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

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**Session:** 227. HIV: Co-morbidities and Co-infections

**Saturday, October 7, 2017: 10:30 AM**

**Background.** People living with HIV (PLHIV) are at increased risk of invasive pneumococcal disease (IPD). 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 2014.

**Methods.** IPD cases, defined as pneumococcal isolation 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, PVP11-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 2011−12 and 2013−14 to the pre-PCV13 baseline (2008−09) by serotype groups.

**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.

**Conclusion.** 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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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 aged 13 years or older 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 = 1,104; 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 < .0001). This increase was driven by non-HIV-related cancers (6.1% of all deaths in 2001 to 15.8% in 2015, p < .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 = 0.9; 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
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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 RNA < 1,000 copies/mL and LDL-cholesterol < 100 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 patients 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 non-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). Hepatitis C, heavy alcohol use and HIV parameters, baseline LFS was independently associated with IP-10 (β = 0.82 p = 0.003) 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
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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 diseases management and improved treatment outcomes.

Methods. The Health and Aging in Africa: A Longitudinal Study of an INDEPTH Community in South Africa (HAALS) is a cohort of 5,059 adults 40 years in Agincourt. The study collects data on demographics, healthcare utilization, height, weight, blood pressure and lipids, HIV status and ART, HIV RNA, CD4 cell count, hepatitis, cancer and other chronic conditions. Baseline data was collected between 2001 and 2003. All adult HIV+ patients were followed up for endpoint events of death, loss to follow-up, or ART withdrawal. ART withdrawal was defined as having an undetectable VL for 3 years and then having an undetectable VL for ≥ 3 years. ART treatment outcomes were assessed using Kaplan-Meier survival analysis.

Results. Of the 5,059 adults enrolled in HAALS, 2,276 were on ART at last contact. The main endpoint event was defined as death. Among those on ART at last contact, 309 (13.5%) died. The median follow-up was 7 years (IQR 5-9 years). The survival rate was 86.5% (95% CI = 84.3 – 88.7%). In both ART and non-ART groups, we observed significant differences in mean systolic blood pressure (SBP) and mean diastolic blood pressure (DBP) at baseline and at 2 years (p < 0.0001). In adjusted linear regression models among those with diagnosed HTN at baseline, having a suppressed VL was associated with lower mean systolic BP (-5.94 mmHg; 95% CI = -7.05 – -4.83) and lower mean DBP (-3.90 mmHg; 95% CI = -5.04 – -2.75) than those without a suppressed VL. However, we did not observe any significant differences in mean systolic blood pressure or mean diastolic blood pressure at 2 years (p = 0.35 and p = 0.05 respectively).

Conclusion. ART has been associated with improved chronic diseases management in this cohort, with the exception of blood pressure.

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