Comparison of Susceptibility-Weighted Imaging and Tracer Kinetic Model Analysis in Brain Tumors

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Introduction

Angiogenesis of forming new blood vessels is known as a pathophysiologic process that is closely related to tumor activity and grading. As tumor is progressing to a higher grade with an increasing proliferation rate, the growth of tumor cells may outstrip so that the local blood supply is no longer adequate for nutrient demands, resulting in a regional hypoxia to induce the new vessel formation. These newly formed vasculatures are hyperpermeable because of their poorly formed endothelial cells. Noninvasive imaging to simultaneously monitor the oxygen supply and leakage profile across these immature vessels is thus of high clinical interest in characterizing tumor cells. The purpose of this study was designed to evaluate the blood oxygenation level dependency (BOLD)-based vasculature and angiogenic status with microvascular proliferation in brain tumors by using susceptibility-weighting imaging (SWI) [1] and the first-pass pharmacokinetic (FPPM) model [2] of T2* MR perfusion-weighted images. We compared SWI and FPPM images with supplementary information from conventional contrast-enhanced T1-weighted (CET1) imaging to find corresponding pathological condition of vascular hyperplasia occurred on tumor proliferations.

Material and methods

Tracer kinetic parameters of the FPPM model [2] were calculated by using the conventional T2* perfusion-weighted MR images (TR/TE=1000/44ms, matrix=128×128, flip angle=90^o at 1-second interval with 60-75 dynamic time points of echo-planar pulse sequence) at 1.5T (Signa HDx, GE). The method used the estimate of vascular contrast medium concentration acquired from normal white matter to allow simultaneous mapping of endothelial permeability (K^{trans}) and the fractional plasma volume (v_p) of brain tissues. The SWI sequence was performed using a three-dimensional, fully velocity-compensated gradient-echo sequence at a 1.5T or 3T (Achieva, Philips Medical System, Best, The Netherlands) scanner (TR/TE=52/40, 67/30; flip angle=25^0, 25^0; slice thickness=2, 1.5mm; in-plane resolution=0.8×0.8, 0.65×0.65 mm^2 for 1.5T and 3T, respectively). The phase images were first unwrapped using a region growing algorithm [3] followed by a high pass filter to obtain filtered phase images. These filtered phase images were then set between zero and unity to get the phase mask and multiplied six times with the magnitude images to enhance the visibility of the venous structures [3]. Venograms were finally computed with use of minimum intensity projection (mIP) technique over five to seven slices. Lesion detection of visible tumor boundaries, abnormal blood vasculature and internal lesion architecture [1] were analyzed in this study by an experienced neuroradiologist (C.J.J) to compare the detecting efficiency of SWI, FPPM, and CET1 images. Score ranging from 0 to 5 was assigned to represent the level of visualizing efficiency of each imaging from the lowest to the highest. Eight patients with brain tumors were included in this analysis.

Results

The preliminary results of observation from one representative case with brain tumor were demonstrated in Fig.1. The enhanced part occurred in the tumor region is suggestive of increased local blood volume content and endothelial permeability shown on FPPM model or an increase in the local deoxyhemoglobin in that area of SWI. It appears to have a high vascular activity in this area which is suggested to be an active part of the tumor. Total scores were summarized in Table 1. Tumor boundaries were easily detected using CET1 technique. SWI is apparently superior to the rest methods in visualization of tumor vasculature of regional blood supply and drainage (Fig.1c, red arrow). The perfusion-based technique of the FPPM model has better detection in internal architecture than SWI and CET1.

Discussion and conclusions

FFPM is a method allowing simultaneous mapping of endothelial permeability and blood volume in brain lesions. It can be used in principle to obtain the blood volume map of the increased microvascularity which is correlated with growing tumors as well as to detect the leakage profile across the destructive BBB areas. Only one case in this study showed worse contrast with FPPM between the lesion and normal areas as compared to SWI and CET1 sequences (not shown). However, the FPPM method relies on blood perfusion efficiency: it has obstacle either in visualizing very small vessels when blood flow is very slow or in differentiating the vasculature from blood products in tumor areas because of its constrained spatial resolution or poor sensitivity to oxygenation changes. The development of SWI allows for improved contrast and detection of both the tumor vasculature and hemorrhage that cannot be clearly delineated by the FPPM method. Furthermore, the BOLD effect because of the lower oxygen saturation in veins makes it feasible to image tumor vascularity without the use of contrast agents. But SWI is the technique which is accessible mainly for venous systems, while the blood supply of incoming arteries of brain tumors is hardly detectable by this susceptibility-weighted technique. SWI serves an important role in revealing complementary information that is otherwise missed by perfusion-weighted imaging. With the accumulation of more cases in the future studies, further insight into the mechanism of the tumor angiogenesis process with regard to patients with specified tumor subtypes should be expected by using these two techniques.

	v_p	K^{trans}	SWI	CET1
Tumor boundary	2.33	1.83	2.33	5
Vasculature	0	0	3	0
Internal lesion architecture	4.17	3.67	2.50	1.83

Table 1. Average scores from all tumor cases evaluated with v_p, K^{trans}, SWI, and CET1 techniques

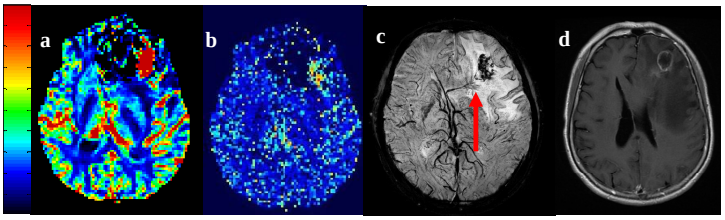

Figure 1. Patient with metastatic papillary carcinoma. (a) v_p with evaluated scores of tumor boundary=4, vasculature=0 and internal lesion architecture=0. (b) K^{trans} with evaluated score of tumor boundary=4, vasculature=0 and internal lesion architecture=5 (c) SWI with evaluated score of tumor boundary=4, vasculature=5 and internal lesion architecture=5 (d) CET1 with evaluated score of tumor boundary=5, vasculature=0 and internal lesion architecture=1.

References: [1] Sehgal V, et al., JMRI 2006;24:41-51. [2] Johnson G, et al., MRM 2004;51:961-8. [3] Rauscher A, et al., JMRI 2003;18:175-180.