Osteoclast-like macrophages drive atherosclerotic plaque calcification, report Chinetti-Gbaguidi et al.

The accumulation of calcium deposits in major arteries is associated with aging and with atherosclerotic plaque formation. Once thought to be a passive process, vascular calcification is now known to occur via the same mechanism as bone formation, suggesting that the balance of osteoblastic (bone-forming) and osteoclastic (bone resorptive) activity becomes somehow skewed. Osteoclasts derive from the same lineage as monocytes and macrophages. And since such cells are abundant in plaques, Chinetti-Gbaguidi and colleagues decided to examine human plaque macrophages for their osteoclastic activity. The researchers specifically focused on cells surrounding calcium deposits and found that, while these macrophages expressed the osteoclastic marker CA2, their expression of a key osteoclastic enzyme involved in bone resorption was abnormally low, as was their resorptive activity in vitro. These osteoclast-like macrophages were of the alternative, or M2, phenotype, which tends to be associated with anti-inflammatory mechanisms and plaque stability. The discovery of these cells with deficient osteoclast behavior, however, suggests that M2 macrophages are not always atheroprotective. The findings also indicate that therapeutic modulation of the osteoclast-like cells may reduce plaque calcification and slow atherosclerosis progression.

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