Changes in invasive pneumococcal disease among adults living with HIV following introduction of 13-valent pneumococcal conjugate vaccine, 2008–2014

Miwako Kobayashi, Centers for Disease Control and Prevention
William Adih, Centers for Disease Control and Prevention
Jianmin Li, Centers for Disease Control and Prevention
Ryan Gierke, Centers for Disease Control and Prevention
Olivia M. Almendares, Centers for Disease Control and Prevention
James Watt, California Department of Public Health
Nisha Alden, Colorado Department of Public Health and Environment
Susan Petit, Connecticut Emerging Infections Program
Monica Farley, Emory University
Lee Harrison, University of Pittsburgh

Only first 10 authors above; see publication for full author list.

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Miwako Kobayashi, MD, MPH; William Adih, DrPH, MPH, MD; Jannain Li, DPE; Ryan Gierke, MPH; Olivia M. Almendares, MSPH; James Watt, MD, MPH; Nisha Alden, MPH; Susan Pettit, MPH; Monica Farley, MD, FIDSA; Lee Harrison, MD; Ruth Lyndfield, MD, FIDSA; Joan Raumbach, MD, MPH, MS; Ann Thomas, MD, MPH; William Schaffner, MD, FIDSA, FSHEA; and Tamara Pilishvili, MPH. 

Background. Introduction of 13-valent pneumococcal conjugate vaccine (PCV13) in children in 2010 reduced adult IPD burden (indirect effects). In 2012, PCV13 was recommended in series with 23-valent polysaccharide vaccine (PPSV23) for adults with immunocompromising conditions, including PLHIV. We evaluated changes in IPD incidence in adults ≥19 years old with and without HIV after PCV13 introduction for children in 2010 and for immunocompromised adults in 2012. PCV13 coverage for adults 19–64 years old with indications was 6% in 2010 and 2011.

Methods. IPD cases, defined as pneumococcal infection from sterile sites, were identified through CDC’s Active Bacterial Core surveillance, with counts projected nationally. HIV status was obtained from medical records. Isolates were serotyped by Quellung reaction or PCR and grouped into PCV13-types, PPV11-types (unique to PPSV23), or non-vaccine types. We estimated IPD incidence (cases per 100,000 people) using national case-based HIV surveillance (for PLHIV) or US Census data (for non-PLHIV) as denominators. We compared IPD incidence in adults ≥19 years old with and without HIV from baseline to 2013–14. 

Results. Overall IPD incidence at baseline was 354.0 for PLHIV and 15.5 for non-PLHIV. From baseline to 2013–14, IPD rates declined in both PLHIV (-36.3%; 95% CI: -38.8, -33.7%) and non-PLHIV (-27.3%; 95% CI: -28.2, -26.5%). The largest reductions were noted in PCV13-type IPD in both PLHIV (Figure 1) and non-PLHIV (Figure 2) for both periods (-46.8% for PLHIV and -45.9% for non-PLHIV in 2011–12; -60.3% for PLHIV and -65.8% for non-PLHIV in 2013–14). Overall IPD rates were 22.8 (95% CI: 22.2, 23.4) times as high in PLHIV compared with non-PLHIV at baseline, and 19.4 (95% CI: 18.8, 20.0) times as high in 2013–2014.

Conclusions. IPD rates declined significantly in both PLHIV and non-PLHIV during the study period due to reductions in PCV13-type IPD; however, IPD rates remained 20-fold in PLHIV compared with non-PLHIV. Similar magnitude reductions in PCV13-type IPD in both groups and low PCV13 coverage in immunocompromised adults suggest that most of the observed decline is due to PCV13 indirect effects from childhood immunization.
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Chitra Ramaswamy, MBBS, DrGO, MPH1; Emily Westheimer, MS2 and Sarah Braunstein, PhD, MPH, Bhr3; 1Bureau of HIV/AIDS Prevention and Control, New York City Department of Health and Mental Hygiene, Long Island City, New York, 2Bureau of HIV Prevention and Control, New York City Department of Health and Mental Hygiene, Queens, New York, 3Bureau of HIV/AIDS Prevention and Control, New York City Department of Health and Mental Hygiene, New York, New York
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Background. With the prolonged life-span of persons with HIV (PWH) due to anti-retroviral therapy, their cancer burden has increased. Cancer continues to be a leading cause of death among PWH. Studying cancer mortality can inform and guide the development of cancer screening and prevention strategies for PWH.

