Background: Vascular calcium (Ca) deposition is an actively regulated inflammatory process. Here we test the hypothesis that vascular inflammation predisposes to subsequent deposition of Ca.

Methods: 137 patients (age 61±13, 48.1%Male, without active cancer or inflammatory disease) underwent 2 PET/CT studies (1 to 4.5 years apart). Arterial inflammation was determined by measuring baseline thoracic aortic FDG uptake on axial sections using PET, as a standardized uptake value (SUV). A separate, blinded investigator assessed arterial segments with or without baseline Ca (BLCa+ve/BLCa-ve) for subsequent Ca deposition as a change (between 2 CT scans) in segmental square root of Ca volume score (using a cut-off value of 2.5), and classified the segments as “stable” or “progressed”. Segmental SUV for each segment was expressed as % difference from over-all mean vessel SUV for each patient (%DIFF).

Results: 42% of BLCa+ve and 3% of BLCa-ve segments progressed. Mean (SD) %DIFF SUV was higher in progressed vs. stable in both BLCa+ve segments (0.92 ±7.05 vs. -4.06 ±10.83, p = 0.003) and BLCa-ve segments (5.34 ±8.87 vs. -1.08 ±9.06, p = 0.004, fig). The likelihood of subsequent Ca deposition increased significantly with each unit increase in SUV. [OR (95%CI), 2.50 (1.04-6.05), p = 0.04].

Conclusion: Baseline inflammation precedes subsequent Ca deposition. This phenomenon is evident in arterial segments with or without baseline Ca, thus linking local vascular inflammation with atherosclerotic Ca deposition.