Viability and effectiveness of large-scale HIV treatment initiatives in sub-Saharan Africa: experience from western Kenya

Kara Wools-Kaloustiana,d, Silvester Kimaiyo,d, Lameck Diero,d, Abraham Siika,d, John Sidleb,d, Constantin T. Yiannoutsosc, Beverly Musickc, Robert Einterzb, Kenneth H. Fifea and William M. Tierneyb

Objectives: To determine the clinical and immunological outcomes of a cohort of HIV-infected patients receiving antiretroviral therapy.

Design: Retrospective study of prospectively collected data from consecutively enrolled adult HIV-infected patients in eight HIV clinics in western Kenya.

Methods: CD4 cell counts, weight, mortality, loss to follow-up and adherence to antiretroviral therapy were collected for the 2059 HIV-positive non-pregnant adult patients treated with antiretroviral drugs between November 2001 and February 2005.

Results: Median duration of follow-up after initiation of antiretroviral therapy was 40 weeks (95% confidence interval, 38–43); 111 patients (5.4%) were documented as deceased and 505 (24.5%) were lost to follow-up. Among 1766 (86%) evaluated for adherence to their antiretroviral regimen, 78% reported perfect adherence at every visit. Although patients with and without perfect adherence gained weight, patients with less than perfect adherence gained 1.04 kg less weight than those reporting perfect adherence ($P = 0.059$). CD4 cell counts increased by a mean of 109 cells/$\mu l$ during the first 6 weeks of therapy and increased more slowly thereafter, resulting in overall CD4 cell count increases of 160, 225 and 297 cells/$\mu l$ at 12, 24, and 36 months respectively. At 1 year, a mean increase of 170 cells/$\mu l$ was seen among patients reporting perfect adherence compared with 123 cells/$\mu l$ among those reporting some missed doses ($P < 0.001$).

Conclusions: Antiretroviral treatment of adult Kenyans in this cohort resulted in significant and persistent clinical and immunological benefit. These findings document the viability and effectiveness of large-scale HIV treatment initiatives in resource-limited settings.


Keywords: HIV, AIDS, antiretroviral therapy, computerized medical records system, sub-Saharan Africa

Introduction

UNAIDS estimates that 2 million of Kenya’s 29.5 million people are currently infected with HIV and that 1.5 million have already succumbed to the disease, resulting in an overall decline in life expectancy of 13 years [1]. This trend is reflected in most of sub-Saharan Africa [2]. HAART has been proven to decrease HIV viral load, increase CD4 cell counts and stem the progression to
AIDS and death in economically developed countries [3–5]. Studies from Senegal, Uganda and South Africa confirm that viral suppression and improvement in immune status can be achieved in African populations [6–8]. Despite these findings, introduction of antiretroviral drugs into sub-Saharan Africa has been slow because of concerns about treatment adherence, emergence of resistance and the impact of antiretroviral therapy on risk behaviors. However, this delay is mostly attributable to inadequate funds for antiretroviral drugs and absence of sufficient infrastructure to dispense and monitor therapy [7,9,10].

The Indiana University School of Medicine has had a collaborative partnership with Moi University in Eldoret, Kenya since 1990 [11]. In response to a significant increase in HIV prevalence noted in 2001 at the Moi Teaching and Referral Hospital (Moi Hospital), the Academic Model for the Prevention and Treatment of HIV/AIDS (AMPATH) was initiated as a joint initiative between Moi University, Moi Hospital and the Indiana University School of Medicine [12,13]. The initial goal of AMPATH was to establish an HIV care system to serve the needs of both urban and rural patients and to assess the barriers to and outcomes of antiretroviral therapy. Details of the development of this system have been described elsewhere [12]. The first urban and rural HIV clinics were opened in November 2001, with six additional clinics opening between January and July of 2004. A computerized medical record system was developed to support clinical care [14]. This study determined the outcomes of patients treated in this care system through analysis of data routinely collected and stored in patients’ electronic medical records.

Methods

Study design

This study was approved by the Indiana University School of Medicine Institutional Review Board and the Moi University Institutional Research and Ethics Committee. This study used data of prospectively enrolled HIV-infected adult patients treated in the AMPATH clinics. The data were stored in computerized medical records and all patient identifiers were removed.

