

# Signal Evolution of Intraplaque Hemorrhage in Asymptomatic and Symptomatic Carotid Plaque: a Long-term In Vivo High-Resolution Magnetic Resonance Imaging Follow-up Study

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## INTRODUCTION:

IPH is a common characteristic of carotid plaque. Previous pathologic studies demonstrated that repeated IPH is detected more frequently in symptomatic plaques than in asymptomatic plaque. To test the hypothesis, we used in vivo multi-contrast high-resolution MRI to consecutively observe signal evolution of carotid IPH every 6 months during a total 3 years period, and investigated the difference in signal evolution between asymptomatic and symptomatic plaque.

## MATERIALS AND METHODS:

Between December 2005 and May 2009, we serially studied 20 recent hemorrhagic plaques in 18 asymptomatic patients (17 men, 1 woman, 72.0±7.5 years) and 16 recent hemorrhagic plaques in 15 patients (14 men, 1 woman, 73.8±3.4 years) with cerebrovascular ischemic events (stroke, transient ischemic attack, or amaurosis fugax) in the territory of index plaques. The cerebrovascular ischemic events were within 1~3 weeks before the baseline high-resolution MRI examination. A recent IPH was identified only if the maximal signal intensity of IPH was >150% than that in the adjacent muscle in all 4 contrast weighted images [1], without disruption of fibrous cap or plaque ulceration, without total occlusion and no luminal thrombus. From the baseline (month 0) to the end point (month 36), each patient were performed total 7 repeated multi-contrast high-resolution MRI examinations on index arteries (1 examination every 6 months). The institutional medical ethical committees approved the study, and all patients were given written informed consent. MRI examinations were performed on a 3.0-T MRI scanner (Signa Excite, General Electric Medical System, Wisconsin, USA). A bilateral 4-channel phased-array surface coil was used. A standardized protocol was used to obtain 4 different contrast MR images: (1) 3D time-of-flight MR angiography (3D-TOF MRA), (2) quadruple-inversion-recovery T1-weighted (QIR T1W), (3) T2-weighted (T2W), and (4) proton density-weighted (PDW). All images were obtained with the following parameters: field-of-view of 14 cm, matrix size of 256 × 256, slice thickness of 2 mm with no inter-slice gap. An eligible IPH at baseline was locked and identically measured throughout all following time points. To track the signal intensity change of IPH, the contrast-to-noise ratio (CNR) was calculated according to equation:  $CNR = (SI_{IPH} - SI_{muscle}) / SD_{noise}$ . The  $SI_{IPH}$ ,  $SI_{muscle}$  and  $SD_{noise}$  presented as the signal intensity of IPH, adjacent reference muscle, and background noise, respectively. The Student unpaired *t* test and Fisher's exact test were applied to estimate the difference in IPH CNR and clinical risk factors between asymptomatic and symptomatic groups at baseline. Repeated measures analysis of variance (ANOVA) was used to estimate the difference in IPH CNR at different follow-up time points in each group and between two groups. Calculations were performed with statistical software (version 15.0, SPSS).

## RESULTS & DISCUSSION:

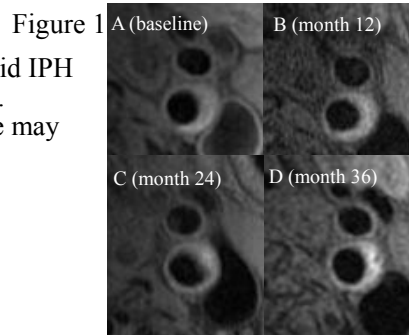
At baseline, there were no significant differences in risk factors of atherosclerosis according to clinical data and in IPH CNR on each contrast imaging between the asymptomatic and symptomatic groups ( $P > 0.05$ ). During repeated measures ANOVA, the IPH CNR change with follow-up time exhibited a markedly significant difference on imaging of 3D-TOF MRA ( $P = 0.021$ ), QIR T1W ( $P = 0.001$ ) and PDW ( $P = 0.003$ ) between the two groups, except for that on T2W imaging ( $P = 0.423$ ). In asymptomatic group, the time significantly influenced the IPH CNR change on all contrast imagings ( $P < 0.05$ ). Compared with no significant change of IPH CNR in symptomatic group, the IPH CNR in asymptomatic group presented as a gradually descending trend on all contrast imagings ( $P < 0.05$ ) (figure 1, 2). These results indicate that IPH signal evolution is multi-temporal in symptomatic carotid plaque, but the evolution is mono-temporal in asymptomatic carotid plaque. Furthermore, the difference in signal evolution between the 2 groups proves that repeated IPH is more common in the symptomatic group than in the asymptomatic group. The exception on T2W imaging may be due to not only the IPH degradation rate, but the content of water and inflammatory cells [2].

## CONCLUSION:

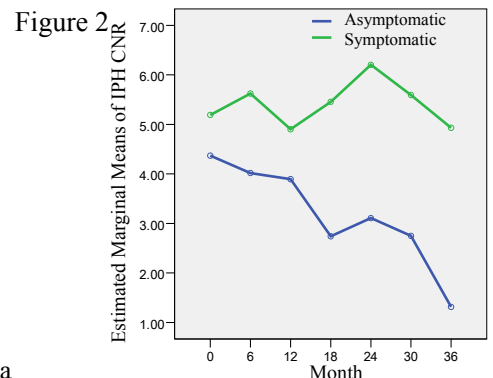
Asymptomatic and symptomatic recent carotid IPH demonstrated different MRI signal evolution. Repeated hemorrhage in symptomatic plaque may be the critical factor for the difference.

## REFERENCES

1. Altaf N, Daniels L, Morgan PS, et al. *J Vasc Surg* 2008;47:337-342
2. Milei J, Parodi JC, Alonso GF, et al. *Am Heart J* 1998;136:1096-1105



A 72-year old male patient had a left carotid plaque with IPH. From baseline to end point, high signal IPH can be always detected. A, B, C, and D are QIR T1W images at different follow-up time points.



The IPH CNR means in asymptomatic group exhibited an obvious descending trend on QIR T1W imaging, however, there was no any significant trend in the symptomatic group.