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INCREASED MICROVASCULAR BLOOD FLOW AND PERMEABILITY ASSOCIATES WITH FDG SIGNAL IN HUMAN ATHEROMA

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Background: Studies have shown the feasibility of imaging carotid plaques with 18F-fluorodeoxyglucose (FDG) and positron emission tomography (PET), a signal that may correlate with presence of macrophages. Yet, macrophages exhibit heterogeneity such that their presence alone may not reflect active inflammation. Moreover, the microvascularization associated with inflamed plaques may increase delivery of tracer to a lesion. The correlation of FDG signal with vascular channels has not been investigated previously. Therefore, this study examined the correlation between FDG uptake and plaque microvascular flow and permeability measured by magnetic resonance imaging (MRI). We further correlate these findings with histologic markers of inflammation and plaque microvascularization.

Methods: 15 patients (median age 73 years, 40% female) with severe carotid stenosis undergoing carotid endarterectomy (CEA) were studied with FDG PET and MRI pre-operatively. We measured FDG uptake (as target to background ratio, TBR) by PET, and Ktrans (reflecting plaque blood flow and permeability) by MRI, and correlated imaging with immunohistochemical markers of macrophage number (CD68+) and activation (MHC Class II+), as well as microvessel density (CD31+) within CEA specimens.

Results: There was a stepwise increase in FDG uptake (maximum TBR) with increasing tertiles of macrophage number (CD68+, p=0.002), activated leukocytes (MHC-II+, p=0.005), and microvessel content (CD31+, p=0.048). FDG uptake in macrophage-rich plaque sections (1.54, IQR 1.26-1.78) exceeded that in plaque regions without inflammation (1.13, IQR 1.04-1.27) (p=0.01). FDG uptake correlated positively with Ktrans (p=0.002). The median Ktrans value in macrophage-rich plaque sections (0.2, IQR 0.14-0.41) was higher than in plaque regions without inflammation (0.054, IQR 0.037-0.136) (p=0.01).

Conclusions: Plaque regions with active inflammation, as determined by macrophage content and MHC-II expression, show increased FDG uptake, which is correlated with increased plaque flow permeability (Ktrans) and neovascularization (CD31+). Thus, FDG signal correlates with neovascularization as well as inflammation.