J HK Coll Radiol. 2008;11:19-23

ORIGINAL ARTICLE

CME

Comparison of 1.5 Tesla and 3.0 Tesla for Skull Base Lesion Enhancement

FJ Londy,[1] S Rohrer,[1] S Kumar,[1] H Parmar,[1] SK Mukherji[1,2]

[1]Department of Radiology, and [2]Department of Otolaryngology Head and Neck Surgery, University of Michigan, Ann Arbor, USA

ABSTRACT

Aim: *To determine whether there are significant differences between 3.0 Tesla and 1.5 Tesla in the degree of contrast enhancement of skull base lesions.*

Patients and Methods: *Fifteen consecutive patients with lesions involving the skull base who had undergone clinical magnetic resonance imaging on both 1.5 Tesla and 3.0 Tesla scanners were identified. Signal intensity and contrast-enhancement ratios were normalised to brain and measured for each abnormality at 1.5 Tesla and 3.0 Tesla. Statistical analysis consisted of paired Student* t *test. Additionally, a mathematical simulation of the influence of field strength on gadolinium enhancement was created.*

Results: *The average percent increase in lesion signal-enhancement ratio identified on the postcontrast enhanced T1-weighted image compared with the non-contrast T1-weighted image was 103% at 1.5 Tesla and 172% at 3.0 Tesla. The increased enhancement identified at 3.0 Tesla compared with 1.5 Tesla was statistically significant (p = 0.001). This result was predicted by the mathematical model.*

Conclusions: *Enhancement of skull base lesions is significantly greater at 3.0 Tesla than at 1.5 Tesla. The enhancement gain is consistent with the known increases in tissue T1 with increasing field strength. Using the selected T1-weighted acquisition settings at 1.5 Tesla and 3.0 Tesla, the impact of a given tissue gadolinium concentration has a predictable greater impact on signal-enhancement ratio at 3.0 Tesla relative to 1.5 Tesla.*

Key Words: *Gadolinium; Magnetic resonance imaging; Skull base*

INTRODUCTION

3.0 Tesla (3.0 T) imaging provides many desirable opportunities that stem from an anticipated increase in signal-to-noise (SNR). Typically, SNR gains are traded for greater spatial resolution, scan speed, and improved lesion conspicuity.[1] The effect of gadolinium contrast enhancement is currently being investigated. Early results indicate that gadolinium administration produces higher contrast between tumour and normal brain at 3.0 T versus 1.5 T.[2]

Imaging of the skull base poses numerous challenges. The complex anatomy of the extracranial head and neck can make it difficult to differentiate normal structure from pathology.[3,4] Contrast administration is routinely performed to help identify a variety of abnormal processes that can involve this region. Asymmetrical enhancement is routinely used to identify infectious, inflammatory, or neoplastic processes.[3] There is relatively little information available on the effect of higher field strength on enhancement of disease processes involving the skull base. The intent of this study was to measure the difference in contrast enhancement between 3.0 T and 1.5 T for diseases involving the extracranial head and neck.

PATIENTS AND METHODS

Patients with skull base abnormalities scanned at the University of Michigan, Ann Arbour, USA, using both a 1.5 T system (General Electric, Milwaukee, USA) and a 3.0 T system (Intera Achieva Philips Medical Systems, Bothell, USA) between April 2003 and June 2005 were enrolled in the study. Fifteen patients with identifiable skull base lesions met these criteria. There were 10 men and 5 women, with an average age of 53 years.

Correspondence: *Dr SK Mukherji, Department of Radiology, B2B311-0030, University of Michigan Health Systems, 1500 E Medical Center Drive, Ann Arbor, MI 48109-0030, USA.*
Tel: (1 734) 936 8865; Fax: (1 734) 764 2412;
E-mail: mukherji@umich.edu

Submitted: 9 November 2007; Accepted: 5 March 2008.

