**ROLE OF MICROPARTICLES IN CARDIOVASCULAR CALCIFICATION**

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Despite clinical evidence linking arterial calcification to risk of acute cardiovascular events, the mechanisms underlying mineral nucleation and growth in atherosclerotic plaques remain unknown. Finite element modeling of stress distribution within plaques indicates that subcellular microcalcifications in a plaque’s fibrous cap can promote plaque rupture. In contrast, large calcifications may stabilize the plaque, but mechanism(s) that give rise to these two morphologies are unclear. We showed that calcific mineral formation results from a series of events beginning with aggregation of calcifying vascular wall cell-derived vesicles (extracellular vesicles) and continuing through microcalcification to large macrocalcification. We developed a three-dimensional controllable collagen-hydrogel model to visualize in vitro the earliest events associated with vascular mineral deposition. Using advanced high-resolution microscopic and spectroscopic analyses of calcified human atherosclerotic plaques and three-dimensional synthetic system, we provided a crucial link between plaque collagen content and calcification morphology—the two determinants of atherosclerotic plaque stability—thus offering novel insight into the mechanisms of plaque integrity.