

Tumor Enhancement in a Brain Glioma Model: An Intra-individual Comparison of Half Dose Gadobenate Dimeglumine vs Full Dose Gadopentetate Dimeglumine at 1.5 and 3 T

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Purpose:

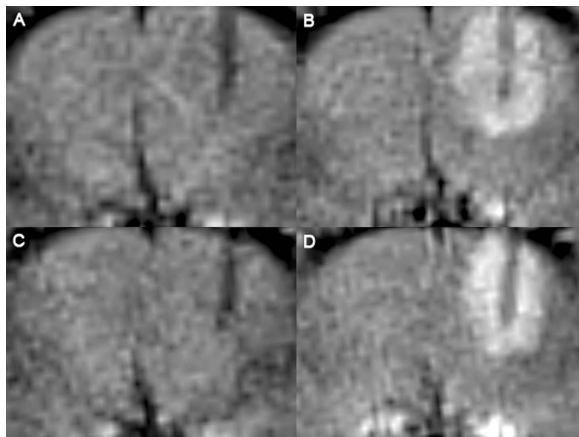
Six standard, extracellular gadolinium chelates have been approved in countries across the world for contrast enhanced MRI of the brain. With gadobenate dimeglumine, a chelate with transient protein binding became available, whose relaxivity is higher in comparison to the standard gadolinium chelates without such protein interaction [1]. Combining a high relaxivity, gadolinium chelate and 3 T offers multiple opportunities for further dose reduction without loss in image quality and thus diagnostic accuracy. Regarding nephrogenic systemic fibrosis (NSF,) the injected dose level becomes very important, since NSF, a severe systematic adverse reaction to unbound gadolinium ions, is reported to be related to gadolinium chelate injection in patients with reduced renal clearance, with a dependency on both chelate stability and cumulative dose [2]. The purpose of this study was to evaluate lesion enhancement with gadobenate dimeglumine at half dose (0.05 mmol/kg BW) compared to gadopentetate dimeglumine at full dose (0.1 mmol/kg BW) in a rat brain glioma model at 1.5 and 3 T.

Material and Methods:

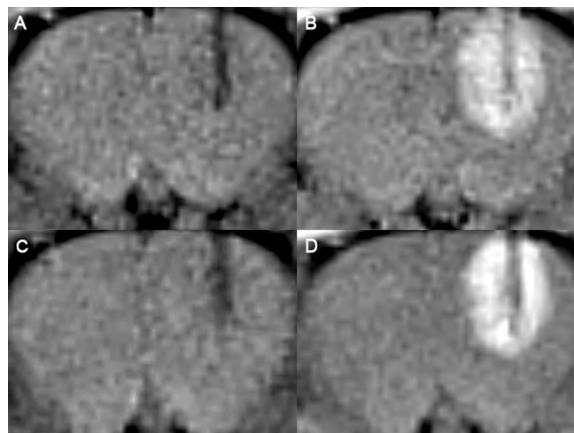
The study included 25 experimental animals, each with an implanted, intra-axial tumor, divided into four groups. In each instance gadobenate dimeglumine (MultiHance) was injected at half dose and gadopentetate dimeglumine (Magnevist) at full dose. Each animal was imaged twice, with the order of studies randomized and the two contrast injections separated by 24 hours to ensure complete clearance of each agent. In groups 1 (n=4), 2 (n=6) and 3 (n=8), gadopentetate dimeglumine and gadobenate dimeglumine were compared at 3 T, with the only difference between the groups being the specific timing of the post-contrast scan. In group 4 (n=7), the two agents were evaluated at 1.5 T, with the timing of the post-contrast scan the same as that used in group 2. A T1-weighted 2D fast spin echo technique (TR/TE 500/16) was used for image acquisition, with voxel dimensions of 0.2 x 0.2 x 2 mm³.

Results:

In groups 1 and 2, at 3 T, half dose gadobenate dimeglumine (group 1: SNR 79±8, CNR 22±7; group 2: SNR 61±13, CNR 21±12) showed comparable tumor SNR and CNR mean values to full dose gadopentetate dimeglumine (group 1: SNR 87±17, CNR 33±17; group 2 SNR 57±7, CNR 16±7). There was no statistically significant difference in either group between the agents, for SNR or CNR, despite the use of half dose for gadobenate dimeglumine. In group 3, at 3 T, a statistically significant difference was able to be demonstrated for contrast enhancement between the two exams, due to lower variability in degree of lesion enhancement across the animal group. This represented however only a gain of 18% in tumor SNR. In group 4, at 1.5 T, there was a marginally statistically significant difference (p < 0.05) in tumor SNR post-contrast between that achieved with half dose gadobenate dimeglumine (31±6) versus that with full dose gadopentetate dimeglumine (37±3).



Illustrated are pre- (a, c) and 3 min. (b, d) post-contrast scans at 1.5 T of an implanted glioma. The magnitude of contrast enhancement was substantially greater after the administration of full dose gadopentetate dimeglumine (b) as compared to gadobenate dimeglumine (d), the latter injected at a dose of 0.05 mmol/kg BW.



Illustrated are pre- (a, c) and 3 min. (b, d) post-contrast scans at 3 T of an implanted glioma. prior to and following administration of 0.1 mmol/kg (b) gadopentetate dimeglumine as compared to (d) gadobenate dimeglumine, injected at 0.05 mmol/kg BW.

Conclusion:

At 3 T, half dose gadobenate dimeglumine, due to transient, weak protein binding, achieves comparable brain tumor enhancement to full dose gadopentetate dimeglumine, with near equivalence demonstrated at 1.5 T. These results support use of half dose in particular in patients with reduced renal function, an important consideration due to the advent of nephrogenic systemic fibrosis.

References:

- [1] Pintaske J et al, Invest Radiol 2006.
- [2] Rydahl C et al, Invest Radiol 2008.