© 2008 Hong Kong College of Radiologists

Routine skull base magnetic resonance imaging (MRI) protocols were performed for each patient at both field strengths, and included pre- and postcontrast axial T1-weighted (T1W) spin-echo sequences. T1W acquisition parameters, according to the pre-existing protocols, were repetition time (TR) = 591 ms (range, 433 to 750 ms) and echo time (TE) = 13 ms (range, 9 to 20 ms) on the 1.5 T system, and TR = 500 ms and TE = 14 ms on the 3.0 T system. Gadopentate (Magnevist; Berlex, Montville, USA) at the standard clinical dose of 0.2 mL/kg was used for each patient at both 1.5 T and 3.0 T. For each patient, a single T1W scan location was identified that contained a skull base lesion and normal brain tissue. Regions of interest (ROIs) were identical between axial pre- and postcontrast T1W sequences in all patients, and were defined to closely match the 1.5 T and 3.0 T scans. All ROIs avoided cystic areas and no patient had multiple lesions. Fat-suppressed images were not evaluated in this study due to the potential for asymmetric fat-suppression. Signal intensity of the lesion normalised to enhancing brain was calculated as:

$$\textit{Lesion-to-brain ratio (LBR)} = \frac{\textit{Signal intensity lesion}}{\textit{Signal intensity normal brain}}$$

for both pre- and postcontrast T1W scans and both field strengths. Normalisation by brain ensures that potential arbitrary scaling of image intensities between scans and across scanners is removed. The degree of contrast enhancement between the pre- and postcontrast images, or signal-enhancement ratio (SER), was calculated as:

$$SER = \frac{LBR\ postcontrast - LBR\ precontrast}{LBR\ precontrast} \times 100\%.$$

SER values were derived for each patient on each field strength. Paired Student *t* test was used to determine whether there was a significant difference between the 1.5 T and 3.0 T LBR and SER ratios. In the mathematical simulation of the equation below, relaxation time values for non-enhancing brain were T1 = 600 ms, T2 = 86 ms at 1.5 T, and T1 = 750 ms and T2 = 75 ms at 3.0 T.

$$SI = Mo[1 - e^{-R_1 \cdot TR}]\, e^{-R_1 \cdot TE}$$

where

$$R_1 = \frac{1}{T_{1o}} + k_1^B \cdot [Gd]; \quad R_2 = \frac{1}{T_{2o}} + k_2 \cdot [Gd]$$

and T_{1o} *= native* T_1*; and* T_{2o} *= native* T_2.

T1 and T2 correspond to white matter values at 1.5 T and 3.0 T, as measured in other studies.[5] In this study, lesion T1s were unknown, therefore assumed values of T1 = 1000 ms and T2 = 100 ms[6,7] were used in this simulation. In addition, the gadolinium T1 relaxivity was adjusted for field strength based on published values of $k_1^{1.5T}$ = 4.74 mMol/second and $k_1^{3.0T}$ = 4.43 mMol/second.[8] T2 relaxivity was held constant at k_2 = 5.7 mMol/second for both 1.5 T and 3.0 T. Since this study relates to T1W contrast, minor inaccuracies in T2 relaxivity should be of little consequence. Lesion to brain signal-intensity ratios were simulated using well-established expressions for spin-echo and relaxation rates as a function of the degree of gadolinium concentration in the lesion.[9]

This retrospective study was approved by the Institutional Review Board of the University of Michigan.

RESULTS

As the accuracy of the study depended on the stability of the lesions between the scans, all chosen lesions measured within the ROIs showed no significant change between time points. A summary of the lesion diagnoses and patient demographics is provided in Table 1.

All patients with malignancies (squamous cell carcinoma [SCC], osteosarcoma, melanoma, and lymphoma; patients 1 to 7) had persistent or progressive disease at the time of each imaging study as determined by characteristic imaging or clinical findings. As shown in Table 1, patients with slow growing tumours (vestibular schwannoma, olfactory groove meningioma; patients 8 to 10) who were being routinely followed-up had the longest time interval between studies. These tumours did not change in size during this interval. One patient had a fourth nerve palsy and a corresponding enhancing mass in the contralateral cavernous sinus (patient 11), which corresponded to the clinical finding. This was presumed to be a schwannoma of the trochlear nerve and surgery was not considered a viable option. Two patients had post-treatment changes without evidence of recurrent disease (patients 12 and 13). Patient 12 underwent surgical resection of an adenocarcinoma of the ethmoid sinus and patient 13 underwent combined chemotherapy and radiation therapy for a nasopharyngeal carcinoma. Patient 14 received a diagnosis of aspergillosis after the second scan, but was untreated between scans. Patient 15 had viral neuritis of the 7th nerve.

Twelve patients underwent initial imaging with 1.5 T followed by 3.0 T, while 4 patients were initially imaged

Table 1. Signal-enhancement ratio by disease.