Study population

Patients were eligible if they were seen in any of the AMPATH clinics between November 2001 and February 2005, were at least 18 years of age, and initiated antiretroviral drug treatment prior to September 2004. Women who were pregnant during the study period were excluded because it is our standard practice to provide a three–drug antiretroviral regimen during the last trimester regardless of CD4 cell count.

The urban clinic is located at Moi Hospital in Eldoret, Kenya, a national referral hospital serving a catchment area of approximately 13 million people. Comprehensive HIV care services were added to the primary care services of one district hospital and six rural satellite health centers. Three of these satellite clinics are 25–35 km from Eldoret while four are located 60–120 km away. HIV care is provided by physicians and clinical officers trained and mentored within the AMPATH model [15].

Clinical procedures

Detailed algorithms consistent with World Health Organization (WHO) guidelines for care of HIV-infected patients were developed locally and followed throughout the study period [16]. Patients receiving antiretroviral drugs were seen 2 weeks after initiation of therapy and every month thereafter. During these visits, patients underwent clinical and adherence assessments and were dispensed antiretroviral drugs. For patients found to be adherent with their medication and clinically stable, visit intervals were sometimes extended to every 2 months.

Laboratory testing was based on local protocols and clinical necessity. Per protocol, a complete blood count with white cell differential count, CD4 cell count and alanine aminotransferase assay were performed at baseline and every 6 months. No viral load testing was performed because of funding limitations. During the early part of the project, when funds were limited, self-pay patients without resources did not have all laboratory testing done at the specified intervals. Patients meeting WHO criteria for treatment started antiretroviral drugs at the sole discretion of their clinician [16]. Because of funding limitations during the first 30 months of the HIV clinic operation, first priority for treatment was given to severely ill patients. During this period, some patients received free antiretroviral drugs and testing through the Maternal to Child Transmission Plus (MTCT-Plus) program, philanthropic donations, and foundation grants, while other patients paid for their medications. By the latter part of this period, significant funding for HIV/AIDS care was available through the President’s Emergency Plan for AIDS Relief (PEPFAR; http://www.usaid.gov/our_work/global_health/aids/pefar.html), and antiretroviral therapy was given free of charge to all patients meeting treatment criteria. Education about antiretroviral drugs was provided by both the clinician and the nurse during the clinic session when the drugs were initiated. The standard antiretroviral treatment regimen consisted of three drugs: stavudine, lamivudine, and nevirapine. To avoid drug interactions, efavirenz was substituted for nevirapine in patients receiving induction therapy for tuberculosis. No patient experienced treatment interruptions owing to stock shortages. A 25% decline in CD4 cell count from the maximum attained after initiation of antiretroviral drugs was considered a regimen failure and triggered a change in patients’ antiretroviral regimen. This level of decline was chosen.
as the definition of antiretroviral therapy failure because Hughes and colleagues [17] documented an average within-subject coefficient of variation for CD4 cell count of 25% among HIV-infected patients in the United States.

Data collection and management
Clinicians completed standard initial and return encounter forms at all AMPATH clinic visits (http://amrs.ui-kenya.org/download/forms). The initial encounter form included standard demographic, historical, psychosocial, physical, and laboratory data as well as the medications provided (antiretroviral drugs and opportunistic infection prophylaxis). Follow-up data were collected on intercurrent symptoms, medication adherence, new diagnoses, laboratory data, and modifications of drug regimens. Dedicated data entry clerks entered this information into the AMPATH Medical Records System, which uses an MS-Access database (Microsoft Corp., Redmond, Washington State, USA) [14].

A standard adherence assessment was added to the follow-up clinic visit form in June 2003, allowing assessment of adherence at every visit. It consisted of one question ‘During the last 7 days, how many of her/his antiretroviral pills did the patient take?’ The available responses were: ‘none’, ‘few’, ‘half’, ‘most’, and ‘all’. In the analyses, adherence was considered as perfect (every response at every visit is ‘all’) or imperfect (any response other than ‘all’ reported at even a single visit). Mortality data were captured using a passive surveillance system.