Methods. We analyzed data for all persons ≥13 years who were diagnosed with HIV from 2001 to 2015 and reported to the New York City (NYC) HIV surveillance registry (HSR). Using the HSR and the underlying cause of death obtained from the NYC vital statistics registry and the National Death Index, we examined age-specific and age-standardized mortality rates from cancer and compared time trends of deaths due to HIV-related cancer to deaths from non-HIV-related cancers.

Results. There were 34,190 deaths reported among 154,688 PWH of whom nearly half (n=16,804; 49.1%) died due to HIV (excluding HIV-related cancers). Among all deaths, HIV was the leading cause, followed by cancer (both HIV and non-HIV-related) (n=5,271; 15.4%) and cardiovascular disease (n=3,724; 10.9%). The top three causes of non-HIV-related cancers were lung cancer (n=1,040; 19.7%), liver cancer (n=552; 10.5%), and colorectal cancer (n=315; 5.6%). Although the mortality rate among PWH increased over time (24.4 to 13.9 per 1,000 person-years from 2001 to 2015), the proportion of deaths attributable to all cancers increased (10.6% in 2001 to 2019 in 2015, p < 0.001). This increase was driven by non-HIV-related cancers (6.1% of all deaths in 2001 to 15.8% in 2015, p < 0.0001). The mean age increased from 2001 to 2015 among the dead (46 to 56 years) and among the censored (35 to 49 years). After controlling for demographic factors, transmission risk, and last CD4 count, the hazard ratio for cancer deaths was higher among people who inject drugs (HR = 1.5; 95% CI = 1.4–1.7) and those with last CD4 count < 200 (HR = 9.3; 95% CI = 8.3–10.5).

Conclusion. Although mortality rates are decreasing in PWH, deaths due to non-HIV-related cancers are increasing. The upward trend in the mean age suggests that aging may be contributing to this increase. Routine screening for liver and colon cancers along with smoking cessation may reduce lung, liver, and colon cancer deaths.

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1813. Fatty Liver Disease in HIV: Predictors and Response to Statin Therapy
Vanessa El Kamar, MD4, Corrilyn O. Hileman, MD5,6,7; Alex, Case Western Reserve University, Cleveland, Ohio, 2MetroHealth Medical Center, Cleveland, Ohio; 3University Hospitals Cleveland Medical Center, Cleveland, Ohio, 4Ohio Childrens and Children's Hospital, Cleveland, Ohio
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Background. Liver disease has emerged as a leading cause of mortality and morbidity in HIV. Much of the current challenge in liver disease is related to nonalcoholic fatty liver disease (NAFLD). In HIV-uninfected populations, statin therapy has been suggested as potential intervention, but no such data is available in HIV. The aims of this study are to investigate the effect of rosuvastatin on hepatic steatosis in HIV infection, measured by Liver Fat Score (LFS) and to assess the natural history and predictors of changes in hepatic steatosis over 96 weeks.

Methods. This is a secondary analysis of the SATURN-HIV trial, in which HIV+ adults on stable ART with HIV-1 RNA < 1,000 copies/mL and LDL-cholesterol < 130 mg/dL were randomized to 10mg daily rosuvastatin or placebo. Changes in LFS and in markers of systemic inflammation and monocyte activation were assessed from entry through week 96. Spearman correlations, multivariable linear regression and logistic regression were used to study relationships among variables.

