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The LipoWatch program from Visionary Health Concepts is designed to support providers and patients with education that integrates a "real world" focus with scientific data. Presentations on cardiovascular risks associated with HIV disease and treatment garnered some of the most attention at this year's Conference on Retroviruses and Opportunistic Infections (CROI.) This issue of LipoWatch addresses several relevant reports and presents an overview of their clinical implications. NOTE: LipoWatch faxes are archived on the web at http://www.vhconcepts.com/edu_progs.cfm.

Studies of cardiovascular events in patients with HIV have produced varying results. Nevertheless, they have advanced our knowledge to the point where there is no longer much controversy as to whether or not HIV disease and treatment are associated with an increased rate of cardiovascular (CV) disease. Questions remain, however, regarding the extent of that increase and the best ways to reduce cardiovascular risks.

The major study on CV risk presented at this year's CROI was the D:A:D study⁽¹⁾, a combination retrospective/prospective observational study of over 23,000 patients in 11 cohorts on three continents. The investigators confirmed the role of traditional CV risk factors but also documented a 26% increase in the CV event rate per additional year of HIV antiviral therapy. The separate contributions of higher cholesterol or triglyceride levels have not been separated from this overall rate of increase. The D:A:D study does not provide comparisons to HIV-infected patients not taking antiviral therapy, so the relative contributions of HIV disease and treatment cannot be determined. The rate of CV events for patients with 4 or more years of antiviral therapy was 5.5/1000 patient years (0.55%). The study will continue to collect data for at least another two years.

Similar results were generated by a group from Johns Hopkins University⁽²⁾. This study found increases in cerebrovascular events and stroke, and comparable increases in cardiovascular events. The study followed 2,671 patients for 7,330 person-years (PY) after January 1, 1996. There were 43 coronary heart disease and 37 cerebrovascular disease events for an incidence rate of 5.9 events/1000 PY (0.59%) and 5.0 events/1000 PY (0.50%), respectively. These events were associated with the use of protease inhibitors and of d4T.

The findings of these two studies contrast sharply with the large, recently published retrospective study of 36,766 patients who received care for HIV infection at Veterans Affairs facilities between January 1993 and June 2001⁽³⁾. This study found no increase in the rate of CV events among veterans using HIV antiviral therapies, underscoring once again the unclear position HAART and HIV itself have with respect to the 'traditional' cardiovascular risk factors. Are they additive or do the traditional risk factors outweigh the contributions of HAART and/or HIV?

Much research in the general population has focused on predictors of CV disease progression, including C-reactive protein (a measure of systemic inflammation) and carotid intima media (CIM) thickness. Two presentations at CROI offered contrasting results on this latter marker. The AIDS Clinical Trials Group⁽⁴⁾ reviewed CIM thickness in 45 triads of one HIV-negative patient, one HIV-positive not taking a protease inhibitor (PI), and one HIV-positive on PI therapy. Patients were matched on CHD risk factors but

those with high risk such as family history of CHD, diabetes, or uncontrolled hypertension were excluded. There were no significant differences among the groups in CIM thickness after two years of follow up. In contrast, a group from San Francisco⁽⁵⁾ who studied 106 HIV-infected subjects found that CIM thickness increased at a rate higher than historical controls. The second study has no concurrent control group, did not exclude patients with some high CVD risk factors, and employed a different methodology for measuring CIM thickness. It remains to be seen whether CIM thickness will clarify the contribution of HIV disease and treatment to CV risk.

Much of the commentary on conflicting results such as these focuses on study design and the need for longer-term follow up. Greater clarity on the contributions of HIV disease or antiviral treatments to the rate of CV events will take years to achieve. In the meantime, clinicians have to manage the cardiovascular risks of their patients. This will be the topic of next month's LipoWatch.

¹Friis-Møller N, Weber R, D'Arminio Monforte A et al. Exposure to HAART Is Associated with an Increased Risk of Myocardial Infarction: The D:A:D Study. Abstracts, 10th Conference on Retroviruses and Opportunistic Infections; February 10-14, 2003, Boston MA, Abstract 130.

²Moore RD, Keruly JC, Lucas G. Increasing Incidence of Cardiovascular Disease in HIV-infected Persons in Care. Abstracts, 10th Conference on Retroviruses and Opportunistic Infections; February 10-14, 2003, Boston MA, Abstract 132.

³Bozzette SA, Ake CF, Tam HK, Change SW, Louis TA. Cardiovascular and Cerebrovascular Events in Patients Treated for Human Immunodeficiency Virus Infection. N Engl J Med 2003;348:702-10.

⁴Currier J, Kendall M, Henry K et al. Carotid Intima-media Thickness in HIV-infected and Uninfected Adults: ACTG 5078. Abstracts, 10th Conference on Retroviruses and Opportunistic Infections; February 10-14, 2003, Boston MA, Abstract 131.

⁵Hsue P, Lo J, Franklin A, Bolger AF, Deeks SG, Waters DD. Increased Atherosclerotic Progression in Patients with HIV: The Role of Traditional and Immunologic Risk Factors. Abstracts, 10th Conference on Retroviruses and Opportunistic Infections; February 10-14, 2003, Boston MA, Abstract 139b.

Next month's LipoWatch will discuss clinical approaches to cardiovascular risk

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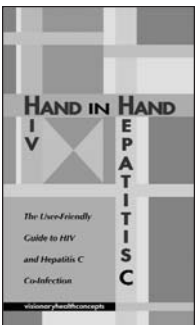
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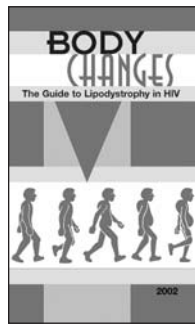


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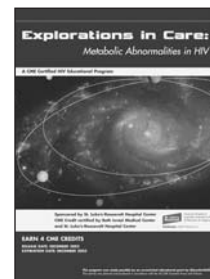


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