Statistical analysis
Frequency tables were produced for all categorical variables and were compared via the Fisher’s exact test. For continuous measures, the median and 95% trimmed ranges (the 2.5th and 97.5th percentiles) were generated and compared between groups using the Kruskal–Wallis test. Time-to-event analyses were performed using the Kaplan–Meier method. The events of interest were the time until a greater than 10% decline in weight and a greater than 25% decline in CD4 cell count compared with the maximum level attained after initiation of antiretroviral drugs. The survival distributions were compared by the log rank test. CD4 cell count and weight changes over time were analyzed via linear and non-linear mixed-effects models [18]. A change-point model was fitted to the square root of CD4 cell counts to account for the expected rapid increase immediately after initiation of therapy followed by a less-steep increase thereafter [19,20]. The location of the change point in time was allowed to vary and the one best reflecting the data was determined through a search among candidate points. The baseline square root CD4 cell count and the slope before and after the change point were allowed to vary from individual to individual (random component of the model) while the effect of other explanatory factors such as gender and adherence was considered fixed. An exponential growth model was fitted to weight data with three components reflecting initial weight, final weight, and rate of weight increase. Interaction of all three with other model predictors was explored. A patient-level random initial weight, final weight, and rate of increase were included. The association between CD4 cell count and weight decline was assessed by Cox regression using as a predictor the most recent CD4 cell count prior to the 10% or 20% decline in weight or the last CD4 cell count for those patients who did not experience an event (these were censored at their last visit). Statistical significance in the Cox model was assessed by Wald-type tests. Analyses were performed by the SAS system version 9.1 (SAS Institute, Cary, North Carolina) and S plus version 6.2 (Insightful, Seattle, Washington State, USA).

Results
Patient characteristics
As of September 2004, 2059 non-pregnant adult patients had initiated antiretroviral therapy. The median time from initial clinic visit to initiation of antiretroviral therapy was 6.9 weeks [95% confidence interval (CI), 6.0–7.1]. Patient characteristics at the first clinic visit are given in Table 1. Median age at enrollment was 37 years [trimmed range (TR): 24.4–57] and approximately 60% of the participants were women. Among patients with available sociodemographic data, approximately two-thirds were married, one third were employed outside the home, and >90% had attended school. The median duration of school attendance was 9 years (TR, 0–16). Sixty-three percent of individuals had disclosed their HIV status to someone, with 25% having disclosed to their sexual partner. The Moi Hospital HIV Clinic (clinic 1) accounted for roughly two-thirds of study patients; the majority of the remaining patients were cared for at the Mosoriot Rural Health Center (clinic 2), the first rural clinic established.

As noted in Table 2, one third of patients presented with asymptomatic disease; however the median CD4 cell count was only 86 cells/μl (TR, 2–395). Women had significantly lower WHO stage and a higher CD4 cell count than men (P < 0.001). The median weight for both men and women at enrollment was < 60 kg.

As of February 2005, the median duration of follow-up after initiation of antiretroviral therapy was 40 weeks (95% CI, 38–43). Pretreatment CD4 cell counts (obtained within 3 months prior to antiretroviral drugs initiation) were available for 1639 patients, with a median CD4 cell count of 82 cells/μl (TR, 2–378). Approximately one quarter (27.2%) of this cohort received treatment for tuberculosis during the study period. There were 111 (5.4%) documented deaths and 505 (24.5%) patients were lost to follow-up (no visit for at least 3 months). The
1- and 2-year estimated loss-to-follow-up rates were 22.4% and 29.7%, respectively. These differed significantly between clinics, approaching 30% at 1 year in clinics 1 and 3, while the rates in clinics 2 and 4 were less than half of that ($P < 0.001$). The more distant rural clinics were not included in this analysis as they had been open for a much shorter period and were responsible for only 6.2% of cohort patients.

By February 2005, 1766 patients with adherence data had been enrolled. More than one adherence assessment was available for 92% of these patients, and 78% reported perfect adherence at all assessments. Of the 22% who reported taking less than all of their medications (imperfect adherence) at one or more visits, 85% reported problems at only one visit. Adherence rates increased as the duration of follow-up increased, with reports of perfect adherence from approximately 93% of patients followed for at least 1 month and nearly 100% of patients followed for 38 months.

