

Clinical value of post-contrast vessel wall imaging with MSDE for patients with cerebral arteriovenous malformation

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Introduction

Vascular wall thickening is a well-known late effect of radiation to vessel walls and can be recognized histologically and also radiologically [1, 2]. Cerebral arteriovenous malformation (AVM) is a congenital vascular malformation often treated with stereotactic radiotherapy or radiosurgery, aiming at obliteration of the nidi of AVMs by the effects of radiation. Complete obliteration after radiotherapy usually takes several years and progresses slowly and indistinctly on conventional MR imaging. It would be helpful to assess therapeutic effects with higher sensitivity more shortly after radiotherapy.

Motion sensitized driven equilibrium (MSDE) preparation is a recently introduced technique to black blood imaging and is expected to highlight thickened vessel walls in atherosclerosis. To our knowledge, however, the utility of vessel wall imaging with a MSDE technique has not been determined on a clinical basis for the evaluation of cerebral AVMs. The purpose of this study was to evaluate the clinical utility of vessel wall imaging with a MSDE technique for patients with cerebral AVM, compared with standard bright blood imaging without MSDE preparation.

Methods

Patient demographics

IRB at our hospital approved this study. Fifteen patients with cerebral AVM (6 male and 9 female patients, aged 24 to 59 years, mean 39.2 years) were recruited. Eight of the patients had not undergone any surgical treatment or radiotherapy for AVM before their participation in this study. The other seven patients had been treated with gamma-knife radiosurgery 2.5 to 5.7 years prior to their participation. All of the patients underwent contrast-enhanced MRI, including post-contrast (A) 3D T1-weighted variable flip angle fast spin echo imaging (a research version of CUBE T1) with MSDE preparation and (B) 3D fast SPGR (FSPGR) without MSDE preparation. Pre-contrast CUBE T1 with MSDE preparation was acquired as a control.

Methods for MR imaging

A 3-T clinical scanner (GE Signa HDxt) was used. We used a standard MSDE preparation scheme (90°_x - 180°_y - 90°_x) [3]. Imaging parameters were as follows: (A) CUBE T1 (with MSDE preparation, motion sensitization gradient = 7 s/mm² in 3-axis composite value, TR/TE = 500/minimum ms, FOV = 22 cm, slice thickness/interslice gap = 0.8/0.4 mm, matrix = 256x256 (512x512 after zero-fill interpolation), and (B) 3D FSPGR (without MSDE preparation, TR/TE = 20/2.2 ms, FA = 20°, FOV = 18 cm, slice thickness/interslice gap = 0.8/0.4 mm, matrix = 256x256 (512x512 after zero-fill interpolation)).

Analyses

Images were assessed on (i) presence or absence of contrast enhancement of thickened vascular walls of feeders or drainers (present or absent) and on (ii) contrast enhancement of intra- and peri-nidal parenchyma with three grades (none, focal perivascular, diffuse parenchymal). Two readers graded the degree of contrast enhancement ((i) and (ii)) on (A) CUBE T1 with MSDE and (B) FSPGR without MSDE, on a consensus basis. We used non-parametric, paired comparison for statistical analyses (McNemar test for (i) and Wilcoxon's signed rank test for (ii)). Statistical significance was set at $P = 0.05$.

Results

Excellent blood signal suppression was recognized on post-contrast, MSDE-prepared, CUBE T1 images [Fig. 1]. The degree of contrast enhancement is higher in (A) CUBE T1 with MSDE than in (B) FSPGR without MSDE on both (i) thickened vascular walls of feeders or drainers ($P = 0.031$) and (ii) intra- and peri-nidal parenchyma ($P = 0.046$) [Fig. 2]. With regard to the status of radiosurgery, (i) vascular wall enhancement was recognized in all post-treatment patients (7/7) and not recognized in all treatment-naïve patients (8/8) in (A) CUBE T1 with MSDE; while contrast enhancement was detected only in one post-treatment patient (1/7) and none of the treatment-naïve patients (0/8) in (B) FSPGR without MSDE. Five of the eight treatment-naïve patients showed contrast enhancement in perivascular parenchyma in the nidi of AVMs in CUBE T1 with MSDE (A) but only one in FSPGR without MSDE. These five treatment-naïve patients whom focal perivascular contrast enhancement was noticed had evidence of prior, either symptomatic or asymptomatic, hemorrhage on MRI.

Conclusion

Our results suggest MSDE preparation can achieve excellent black blood capability in post-contrast CUBE T1 and improve detectability of contrast enhancement of abnormal vessels of cerebral AVMs either in pre-treatment or post-treatment status, compared with standard, post-contrast, 3D gradient-recalled echo imaging without MSDE preparation. These advantages of CUBE T1 with MSDE may help us to confirm therapeutic effects on cerebral AVMs earlier after radiotherapy. Post-contrast T1 CUBE with MSDE showed intranidal perivascular enhancement more clearly in association with prior hemorrhage suggesting higher risks of rupture of cerebral AVMs, which may offer another benefit to prompt treatment.

References

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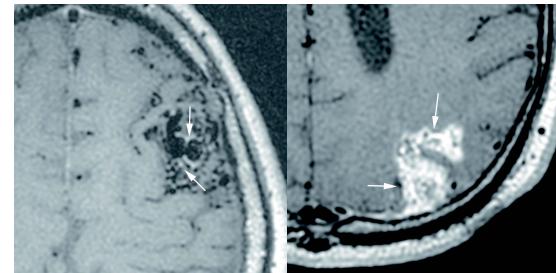


Fig. 1 Post-contrast CUBE T1 images with MSDE: focal perivascular intranidal enhancement (left) and diffuse parenchymal enhancement (right)

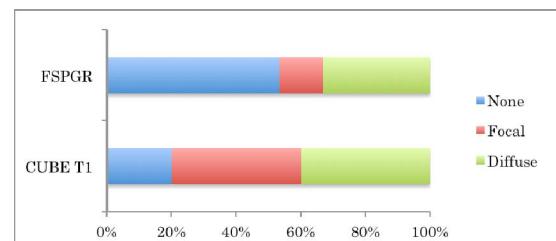


Fig. 2 Stacked bar chart of the degree of contrast enhancement of intra- and peri-nidal parenchyma