Patient number	Lesion type	Age (years)	Sex	Scan interval (days)	Signal-enhancement ratio		
					1.5 T	3.0 T	3.0 T/1.5 T
1	SCC maxillary sinus	70	M	128	106.7	161.4	1.5
2	SCC maxillary sinus	73	M	7	143.8	286.3	2.0
3	SCC nasopharynx	57	M	80	106.4	143.1	1.3
4	Calvarial metastases from SCC skin	44	M	38	127.9	173.1	1.4
5	Melanoma maxillary sinus	69	M	85	107.5	251.6	2.3
6	Lymphoma	50	F	2	107.4	349.2	3.3
7	Osteosarcoma	46	M	102	112.1	147.9	1.3
8	Olfactory groove meningioma	48	F	368	54.4	116.0	2.1
9	Vestibular schwannoma	82	F	596	220.7	209.5	0.9
10	Vestibular schwannoma	52	F	454	36.4	29.9	0.8
11	Presumed right cavernous sinus schwannoma	28	M	12	61.3	156.4	2.6
12	Post-treatment granulation tissue	48	M	89	171.8	238.5	1.4
13	Post-treatment granulation tissue	62	M	210	71.2	64.3	0.9
14	Aspergillosis skull base	63	M	58	68.1	120.7	1.8
15	Viral neuritis cranial nerve VII	6	F	75	55.0	139.4	2.5
	Average	53.2		153.6	103.4	172.5	1.7

Abbreviations: SCC = squamous cell carcinoma; T = Tesla.

with 3.0 T followed by 1.5 T (patients 4, 5, 6, and 12). The average time between imaging performed at 1.5 T and 3.0 T was 154 days (range, 2 to 596 days).

Calculation of LBR and subsequent SER demonstrated that 12 of the 15 lesions had increased degrees of enhancement at 3.0 T compared with 1.5 T. Three lesions (both vestibular schwannomas and the post-treatment granulation tissue [patient 13]) had significantly reduced SER on 3.0 T compared with 1.5 T ($p = 0.001$). The average percent increase in lesion signal after contrast administration on the T1W scans was 103% at 1.5 T and 172% at 3.0 T (Table 1). The LBR for the non-contrast T1W sequences between 1.5 T and 3.0 T was not significantly different ($p = 0.915$). If the biological properties of the lesions and agent distribution kinetics are assumed to be largely unchanged between scan dates, differences in SER between field strengths are not likely to be due to variable gadolinium concentration. Therefore, the difference in postcontrast signal intensity is most likely due to field strength.

In the mathematical simulation of the SER index for an enhancing lesion relative to a non-enhancing lesion of the brain at 1.5 T and 3.0 T, an SER increase at 3.0 T was predicted, which is consistent with that observed with the measured lesions. The measured average SER values (103% for 1.5 T and 172% for 3.0 T) are reasonably consistent with those illustrated in Figure 1 for a gadolinium concentration of ≥0.8 mMol. While gadolinium relaxivity decreases with increasing field strength (longitudinal relaxation rate decreases by 6.5% for 3.0 T vs 1.5 T), the increase in native longitudinal relaxation time of the lesion (i.e., pre-gadolinium) with field strength supersedes the slight decrease in R1 and leads to greater gadolinium impact at a given gadolinium concentration at the higher field strength.

DISCUSSION

The results of this study are consistent with previous studies that reported increased enhancement in a variety of intracranial abnormalities when imaged at 3.0 T versus 1.5 T.[2,10-12] Nöbauer-Huhmann et al[2] and Tratting et al[10] reported significantly higher tumour-to-brain ratios for primary brain tumours and intracranial metastases when imaged at 3.0 T compared with 1.5 T. Sicotte et al reported that 3.0 T imaging detected a 21% increase in the number of enhancing lesions in patients with multiple sclerosis compared with imaging at 1.5 T.[11] Barth et al reported that contrast-enhanced

