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Circulating CD14+CD16- classical monocytes do not associate to a vulnerable plaque phenotype, nor prognosticate secondary events in atherosclerotic patients.

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Introduction: Mouse studies have established distinct monocyte subtypes that participate in the process of atherosclerotic lesion formation. The pro-inflammatory Ly6Chigh monocyte subtype actively contributes to murine plaque progression and destabilization. Also in humans different monocyte subtypes have been identified, of which the CD14+CD16- classical monocyte is suggested to display similar pro-atherosclerotic properties as the murine Ly6Chigh subtype.

Purpose: We aimed to investigate if circulating CD14+CD16- classical monocytes reflect a vulnerable plaque phenotype and if they associate with the risk of secondary adverse manifestations.

Methods: We enrolled 175 carotid endarterectomy patients of the Athero-Express biobank in our study. Just prior to surgical procedure, blood was collected and peripheral blood mononuclear cells were isolated. Characterization of monocyte subsets was performed by flow cytometry. A vulnerable plaque phenotype was defined as increased presence of macrophages, smooth muscle cells and fat, or decreased collagen deposition, calcification, neovascularization and intraplaque haemorrhage. To investigate the prognostic value for recurrent cardiovascular events of circulating CD14+CD16- classical monocytes over time, we used Cox regression models.

Results: We observed no correlation between plaque macrophages and absolute numbers of blood derived classical monocytes (R² = 0.004, p = 0.456). In addition, all other features of a vulnerable plaque phenotype including low amounts of collagen and smooth muscle cells, and increased fat content, neovascularization and intraplaque haemorrhage, were not associated with differential levels of peripheral classical monocytes or other monocyte subsets (p > 0.05). Total counts of peripheral monocytes, as well as CD14+CD16- classical and other monocyte subtypes were not different between patients with and without follow-up events. Also in Cox regression models, circulating monocyte subsets were not associated with the risk of secondary cardiovascular events over time.

Conclusion: Circulating CD14+CD16- classical monocytes do not associate to specific vulnerable plaque characteristics. In addition, they do not predict recurrent adverse manifestations. This suggests that circulating monocytes do not reflect plaque phenotype and have only limited value in identifying patients at risk for future cardiovascular events.