Journal of the International AIDS Society

Oral presentation

O212 Rate of change in CD4 counts in patients with stable HIV viremia

AN Phillips[1], B Ledergerber[2], JR Bogner[3], K Lacombe[4], A Wiercinska-Drapalo[5], P Reiss[6], O Kirk[7], JD Lundgren[8] and A Mocroft*[1]

Address: [1]Royal Free and University College Medical School, London, UK, [2]University Hospital, Zurich, Switzerland, [3]Medizinische Poliklinik, Munich, Germany, [4]Hospital Saint-Antoine, Paris, France, [5]Medical University, Bialystok, Poland, [6]Academisch Medisch Centrum bij de Universiteit van Amsterdam, Amsterdam, Netherlands, [7]Copenhagen HIV Programme, Panum Institute, Copenhagen, Denmark and [8]Copenhagen HIV Programme, Panum Institute and Centre for Viral Disease KMA, Rigshospitale, Copenhagen, Denmark

* Corresponding author

from Ninth International Congress on Drug Therapy in HIV Infection
Glasgow, UK. 9–13 November 2008

Published: 10 November 2008

Journal of the International AIDS Society 2008, **11**(Suppl 1):O17 doi:10.1186/1758-2652-11-S1-O17

This abstract is available from: http://www.jiasociety.org/content/11/S1/O17

Purpose of the study

The majority of patients currently treated with cART live in resource-limited settings and many patients are left on a virologically failing regimen, typically an NNRTI-containing regimen. The aim was therefore to identify the level of viremia at which CD4 counts significantly decrease in patients on cART and the factors, including regimen type, associated with CD4 count changes.

Methods

Annual CD4 slopes were calculated from three consecutive CD4 measurements whilst the viral load was stable, defined as <0.5 $\log_{10}$ copies/ml difference between the highest and lowest viral loads measured at the same three time-points. Generalised linear models, with adjustment for repeated measurements within patients, were used to model CD4 slopes.

Summary of results

A total of 7,231 patients were included in analyses contributing 58,929 CD4 slopes. There was a median of six slopes per patient included in analyses (IQR 3–12), and the CD4 slope was calculated over a median time of 6.6 months (IQR 5.5–8.1 months). The median date of inclusion into this study was May 2000 (IQR September 1998–May 2003) and median CD4 at this date was 354/mm^3 (IQR 220–529/mm^3). Figure 1 illustrates the highly significant differences in mean CD4 slope after stratification by current viral load and current cART treatment status. Patients on cART had more favourable (more positive or less negative) mean CD4 slopes than those off cART for any given level of viremia, whilst antiretroviral-naïve patients had more favourable CD4 slopes than patients who had previously started cART but were currently undergoing a treatment interruption.

Among patients on cART, adjusting for relevant confounders, patients on a single PI-based cART regimen (mean CD4 slope [SE] 43.3/mm^3 [2.4]) or a ritonavir-boosted PI (46.9/mm^3 [2.3]) had more positive CD4 slopes compared to patients taking a non-nucleoside reverse-transcriptase inhibitor (31.1/mm^3 [1.7]) or a triple nucleoside regimen (30.2/mm^3 [4.0], $p < 0.0001$).

Conclusion

The results from our study would suggest that some treatment with cART is better than none, but also that better CD4 count increases are obtained by using a PI or ritonavir-boosted PI-based regimen than by using an NNRTI-based regimen. Patients were not randomised to treatment and confounding by indication cannot be ruled out. Patients with stable viremia unable to fully suppress HIV replication should continue therapy and consideration should be given to the use of a PI-based regimen.

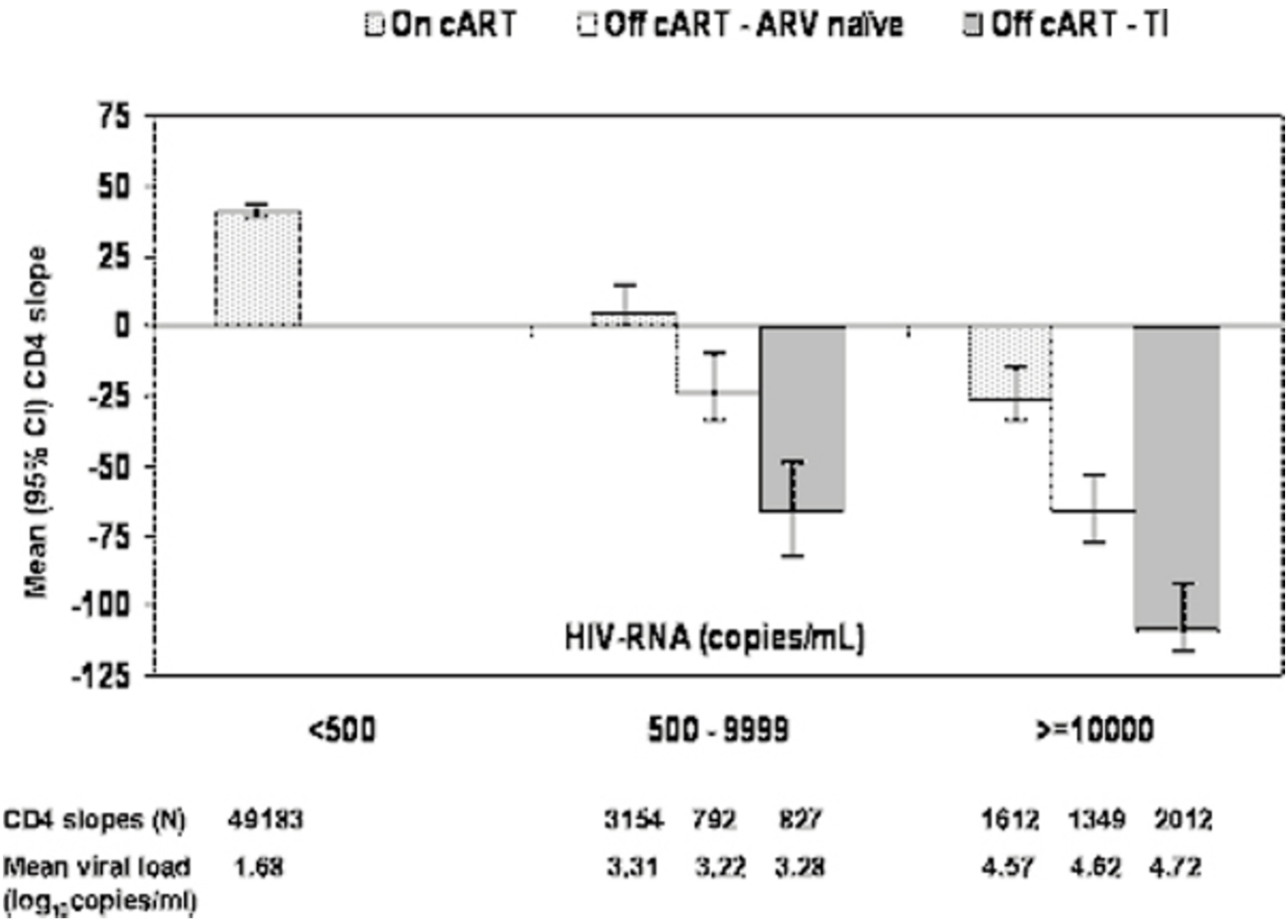

Figure 1