### Longitudinal analysis of patient weight and CD4 cell count

Using a non-linear growth model, weight increased significantly after initiating antiretroviral therapy and then tapered over time (Fig. 1). The average increase in weight over the 3 years of observation was 4.42 kg ($P < 0.001$). Weight loss of $>10\%$ from maximal weight was experienced by 27% and of $>20\%$ by 5% of all patients within 1 year after antiretroviral drug initiation. The median time to a 10% weight loss was 88 weeks (95% CI, 85–97).

The median time to the initial CD4 cell count measurement after starting antiretroviral drugs was 34 weeks. CD4 cell counts rose rapidly for the first 6 weeks after therapy initiation (prior to the change point), resulting in an overall mean CD4 cell increase of 109 cells/µl (Fig. 2). CD4 cell count increased more slowly thereafter, resulting in estimated increases of 160, 225 and 297 cells/µl at 12, 24, and 36 months, respectively. Based on the Kaplan–Meier analysis of 3 years of observation, <50% of patients experienced a presumed antiretroviral failure (greater than 25% decline from maximal CD4 cell count after initiation of antiretroviral drugs; Fig. 3).

### Table 1. Sociodemographics at enrollment.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Total No.</th>
<th>Median age [years (trimmed range)] ($n = 1878$)</th>
<th>Female [No. (%)]</th>
<th>Ethnic groups [No. (%)] ($n = 2019$)</th>
<th>Married [No. (%)] ($n = 2035$)</th>
<th>Employed [No. (%)] ($n = 1405$)</th>
<th>Education</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total No.</td>
<td>2059</td>
<td>37.3 (24.4–57)</td>
<td>1247 (60.6)</td>
<td>1</td>
<td>913 (45.2)</td>
<td>1303 (64.0)</td>
<td>Attended school</td>
</tr>
<tr>
<td>Median age</td>
<td>37.3 (24.4–57)</td>
<td></td>
<td>1247 (60.6)</td>
<td>1</td>
<td>913 (45.2)</td>
<td>1303 (64.0)</td>
<td>No. years</td>
</tr>
<tr>
<td>trimmed range balanced</td>
<td>2.5th and 97.5th percentile of available observation.</td>
<td></td>
<td>1247 (60.6)</td>
<td>1</td>
<td>913 (45.2)</td>
<td>1303 (64.0)</td>
<td></td>
</tr>
</tbody>
</table>

### Table 2. Clinical characteristics at enrollment by gender.

<table>
<thead>
<tr>
<th>Clinical characteristic</th>
<th>Total</th>
<th>Male</th>
<th>Female</th>
<th>$P$ value$^a$</th>
</tr>
</thead>
<tbody>
<tr>
<td>WHO stage [No. (%)]$^b$</td>
<td></td>
<td></td>
<td></td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Total No.</td>
<td>2059</td>
<td>812</td>
<td>1247</td>
<td></td>
</tr>
<tr>
<td>Stage 1</td>
<td>685 (33.3)</td>
<td>239 (29.4)</td>
<td>446 (35.8)</td>
<td></td>
</tr>
<tr>
<td>Stage 2</td>
<td>243 (11.8)</td>
<td>84 (10.3)</td>
<td>159 (12.8)</td>
<td></td>
</tr>
<tr>
<td>Stage 3</td>
<td>775 (37.6)</td>
<td>318 (39.2)</td>
<td>457 (36.7)</td>
<td></td>
</tr>
<tr>
<td>Stage 4</td>
<td>356 (17.3)</td>
<td>171 (21.1)</td>
<td>185 (14.8)</td>
<td></td>
</tr>
<tr>
<td>CD4 cell count [No. assessed]</td>
<td>1763</td>
<td>694</td>
<td>1069</td>
<td>&lt; 0.001$^b$</td>
</tr>
<tr>
<td>Median [cells/µl (trimmed range)]</td>
<td>86 (2–395)</td>
<td>77 (1–366)</td>
<td>91 (2–430)</td>
<td></td>
</tr>
<tr>
<td>Weight [No. assessed]</td>
<td>2035</td>
<td>804</td>
<td>1231</td>
<td>&lt; 0.001$^b$</td>
</tr>
<tr>
<td>Median [kg (trimmed range)]</td>
<td>54 (36.0–80.5)</td>
<td>56.6 (41.0–80.2)</td>
<td>51 (35.0–80.5)</td>
<td></td>
</tr>
<tr>
<td>Body mass index [No. assessed]</td>
<td>1807</td>
<td>717</td>
<td>1090</td>
<td>&lt; 0.001$^b$</td>
</tr>
<tr>
<td>Median (trimmed range)</td>
<td>19.2 (13.7–28.4)</td>
<td>18.9 (14.0–26.7)</td>
<td>19.5 (13.5–29.5)</td>
<td></td>
</tr>
</tbody>
</table>

