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HIV POST SCD STUDY: 80% HIGHER RATE OF AUTOPSY-DEFINED SUDDEN ARRHYTHMIC DEATH IN HIV

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Background:

Persons living with HIV have higher rates of CVD including acute MI, heart failure, and our group first reported high rates of out-of-hospital presumed sudden cardiac death (SCD) using World Health Organization (WHO) criteria. However, the precise incidence of actual sudden arrhythmic deaths (SAD) in HIV remains unknown.

Methods:

Between 2011 to 2016, we prospectively identified all incident deaths attributable to out-of-hospital cardiac arrest among individuals with and without HIV aged 18-90 in SF County for medical record review and comprehensive autopsy, toxicology, and histology via medical examiner surveillance of consecutive out-of-hospital deaths. Autopsy-defined SAD had no extracardiac cause of death or acute heart failure. Final cause was adjudicated by a committee of pathologists, cardiologists, HIV clinicians, and electrophysiologists.

Results:

126 out-of-hospital HIV-infected deaths were identified, and 47 of these met WHO SCD criteria. The mean age was 65.6 years, 94% male, and 57% white. Compared to uninfected WHO-defined (presumed) SCDs (N=505), SCDs with HIV were more likely to have a history of MI, psychiatric disorder, cigarette smoking, and substance abuse. Similar to the general population, about half of WHO-defined SCDs in HIV were autopsy-defined SADs; the remainder were non-cardiac and included 16 due to occult overdose. Presumed SCDs with HIV were more likely to be due to occult overdose (13% vs 34%, $p<0.0001$) and renal failure (1% vs. 6%, $p=0.003$) as compared to uninfected presumed SCDs. Adjusted incidence ratios for WHO (presumed) SCD and autopsy-defined SAD were both significantly higher in HIV (IRR 1.82, 95%CI 1.4-2.4, $p<0.0005$ and IRR 1.83, 95%CI 1.2-2.8, $P=0.006$, see Figure). After adjustment for age, gender, heart disease and CAD, SCDs with HIV had 60% higher interstitial fibrosis by myocardial trichrome staining compared to uninfected SCDs.

Conclusion:

In this countywide postmortem study, 1/3 of apparent SCDs in HIV over a 5-year period were due to occult overdose. However, adjusted rates of both presumed SCDs and autopsy-defined SAD were 82% and 83% higher respectively in HIV compared to the uninfected population. Higher levels of cardiac fibrosis in HIV, a known substrate for SAD in the general population, may underlie the mechanism by which HIV increases risk for SAD. Development of criteria and evaluation for implantable defibrillators should be carefully considered in HIV as a means to prevent SAD in this high-risk population.