T1 Gadolinium Enhancement of Intracranial Atherosclerotic Plaques Associated with Symptomatic Stenoses

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Purpose

Inflammation in atherosclerotic plaques locally alters vascular endothelium permeability, allowing gadolinium (Gd) contrast uptake and visualization on T1 weighted MR imaging. Atherosclerotic plaque enhancement in the cervical carotid arteries has been correlated with a higher incidence of stroke (1). However, only a paucity of research has examined this relationship within the intracranial vasculature (2). We studied the relationship between intracranial plaque enhancement and acute symptomatic presentations of intracranial atherosclerotic disease.

Materials and Methods

Using our institution's PACS and medical record databases, we retrospectively identified and analyzed 11 patients with high-grade atherosclerotic plaques (>70% stenosis) within the proximal intracranial vasculature (supraclinoid ICA, A1-A2 ACA, M1-M2 MCA, vertebral-basilar, P1-P2 PCA) that met inclusion criteria: 1) MRI/MRA 3D time-of-flight (TOF) imaging at time of diagnosis or symptomatic presentation and 2) visualization of intracranial plaques in vessels perpendicular to the imaging plane to minimize partial-volume averaging. Plaques were classified as asymptomatic or symptomatic if they induced acute physiological symptoms (as determined by neurological exam on presentation) and/or demonstrated DWI positive infarcts in the vascular distribution of the stenosed vessel. Two neuroradiologists conducted independent and blinded evaluation of each intracranial plaque using axial 3D TOF and T1 spin echo imaging (pre- and post- contrast injection) acquired as part of the initial neurovascular MR imaging evaluation. Scoring was performed on a five point scale (1-5) and results were subgrouped based on the probability of plaque enhancement (1-2 unlikely to be enhancing, 3 equivocal, 4-5 likely to be enhancing). Contralateral non-stenotic vessels served as internal controls.

Results

In 11 patients, 8 symptomatic and 3 asymptomatic intracranial plaques were subgrouped. In addition, 7 intracranial plaques were scored as intermediate to highly enhancing (score of 4-5) and 4 plaques were scored as likely non-enhancing (score of 1-2). We found that 6/7 (86%) enhancing plaques versus 2/4 (50%) non-enhancing plaques were symptomatic. We observed no associated vessel enhancement in the contralateral control symptomatic vessels. Figure 1a-b depicts pre- and post- contrast T1 SE images of a typical plaque that was scored as highly enhancing (score 5) and symptomatic involving the A2 ACA segment.

Conclusion

Our preliminary study demonstrates that T1 Gd-enhancing plaques may be an indicator of progressing or symptomatic intracranial atherosclerotic disease. We report an association between intracranial plaque enhancement and incidence of acute symptoms from severe intracranial stenoses. Our findings suggest that acute inflammation of intracranial stenoses may exacerbate luminal narrowing and/or plaque instability leading to symptomatic presentations.

References 1) Kerwin et al Radiology 2007. 2) Ryu et al, Cerebrovasc Dis 2009.

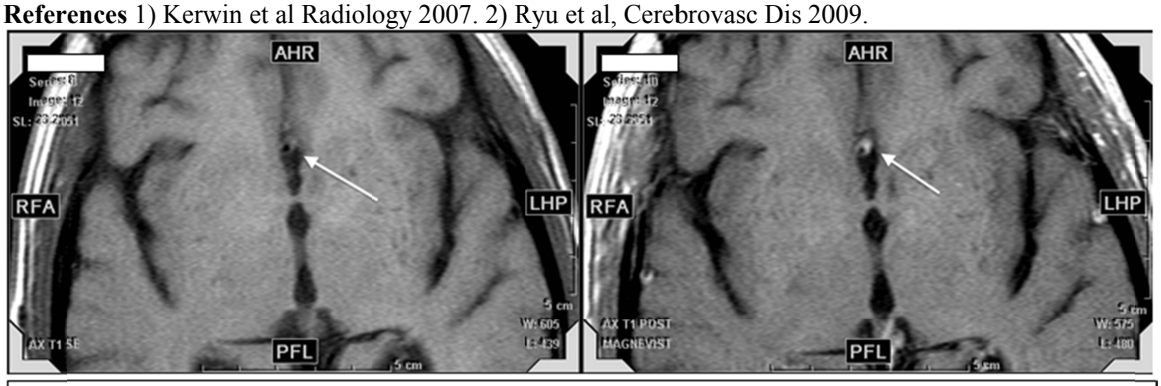

Figure 1 Pre (a) and Post (b) T1 SE images of an intracranial plaque in the A2 segment that is enhanced after contrast injection (b-arrow).