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Title: The impact of airway infection on cardiovascular risk during COPD exacerbations

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Body: Arterial stiffness, a validated measure of cardiovascular risk, increases from stable COPD to exacerbation (Patel et al, ERS 2011). We hypothesised this increased cardiovascular risk was mediated by airway infection. We measured aortic pulse wave velocity (aPWV) in the stable state, at exacerbation and during recovery at 3,7,14 and 35days thereafter. Infective exacerbations were defined by a potentially pathogenic microbe (PPM) in exacerbation sputum by culture or positive PCR for H. influenzae, S. pneumoniae, M. catarrhalis, or human rhinovirus. Differences in the area under the curve (AUC) adjusted for stable aPWV level between groups were compared by unpaired t-test. 55 COPD patients (32 male, 11 current smokers) had a mean±SD FEV_1 of 1.14±0.41L (46.7±18.5%predicted) and FEV_1/FVC ratio 0.46±0.14. Two-thirds of them (36/55, 65%) produced a sputum sample at exacerbation. Two-thirds of these events (24/36, 67%) had an identifiable PPM. Patients with an infective exacerbation had a greater rise in arterial stiffness from stable state to exacerbation (1.4±1.7ms-1 vs 0.4±1.0ms-1, p=0.050). Arterial stiffness was also higher during the recovery period in those with an infective exacerbation (AUC 37.4±55.2ms-1days vs 11.4±44.3ms-1days, p=0.036).

The increase in arterial stiffness during COPD exacerbations appears to be driven by airway infection and may explain the association between these infective events and increased cardiovascular risk.