Results. Overall, 147 participants were randomized to rosuvastatin n=72 to rosuvastatin n=75 to placebo); 78% were male, 68% African Americans, 8% had chronic hepatitis C and mean age and BMI were 46 years and 29/kg/m2. A significant increase in LFS over 96 weeks was seen in both the placebo and statin arms (p = 0.01 and p < 0.01 respectively, p = 0.49 between groups). Furthermore, the progression from no-steatosis (LFS ≤ -0.64) at baseline to steatosis (LFS > -0.64) at week 96 was higher in rosuvastatin arm (OR = 4.3, p = 0.03), and remained statistically significant after adjusting for demographics, HOMA (baseline and change over 96 weeks), hepatitis C, heavy alcohol use and HIV parameters. Baseline LFS was independently associated with IP-10 (β = 0.82, p = 0.03) and sCD163 (β = 0.43, p = 0.005), and the increase in LFS over 96 weeks was independently associated with IP-10 (β = 2.85, p = 0.02).

Conclusion. In HIV+ subjects on ART, hepatic steatosis increased over time, regardless of statin treatment, and was independently associated with markers of immune activation. The progression from non-steatosis to hepatic steatosis was greater on statin. Despite its effective role in reducing cardiovascular disease risk and inflammation, statin therapy does not appear effective in hepatic steatosis.

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1814. Leveraging the ART Advantage: diabetes and hypertension along the HIV care cascade in rural South Africa
Jennifer Manne-Goeher, MD, DSc, ScM1; Mark Siedner, MD, MPH2; Pascal Cote-Hermand, MBChB3; Gary Harling, ScD4; Livia Montana, DSc, MA5; Julia Rakotozafy, PhD, Xavier Gomez-Olive, MB, MPh, MSc6, Alisa Wade, MBBS, MD PhD7; Justine Davies, MD2 and Till Barnighausen, MD, DSc, Msc5; Beth Israel Deaconess Medical Center, Boston, MA; Center for Global Health, Massachusetts General Hospital, Boston, Massachusetts; 2Department of Global Health & Population, Harvard T. H. Chan School of Public Health, Boston, Massachusetts; 3Research Department of Infection and Population Health, University College London, London, United Kingdom; 4Harvard Center for Population and Development Studies, Cambridge, Massachusetts; 5School of Public Health, University of the Witwatersrand, Johannesburg, South Africa; 6King's College London, Centre for Global Health, London, UK; 7Harvard T.H. Chan School of Public Health, Boston, Massachusetts
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Background. Participation in antiretroviral therapy (ART) programs has been associated with greater utilization of care for diabetes and hypertension in rural South Africa. However, there is limited data about whether this apparent ART advantage translates into improved chronic conditions improvement in rural South Africa.

Methods. The Health and Aging in Africa: a Longitudinal Study of an INDEPTH Community in South Africa (HAALSI) is a cohort of 5,059 adults >40 in Agincourt. The study collects data on demographics, healthcare utilization, height, weight, blood pressure (BP), and blood glucose. HIV, viral load (VL) and ART drug levels are tested via dried blood spots. We defined hypertension (HTN) based on measured BP or self-report of diagnosis by a healthcare provider or use of antihypertensive medication and diabetes (DM) by measured glucose or self-report of diagnosis by a healthcare provider or the use of DM medications. Our primary predictor of interest was stage along the HIV care cascade (HIV-, HIV+ not on ART, ART with a detectable VL, and with a suppressed VL). We compared the proportion in each subgroup who were aware of and treated for their hypertension or diabetes diagnosis, and fit adjusted linear regression models to estimate differences in systolic BP and glucose among those with diagnosed HTN or DM.

Results. Rates of HTN and DM were higher in HIV- than those with a suppressed VL (HTN: 68.4% v. 46.4%; DM: 12.9% v. 8.8%, respectively). However, the suppressed VL group had higher crude rates of awareness of HTN diagnosis and treated HTN as compared with the HIV group (Aware: 69.9% vs. 65.2%, p = 0.18; Treated: 50.2% vs. 46.4%, p = 0.002). There were no significant differences in awareness or treatment rates for DM. In adjusted linear regression models with those diagnosed HTN or DM, having a suppressed VL was associated with lower mean systolic BP ( -5.94 mmHg, 95% CI: -9.68 – -2.20) and lower mean glucose ( -3.74 mmol/L, 95% CI: -5.95 – -1.50), compared with being HIV+.

Conclusion. This was preserved in models restricted to overweight and obese participants.

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