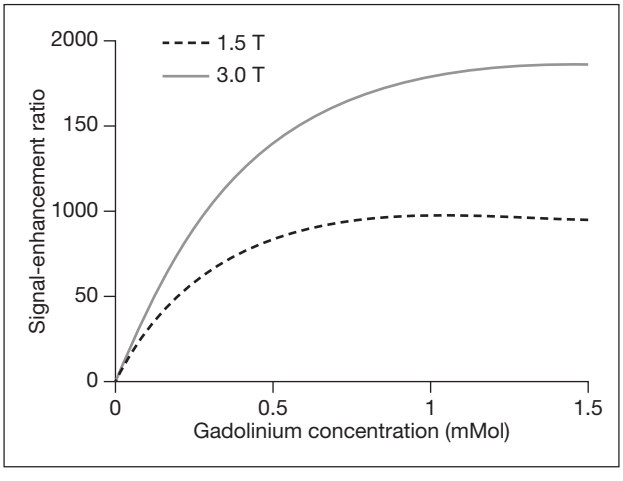

Figure 1. Mathematical simulation of predicted changes in signal-enhancement ratio by gadolinium concentration.

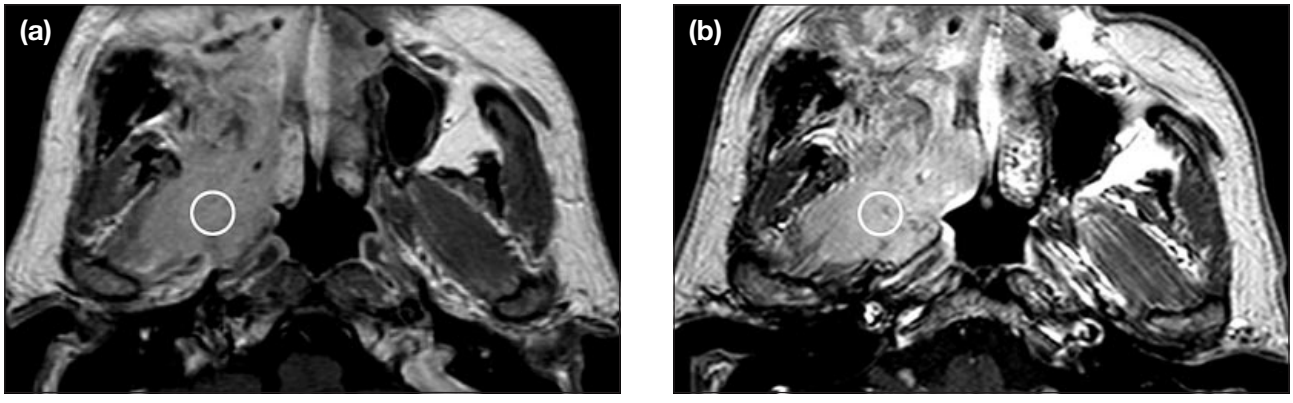

Figure 2. Imaging of a patient with an osteosarcoma at (a) 1.5 Tesla; and (b) 3.0 Tesla. These postcontrast T1-weighted scans show an increase of 36% of the signal-enhancement ratio of the lesion when scanning at 3.0 Tesla. The circle represents the lesion region of interest.

magnetic resonance venography at 3.0 T showed more peritumoural venous anatomy compared with a similar study performed at 1.5 T.[12] However, these studies only evaluated intracranial pathology. This comparative study specifically evaluated skull base pathology images at both 3.0 T and 1.5 T.

In this study, a significant gain in SER was noted for 3.0 T relative to 1.5 T. The source of this increase is predictable, based on known increases in tissue T1s at higher fields (Figure 2). Other researchers have measured an increase in T1 of approximately 25% to 40% in brain parenchyma at 3.0 T relative to 1.5 T.[8]

The ability of 3.0 T to increase lesion enhancement raises some interesting issues. There is debate as to whether the amount of contrast administered at 3.0 T can be reduced, such that the enhancement changes are similar to those depicted at 1.5 T. An alternative approach would be to administer the same amount of contrast at 3.0 T and consider the increased conspicuity a 'benefit' of the higher field strength. For patients who have undergone serial imaging, the 2 main features that are assessed are changes in lesion size and enhancement. Based on the findings of this study, it may be difficult to accurately incorporate changes in enhancement as a measure of disease progression if a patient has undergone scanning at different field strengths.

