort, more than 50% of HIV-1 women were still alive.
Finally, this study shows that PVL and CD4% are independent and significant predictors for mortality in HIV-2 in West Africa, confirming the results of an earlier community-based study in Guinea-Bissau and a clinic-based study in The Gambia. HIV-2 infected women with normal CD4% had a significantly lower mortality than HIV-1 infected women with normal CD4%, but among women with low CD4% there were no significant differences in mortality rate between the two HIV types. These findings are in agreement with findings from a clinical cohort in The Gambia.

**Possible biases**

A possible source of selection bias is that women were pregnant when they were recruited, and fertility is reduced among HIV-infected women. As HIV-associated subfertility affects recently and long infected women to a similar degree, this is unlikely to have lead to underestimates of mortality rates, or overestimates of survival probabilities.

Up to 18 months after delivery, the women were visited quarterly and the loss to follow-up was limited. The next visit was 4-7 years later, and this long time gap and the associated loss to follow-up could be a source of bias. However, there were no significant differences in the key baseline variables age, CD4%, PVL, and parity between those lost to follow-up and those who were not.

We had anticipated that many women would wish to obtain their HIV status after being counselled a second time, but this did not transpire. Potential reasons for the poor uptake could be the stigma attached to the disease, lack of effective anti-retroviral treatment on offer, and counselling by male field workers. Voluntary counselling and testing is considered to be a valuable tool in the fight against HIV and AIDS, but this low acceptance ratio of HIV tests suggests that the uptake may be very low in societies where HIV disease is stigmatised and effective antiretroviral therapy unavailable.
HIV-2 in West Africa

Pathogenesis

This study shows that, like in HIV-1, PVL is also a key predictor of mortality in HIV-2 infection. Although in univariate analysis the mortality of HIV-2 was much lower than in HIV-1, HIV type was not an independent predictor of mortality after adjusting for PVL, CD4%, age group and time period. This suggests that the lower mortality of HIV-2 can be explained by the generally lower PVL and higher CD4% in HIV-2, and indicates a lower virulence of HIV-2.

An intriguing question remains why some people infected with HIV-2 develop a high PVL and rapid decay of the immune system, and others not. This is not due to subtype (all HIV-2 patients in The Gambia have subtype A), but could be due to intra-subtype variations. The transmission route, infecting dose, genetic factors such as HLA type, and the HLA type of the infecting partner may be important as well. Further studies to elucidate this interaction between agent, host and environment are needed.

Implications

This study indicates that PVL and CD4% are of great value in predicting the outcome of infection: women with a normal baseline CD4% and a baseline PVL < 500 copies/ml had a mortality rate that was not significantly different from that of seronegative women (p=0.6). Since clinical markers alone do not reliably predict mortality, baseline CD4% or PVL measurement may be important to guide decisions to start ART. Currently there are no guidelines for the treatment of HIV-2. Our data suggest that the same clinical, virus load, and CD4 criteria should be applied in cases of HIV-2 infection as in HIV-1 when considering the use of ART. Most HIV-2 infected people live in Africa, but unfortunately few HIV-2 infected patients in Africa are receiving ART.

Acknowledgements

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References

HIV-2 in West Africa


