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COPD AND THE RISK FOR MYOCARDIAL INFARCTION BY TYPE IN PEOPLE LIVING WITH HIV

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Background:
People living with HIV (PLWH) are at increased risk for chronic obstructive pulmonary disease (COPD) compared to uninfected persons, in whom COPD is a known risk factor for cardiovascular disease such as myocardial infarction (MI). However, the relationship between COPD and MI in PLWH is less well understood. MIs have been classified into types including type 1 (T1MI, atherothrombotic coronary plaque rupture) and type 2 (T2MI, supply-demand mismatch as with sepsis), with a much higher proportion of T2MI in PLWH than the general population. We hypothesized that COPD would be associated with increased MI risk among PLWH, particularly for T2MI.

Methods:
We utilized data from six sites in the CFAR Network of Integrated Clinical Systems (CNICS) cohort. MIs were centrally adjudicated by two reviewers (3 if discrepancies) and also categorized by type and cause of T2MI. COPD was defined based on an algorithm we previously validated against spirometry requiring COPD diagnosis codes and ≥90-day continuous supply of long-acting COPD controller medications. Time to MI was assessed using Cox proportional hazards models. Models were adjusted for baseline age, sex, race/ethnicity, HIV viral load, nadir CD4 count, diabetes, hypertension, statin use, and CNICS site. We subsequently examined whether associations were attenuated by adjustment for smoking status (ever vs. never), as this was potentially an important confounder.

Results:
In total, 25,509 PLWH were included, of whom 423 met our definition of moderate-to-severe COPD. There were 698 PLWH who had MIs (339 T1MI [54%], 294 T2MI [46%]). COPD was associated with a significantly increased risk of MI [adjusted hazard ratio (aHR) 2.09 (95%CI 1.50-2.91)] even after adding smoking [aHR 1.88 (95%CI 1.34-2.63)]. COPD was significantly associated with T1MI and T2MI in unadjusted analyses, but only T2MI in adjusted analyses, and this was only minimally attenuated by smoking (Table); this association was particularly notable for T2MI due to sepsis/bacteremia.

Conclusion:
COPD is independently associated with an increased risk for MI in PLWH, particularly T2MI in the setting of sepsis/bacteremia. COPD severity, inadequate disease control and/or exacerbations can contribute to supply-demand mismatch, and COPD increases risk for pneumonia, a common cause of sepsis. Further investigation is required to understand mechanisms for this association and to optimize preventative and therapeutic strategies.