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.

Journal Title: Open Forum Infectious Diseases
Volume: Volume 4, Number suppl_1
Publisher: Oxford University Press (OUP) | 2017-10-04, Pages S57-S58
Type of Work: Article | Final Publisher PDF
Publisher DOI: 10.1093/ofid/ofx162.134
Permanent URL: https://pid.emory.edu/ark:/25593/s6fbx

Final published version: http://dx.doi.org/10.1093/ofid/ofx162.134

Copyright information:
© The Author 2017. Published by Oxford University Press on behalf of Infectious Diseases Society of America. This is an Open Access work distributed under the terms of the Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Accessed October 5, 2019 5:35 PM EDT

Miwako Kobayashi, MD, MPH; William Adih, DrPH, MPH, MD; Jianmin 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

Centers for Disease Control and Prevention, Atlanta, Georgia; Respiratory Diseases Branch, Centers for Disease Control and Prevention, Atlanta, Georgia; Georgia Department of Public Health, Sacramento, California; Colorado Department of Public Health and Environment, Denver, Colorado; Connecticut Emerging Infections Program, New Haven, Connecticut; Department of Medicine, Emory University School of Medicine and Atlanta VA Medical Center, Atlanta, Georgia; University of Pittsburgh, Pittsburgh, Pennsylvania; Minnesota Department of Health, St. Paul, MN; New Mexico Department of Health, Santa Fe, New Mexico; Oregon Public Health Division, Portland, Oregon; Vanderbilt University School of Medicine, Nashville, Tennessee; National Center for Immunizations and Respiratory Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia.

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 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 provided 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 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.

Disclosures. All authors: No reported disclosures.
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, statoric 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 patients were randomized 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 29.4±7.7 kg/m². A significant increase in LFS over 96 weeks was seen in both the placebo and statin arms (ρ = 0.01 and ρ = 0.01 respectively, p = 0.49 between groups). Furthermore, the progression from non-steatosis (LFS ≤ -0.64) to baseline at 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 = 0.003) 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 hepatitis steatosis was greater on statin. Despite its effective role in reducing cardiovascular disease risk and inflammation, statoric therapy does not appear effective in hepatic steatosis.

Disclosures. C. O. Hileman, Gilead; Medical Advisory Board, Research support; G. Mccomsey, Gilead; Consultant, Consulting fee and Research support; BMS: Consultant, Consulting fee and Research support; GSK/ViV: Consultant, Consulting fee and Research support; ICON: Consultant, Consulting fee; Merck: Investigator, Research support.