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## **Long-term HIV treatment doesn't damage kidney function**

**Michael Carter**, Thursday, November 26, 2009

Long-term HIV treatment has not caused a deterioration in kidney function among French patients, investigators report in the December 15<sup>th</sup> edition of *Clinical Infectious Diseases*. "Renal function is remarkably stable over seven years among combination antiretroviral therapy-treated patients", they comment.

Although the investigators did find some evidence of deterioration in kidney function, they note that this was to be expected in an ageing population. They believe the long-term stability of kidney function in the majority of patients can be attributed to good control of HIV replication.

Treatment with the protease inhibitor indinavir (*Crixivan*) was also associated with a deterioration in kidney function during the first 16 months of antiretroviral therapy. Although this drug is now rarely used, the investigators believe that this finding still has relevance and recommend "clinicians should closely monitor renal function in combination antiretroviral therapy-treated patients, especially those who have been exposed to indinavir."

HIV-positive patients have an increased risk of kidney disease. HIV itself can be a cause, and for this reason timely initiation of antiretroviral therapy is recommended for patients with evidence of renal dysfunction, or a significant risk of this developing.

HIV treatment, however, especially the first-line drug tenofovir (*Viread*, also in the combination pills *Truvada* and *Atripla*) have also been associated with a deterioration in kidney function.

The French ANRS CO8 APROCO-COPILOTE cohort involves over 1000 patients who initiated triple-drug antiretroviral therapy between 1997 and 1999 and therefore provides seven years of information about evolving kidney function in the context of HIV treatment.

Glomerular filtration rate (GFR), an important marker of kidney function, was monitored in 1121 patients at four monthly intervals.

Most of the patients (77%) were men, the median age at baseline was 37 years, 10% were of African origin and 21% had progressed to AIDS.

Median creatinine level at the start of the study was 81 mmol/l, and median GFR was 93 ml/min/1.73m<sup>2</sup>.

The median duration of follow-up was seven years. During this time, CD4 cell count increased from a median of 251 cells/mm<sup>3</sup> to 524 cells/mm<sup>3</sup> and viral load fell to a median of 50 copies/ml.

Hypertension, an established risk factor for kidney disease, was present in 18% of patients. The most frequently used protease inhibitor was indinavir, a drug that was taken by 40% of individuals. The median duration of therapy with this drug was 21 months. A total of 214 patients received tenofovir for a median of 20 months.

After two years of HIV treatment, GFR had increased to a median of 97 ml/min/1.73m<sup>2</sup> and after six years of follow-up was 93 ml.min/1.73m<sup>2</sup> - the same value as at baseline.

The proportion of patients with impaired GFR (below 60 ml/min/1.73m<sup>2</sup>) at baseline was 39% and this remained

unchanged over the study. Overall, 5% of patients had two or more consecutive measures below this value.

Mortality for patients with GFR of below 60 ml/min/1.73m<sup>2</sup> was 4.1 per 100 person years. This fell to 1.6 per 100 person years for individuals with GFR between 60-90 ml/min/1.73m<sup>2</sup> and 1.8 per 100 person years for those with GFR over 90 ml/min/1.73m<sup>2</sup>.

Baseline GFR was significantly lower in patients with a baseline CD4 cell count below 200 cells/mm<sup>3</sup> (p = 0.029).

Treatment with tenofovir did not have a detrimental effect on the evolution of GFR during follow-up.

Statistical analysis that controlled for potential confounding factors showed that poorer evolution of GFR during the first 16 months of antiretroviral therapy was associated with male sex (p < 0.01), lower baseline body mass index (p < 0.001), and treatment with indinavir.

After this time, poorer GFR was associated with African race and a baseline CD4 cell count above 200 cells/mm<sup>3</sup>. No antiretroviral drug was significantly associated with the poorer evolution of GFR after 16 months of HIV therapy.

"This is the longest follow-up report, to our knowledge, involving treated European HIV-infected patients indicating stability of renal function over time", comment the investigators.

They add, "our long-term data further suggest that the favourable evolution in renal function during prolonged combination antiretroviral therapy might primarily be related to long-term control of HIV replication."

The investigators suggest that any deterioration in GFR during follow-up "was consistent with those observed in the context of the natural evolution of GFR (-0.5 ml/min/1.73m<sup>2</sup> per year) in an ageing uninfected population."

## Reference

Leport C et al. *Long-term evolution and determinants of renal function in HIV-infected patients who began receiving combination antiretroviral therapy in 1997-99, ANRS CO8 APROCO-COPILOTE*. Clin Infect Dis 49: 1950-54, 2009.

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