

Characterization of carotid plaque by using an inversion-recovery based T1-weighted imaging in correlation with ipsilateral ischemic events

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BACKGROUND Atherosclerotic carotid stenosis is a major cause of cerebral ischemia. However, ischemic events in the territory of the carotid artery are not necessarily related to the severity of stenosis, and treatment of moderate carotid stenosis is controversial. The aim of this study was to assess prevalence of ischemic events ipsilateral to the carotid artery characterized by a magnetic resonance imaging according to the severity of stenosis.

METHODS Between December 2001 and June 2004, 222 patients with suspected or proven atherosclerosis of carotid artery underwent plaque imaging by using a three-dimensional inversion-recovery based T1-weighted imaging (MPRAGE) on a 1.5tesla clinical system. After exclusion of 45 occluded arteries, 392 atherosclerotic carotid arteries were enrolled in the study. High signal of carotid plaque on MPRAGE was defined as over 200% signal of adjacent muscle (typically sternocleido mastoid muscle), and correlated with ipsilateral ischemic events within past six months. Forty arteries from 25 patients were studied repeatedly with interval of 3 to 790 days (median 161 days).

RESULTS Ipsilateral ischemic events were significantly more prevalent in carotid arteries with MPRAGE high signal than in carotid arteries with low signal in each of 0-29, 30-69, and 70-99% stenosis groups ($P < 0.05$, 0.001, 0.05 respectively). Prevalence of ischemic events in 0-29 and 30-69% stenoses with MPRAGE high signal was similar to prevalence in 70-99% stenosis with low signal. In the follow-up study, numbers of high and low signal carotid arteries were 18 and 22 respectively, in which only two of high signal carotid arteries changed significantly in MPRAGE signal, and five of the 18 carotid arteries with high signal were followed by ipsilateral ischemic events (Fig 1).

DISCUSSION The results of the present study that the high intensity did not change significantly in the follow-up interval suggest that MPRAGE high intensity is not due to acute or subacute thrombus or hematoma. Therefore, MPRAGE high signal may not be the result of plaque rupture, and could a risk of Thromboembolism. Following ex vivo study of correlation between MRI and histology, MPRAGE high signal corresponded to lipid-rich necrotic core mixed with hemorrhage (unpublished data).

CONCLUSION High signal in carotid plaque with a T1-weighted magnetic resonance imaging was highly associated with ipsilateral ischemic events, and stable over months, and could be a risk of subsequent ischemic events.

Fig 1. Follow-up of a right carotid plaque

Top row: initial MRI

Bottom row: 5 months after

The plaque did not change significantly

in the follow-up interval.