Trimmed range, 2.5th and 97.5th percentile of available observations.

$^a$P value from Kruskal–Wallis test except for WHO stage, which was from Fisher’s exact test.

$^b$Calculated from intake data when clinician assessment was unavailable.
CD4 cell count was a statistically significant predictor of subsequent 10% and 20% weight loss ($P < 0.001$ in both). Among two individuals with a 100 cell/$\mu$L count difference, the individual with the lower CD4 cell count had one-third higher risk of a 10% weight decline and a 52% greater risk for a 20% weight decline. The risk of death for a patient with a CD4 cell count at the time of treatment initiation $< 100$ cells/$\mu$L was over three times higher than that for a patient with CD4 cell count $> 100$ cells/$\mu$L (log rank test $P < 0.001$).

A few patients (4.9%) initiated an antiretroviral regimen other than the three-drug standard but were subsequently converted to the standard regimen. A regimen change from the standard was made in 242 (11.9%) patients after a median of 150 weeks. The time to initiation of second-line therapy was slightly shorter for patients who initiated treatment on a non-standard regimen (median 117 weeks; $P = 0.073$).

The effect of adherence on patient outcomes

Patients with imperfect adherence experienced somewhat lower average weight gains (1.04 kg less) than those reporting perfect adherence ($P = 0.059$; Fig. 1). Perfectly adherent patients were significantly less likely to experience a 10% decline in weight compared with those with imperfect adherence ($P = 0.011$).
Adherence was a strong predictor of overall CD4 cell response. During the first 6 weeks of therapy, a mean increase of 112 cells/μl was seen among perfectly adherent patients compared with 108 cells/μl among those reporting imperfect adherence ($P = 0.032$; Fig. 2). Thereafter, the rate of CD4 cell increase in this latter group was roughly half that seen in the perfectly adherent group, giving rise to a CD4 cell increase at 1 year that was 43 cells/μl lower than the increase in the perfectly adherent group ($P < 0.001$; Fig. 2). Imperfect adherence was also associated with shorter time until a presumed antiretroviral therapy failure ($P < 0.001$; Fig. 3).

The effect of gender on patient outcomes
Males were significantly more likely to be lost to follow-up than were females ($P = 0.004$). Among individuals continuing care, gender was not associated with level of adherence. Men were heavier by an average of 5.35 kg and had a significantly faster rate of weight gain than did women ($P < 0.001$; Fig. 1). CD4 cell count increases were greater in women than men during the initial 6 weeks of antiretroviral therapy ($P = 0.007$). After the initial 6 weeks, women experienced CD4 cell count increases nearly double those of men ($P = 0.003$). Time to antiretroviral therapy failure was also slightly longer in women than in men ($P = 0.082$).

Discussion

We have established that it is possible to administer antiretroviral therapy and to achieve substantial clinical responses within a public sector setting in western Kenya. This cohort has demonstrated clinical benefit in terms of both CD4 cell count and weight increases well into the third year of follow-up. In the absence of viral load data, a direct comparison with previous studies is difficult. However, CD4 cell increases attained on antiretroviral therapy are consistent with those seen elsewhere [21–23]. Our findings of a rapid rise in CD4 cell counts during the initial few weeks of therapy, followed by a slower rise, are consistent with earlier reports on CD4 cell kinetics [19,20]. In addition, the median CD4 cell increase seen at 52 weeks after antiretroviral therapy initiation is similar to the response seen in studies conducted in the United States and Europe [21–23]. One limitation of this analysis is that regimen changes were not taken into account. As such, the mean CD4 cell increase seen at later time periods reflect responses to both primary and secondary regimens (as 11.9% of our patients switched to second-line therapy).