Three patients did not demonstrate increased enhancement on 3.0 T versus 1.5 T. Two of the patients had vestibular schwannomas and 1 patient had post-treatment changes following combined chemotherapy and radiation therapy for nasopharyngeal carcinoma. It is possible that the lack of increased enhancement could be due to substantial biophysical changes in the lesion secondary to treatment, which violates the assumption that lesion gadolinium concentration is essentially the same between 1.5 T and 3.0 T scans. Vestibular schwannomas (patient 13) tend to be slow growing lesions with a lower overall SER at both 1.5 T and 3.0 T. The enhancement of schwannomas can be variable, which may be due to heterogenous histology due to the relative amounts of Antoni A and B cells within the lesion. In addition, the degree of enhancement may also depend on the time interval between the administration of contrast and imaging. Similarly, there may be reduced vascularity to the treated tissue following chemotherapy and radiation therapy. The imaging of this patient was performed 2 months (1.5 T) and 9 months (3.0 T) after completion of therapy. It is interesting to note that the only patient treated with primary surgery of an adenocarcinoma of the ethmoid sinus demonstrated increased enhancement. This may be due to hypervascular granulation tissue that is typically seen at surgical margins.

This study had several limitations. The investigation was a retrospective evaluation. Various pathologies, including neoplastic, infectious, and inflammatory pathologies were evaluated, as opposed to focusing on a single pathology. In addition, the time interval between the 1.5 T and 3.0 T studies was not consistent. Selection of acquisition parameters to achieve T1 weighting at both field strengths was established independent of this study and were not modified for the study. Therefore, it is possible that greater T1 weighting could be achieved at one of the field strengths, which would affect the results. However, these results do represent an initial attempt to comparatively evaluate contrast enhancement of skull base lesions at different field strengths in the same patient.

The results of the investigation were highly significant and demonstrate clear trends that are consistent with

previously published reports regarding increased lesion enhancement at 3.0 T versus 1.5 T. These initial results need to be confirmed by future prospective comparative studies.

REFERENCES

1. Frayne R, Goodyear BG, Dickoff P, Louzon L, Sevick RJ. Magnetic resonanace imaging at 3.0 Tesla: challenges and advantages in clinical neurologic imaging. Invest Radiol. 2003;38:385-402.
2. Nöbauer-Huhmann IM, Ba-Ssalamah A, Mlynarik V, et al. Magnetic resonance imaging contrast enhancement of brain tumors at 3 Tesla versus 1.5 Tesla. Invest Radiol. 2002;37:114-9.
3. Ginsberg LE. MR imaging of perineural tumor spread. Neuroimaging Clin N Am. 2004;14:663-77.
4. Chong VF, Khoo JB, Fan YF. Imaging of the nasopharynx and skull base. Neuroimaging Clin N Am. 2004;14:695-719.
5. Lu H, Nagae-Poetscher LM, Golay X, Lin D, Pomper M, van Zijl PC. Routine clinical brain MRI sequences for use at 3.0 Tesla. J Magn Reson Imaging. 2005;22:13-22.
6. Bottomley PA, Foster TH, Argersinger RE, Pfeifer LM. A review of normal tissue hydrogen NMR relaxation times and relaxation mechanisms from 1-100 MHz: dependence on tissue type, NMR frequency, temperature, species and age. Med Phys. 1984;11:425-48.
7. Bottomley PA, Hardy CJ, Argersinger RE, Allen-Moore G. A review of ^{1}H nuclear magnetic resonance relaxation in pathology: are T_1 and T_2 diagnostic? Med Phys. 1987;14:1-37.
8. Bernstein MA, Huston J, Lin C, Gibbs GF, Felmlee JP. High-resolution interacranial and cervical MRA at 3.0T: technical considerations and initial experience. Magn Reson Med. 2001; 46:995-62.
9. Hendrick RE, Haacke EM. Basic physics of MR contrast agents and maximization of image contrast. J Magn Reson Imaging. 1993;3:137-48.
10. Tratting S, Ba-Ssalamah A, Nöbauer-Huhmann IM, et al. MR contrast agent at high-field MRI (3 Tesla). Top Magn Reson Imaging. 2003;14:365-75.
11. Sicotte NL, Voskuhl RR, Bouvier S, Klutch R, Cohen MS, Mazziotta JC. Comparison of multiple sclerosis lesions at 1.5T and 3.0T. Invest Radiol. 2003;38:423-7.
12. Barth M, Nöbauer-Huhmann IM, Reichenbach JR. High-resolution three-dimensional contrast-enhanced blood oxygenation level-dependent magnetic resonance venography of brain tumors at 3 tesla: first clinical experience and comparison with 1.5 tesla. Invest Radiol. 2003;38:409-14.