Previous clinical trials assessing durability of response to regimens based on non-nucleoside reverse transcriptase inhibitors (NNRTI) have used either detectable HIV RNA or the combination of detectable HIV RNA and regimen change as the outcome measure defining treatment failure [7,24,25]. The 3-year treatment failure rates documented in these studies range from 20 to 48%. Rather than virological failure, immunological failure was used to define treatment failure in our cohort. This outcome likely follows virological failure by several months, which may indicate that our population is experiencing treatment failure (as defined by detectable HIV RNA) at a slightly earlier point than seen in prior studies. This is not unexpected given that the patients described here are part of a clinic cohort rather than participants in a clinical trial. As such, the median time to failure of an NNRTI-based first-line therapy within a public sector clinic in sub-Saharan Africa appears to be approximately 3 years.

Because of the cross resistance between the non-nucleoside reverse transcriptase inhibitors, second-line regimens will need to be protease inhibitors based. In addition, in the absence of genotyping data, two nucleoside reverse transcriptase inhibitors with minimal cross resistance to those used in the initial regimen will also be required. Use of a protease inhibitor in the second-line regimen has significant cost and monitor implications that must be addressed as HIV treatment programs are extended in the developing world. We believe that viral load data would be invaluable for the early identification of patients experiencing antiretroviral therapy failure and would assist in preventing the development of multiple drug resistance mutations. However, at current cost, this technology is unlikely to have wide application in resource-poor settings such as ours. Therefore, we strongly support development of low-cost technologies for viral load determination as part of the policy for providing antiretroviral drugs in sub-Saharan Africa.

Since measurements of CD4 cell counts are not routinely available in resource-poor settings, and because weight is an independent predictor of HIV progression, weight was assessed as one of the clinical outcomes [26–28]. We found that mean weight gains mirrored CD4 cell increases immediately following antiretroviral therapy initiation. However, weight was found to have a within-subject coefficient of variation of 20%. This may have contributed to the discrepancy seen between our finding of consistent mean weight increases throughout the study period and the Kaplan–Meier model, which demonstrated that a significant percentage of the study cohort experienced a greater than 10% decline in weight. We initially attributed the substantial variability in weight to patients’ inability to access food consistently. However, further analysis showed that weight declines correlated with CD4 cell count, indicating that the at least some of the variability in weight reflects variations in clinical status. Because of the discrepancies between the two models, noted above, we believe that, in the absence of a reliable food supply, weight should be used with caution in assessing individual responses to antiretroviral therapy.
Studies conducted in the United States have shown that adherence to an antiretroviral regimen is a significant predictor of viral suppression and clinical response [29,30]. More than three-quarters of our patients claimed perfect adherence at all visits. We found that the use of this single item to assess patient adherence was predictive of immunological and clinical responses in this cohort. This finding warrants further exploration, with particular attention to the sensitivity of brief adherence assessments in a busy clinic setting.

We acknowledge that the 24.5% loss to follow-up rate for our study population might have had a significant impact on our findings. In the presence of a passive reporting system for deaths, we believe that much of our loss to follow-up is from unreported deaths. This assumption is based on the low death rate (5.4%) for this population in which 55% of individuals have WHO stage 3 or 4 disease. Other likely reasons for loss to follow-up include patients moving outside the catchment area and problems with transportation access. Deaths likely represent poor responses to therapy, which, if captured, would negatively impact both weight and CD4 cell trends. This could result in the introduction of a potentially serious bias and possible overestimation of both CD4 cell count and weight response to therapy. The impact of data from patients who have been lost to follow-up for reasons other than death is difficult to predict. Because the median duration of follow-up for this cohort is approximately 1 year, all models discussed above are limited by the relatively small number of data points in the later time periods.

Despite the limitations of this study, we have documented significant clinical and immunological responses in this population that appear to persist for at least 3 years after initiation of antiretroviral therapy. These findings provide significant support for the viability of large-scale HIV treatment initiatives in resource-limited settings. However, in order to ensure the long-term success of treatment programs, there remains a significant need to identify feasible and reliable tools to assess adherence, methods to identify and locate patients who are lost to follow-up, low-cost monitoring technologies, and rational second-line drug regimens.

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